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Histologic Evaluation of the Effect of Formocresol on the Furcation Area of Dog Molars

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HISTOLOGIC EVALUATION OF THE EFFECT OF FORMOCRESOL ON THE FURCATION AREA OF DOG MOLARS

By

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DEDICATION

To Patty, Mila, and Ricky

without whom this would be pointless

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VITA

The author, Richard Albert Munaretto, was born to his parents Frank and Adeline in Chicago, Illinois, U.S.A. on December 5, 1943.

He obtained his elementary education at Our Lady of the Angels School (Chicago, Illinois), and his secondary education at St. Philip Basilica High School (Chicago, Illinois), graduating with honors in June of 1961.

In September of 1961 he began pursuing a bachelor of science degree, at Loyola University of Chicago, completing those requirements and graduating in June, 1965. That September he entered Loyola University School of Dentistry seeking the degree of Doctor of Dental Science, which he received in June of 1969.

After serving in the United States Army in Germany for three years, he entered private practice in the Chicago area.

In September 1977 he entered Loyola University School of Dentistry in a dual course of study leading to the degree of Master of Science in Oral Biology and a Certificate of Specialty Training in Endodontics.

The author married Patricia Ruth McCormick on August 3, 1968 and they have two children, Mila and Richard.

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INTRODUCTION

Many years have passed since formocresol was introduced to the dental profession. Since that time, it has become one of the most widely utilized and clinically accepted medicaments used in treating pulp tissue. In a field where science is making almost daily progress and evolving new techniques, this empirically derived medicament and its application have remained basically the same since its inception. After many clinical and histologic studies, it seems that the only recommended alterations have been a mild dilution of the product and method of application in the tooth.

According to the available dental literature, lateral canals appear to range from "sparse" to "profusely evident". This paradox is made more interesting since some authors link lateral canals with combined endodontic-periodontic problems while others claim that no interrelationship exists.

Formocresol is routinely placed on a cotton pellet and sealed into the pulp chamber during a pulpotomy procedure or between visits during endodontic therapy as an interappointment medication. Therefore, this study was undertaken to investigate the effects of these medications on the tissues in lateral canals and in the furcation area of treated teeth.

CHAPTER II

REVIEW OF THE LITERATURE

EMBRYOLOGY OF THE FURCATION

At the margins of the bell-shaped enamel organ, the inner and outer layers of the enamel epithelium proliferate to give rise to Hertwig's epithelial root sheath. This sheath outlines the dentinocemental junction and acts as the blueprint for the shape, size, and length of the roots.[1]

A marked difference exists in the development of Hertwig's sheath in a single rooted teeth as compared with multi-rooted teeth. In multi-rooted teeth, differential growth of the epithelial diaphragm causes a division of the root trunks into two or more roots. Thus, during the general growth of the enamel organ the expansion of its cervical opening occurs in such a way that long, tongue-like extensions of the horizontal diaphragm develop. In general two such extensions are found in the germinal tissue of lower molars, whereas three are present in the germs of upper molars. Before root trunk division occurs, the free ends of these horizontal epithelial flaps grow toward each other and fuse, dividing the single cervical opening of the coronal enamel organ into two or three openings. On the pulpal surface of the dividing epithelial bridges, dentin formation begins, and on the periphery of each opening, root development follows as in single rooted teeth.

If the continuity of Hertwig's sheath is broken or not established prior to dentin formation, a defect in the dentinal wall of the pulp results, accounting for the development of lateral or accessory root canals opening to the periodontal ligament. Such defects are found in the pulpal floor corresponding to the furcation, or at any point on the root itself, if the fusion of the horizontal extensions of the diaphragm remains incomplete.[2]

LATERAL CANALS

As listed in the glossary of terms, second edition, published by the American Association of Endodontists, a lateral canal is "a lateral branch of the main root canal which is approximately perpendicular to it".[3] These are generally found in the coronal two-thirds of the root and do not include apical canals.

Cahn was the first to postulate that periodontal disease could lead to pulpal degeneration via exposed lateral canals.[4] In his 1925 article he noted that Professor C.F. Bodecker, then the head of the histo-pathology laboratory at Columbia Dental School, believed that lateral canals are of embryonic origin. Barrett's study[5] of the previous year explains their origin as due to the interference by persistent blood vessels with the downward growth of the enamel organ, known by many as the sheath of Hertwig.

In a more recent work, Scott and Symons[6] agreed, stating that these aberrant openings are presumed to be caused by a localized failure in the formation of Hertwig's sheath, with a consequent lack of odontoblastic

differentiation and dentin formation at this point, so that the pulp remains in contact with the follicular or periodontal tissue. The gap in Hertwig's sheath is probably produced by the persistence of abnormally placed blood vessels reaching the pulp. Stallard confirms this view.[7]

A review of the literature provides a general concensus that a great deal of communication exists between the intraradicular and extraradicular tissues via lateral canals.

In 1901, Preiswork,[8] studying Wood's metal* castings of root canals demonstrated that the root canals of some teeth, especially molars, possessed lateral branchings and anastomoses.

A few years later, Fischer[10] macerated the pulp tissue of several teeth, dried them, and placed a celluloid-acetone solution into the pulp chamber. By dissolving the teeth he disclosed a "high percentage" of branchings in posterior teeth but less in anterior teeth. His study led him to believe that the number of branchings increased with age.

Fasoli and Arlotta[11] using Preiswork's method of forcing Wood's metal into the empty canals of teeth, were able to demonstrate these branchings radiographically and also by placing the teeth in a clearing solution.

Hess[12] used vulcanite perfusion instead of celluloid, but like

* Wood's metal[9] - a metal used in making casts of blood vessels: bismuth 50%, lead 25%, tin 12.5%, and cadmium 12.5%.

Fischer, he believed that the number of accessory canals (in multi-rooted teeth) increased with age. Of 300 teeth studied, he found "marrow canals" in approximately seventeen per cent. "In young teeth either a few marrow canals were present or none; whilst at ages from twenty to 40 a large percentage of marrow canals were seen in teeth with one root, as incisors or canines, but the numbers declined in teeth of patients 40 to 50 years of age; in teeth with several roots the number of marrow canals increased with advancing age".

Contrarily, Rottenbitter[13] using Hess's method of obtaining casts and by histological sections of 600 teeth with vital pulps found these ramifications only twice. All types of teeth were equally represented and the patients ages ranged from five to 60 years.

Barrett[5] stated that the findings of Fisher and Hess have been corroborated, in part at least, by Callahan, Howe, Grove and others. He wrote "an examination of the tracings of Preiswork, Fasoli and Arlotta will show that their methods have not demonstrated that the lateral canals connect the main canal with the outer surface of the tooth. The same we feel is true of the work of Fischer and Hess". He claimed that his study clearly demonstrated that age of the individual is irrelevant as the number of "branches" is entirely the result of a developmental process and the only change produced by aging may be a slight lengthening of the canals as the apical cementum thickens. A decrease in the number of canals may occur with advancing age due to partial or complete calcification. "The importance of these lateral

processes should be apparent, inasmuch as we have found suppurating processes about the small foramina".

After a twenty-three year hiatus, the subject again was brought to light by Johnston and Orban,[14] who independently observed obscure pathologic conditions occurring in the furcations of endodontically treated multi-rooted teeth. Orban described three failing cases several years after endodontic therapy completion, during which time they were regarded as successful. The problems only involved the interradicular bone. Johnston, who revised Callahan's chloroform-gutta-percha technique of obliterating root canals, did several studies on root canal shapes and demonstrated many lateral canals.[15] In this article,[14] he showed two of several cases where complete healing of an interradicular area following root canal treatment. These cases, among many others, led the authors to consider the possibility of lateral canals in this region to be the cause of the furcation pathosis. Orban's classic histological section demonstrating a large furcation canal, as well as Johnston's much copied radiograph of the obliteration of such a canal, may be seen here.

In 1956 Russell and Kramer,[16] studying the vascular architecture of the pulp, reported numerous lateral canals usually containing a pair of vessels, one large and one small.

Saunders[17] used a contact microradiographic technique to demonstrate lateral canals in the pulp chamber floor of a human molar showing the presence of numerous blood vessels coursing between the pulp and the periodontal ligament.

Everett, Jump, Holder and Williams[18] studied the morphology of the bifurcation of the lower first molar and described a ridge in that area which originated on the mesial surface of the distal root, ran across the bifurcation, and ended high up on the mesial root. They noted this ridge as prominent in 47%, visible in 29%, and absent in 24%. Nutrient canals were often seen at the buccal and lingual margins of this ridge.

A 1960 article by Kramer[19] described a vascular injection technique using India ink perfusion. He was able to demonstrate paired vessels (one larger than the other) in lateral canals of a human molar tooth located in the midradicular region between the apex and the pulpal floor. This specimen exhibited six lateral canals in one of the roots, all containing arteries and veins.

A preliminary study by Winter[20] in the same year found a ten percent incidence of accessory root canals in primary molar teeth with a periapical (or furcation) abscess. This abstract was followed in 1962 by a detailed article describing his complete study.[21] Using 2% methylviolet dye under suction after the teeth were treated with proteolytic enzymes, Winter found a 23% incidence of accessory canals leading to the interradicular root surface in primary molar teeth. These canals were found only on the interradicular surface and commonly opened on to the web of dentin between the root canals. Howship's lacunae covered by a thin layer of calcified repair tissue could be seen lining the wall of the canal in each case, showing that phases of active resorption and

repair had taken place. The radiographic change noted was generally one of rarefaction which appeared to begin at the furcation of the roots and gradually extended to involve all of the interradicular bone. Six per cent of the teeth also exhibited areas of macroscopic resorption which would have constituted aberrant channels of communication between the pulp and the surrounding periodontal tissues. Whether these were the initial sites of lateral canals or not is unknown. He noted that Applebaum demonstrated that root resorption may start in the walls of the accessory canals, and he agreed.

In 1963 a study by Nichols[23] of 228 pulpless root canals (221 teeth) with radiographic evidence of "obvious alveolar destruction due to pulpal disease", indicated ten teeth (4.4%) were associated with a separate lateral radicular lesions thought to be due to a lateral canal. Following nonsurgical root canal therapy using gutta-percha or silver cones as the filling material, eight teeth were re-examined, six of which were judged successful, while two were judged uncertain. He concluded that the treatment rendered to the main canal was sufficient to eliminate the associated lateral canal as a source of irritation to the adjacent alveolar bone.

That same year, Seltzer, Bender and Ziontz[24] described their study of the interrelationship of pulp and periodontal disease. Altogether, 95 periodontally involved teeth were examined. Lateral canals "were found in profusion in the roots of posterior teeth and occasionally in anterior teeth". They were also profusely evident in the bifurcation or trifurcation

regions. In a few instances, they could be seen at different levels, reaching from the interradicular region of the root into the coronal pulp. Some canals in the furcation were seen to traverse the root and enter the root canal. These canals contained capillaries, pulp cells, ground substance, and fibers; and this tissue was confluent with the pulp tissue. In many teeth, however, the width of the accessory foramina or lateral canals was exceedingly small, permitting the presence of only small caliber vessels and their supporting stroma. Completely calcified canals were never discovered histologically. Deep periodontal lesions were frequently found exposing the lateral canals, and in the more advanced lesions, necrotic pulp tissue was discovered in the larger lateral canals which were exposed. In cases in which the pulp was severely inflamed, inflammation was readily spread to the periodontal membrane through these canals.

The following year an article by Simring and Goldberg[25] on retrograde periodontitis postulated that pulpal disease could cause periodontal disease, contribute to it, and prevent healing following periodontal treatment. They found that in many cases, periapical extension of pathosis may never occur because lateral drainage may be adequate to relieve the flow of exudate, allowing the apical portion of the pulp to remain vital. Lateral canals are important, they said, not for the minute amounts of necrotic material they may harbor, but as channels of communication for a large bulk of necrotic material which may pass in either direction.

Also in 1964, Mazur and Massler[26] concluded that morphologic changes

in the pulp are not related to changes in the periodontium, but probably more directly related to the systemic condition of the patient.

The next year, Winter and Kramer[21] surgically exposed the pulp of each lower second primary molar in thirteen crossbred kittens, leaving them open to the oral cavity. The first radiographic changes occurred at fourteen days showing a definite thickening of the interradicular periodontal shadow. An area of interradicular rarefaction was evident on the fifteen day animal, and was maximal at the furcation. Finally, at twenty days the interradicular rarefaction involved most of the bone beneath the exposed molars, with a maximum intensity at the bifurcation in each case. Tooth substance had also been resorbed.

Histologic examination showed an unexpectedly high proportion of the operated teeth had a canal (31 of 45 teeth plus three where extensive resorption did not allow determination of a canal) running through the dentin and cementum from the floor of the pulp chamber to the furcation. Where tissues were sufficiently normal, it could be seen that each such canal carried a relatively large blood vessel plus a small amount of loose connective tissue. The vessel passed from the pulp through the canal and into the interradicular bone. Four teeth possessed two canals. In the teeth examined, these canals were clearly formed as a result of the vascular pattern that existed prior to dentinogenesis.

Resorption of the crest of the interradicular bone, of such a degree that undoubtedly exceeded normal remodeling activity, was first detected microscopically in the five day animal. In the floor of the

pulp chamber, resorption was progressive and it could be destroyed in as little as ten days. This resorption must have occurred mainly from the root surface for, except in the earliest stages, it is unlikely that there was surviving tissue in the accessory canal to support the osteoclastic activity on the walls of the canal. They concluded that where an accessory canal runs from the floor of the pulp chamber to the furcation, this canal facilitates the spread of inflammation from the pulp to the furcation. There, an inflammatory focus may develop resulting in a rapid, progressive resorption of the dental tissues and of interradicular bone, which in later stages is demonstrable radiographically as an area of rarefaction.

In 1965, Rubach and Mitchell[27] published their study which was designed to illuminate the effects of periodontal disease on the pulp, and to determine the relative frequency and importance of pulp exposure through periodontal recession to lateral canals. Microscopic study of the histologic sections of 74 teeth, which were first ground until the outline of the pulp was visible (thus allowing study of only one-half of the tooth) revealed eight (10.9%) lateral canals so located that connection with periodontal pockets was demonstrable in five of the specimens. Periodontal granulation tissue in the bifurcation had progressed into the canals. They concluded that the periodontist should be aware of this potential source of contamination since surgical exposure of accessory canals would result in pulp exposure.

The same year Moss, et al.,[28] presented their investigation in

which they tried to determine if necrotic material could leave the pulp chamber directly through the pulpal floor into the interradicular region either through lateral canals or through the dentin and cementum, whose porosity may be altered in the presence of infection; and to study histologically the pulpal floor of the primary molar and the histopathologic changes found in infected primary molars that show radiographic pathologic change directly below the pulpal floor. They noted that infected primary molars displayed radiographic phenomena not observed in permanent molars. In the former, pathologic changes are evidenced by a radiolucent zone between the roots of the tooth, whereas the latter show radiographic change at the root apex.

No significant difference in permeability to methylene blue dye was seen between infected and control teeth that had accessory canals seen on histologic examination. However, comparisons of infected and noninfected teeth without such anatomic channels showed a significant increase in permeability of all the infected teeth. Of the 56 teeth studied, twenty per cent had accessory canals entering the pulp chamber in the region of the furcation. The majority of the canals entered the region of least thickness, where the floor of the pulp chamber slopes into the root; others entered directly through the height of greatest curvature in the center of the pulp chamber. The canals were smooth-walled, five to seven microns in diameter, and traveled through dentin and cementum. Changes were noticed in both the dentin and cementum suggesting decalcification. They concluded that transport of material may occur in and out of infected and noninfected teeth via lateral canals.

In 1966 Hiatt[29] noted that although furcation lesions generally are of periodontal origin, it is not uncommon that pulpal inflammation may reach the furcation via an accessory canal. He cited Coolidge,[30] who stated in his text that an additional source of repair from the periodontal tissues in which lateral and apical canals have either filled or partially filled with hard substance after root canal therapy.

Abstracts by Amen, Ingle, and Weiner[31] published in 1967 indicated that these authors consider lateral canals to be significant pathways for irritants to reach the pulp or furcation tissues in either direction. Weiner states that when periodontal disease affects the furcation alone, it should be remembered that as many as eighteen per cent of such teeth have a lateral canal extending from the pulp chamber to this area.

Binns and Escobar[32],1967, studied the effects of endogenous infection in the pulps of primary teeth on the permanent tooth germs at different stages of tooth formation. Evidence was obtained indicating that the infection of a primary tooth can and does affect the permanent tooth germ and surrounding structures. They stated that since horizontal or vertical bone loss occurred in most cases rather than periapical bone loss, this indicated that the dog's primary molars, like those of humans, may possess accessory canals which extend from the pulpal chamber into the interradicular area. The importance of accessory canals should not be overlooked since they provide a short and easy path for the extension of infection. Changes occurred in the permanent tooth bud with little evidence of the fact clinically or radiographically.

The same year, Seltzer, Bender, Nazimov and Sinai[33] published the results of a study to determine if the development of periodontal lesions could occur by inflammatory or degenerative products reaching the interradicular area through lateral canals. They had observed previously that there is a fairly high incidence of lateral canals which communicate between the floor of the pulp chamber and the periodontal tissues in the posterior teeth of both dogs and monkeys.

Alterations of the interradicular tissues were detected in 21 of the 100 teeth examined. Within seven days, an inflammatory infiltrate was seen in the periodontal ligament near the alveolar crest of seven teeth, mainly in the vicinity of lateral canals. Resorption of the cementum, dentin, and crestal bone in the furcation occurred in all of these specimens. The persistence of periodontal inflammation appeared to coincide with the persistence of coronal pulp inflammation or necrosis. Lateral canals were detected either in the floor of the pulp chamber or in the cementum and dentin of the furcation regions in most, but not all, of the involved cases, though they may have been present.

In 1969 Bender and Seltzer[34] noted that histologic observations demonstrating the presence of lateral canals have been reported by numerous investigators as a rather uncommon finding. They stated that some texts even omitted mention of lateral canals. This neglect might be attributed to the fact that serial sections of the teeth were not inspected so that "the ubiquitious distribution of the lateral canals was not recognized". In a histologic study of 178 human teeth, they "found lateral canals in

perfusion within the roots of posterior teeth, and occasionally in anterior teeth". They cited one case where arsenical necrosis developed in the furcation region of a lower first molar following application of an arsenical dressing in the pulp chamber. In other cases, resorption of the alveolar crest occurred without the apparent presence of lateral canals.

A 1972 article by Stallard[7] concerning endodontic-periodontic relationships states that "a secondary vascular supply exists in many teeth by way of the lateral or accessory canals". While these canals contribute to the overall nutrient supply, they are inadequate as a major collateral circulation during pulp pathosis, but inflammation may spread in either direction through these channels. Statistically, endodontic success increases with the patient's age, since continued dentin formation reduces the lumen of the pulp chamber in addition to obliterating numerous accessory canals.

Mandi[35], studying pulp changes as related to periodontal diseases, reported in 1972 that eighteen of the 38 intact (free of caries or restorations) teeth examined had chronic inflammatory pulp lesions, two contained microorganisms; in two cases inflammation proceeded from a lateral canal to the radicular pulp tissue and one had osteoclasts in the dentin of the lateral canal. Four teeth were necrotic, one of which contained a lateral canal with necrotic tissue. However, the severity of the pulpal disease did not always correspond to the severity of the periodontal disease.

In the same year, Pineda and Kuttler[36] disclosed the results of their radiographic investigation of 7,275 teeth. No ramifications in the furcations of multi-rooted teeth were noted.

Lowman, Burke and Pelleu,[37] 1973, reported a study of the incidence of patent accessory canals in the molar furcation region. Forty-six extracted maxillary and mandibular first and second molars were studied. After removing the pulp tissue, root planing was done to simulate a clinical situation. Methylene blue dye in Hypaque was drawn through the canal system by a vacuum, and the teeth were radiographed in mesio-distal and bucco-lingual planes. By this method they observed that 59% had accessory canals in the coronal-and middle-thirds of the molar roots, with no significant difference between maxillary and mandibular molars. However, in preliminary studies, such canals could not be consistently demonstrated in teeth which had not been root planed.

The following year Koenigs, Brilliant, and Foreman[38] published their scanning electron microscopic investigation of accessory foramina in human molar teeth. The foramina varied in size from four microns to 250 microns and occupied random positions in the cementum web. A larger number of accessory canals and greater canal diameters were found in the maxillary molar. At the same time dentinal tubules were examined, revealing that tubule numbers per unit surface area were similar, but orifice size was not. In the root canal area just apical to the furca, the tubules measured approximately one to two microns, whereas those of the pulpal floor measured approximately two to four microns. Surface morphology and texture of the two areas were also very different. Although this premilinary study did not disclose the numbers reported by Kramer[19] and Moss[28], who studied primary molars, the presence of lateral

canals in the furca was confirmed.

In 1974 Vertucci and Williams[39] presented their study of furcation canals in human mandibular first molars. One hundred of these, randomly selected, were decalcified and internally stained with hematoxylin dye before observation with a dissecting microscope. Forty-six per cent of these teeth exhibited lateral canals in three distinct patterns. In thirteen per cent of the specimens a single lateral canal extended from the floor of the chamber to the interradicular region. Of these 57.1 per cent exited from the center of the pulpal floor, 28.5 per cent from the mesial, and 14.4 per cent from the distal. In the coronal one-third of the root, 23 per cent were found to have lateral canals extending to the furcation. These usually occurred singly, 80 per cent from the distal root, and twenty per cent from the mesial. Finally, ten specimens (ten per cent) manifested both lateral and furcation canals, of which in half of the specimens the canals merged before exiting into the furca.

The floor of the chamber averaged 3.9 mm in depth with a range of 2.5 to 7.0 mm. Interestingly, in most cases the greater the thickness, the higher the incidence of both lateral and furcation canals.

The same year, Burch and Hulen[40] published their study of the foramina and topography of molar furcations in 95 maxillary and 100 mandibular permanent molars which were stained with permanent blue ink and examined with a dissecting microscope. Seventy-six per cent were found to have furcation foramina (147 of 195 teeth). Of these, maxillary molars average 2.51 foramina per tooth, while mandibular molars

averaged 2.14. The range was from one to fourteen foramina in mandibular molars, and between one and ten in maxillary molars. The findings of Everett, et al.,[18] concerning the bifurcational ridge in mandibular molars were also confirmed.

In 1975 De Deus[41] presented his work concerning the frequency, location, and direction of all types of accessory canals found in 1,140 dyed permanent human teeth. He found that 27.4 per cent had accessory canals, with 10.4 per cent displaying lateral canals. In the furcation areas of molars and premolars, only 2.3 per cent demonstrated lateral canals, none of which emanated from the pulp chamber.

Hattler, Snyder, Listgarten and Kemp[42], 1977, studied pulpal pathosis in rice rats with the periodontal syndrome. Their results agreed with Mazur and Massler[26] that periodontal disease did not affect the health of the pulp, and noted the incidence of lateral canals to be twelve per cent.

In 1978, Gutmann[43] published his study of accessory canals in molar furcation regions. Safranin dye was drawn from the pulp chamber to the external root surfaces by vacuum on 102 maxillary and mandibular first and second molars. Twenty-nine teeth (28.4%) exhibited patent lateral canals in the furcation region; while in 25 of these (24.5%) he found lateral canals in the furcation. Canals on lateral root surfaces averaged 10.2% of the sample. No significant difference was noted between maxillary and mandibular molars.

The same year Walton and Langeland[44] presented a study on the

migration of materials in the dental pulps of four monkeys. Sections of sixteen teeth did not reveal any lateral canals in the cervical one-third of any of the teeth. They did not state whether or not complete serial sections were viewed.

Other factors

Two other possible factors concerning communications with the furcation area require consideration: dentinal tubules and migration.

Cahn[4] concluded in 1926 that although a dentinal-cemental communication may not always exist, it must occur sufficiently often to justify stating that a protoplasmic connection between the periodontium and the pulp is more than an accidental occurrence and must be taken seriously into consideration as an existing condition.

In 1933 Fish[45] published a work in which he placed methylene blue dye into the pulp chamber of extracted teeth at 37°C. for 24 hrs. Sections showed penetration of the dye only to the dentinal-cemental junction.

Marshall, Massler, and Dute[46] used radioisotopes in 1960 to study dentin permeability. Although their results only demonstrated significant permeability changes due to canal preparation or medicaments, none of the isotopes penetrated beyond the dentino-cemental junction. They cited Going's thesis[47] which showed that isotopes placed outside the tooth penetrated the cementum only to the cemento-dentinal junction. However, Wassermann et al.,[48] in 1941 studied the penetration of radioactive phosphorous in vivo and found that the cemento-dentinal junction did not

act as a barrier.

In 1963 Stahl[49] studied the pulpal response to gingival injury and and demonstrated irregular dentin-formation at the pulpal surface opposite the site of injury in 21 of 69 adult rats. He postulated that this was associated with the irritation of odontoblastic processes possibly by inflammatory products penetrating permeable cementum and dentin.

The next year Moss et al.,[28] concluded from his study that in infected cases the dentin and cementum was histologically changed and allowed a constant flow of material directly through the pulpal floor.

Koenigs, Brilliant, and Foremen[38], studying the furcations of human permanent molars in 1974, measured the tubules and found that apical to the furca the tubules were one to two mm. in diameter, but those in the furcal region measured two to four mm.

In 1978 Gutmann's[43] dye study of molar furcation canals demonstrated that the dye also penetrated the dentinal tubules, but never passed through the dentino-cemental junction.

Later that year, Walton and Langeland[44] confirmed the work of other investigators who demonstrated that a variety of material and substances, when placed in direct contact with pulpal tissue, will migrate within the pulp and through the apical foramen to adjacent supporting tissues and even into the systemic circulation. One interesting finding was the presence of Kerr's (Rickert's Formula) Sealer particles in the coronal periodontal ligament and gingiva. No particles were seen in the dentinal tubules and no accessory canals were found. They postulated that these

particles were transported there through pulpal lymph channels.

FORMOCRESOL

The use of formocresol in dentistry began in 1904 when Buckley[50] introduced it for the treatment of putrescent pulps, claiming that this mixture would convert the necrotic pulpal contents into a sterile, stable and odorless mass. He mentioned two formalae for formocresol: one was prepared by mixing tricresol and formalin in equal amounts (for the treatment of putrescent pulps) while the other consisted of two parts tricresol and one part formalin. The latter, known today as "Buckley's Formocresol" was used to treat abcesses without sinus tracts.

In order to select drugs rationally to treat these conditions, he picked those agents able to unite chemically with the end products of pulp decomposition. One such agent is formaldehyde, which when united with ammonia forms a solid colorless and odorless compound, urotropin. Formaldehyde also chemically binds hydrogen sulfide and basic ptomaines to form nonaromatic compounds. However, he said, formalin was too strong for general use and fatty acid metabolic end products remained unchanged. A mixture of ortho-, meta-, and para-cresol (tricresol) was selected to dilute the formalin since they mix easily, form a good germicide (phenol coefficient of 3) and acts chemically on the fatty constituents of the pulp. However, he stressed that the formaldehyde must be confined to the tooth, as it one of the most irritating agents known.

Buckley had intended that formocresol be used only on nonvital tissue to clear up infection and prevent its recurrence. Vital exposures

were still being devitalized with arsenic at that time. In 1922 Jordan[51] discussed the treatment for pulpally involved primary molars and recommended formocresol application after devitalization with arsenic. Prior to this, vital exposures were devitalized with arsenic followed by application of Gysi' Triopaste, a paraformaldehyde (polymerized formaldehyde) - tricresol mixture.

Several forms of formaldehyde had been in use for many years previously. Bonnecker[52] converted to vital pulp amputation in 1893, using a formalin-thymol mixture in the treatment of infected pulps. He found that the formaldehyde penetreated to the root apex in two to twelve hours, changing the pulp into a stiff fibrous tissue resistant to infection. Lepkowski[53] (1897) treated 4,679 teeth with "Formagen", a paraformaldehyde compound, and claimed less than one per cent failure. He reported that the pulp became quite hard and insensitive after such treatment.

In 1897 Bonnecker[52] reported 500 cases treated with formaldehyde solutions after arsenical cauterization. After removing its contents, the pulp chamber was washed with a five to ten per cent solution of formaldehyde and he then filled the chamber with a formalin paste. He said the formaldehyde caused a coagulation necrosis and death of the pulp tissue in the canals.

Gysi's Triopaste was introduced in Switzerland in 1899 for the treatment of gangerenous pulps; it consisted of tricresol, creotin, glycerin, paraformaldehyde, and zinc oxide.[54] Gysi's Triopaste, a forerunner of formocresol, was widely used.

Many years later, in 1929, Hess[55] reported on a series of experiments

performed under his direction by Meyer in 1917. Pulps of dog's teeth were devitalized with arsenic and treated with Gysi's Triopaste for one year. After histological examination he concluded that the devitalized and mummified pulp remained as a thoroughly impregnated, structurally intact tissue without shrinkage. The necrobiotic zone consisted of granulation tissue infiltrated by leucocytes and lymphocytes, while cementum was laid down in the apical foramen; the area was sterile. Mueller[56] reported similar results in 1919.

A few years later Sweet[57] (1923) discussed the treatment of pulpally involved primary teeth. He recommended repeated treatments with formocresol and instrumentation of the occlusal two-thirds of the canal, finally filling the canal with a creamy paste of carbo-eugenol and silver nitrate.

That same year Lutz[58] reported his histological finding on amputated pulps cauterized with cobalt and covered with Gyri's Triopaste. Sixty-two human teeth examined histologically one to six years after treatment demonstrated that the pulps were replaced by granulation tissue from the periapical tissue through the foramen. Deposition of secondary cementum on the canal walls resulted in closure of the foramen and he found that the pulps were all sterile.

In 1930 Sweet[59] revised his technique suggesting that in exposed primary teeth only the pulp in the chamber be removed, the stumps treated with formocresol and sealed with zinc oxide and eugenol. Treatment of teeth with putrescent pulps were similarly handled.

Two years later Coolidge[60] published his results on the reaction of dog tissue to root canal medications. In some cases formalin caused considerable cellular infiltration of the periapical tissue, while formocresol reaction consisted of a small localized coagulated area in the periapical tissue at the apical foramen with very little cellular infiltration. It seemed to Coolidge that the protein-coagulating drugs had less penetrating ability than the non-coagulating medicaments, and thus the damage was less intense.

The next year Orban[61] reported on his histological examination of fourteen dog teeth which had paraformaldehyde applied to the pulp. The pulps underwent necrotic changes and inflammation extended into the periapical tissues. When a ten per cent paraformaldehyde-zinc oxide mixture was sealed in the cavity, secondary dentin formation was stimulated with no pulp damage. However, a 25 per cent paraformaldehyde-zinc oxide mixture sealed in the cavity changed the pulp to fibrous connective tissue.

By 1937 Sweet[62] abandoned arsenic as a devitalizer, and used local anesthesia to remove the coronal pulp. Two separate treatments of formocresol, each for two to three days, were performed followed by a creamy covering of zinc oxide and eugenol, followed by a zinc phosphate cement base and a permanent restoration.

After many years of little work on the subject, Low and Krasnow[63] (1950) recommended that paraformaldehyde (1%) be incorporated into a zinc oxide-eugenol paste for treatment of exposed vital pulps. They believed that a low concentration of paraformaldehyde would be antiseptic and induce secondary dentin formation rather than mummify the pulp.

They reported a clinical success rate of 90.7 per cent for primary teeth and 88.7 per cent for permanent teeth so treated.

In 1956 Nacht[64] treated 456 primary teeth with Oxpara (made from: a liquid containing phenol, formalin, cresote and thymol; and a powder made of barium sulfate, iodine, and paraformaldehyde). In a one visit treatment he removed the majority of the pulp under anesthesia, applied a thick layer of the formaldehyde paste, and covered this with a cement base and amalgam. Thirty of the 456 cases were lost due to acute symptoms. In general, radiographic evidence demonstrated a progressive loss of bone in the furcation after two years, resulting in early exfoliation and eruption of the secondary teeth.

Two years later Handler[42] presented the results of his study of formocresol in contact with vital pulp tissue. That pulp in contact with the formocresol for a short time (about three minutes) demonstrated acute inflammation, while prolonged contact (over fourteen days) resulted in amyloid degeneration of the entire pulp.

The same year Wong[66] reported his study of the effects of paraformaldehyde paste on the pulp and periapical tissues of Rhesus monkeys. Six permanent and 28 primary teeth were treated by pulpotomy and examined nine, 37, 48 and 84 days after treatment. All teeth exhibited pulpal changes ranging from inflammation and atrophy at the contact zone to degenerative changes in the surrounding bone. He noted that the pulp responded differently in the primary teeth, exhibiting fibrosis, while in permanent teeth he found bone growing into the canal as well as dentin deposition on the canal walls. If in contact, the paste could destroy the

developing follicle.

In a 1959 master's thesis, Mansukani[67] investigated the effects of formocresol-saturated pellets on the pulps of 43 human primary and permanent teeth. He reported that the pulpal surface immediately under the formocresol (Buckley's formula) became fixed within a few minutes after application. After seven to fourteen days exposure to the formocresol, three distinct zones became evident: (1) a broad acidophilic zone of fixation, (2) a broad pale-staining zone with diminished numbers of cells and fibers indicating atrophy, (3) a broad zone of inflammatory cells concentrated at the junction with the pale staining zone and deeply diffusing into the underlying tissue to the apex. Mansukani concluded that formocresol produced a progressive fixation of the human pulp when applied for seven days or more, and a complete fibrosis when applied for 60 days or more.

Emmerson, Miyamoto, Sweet and Bhatia[68] (1959) studied the effects of formocresol on the pulp tissue of fifteen asymptomatic human primary teeth for up to eight weeks. Changes in the primary pulps varied with application time from surface fixation to complete calcific degeneration. These investigators concluded that the use of formocresol in pulp therapy may be classified as either a vital or non-vital technique depending on the length of time of formocresol application.

Also in 1959 Schilder and Amsterdam[69] tested the inflammatory potentials of several root canal medicaments. Formocresol, among others, produced severe inflammation when injected intradermally in rabbit abdomens, or when deposited in the conjunctive of the rabbit eye.

Two years later Dietz[70] described the effects of formocresol placed over the pulp stumps of primary cuspids for seven days followed by zinc oxide and eugenol for 24 hours to 16 weeks. He noted that the pulpal tissue seemed to be separated from the surface necrosis by the formation of an acellular collagenous band. This was first seen at 24 hours, widened in successive time periods, and began degenerating along the pulpal canal. No secondary dentin was found and inflammatory responses were not seen until the eighth week and then only to a mild degree. The pulpal tissue seemed to produce a new pulpal network of proliferating young fibroblasts. This network was found immediately below and infiltrating into the collagenous band at the second week; however, by the sixteenth week, most of the pulp was necrotic.

Also in 1961, Torneck[71] published his study of the reaction of hamster connective tissue to various root canal medication. Using punctured polyethlene tubes implanted in hamster abdomens, he noted a purulent exudate at the tube border with an intense, active inflammation after 48 hours. Necrosis of connective tissue occurred near the punctures. At 96 hours, an extensive subacute inflammation around the tube was still present with some coagulation necrosis of connective tissue noted in an area next to the puncture.

The same year Doyle[72] published his master's thesis comparing formocresol and calcium hydroxide pulptomomy techniques on 65 human primary teeth in a study ranging from four to 388 days. The formocresol was sealed in the pulp chamber from four to seven days, and at the second

appointment the chamber was filled with zinc oxide mixed with equal parts of formocresol and eugenol. He found that radiographically 93 per cent were successful, while only 71 per cent were histologically successful. No calcific degeneration as reported by Emmerson, et al.,[68] was observed, and in fact, the time span of formocresol application did not appear significant, as little difference in effect was seen in those treated four days or those treated for more than a year.

Two years later, Spedding[73] reported on formocresol versus calcium hydroxide pulp therapy in monkeys. Of twenty primary teeth subjected to pulpotomy, formocresol was applied for five minutes, then the pulp stumps were covered with zinc oxide mixed with formocresol. Histological examination after seventeen to 286 days of treatment indicated that all twenty teeth had evidence of vital tissue in the apical one-half or one third. The other region appeared fixed. However, some of the pulps exhibited leucocytic infiltration and osteodentin formation in the apical region.

In 1965 Berger[74] reported the reaction of pulp tissue to formocresol and zinc oxide-eugenol. In 35 primary teeth with cariously involved vital pulps, the coronal pulp was removed, formocresol applied for five minutes and a sub-base of formocresol, eugenol and zinc oxide placed. Histologic examination was begun at three weeks and concluded at 38 weeks. Beginning in the seventh week, granulation tissue was seen growing through the apex and progressing coronally in time until it approached the amputation site. Ninety-seven per cent were judged radiographically successful.

Berger reported that this granulation tissue might even change to osteodentin and narrow the canal lumen. He concluded that the clinical success of formocresol is related to the ability of formaldehyde to bind to tissue and render it incapable of autolysis, but still able to be replaced by granulation tissue.

The next year Beaver, Kopel and Sabes[75] reported on the effects of zinc oxide-eugenol cement on pulps that had been treated with formocresol. They did not observe the ingrowth of granulation tissue as described by Berger[74], but reported a morphologic change in the pulp tissue to a more fibrotic appearance (as in aging). The authors concluded that once the formocresol initiated a pulpal response, it was no longer necessary to incorporate it into the sub-base.

The effect of formocresol on hamster connective tissue cells was studied by Straffon and Han[76] in 1960 through the examination of sponge implants in femur wounds using proline-H^3 injection and quantitative radioautography. In all animals (24) treated with formocresol, a definite reduction in the number of inflammatory cells was noted. By the tenth day both the experimental and control areas exhibited comparable healing, leading the authors to conclude that formocresol does not interfere with prolonged recovery of connective tissue and might suppress initial inflammatory response significantly.

In 1970 the same investigators[77] studied the effects of an alternate concentration of formocresol on RNA synthesis of rat connective tissue. The one-fifth dilution of formocresol had the same effect as full strength

in suppressing the RNA synthesis of fibroblasts in connective tissue, but concentrations lower than 1/25th were not effective in fixing the tissue. Combining results of this and the previous study, they concluded that formocresol at full concentration exerts its clinical effect by a thorough fixation of the tissue which blocks the synthesis of the connective tissue proteins and ribonucleic acids as well as suppressing all of the respiratory enzymes. Similar cytotoxic effects can be produced at a one-fifth concentration, but with functional recovery of the affected tissue in fourteen days.

Loos and Han[78], in the following year, conducted an enzyme-histochemical study of the effects of various concentrations of formocresol on connective tissue. They noted that the enzymes involved in cellular respiration are far more sensitive than those related to hydrolytic activities, which seem to survive the cytotoxic effects of formocresol. Little differences are noted between the full concentration of formocresol and a one-fifth dilution in terms of initial effects of tissue fixation; however, a one-fith dilution resulted in an earlier recovery of enzyme activities.

In a 1972 in vitro study of the antibacterial efficacy and cytotoxicity of three endodontic drugs, Vander Wall, Dowson and Shipman[79] sealed formocresol, cresatin, and camphorated para-mono-chlorophenol in endodontically prepared teeth and placed them in contact with the bacterial growth on culture dishes. Formocresol was the most effective antibacterial drug and the only effective drug when not in contact with the bacteria. The cytoxicity section of the study indicated that clinical

doses of formocresol, if confined to the pulp chamber of the tooth were relatively non-toxic.

In the same year, Boller[80] reported on the reaction of pulpotomized teeth to zinc oxide and formocresol in both humans and monkeys. At the time of extraction the radiographic appearance of the human teeth in the one, three and six month groups appeared to be unchanged; but at twelve and eighteen months the relative size of the periapical and interradicular radiolucencies (22 of 57 had preoperative radiolucencies) appeared to have enlarged. Additional radiolucencies did not appear. The monkey teeth preoperatively and prior to extraction did not disclose any radiographic evidence of decay, periapical or interradicular resorption or rarefaction.

The following year, 1973, Kelley, Bugg and Skjonsky[81] compared formocresol and Oxpara pulpotomies in Rhesus monkeys. Regardless of the pulpal contact time, results were the same for both materials. Initial samples showed the coronal one-third of the pulp to be well fixed, blending into a necrotic middle one-third, while granulation tissue occupied the apical one-third. However, by 260 days the canals were completely filled with granulation tissue.

Also, in 1973 Kennedy, El-Kafrawy, Mitchell and Roche[82] studied formocresol pulpotomies in dog's teeth with induced pulpal and periapical pathosis. They found radiographic interpretation possible only with mandibular teeth. Of these, five of ten permanent teeth had radiolucencies prior to the application of formocresol. However, after sacrifice,

seven of these ten were termed failures due to persistent or increased radiolucencies.

Histologically, furcation pathosis (usually foci of chronic inflammatory cells) was seen in ten permanent (out of twenty-six) and in all sixteen primary teeth. Microscopic accessory canals were detected in two primary teeth. Thus, Kennedy and associates concluded that the radiographic and histologic alveolar bone levels and the low incidence of lateral canals indicated that the furcation lesions were due to periodontal disease.

Two years later, Morawa, Straffon, Han and Corpron[83] clinically evaluated pulpotomies using a 1:5 dilution of formocresol made by first preparing the dilutent (three parts glycerin, one part distilled water), and then mixing four parts dilutent with one part Buckley's formocresol. They claimed that based on routine clinical and radiographic examinations the pulpotomies using a one-fifth concentration of formocresol were as effective as the pulpotomies utilizing the full strength agent. They were not surprised as both concentrations are potent cytostatic medicaments exceeding the concentration necessary for the histological preparation of tissue.

In 1975 Ranly, Montgomery and Pope[84] studied the <u>in vitro</u> loss of ^{3}H-formaldehyde from zinc oxide-eugenol-formocresol cement. Their results indicated little or no chemical binding of formaldehyde by zinc oxide-eugenol, since up to 80% can be lost with only minimal loss of the eugenol matrix. Apparently, the zinc oxide-eugenol cement acts as

a physical matrix indicating that application of formocresol by saturated pellet may be an unnecessary step.

A 1976 article by Willard[85] noted that the most common radiographic change in primary molars after formocresol pulpotomy was calcification of the root canals (twenty-four of thirty). He reasoned that fixation of vital pulp tissue with formocresol probably does not cause complete loss of vitality.

Also in 1976 Ranly and Fulton[86] reported the reaction of rat molar pulp tissue to formocresol, formaldehyde, cresol, or glycerol (control) after five minute applications to mechanical exposures. These sites were then covered by ZOE and amalgam. Formaldehyde treated pulps healed as rapidly as control teeth, cresol delayed recovery, and formocresol recovery was in between the two. The investigators speculated that this was consistent with the property of cresol not to make permanent bonds, but rather to move from site to site.[87] In the case of formocresol, the formaldehyde's cross linking property apparently prevents the full penetration of the cresol.

The following year Pruhs, Olen and Sharma[88] studied the relationship between formocresol pulptomies on primary teeth and enamel defects on their permanent successors.[32] They noted that Binns and Escobar proposed that periapical and interradicular infection in the supporting tissue of primary teeth was a causative factor in enamel hypoplasia or the discoloration of permanent tooth enamel. The results of Pruhs, et al., indicate a definite relationship ($P<.01$) between formocresol pulpotomies in human primary teeth and enamel defects on their permanent

successors.

Towards the end of 1977 Block, et al.,[89] hypothesized that formocresol acted as a hapten which, when combined with pulp tissue, altered it into an immunogen. Their results agreed with Eleazer, Farber, and Seltzer[90] who showed that untreated root canal byproducts failed to stimulate lymphocytes. However, when pulp tissue was incubated with formocresol, noticeable lymphocyte production could occur. In another article, Block, et al.,[91] demonstrated similar results with paraformaldehyde. These investigators therefore questioned the biologic compatibility of formocresol and paraformaldehyde.

Myers and associates[92], 1978, reported on the distribution of ^{14}C-formaldehyde after formocresol pulpotomies on Rhesus monkeys. Five minute application of the ^{14}C-formocresol resulted in the systemic absorption of approximately one per cent of the dose. Although two hour exposure did not increase this systemic uptake, multiple sequential pulpotomies did result in a higher systemic absorption of ^{14}C-formaldehyde. The uptake of ^{14}C-formaldehyde peaked after thirty minutes, indicating that the absorption is a self-limiting brief, but rapid uptake in the first few minutes until vessel thrombosis occurs. This is borne out by a low uptake of ^{131}I from these sites, indicating a compromised microcirculation.

Radioautography of ^{14}C-formocresol treated teeth disclosed extensive concentrations of ^{14}C-formaldehyde in the pulp, dentin, periodontal ligament, and bone. These investigators recommend consideration of these factors when selecting agents for vital pulp therapy.

METHODS AND MATERIALS

In order to reduce the risk of the congenital absence of lateral canals in a litter or a breed, four unrelated, non-beagle mongrel dogs of approximately two years of age were obtained through the Animal Research Facility (ARF) at the Loyola University Medical Center. According to Hattler,[42] beagle dogs are prone to periodontal disease. Upon arrival the dogs were examined by the attending veterinarian to ensure their good health and later by the author to inspect their dental and gingival conditions. The dogs were kept in separate cages and fed a standard dry pellet food with water ad libitum. Their feeding and care were provided by the staff at the ARF, along with constant veterinary supervision. The dogs were identified by round tags marked with four digit numbers (2446, 2475, 2489, or 2568) which were tied around their necks. In addition the cages were so marked.

Prior to any work done on the animals all instruments were washed and autoclaved and the operating room, reserved previously, was cleaned and prepared for the dogs work. A fresh CO_2 tank was attached to the portable dental unit; and the water tank was filled.

Upon arrival at the ARF for the planned days work, injection syringes were prepared. Two milliliters of Inovar Vet*, a tranquilizer, was

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

injected IM into the hindleg, followed immediately with 1.0 ml (0.5 mg/ml) of Atropine Sulfate Injection, U.S.P., to stem salivary secretions. While these drugs became effective, the room was prepared for the days work. After fifteen minutes the animal was well tranquilized, and was easily handled. The dog was weighed, the foreleg sheared with animal clippers, and the animal carried to the prepared operatory. Here, an IV injection of Sodium Pentobarbital** (65 mg/ml) was administered. An initial dose of 3 ml. was given with additional doses of 2 ml. given when needed to maintain the required depth of anesthesia. The depth of anesthesia was checked by brushing the eye lashes, watching for a lack of corneal reflex, and by pinching the dogs toe pads.

As soon as the dog was properly sedated preoperative radiographs were taken of the premolars to observe tooth and periodontal anatomy, especially that of the furcation. Radiographs were also taken postoperatively and prior to sacrifice. Occlusal film*** was placed beneath the arch to be radiographed, the dog lying on its side, its mandible extended with a spring loaded device attached to the maxillary and mandibular canines of the contralateral arches being radiographed. Following the technique for lateral jaw radiographs, the portable, hand-held General Electric x-ray generator giving 60 KVP at 20MA was aimed from the opposite

* Med Tech, Inc. Elwood, Kansas
** W.A. Butler Co., Columbus, Ohio
*** Kodak DF-46, Eastman Kodak Do., Rochester N.Y.

side. A 0.2 second exposure time was used. After the four films were taken each was developed for 20 seconds in Insta-Neg*, rinsed, fixed for 40 seconds in Instra-Fix*, and washed for five minutes, all in a portable, light tight box.

Sedation and Anesthesia

Prior to the procedure, each animal was tranquilized with 2.0 ml. of Inovar Vet**, which was immediately followed by 1.0 ml. (0.5 mg./ml.) of Atropine Sulfate Injection,*** to stem salivary secretions.

In fifteen minutes, when the animal was well tranquilized, it was weighed, the foreleg sheared, and an IV injection of Sodium Pentobarbitol**** (65 mg/1 ml.) was administered. An initial dose of 4.0 ml. was given to each dog, with an additional dose of 2.0 ml. given when needed to maintain anesthesia of surgical depth.

At this time a complete periodontal profile was recorded to establish the sulcular (or pocket) depth of six areas of each tooth included in this study. The mandibular second, third, and fourth premolar and the maxillary second and third premolars and first molar teeth were listed as either control, operated control, or experimental tooth.

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* Micro-Copy, Culver City, California
** Pitman-Moore, Inc. Washington Crossing, N.J.
*** Med Tech, Inc. Elwood, Kansas
**** W.A. Butler Co., Columbus, Ohio

One tooth per animal was listed as the control tooth and as such was untouched. In each quadrant one tooth was selected as the operated control and was treated as follows. After pumicing, the tooth was washed, wiped with alcohol and isolated. The occlusal surface was ground to near gingival height, the pulp chamber opened with a sterile #556 tapered fissure bur, and the pulp removed with barbed broaches. All the canals were filed in a circumferential manner, two sizes beyond the first file to bind apically. Sterile saline was used as the irrigant. After the canals were dried with paper points, a cotton pellet moistened with saline and squeezed dry was placed in the chamber, and the access cavity was sealed with amalgam.

The remaining 26 teeth (the mandibular second pre-molars of the animal tagged #2446 had fused roots-no furcation-and were excluded from the study) termed experimental were treated exactly as the operated controls except that Buckley's formocresol* was sealed in the chamber instead of saline.

The pattern of control, operated control and experimental teeth in each dog is shown below.

E	O	E		O	E	E
1	3	2		2	3	1
4	3	2		2	3	4
O	E	E		E	C	O

E = experimental
O = operated control
C = control

* King's Specialty Co., Fort Wayne, Ind.

One animal was sacrificed at each of the following periods: 7 days (#2568). 30 days (#2489), 90 days (#2475), and 150 days (#2446).

At the time of sacrifice the animals were prepared as before. The restorations and the cotton pellets were removed from the teeth and final radiographs were taken before transferring the animal to the necropsy room. An intravenous injection of 10 c.c. of Beuthanasia - D*, a highly concentrated solution of sodium pentobarbitol, produced a rapid, painless lethal overdose. Immediately, the tissue was stripped from the jaws and the areas to be studied were removed with electric bone saws and placed individually in pre-marked quart bottles containing approximately 850 ml. of 10% buffered formalin. These block sections were then further reduced using a high speed handpiece and #557 bur, to aid rapid diffusion of the fixative.

Fixation continued for fourteen days, with the solution changed each day. After rinsing the jaw sections for 24 hours under running water, the specimens were decalcified for approximately 35 days in a solution of equal parts of 50 percent formic acid and 20 percent sodium citrate. When the blocks were decalcified, razor blades were used to trim the blocks and separate the teeth, each of which was trimmed as small as possible to allow the greatest numbers of sections per slide. The buccal of each tooth was marked with India Ink and each tooth numbered and placed into the decalcifying solution for a few more days. After dehydration in increasing concentrations of alcohol, the individual blocks were embedded in paraffin,

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

seven micron sections were cut, and these mounted on glass slides, and stained with hematoxylin and eosin for light microscopic examination.

RESULTS

Gross observations

All of the animals remained in good physical condition and weight gains were noted for the 90 (0.8 Kg) and 150 day (3.6 Kg) subjects. Another dog originally slated to be the 90 day animal expired two days postoperatively. The loss was attributed to starting the experiment before the dog could adapt to its new environment. After this occurence, operations were postponed for at least two weeks following each dog's arrival, to allow for adjustment to the new environment and diet.

Periodontal conditions did not change in any of the instrumented teeth. However, a furcation defect was noted on a control tooth (maxillary right second premolar) of the 90 day animal.

At the time of the sacrifice of the seven day animal, the maxillary left third premolar (saline) was found to be fractured through the furcation, probably due to the dog's chewing on the bars of the cage, as several of the amalgam restorations on the other teeth appeared worn. Also, a sinus tract was noted on the 150 day animal, labial to the maxillary right second premolar (formocresol). The remaining gingival tissues appeared healthy; firm marginal gingiva with normal epithelial attachments.

Radiographic Observations

As could be expected, no radiographic changes were noted in the seven and 30 day animals.

Ninety day animal

Initial radiographs indicated a furcation periodontal defect on the maxillary right second premolar (control). No alterations were noted after 90 days. The maxillary left second premolar developed an apparent furcation radiolucency. However, this could not be demonstrated histologically. Periapical radiolucencies were noted on the maxillary right third premolar and first molar, maxillary left third premolar and first molar, and the mandibular right fourth premolar.

One hundred fifty day animal

All operated teeth in the 150 day animal demonstrated periapical radiolucencies; however, all of the furcation areas appeared normal. External root resorption in the periapical area was seen on the maxillary right second (formocresol) and third (saline) premolars, maxillary left second premolar (formocresol), mandibular left third premolar (formocresol), and the mandibular right third premolar (formocresol). The periapical lesions of both maxillary third premolars appeared to have coalesced, encircling both roots.

Histological Results

Lateral Canals

Despite viewing complete serial sections of the 45 teeth involved in this study no evidence of a lateral canal could be found. The canal walls in all sections were perfectly straight and even as was the floor of the pulp chamber. Many artifacts were observed and disqualified.

Pathology

Inflammation in the furcation could not be detected in any of the teeth studied. All sections demonstrated completely healthy periodontal tissues in the occlusal one-half of the roots, exactly as in the controls, regardless of whether saline or formocresol was placed in the tooth. Only the 150 day animals' teeth demonstrated pathosis which approached the furca, and all of these could be traced to periapical causes (Figures 1-4).

Seven Day Animal

Sections through any of the studied teeth presented the following: an even thickness of acellular cementum lined by cementoblasts was seen adjacent to a normal periodontal ligament containing abundant numbers of spindle and stellate-shaped fibroblasts, numerous bundles of interradicular fibers, epithelial rests, and many capillaries in loose connective tissue (Figure 5). The blood vessels were not engorged and margination was not seen. Adjacent, a layer of osteoblasts lined bundle bone with a few osteoclasts interspersed demonstrating normal interradicular bone. Myeloid cells were seen in the marrow spaces in several teeth.

Thirty Day Animal

Only two teeth in this animal differed from the control tooth in the 30 day specimen. The maxillary second premolar (saline) displayed a few inflammatory cells in some slides and the maxillary right third premolar (formocresol) demonstrated a dilated furcation vessel with some hemorrhage into the surrounding marrow.

Ninety Day Animal

The maxillary left second premolar (formocresol) demonstrated a mild gingivitis with plasma cell infiltration into the periodontal ligament at the furcation (Figures 6-11). The mandibular right quadrant demonstrated an increased amount of autolysis and degeneration, especially in the fourth premolar (Figure 12). Also, some scattered plasma cells were noted in the second (saline) and third (formocresol) premolars.

One Hundred Fifty Day Animal

In this animal the only inflammation noted was due to coronal spread from the root apices (Figure 1), especially in the maxillary left third premolar (formocresol) and the mandibular left fourth premolar (saline).

DISCUSSION

The literature indicates that lateral canals allow for the interchange of irritants between the pulp canal system and the periradicular tissues. Therefore, products of pulp degradation may escape through these canals into the periodontal ligament and surrounding tissues without going the extended distance to the apex of the tooth. Similarly, inflammatory products in the periodontal ligament may reach the pulp by traveling through lateral canals.

Therefore, in treating any pulpal conditions, the medications used may traverse any present lateral canals and exit into the adjacent periodontal structures. As it is common practice to place formocresol in the pulp chamber when performing pulpotomies and pulpectomies, it is possible that this potentially irritating fixative might have some effect on the interradicular tissues either because the lateral canal acts as a highway for the formocresol itself, or for the products of the effects of formocresol. Observing this relationship was the objective of this study. Thus, the complete absence of lateral canals in the forty-five teeth studied was very disturbing. Although a lateral canal (a branch of the main root canal which is approximately perpendicular to it) could not be found, several sections displayed apical canals (a branch of the main root canal in the apical one-third of the root). According to Seltzer, Bender, and Ziontz[24] a lateral canal contains capillaries, pulp cells, ground substance, and fibers. However, many canals are very small and contain only small caliber vessels and their supporting stroma. Thus,

these structures could easily be differentiated from artifacts such as cracks, gouges, and tears resulting from slide preparation, which were seen in many sections. Moss, et al.,[28] reported the lateral canals they found measured five to seven microns in diameter. Something of this size could not be missed in serial sections. Somewhere along its course from the pulp canal to the surface of the tooth at least one section of the canal would have been noticed, as the apical canals were noticed.

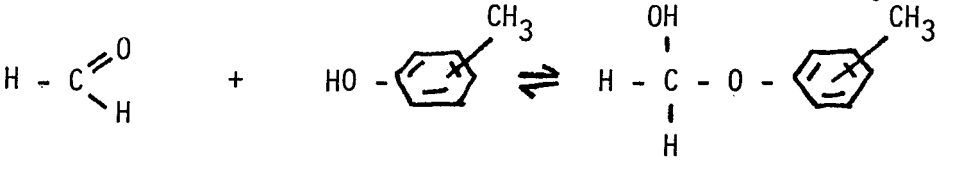
Based on statements by Seltzer, Bender, Nazimov, and Sinai,[33] we had expected to find several examples of lateral canals in each animal. As the lateral canal was a basic aspect of this study, a great deal of time and expense was wasted in sectioning, slide preparation and the viewing of these slides. Our confidence in finding the promised lateral canals led us to choose vertical sections (mesio-distal). However, in hindsight, much effort could have been saved by screening the animals to be studied. Representative teeth taken from each animal could be cleared, stained, and examined for lateral canals. If none were found in that animal, that animal would be eliminated from the study. One or two horizontal sections of the area between the pulp chamber and the furcation should reveal any furcation canals. If such a canal was found this tooth could then be sectioned vertically to demonstrate the lateral canal and furcation together. Another possibility was to examine one quadrant per animal, eliminating any animal where lateral canals were not found.

The shepherd-type mongrel dogs used in this study were easily handled, and their teeth were large enough to instrument with a #25 file as

the initial instrument. The coronal segment, although narrow, allowed direct access to straight canals with "dead-end" stops at the apical delta. The dogs tolerated the procedures well, if left to adjust to their new surroundings for several days before beginning the experiments. Experimental trauma, added to the trauma of a new environment, and new diet resulted in the demise of one animal two days postoperatively.

According to Hattler, et al.,[42] very few laboratory animals develop inflammatory periodontal disease readily. Monkeys, marmosets, beagle dogs, miniature pigs, and rice rats are among those susceptible to such a problem. Non-beagle dogs were ordered as a healthy periodontium, especially at the furcation, was a requirement. Regardless of the advantages of the experimental model the lack of lateral canals negated their value. Other animal models though not as easy to operate because of their small size would be much less expensive to keep and to prepare histologically (smaller teeth mean fewer slides). Winter[20] studied kittens with success, while Mansukani[67] used white rats successfully. Using these animals as the experimental model would allow greater numbers to be operated, thus increasing the probability of finding lateral canals.

Formocresol is a mixture of formaldehyde (CH_2O) and tricresol (methylphenol). Together they react to form a much larger hemiacetal molecule, which reduces the fixatives diffsuion ability.



Formaldehyde Tricresol Hemiacetal

"A satisfactory fixation requires an excess of formocresol and a long period of interaction. Either of these requirements results in undesireable effects."[93]

Was formocresol different than saline? Histologically, no difference could be demonstrated in the furcation. Clinically, radiographically, and histologically the furcation areas were normal in all of the samples. In some teeth, necrotic tissue remnants were visable in fine areas of the canals; however, all were in a necrotic, amorphous state. Mansukani's[67] picture of formocresol fixation [(1) acidophilic zone of fixation followed by (2), a pale staining zone of a few cells indicating atrophy, and (3), a zone of inflammatory cells] was not noted since very little tissue remained, and it was not in direct contact with the formocresol.

Radiographically, external root resorption was noted on five teeth on the 150 day subject, four of these were treated with formocresol, one with saline. Unfortunately, this could not be demonstrated histologically, as most of the apices were trimmed away in order to allow faster fixation, center attention in the furcation, and to place more sections on each slide. This seemed to be very important when we were contemplating the number of slides to be viewed. The importance of saving all histological material related to the experiment must be emphasized, as the point of view of the study may require change.

Initially, we had thought that radiographs of the animals were going to take an extraordinary amount of time, especially after attempting a few intraoral periapical radiographs taken with a hand-held X-ray

generator. This led us to attempt the extraoral views using occlusal film, which facilitated the radiographic section of this study in addition to providing images of good quality.

SUMMARY AND CONCLUSIONS

In an effort to discover the effects of formocresol on the furcation area of dog's molar teeth and any role that lateral canals in this area may play, pulpectomies were performed on 42 teeth of four dogs, and either formocresol or saline was placed in the pulp chamber. One tooth in each dog served as the control. The animals were sacrificed at seven, 30, 90, and 150 postoperative days. The material was then prepared for microscopic examination. These results were obtained:

1. No lateral canals were found in any of the teeth studied, despite serial sectioning.
2. No effect on the furcation could be found due to any of the procedures.

The following conclusions have been drawn:

1. Lateral canals are not "profusely evident" as indicated by Seltzer et al.[24]
2. If lateral canals are not present the furcation area is unaffected by the pulpectomy procedure; also neither formocresol nor saline placed in the chamber affected the furcation area.
3. If one wishes to study roles played by lateral canals, one should screen the model to be sure of finding lateral canals.
4. It is vitally important to save all histological material related to the experiment, as one may wish to change the point of view of the study.

REFERENCES

1. Orban's Oral Histology And Embryology, 7th ed., Sicher, H. and Bhaskar, S.N., editors. C.V. Mosby Co., St. Louis, 1966, p. 31.

2. Schour, I. and Massler, M. Studies in tooth development. J.A.D.A. 27:1785, 1918, 1940.

3. An Annotated Glossary Of Terms Used In Endodontics, 2nd ed., Maurice, C.G. Amer. Assoc. of Endodontists. Atlanta, 1973, p. 7.

4. Cahn, L.R. Abnormally large channels seen in pyorrhetic teeth. Dent. Items of Interest 48:477, 1926.

5. Barrett, M.T. The internal anatomy of the teeth with special reference to the pulp with its branches. Dental Cosmos 67:581, 1925.

6. Scott, J.H. and Symons, N.B.B. Introduction To Dental Anatomy. E.S. Liningstone, Edinburgh and London, 1971, p. 231.

7. Stallard, R.E. Periodontic-endodontic relationships. Oral Surg. 34:314, 1972.

8. Prieswork, G. Die Pulpamputation, eine klinische, patho-histologische und bakteriologische studie. Oesterr-ung. V.f. Zahnheilkunde, 17:145, 1901.

9. Dorland's Illustrated Medical Dictionary, 24th ed., W.B. Saunders Co. Philadelphia, 1965.

10. Fischer, G. Uber die feinese Anatomie der Wurzel-kanale menschlicher Zahne. (Vorlaufige Mitteilung). Deutsche. Monats. fur Zahnheilkunde, 25:544, 1907.

11. Fasoli and Arlotta. Sull Anatomica dei canali radicolari dei denti umani. La Stomatologia, 6:409, 1913.

12. Hess, W. The Anatomy Of The Root Canals Of The Permanent Dentition. William Wood Co. N.Y. 1916.

13. Rottenbitter: Zur Frage der Wurzelkanalramificationen. Oesterr-ung. V. F. Z. 34, 1918.

14. Johnston, H.B. and Orban, B. Interradicular pathology as related to accessory root canals. J. Endodontia 3:21, 1948.

15. Weine, F.S. Personal Communication.

16. Russell, L.H. and Kramer, I.R.H. Observations on the vascular architecture of the dental pulp. J.D. Res. Abst. 35:957, 1956.

17. Saunders, R.L. X-ray microscopy of human dental pulp vessels. Nature 180:1353, 1957.

18. Everett, G.G., Jump, E.G., Holder, T.D. and Williams, G.C. The intermediate bifurcational ridge: a study of the morphology of the bifurcation of the lower first molar. J. Dent. Res. 37:162, 1958.

19. Kramer, I.R.H. The vascular architecture of the human dental pulp. Arch. Oral Biol. 2:177, 1960.

20. Winter, G.B. A preliminary investigation into abcess formation in deciduous teeth. J. Dent. Res. 39:112, 1960, (Abstract).

21. Winter, G.B. and Kramer, I.R.H. Changes in periodontal membrane and bone following experimental pulpal injury in deciduous molar teeth in kittens. Arch. Oral Biol. 10:279, 1965.

22. Appelbaum, E. Internal resorption of teeth. Dental Cosmos 76:847, 1934.

23. Nicholls, E. Lateral radicular disease due to lateral branching of the root canal. Oral Surg. 16:389, 1963.

24. Seltzer, S., Bender, I.B. and Ziontz, M. The interrelationship of pulp and periapical disease. Oral Surg. 16:1474, 1963.

25. Simring, M. and Goldberg, M. The pulpal socket approach: retrograde periodontitis. J. Periodontol. 35:22, 1964.

26. Mazur, B. and Massler, M. Influence of periodontal disease on the dental pulp. Oral Surg. 17:592, 1964.

27. Rubach, W.C. and Mitchell, D.F. Periodontal disease, accessory canals, and pulp pathosis. J. Periodontol. 36:34, 1965.

28. Moss, S.J., Addelston, H. and Goldsmith, E.D. Histologic study of the pulpal floor of deciduous molars. J.A.D.A. 70:372, 1965.

29. Hiatt, W.H. Regeneration via flap operation and the pulpal periodontal lesion. Periodontics 4:205, 1966.

30. Coolidge, Edgar D. Endodontia, Lea and Febiger, Philadelphia, 1950.

31. Amen, C.R., Ingle, J. and Weiner, L. When is the condition of the pulp an important consideration in periodontal disease? Periodontal Abstracts 15:7, 1967.

32. Binns, W.H. and Escobar, A. Defects in permanent teeth following pulp exposure of primary teeth. J. Dent. Child 4:14, 1967.

33. Seltzer, S., Bender, I.B., Nazimov, H. and Sinai, I. Pulpitis-induced interradicular periodontal changes in experimental animals. J. Periodontol. 38:124, 1967.

34. Bender, I.B. and Seltzer, S. The effect of periodontal disease on the pulp. Oral Surg. 33:458, 1972.

35. Mandi, F.A. Histologic study of the pulp changes caused by periodontal disease. J. Brit. Endodont. Soc. 6:80, 1972.

36. Pineda, F. and Kuttler, Y. Mesio-distal and bucco-lingual x-ray investigations of 7275 root canals. Oral Surg. 33:101, 1972.

37. Lowman, J.V., Burke, R.S. and Pelleu, G.B. Patent accessory canals: Incidence in molar furcation region. Oral Surg. 36:580, 1973.

38. Koenigs, J.F., Brilliant, J.D. and Foreman, D.W. Preliminary scanning electron microscope investigations of accessory foramina in furcation areas of human molar teeth. Oral Surg. 38:773, 1974.

39. Vertucci, J. and Williams, R.G. Furcation canals in the human mandibular first molar. Oral Surg. 38:308, 1974.

40. Burch, J.G. and Hulen, S. A study of the presence of accessory foramina and the topography of molar furcation. Oral Surg. 38: 451, 1974.

41. DeDeus, Z.D. Frequency, location, and direction of the lateral, secondary, and accessory canals. J. Endod. 1:361, 1975.

42. Hattler, A.B., Snyder, D.E., Listgarten, M.A. and Kemp, W. The lack of pulpal pathosis in rice rats with the periodontal syndrome. Oral Surg. 44:939, 1977.

43. Gutmann, J. Prevalence, location, and patency of accessory canals in the furcation region of permanent molars. J. Periodontol. 49:21, 1978

44. Walton, R.E. and Langeland, K. Migration of materials in the dental pulp of monkeys. J. Endod. 4:167, 1978.

45. Fish, E.W. An Experimental Investigation Of Enamel, Dentine, And The Dental Pulp. John Bale, Sons, and Danielsson, Ltd. London, 1933.

46. Marshall, F.J., Massler, M., and Dute, H.L. Effects of endodontic treatments on permeability of root dentine. Oral Surg. 13:208, 1960.

47. Going, R.E. Marginal penetration of dental restorations as studied by dyes and radioactive isotopes. M.S. Thesis, U. of Illinois, Chicago, 1959.

48. Wassermann, F., Blayney, J.R., Sroetzinger, G. and DeWitt, T.G. Studies on the different pathways of exchange of minerals in teeth with aid of radioactive phosphorous. J.D. Res. 20:389, 1941.

49. Stahl, S.S. Pulpal response to gingival injury in adult rats. Oral Surg. 16:1116, 1963.

50. Buckley, J.P. The chemistry of pulp decomposition, with a rational treatment for this condition and its sequelae. Am. Dent. J. 3: 764, 1904.

51. Jordan, M.E. The treating and filling of deciduous teeth. Pacific Dental Gazette 30:481, 1922.

52. Boennecker, P. Modern methods of treatment of diseased pulp. Ohio Dent. J. 19:183, 1899.

53. Lepkowski, A. New inventions: Formagen. Brit. Dent. Assn. J. 18: 525, 1897.

54. Miller, S. Formocresol - a review. Bull. Phila. City Dent. Soc. 36:14, 1971.

55. Hess, W. Pulp amputation as a method of treating root canals. Dent. Items of Interest 51:596, 1929.

56. Mueller, O. Histological and bacteriological findings after pulp amputation. Dent. Cosmos 71:57, 1929.

57. Sweet, C.A. Root canal treatment in deciduous teeth, including pulp exposures. Pacific Dental Gazette 31:719, 1923.

58. Lutz, K. Histologische and Bakteriologische Untersuchungen uber die Wirkung der Triopaste bei der Pulpa Amputation. Schweiz. Mschr. fur Zahnheilk. 33:81, 1923.

59. Sweet, C.A. Treatment for deciduous teeth with exposed pulps. Wash. Univ. Dent. J. 3:78, 1936.

60. Coolidge, E.D. Reaction of dog tissue to drugs used in root canal treatment. J.A.D.A. 19:747, 1932.

61. Orban, B. Action of paraformaldehyde on dental pulp. J. Dent. Res. 13:215, 1933.

62. Sweet, C.A. Treatment for deciduous teeth with exposed pulps. Review of Dentistry for Children 4:11, 1937.

63. Low, M. and Krasnow, F. Pulpotomy - a successful pulp treatment for deciduous and young permanent teeth. N.Y. Dent. J. 16:59, 1950.

64. Nacht, M. A devitalizing technic for pulpotomy in primary molars. J. Dent. Child. 23:45, 1956.

65. Mandler, W. Preliminary report on the effects of formolcresol on vital human pulps. Presented before the American Academy of Pedodontics at St. Louis, Aug. 1958.

66. Wong, K. Effects of paraformaldehyde preparations on the periapical tissues in non-vital pulpotomy procedures. North-w. Univ. Bull. Dent. Res. 59:13, 1958.

67. Mansukhani, N. Pulpal reactions to formocresol. Master's thesis, U. of I. Chicago, 1959.

68. Emerson, C.C., Miyamoto, O., Sweet, C.A., Sr. and Bhatia, H.L. Pulpal changes following formocresol applications on rat molars and human primary teeth. J. South Calif. Dent. Assoc. 27:309, 1959.

69. Schilder, H. and Amsterdam, M. Inflammatory potential of root canal medicaments. Oral Surg. 12:211, 1959.

70. Dietz, D.R. A histologic study of the effects of formocresol on normal primary pulpal tissue. M.S. Thesis U. of Wash., Seattle, 1961.

71. Torneck, C.D. Reaction of hamster tissue to drugs used in sterilization of the root canal. Oral Surg. 14:730, 1961.

72. Doyle, W.A. A comparison of the formocresol pulpotomy technique with the CaOH technique. M.S. Thesis, Indiana U. School of Dentistry, Indianapolis, 1961.

73. Spedding, R.H., Mitchell, D.F. and McDonald, R.E. Formocresol and calcium hydroxide therapy. J. Dent. Res. 44:1023, 1965.

74. Berger, J.E. Pulp tissue reaction to formocresol and zinc oxide-eugenol. J. Dent. Child 32:13, 1965.

75. Beaver, H.A., Kopel, H.M. and Sabes, W.R. The effect of zinc oxide-eugenol cement on a formocresolized pulp. J. Dent. Child 33: 381, 1966.

76. Straffon, L.H. and Han, S.S. The effect of formocresol on hamster connective tissue cells; a histologic and quantitative radio-autographic study with proline-H^3. Arch. Oral Biol. 13:271, 1968.

77. Straffon, L.H. and Han, S.S. Effects of varying concentrations of formocresol on RNA synthesis of connective tissues in sponge implants. Oral Surg. 29:915, 1970.

78. Loos, P.J. and Han, S.S. An enzyme histochemical study of the effect of various concentrations of formocresol on connective tissue. Oral Surg. 31:571, 1971.

79. Vander Wall, G.L., Dowson, J. and Shipman, C. Jr. Antibacterial efficacy and cytotoxicity of three endodontic drugs. Oral Surg. 33:230, 1972.

80. Boller, R.J. Reactions of pulpotomized teeth to zinc oxide and formocresol type drugs. J. Dent. Child 39:298, 1972.

81. Kelley, M.A., Bugg, J.L. and Skjonsky, R.S. Histologic evaluation of formocresol and Oxpara pulpotomies in rhesus monkeys. J.A.D.A. 86:123, 1973.

82. Kennedy, D.B., El-Kafrawy, A.H., Mitchell, D.F. and Roche, J.R. Formocresol pulpotomy in teeth of dogs with induced pulpal and periapical pathosis. J. Dent. Child 40:193, 1973.

83. Morawa, A.P., Straffon, L.H., Han, S.S. and Corpron, R.E. Clinical evaluation of pulpotomies using dilute formocresol. J. Dent. Child 42:360, 1975.

84. Ranly, D.M., Montgomery, E.H. and Pope, H.O. The loss of ^3H-formaldehyde from zinc oxide-eugenol cement: an in vitro study. J. Dent. Child 42:128, 1975.

85. Willard, R.M. Radiographic changes following formocresol pulpotomy in primary molars. J. Dent. Child 44:414, 1976.

86. Ranly, D.M. and Fulton, R. Reaction of rat molar pulp tissue to formocresol, formaldehyde, and cresol. J. Endod. 2:176, 1976.

87. Gurney, B.F. Substituted phenols: Part two - cresols, cresylacetate, formocresol. Dental Digest 78:314, 1972.

88. Pruhs, R.J., Olen, G.A. and Sharma, P.S. Relationship between formocresol pulpotomies on primary teeth and enamel defects on their permanent successors. J.A.D.A. 94:698, 1977.

89. Block, R.M., Lewis, R.D., Sheats, J.B. and Fawley, J. Cell-mediated immune response to dog pulp tissue altered by formocresol within the root canal. J. Endod. 3:424, 1977.

90. Eleazer, P.D., Farber, P.A. and Seltzer, S. Lack of lymphocyte stimulation by root canal products. J. Endod. 1:388, 1975.

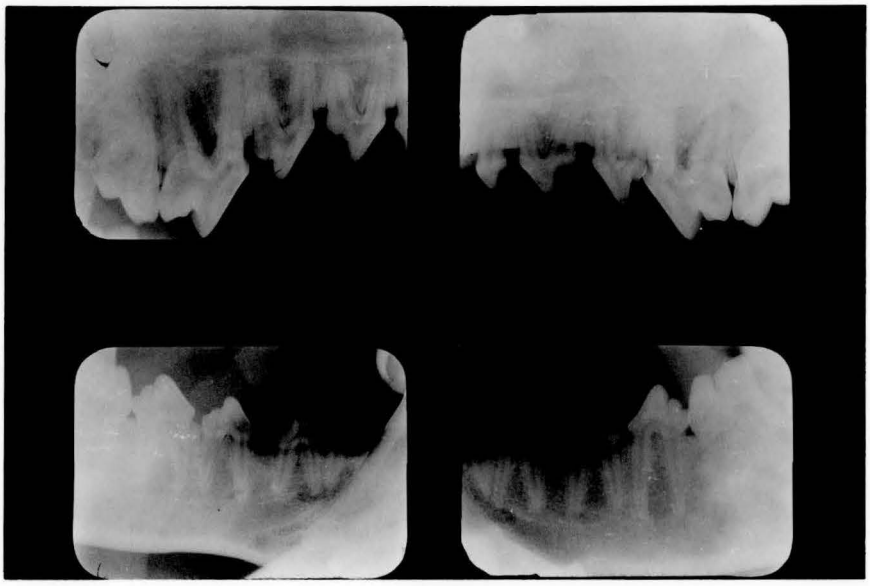
91. Block, R.M., Lewis, R.D., Sheats, J.B. and Burke, S. Cell-mediated immune response to dog pulp tissue altered by 6.5% paraformaldehyde via the root canal. J. Endod. 4:346, 1978.

92. Myers, D.R., Shoaf, H.K., Dickson, T.R., Pashley, D.H., Whitford, G.M. and Reynolds, K.E. Distribution of ^{14}C-formaldehyde after pulpotomy with formocresol. J.A.D.A. 96:805, 1978.

93. 's-Gravenmade, E.J. Some biochemical considerations of fixation in endodontics. J. Endod. 1:233, 1975.

Figure 1: Preoperative radiographs of seven day animal. Note fully developed teeth with normal periodontal tissues.

Figure 2: Preoperative radiographs of 30 day animal. Note normal dental anatomy.



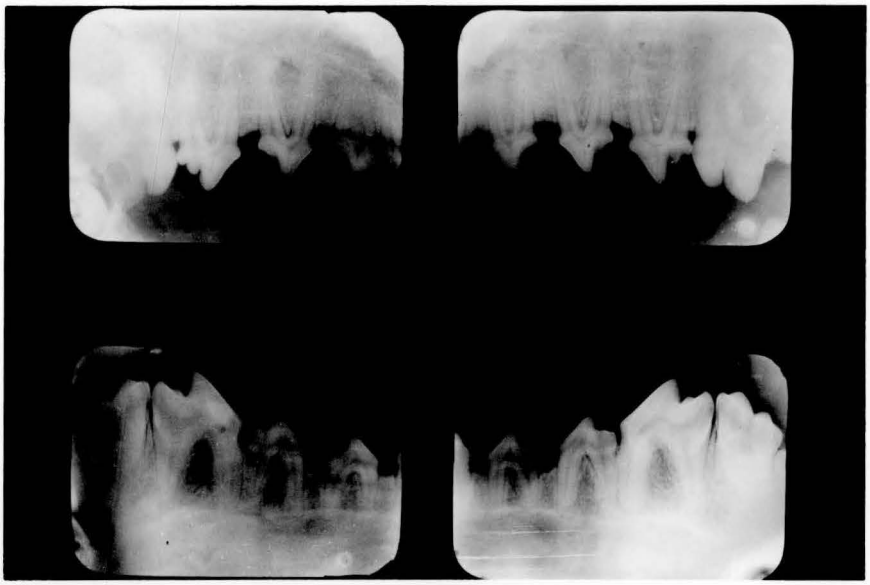
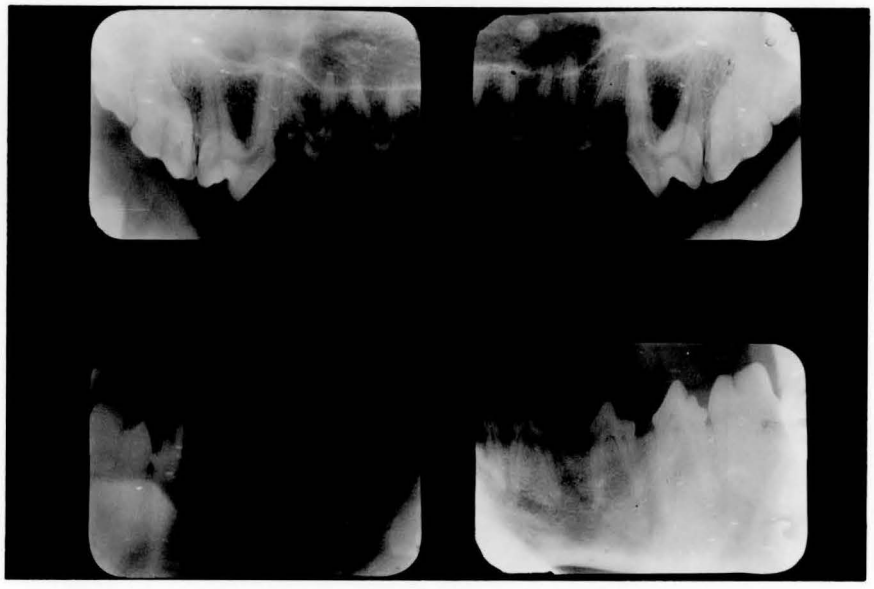


Figure 3: Preoperative radiographs of 90 day animal exhibiting normal dental anatomy.

Figure 4: Preoperative radiographs of 150 day animal. Note fully developed and normal calcified tissues.



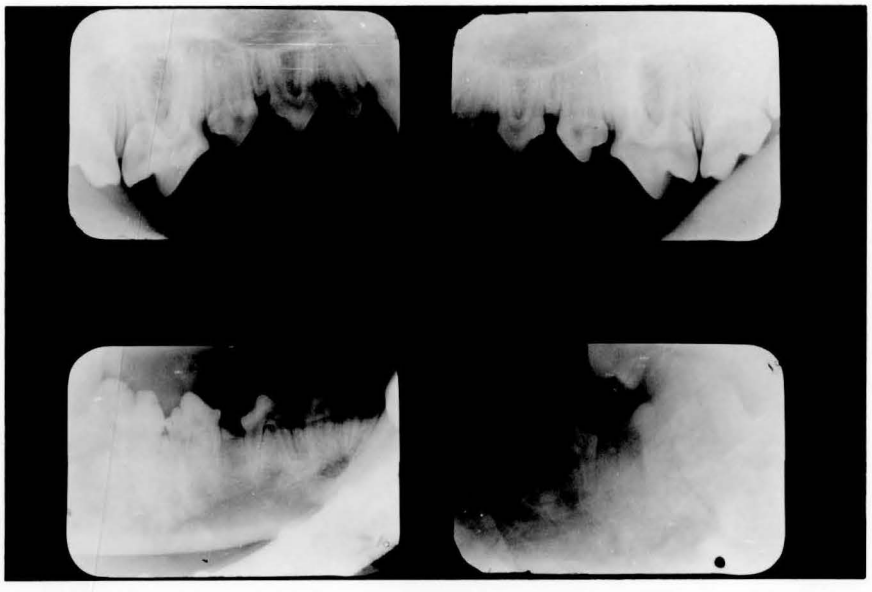
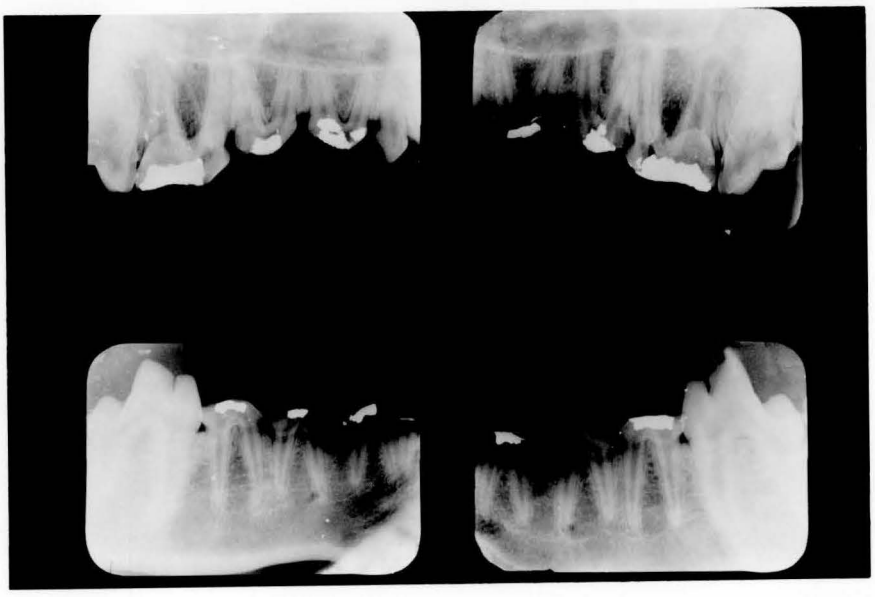


Figure 5: Postoperative radiographs of 7 day animal. Note prepared canals, amalgam restorations, and occlusal reduction.

Figure 6: Radiographs taken seven days postoperatively; immediately prior to sacrifice. Note lack of radiographic change, except for lost restoration and fracture through the furcation of the maxillary left third premolar.



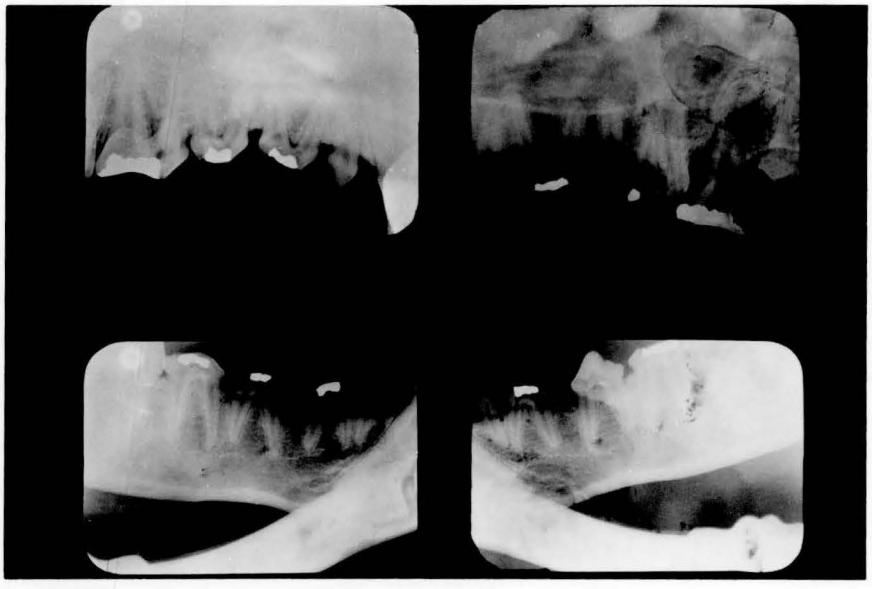
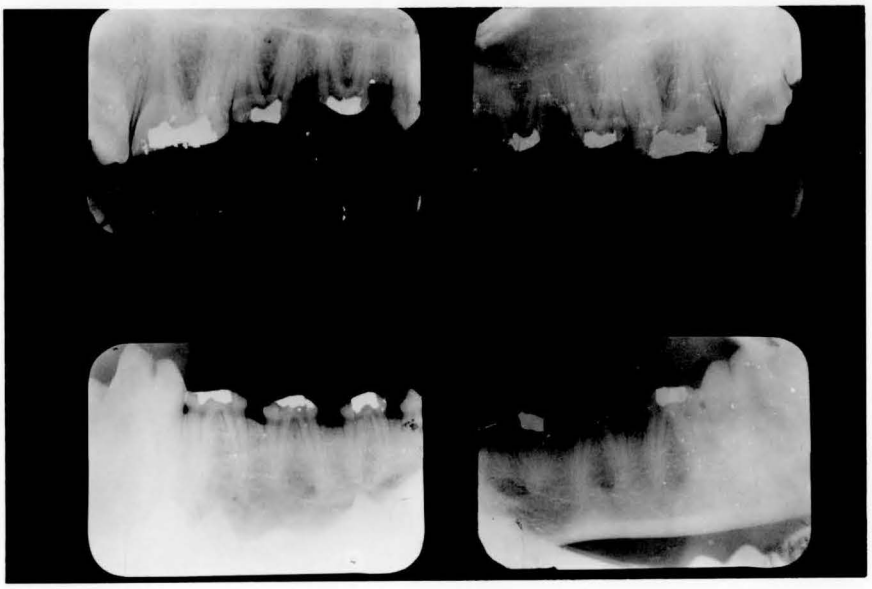


Figure 7: Radiographs taken immediately after operative procedures were performed on the 30 day animal.

Figure 8: Thirty day postoperative radiographs taken immediately prior to sacrifice. The maxillary left second premolar (saline) appears to have a widened periodontal ligament in the furcation area. Also note a loss of radiographic density in the furcation of the maxillary first molar (formocresol).



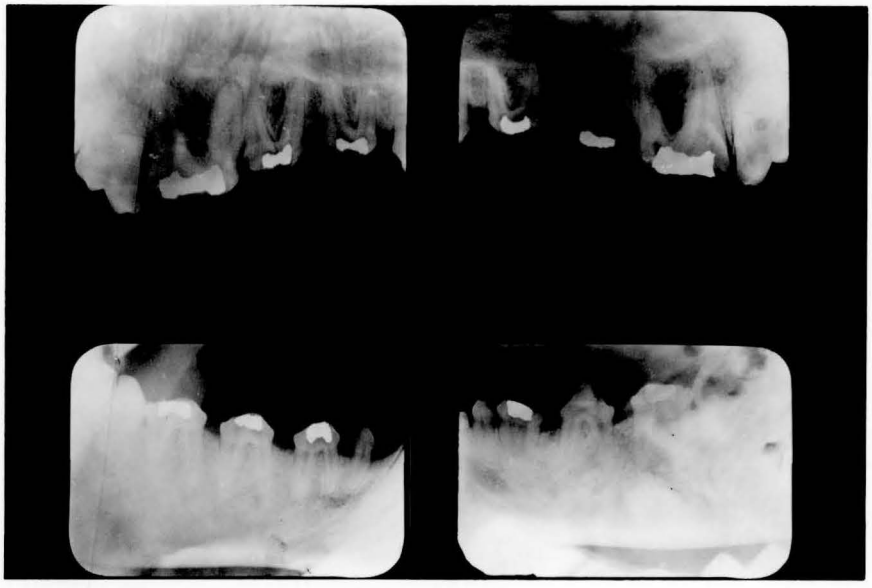
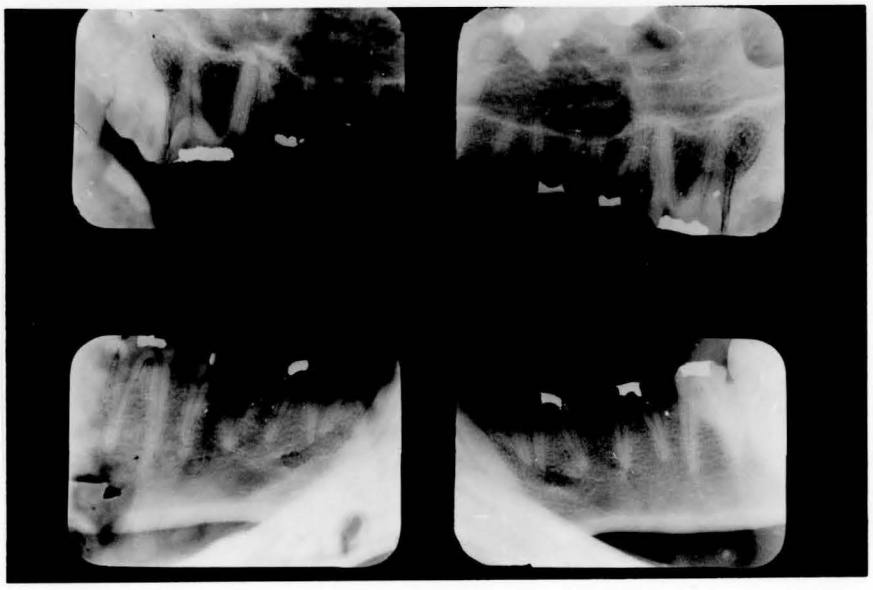


Figure 9: Immediate postoperative radiographs of the 90 day animal.

Figure 10: Presacrifice radiographs of 90 day dog. Maxillary second premolars both appear to have furcation defects.



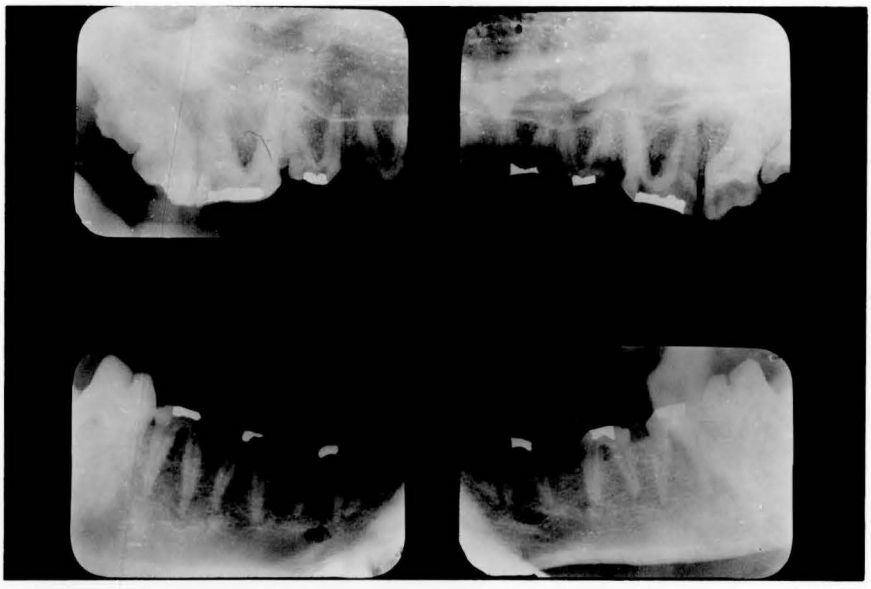
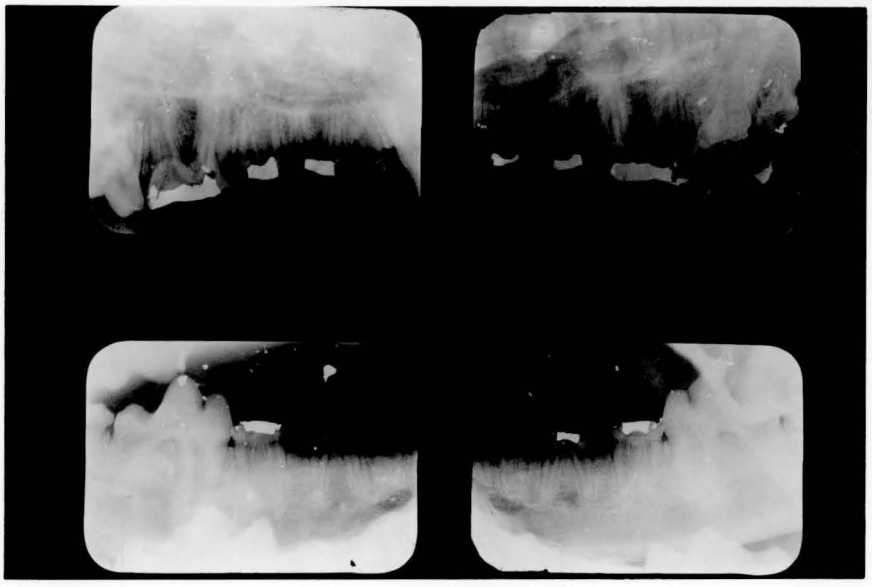


Figure 11: Postoperative radiographs of the 150 day animal.

Figure 12: Radiographs of 150 day animal taken immediately before sacrifice. Note periapical radiolucencies and resorption especially the mandibular left. Also note the lack of changes in the bifurcation. Restorations appear to be in good shape.



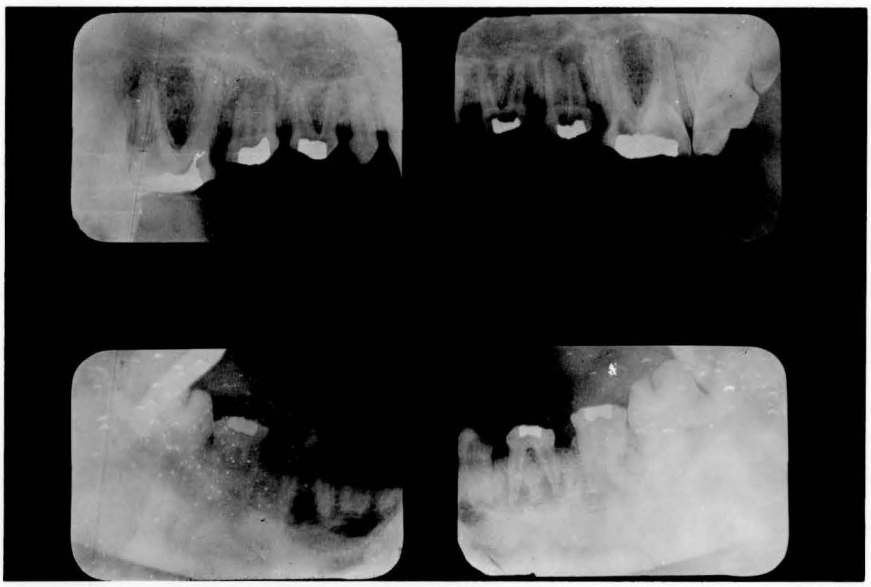
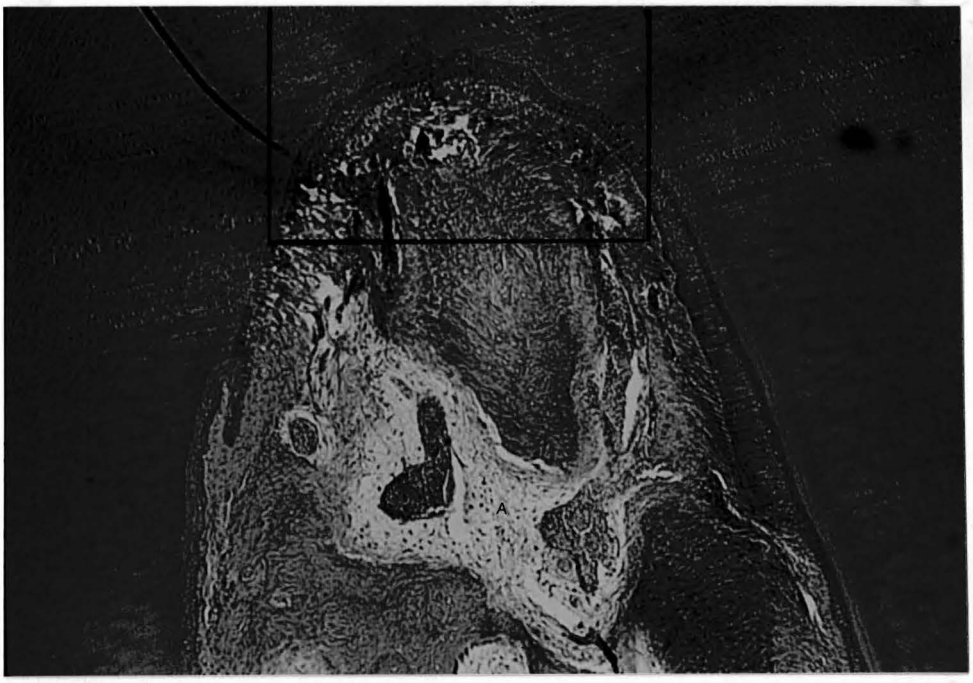


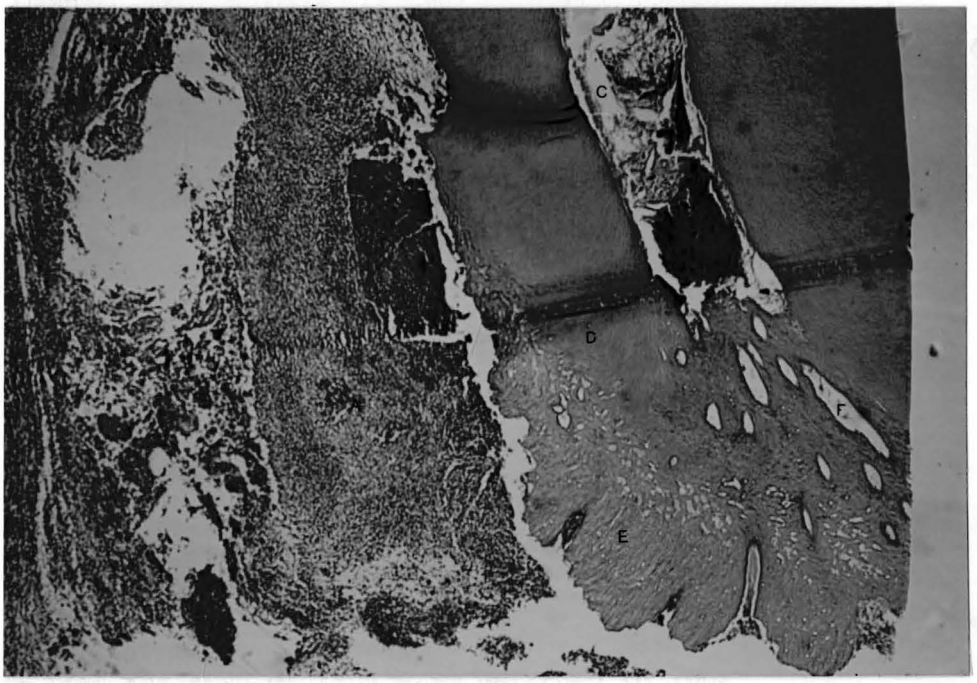
Figure 13: Furcation area of maxillary left first molar treated with formocresol for 150 days. Note normal furcation area, devoid of lateral or auxiliary canals, and with no inflammatory cells present. (40X, H&E stain).

Figure 14: Enclosed box of Fig. 13 indicating normal furcation architecture. (Marrow cavity (A), crestal bone (B), dentin (C), cellular cementum (D), periodontal ligament (E).) (100X, H&E stain).


A


C
E
B

Figure 15: Apex of maxillary left first molar, treated with formocresol for 150 days demonstrating chronic apical periodontitis. Chronic inflammatory infiltrate (A), external root resorption (B), necrotic pulp tissue (C), dentin (D), cellular cementum (E), apical canals (F). (25X, H&E stain).


C
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F
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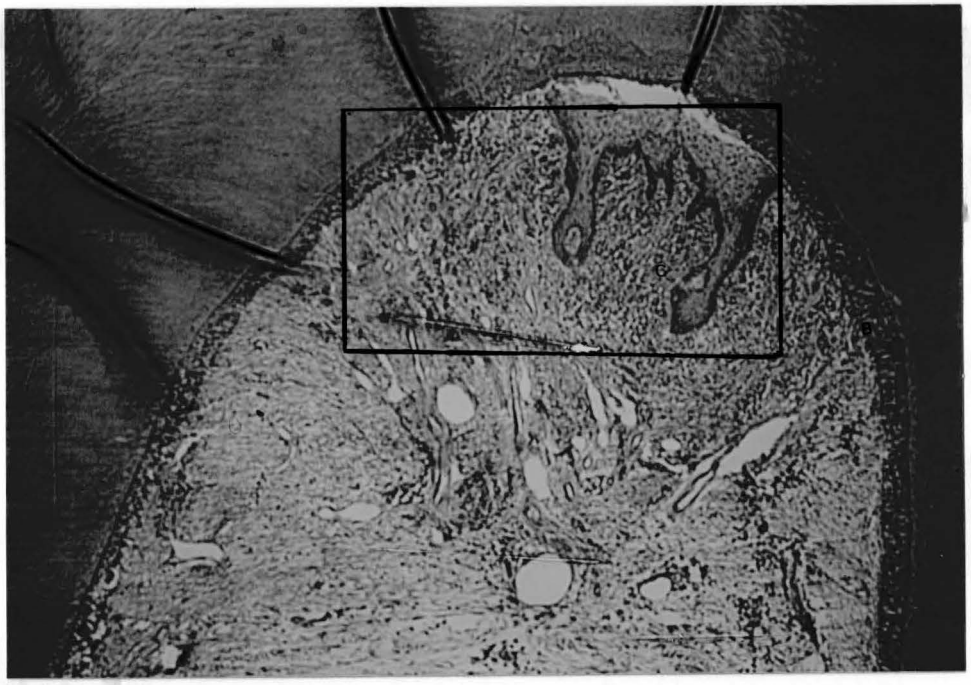
Figure 16: Furcation area of maxillary left second premolar treated with formocresol for 90 days. Note chronic periodontitis. Sulcular epithelium (A), cementum (B), inflammatory infiltrate (C). (40X, H&E stain).

Fig. 17: Box area of figure 16 enlarged to 100X.



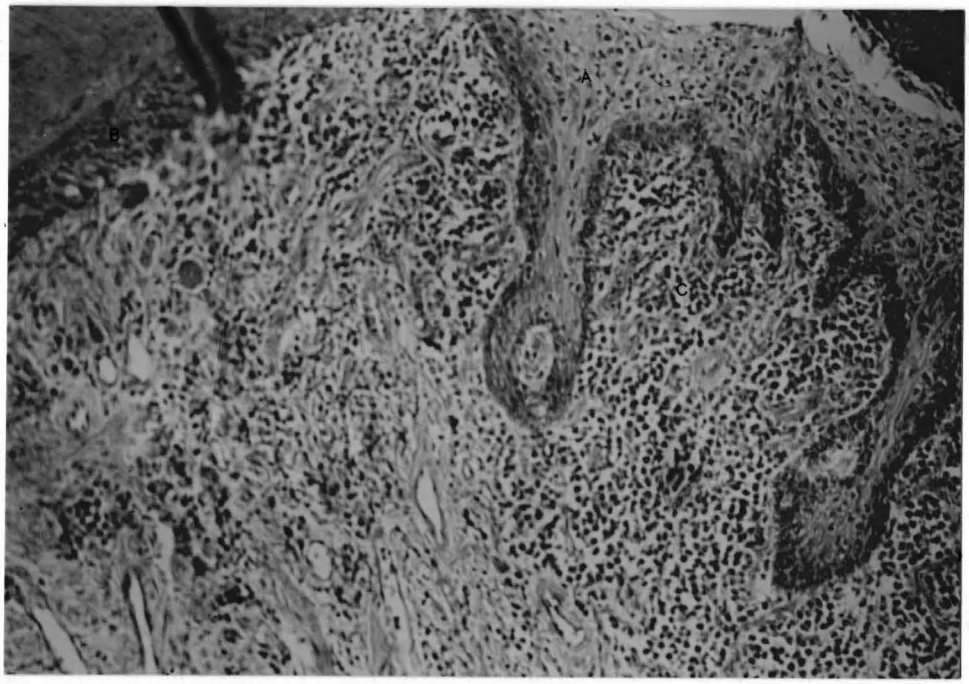

A
B
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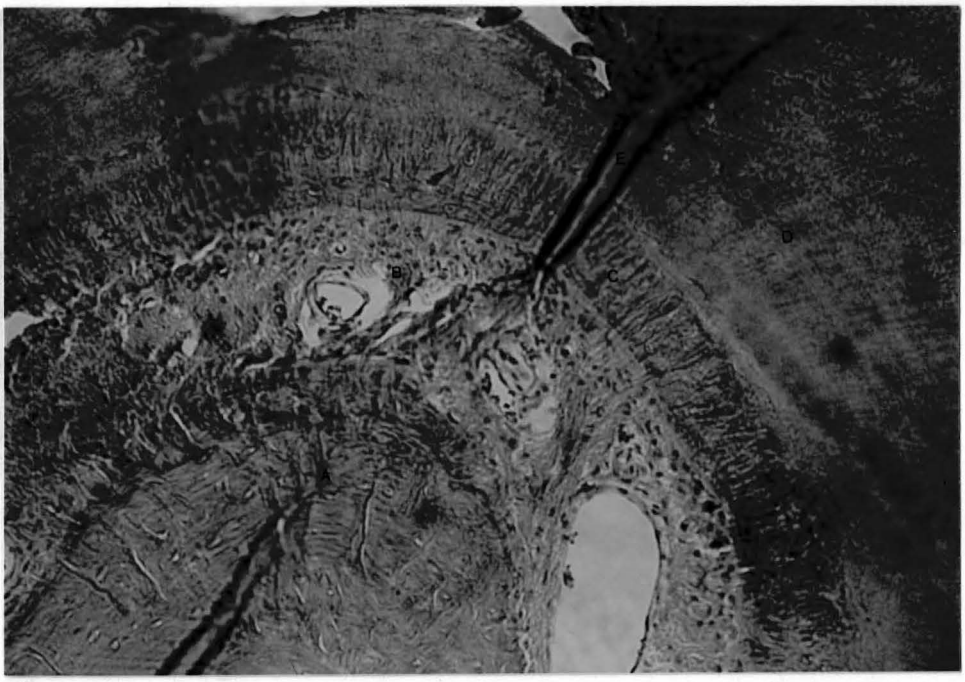
Figure 18: Furcation area of untreated (control) mandibular right third premolar, 150 day animal. Crestal bone (A), periodontal ligament (B), cellular cementum (C), dentin (D), artifact (E). (100X, H&E stain).

Figure 19: Furcation area of mandibular left third premolar treated for 150 days with formocresol. Dentin (A), cellular cementum (B), crest of interradicular bone (C), periodontal ligament (D), marrow spaces (E). (25X, H&E stain).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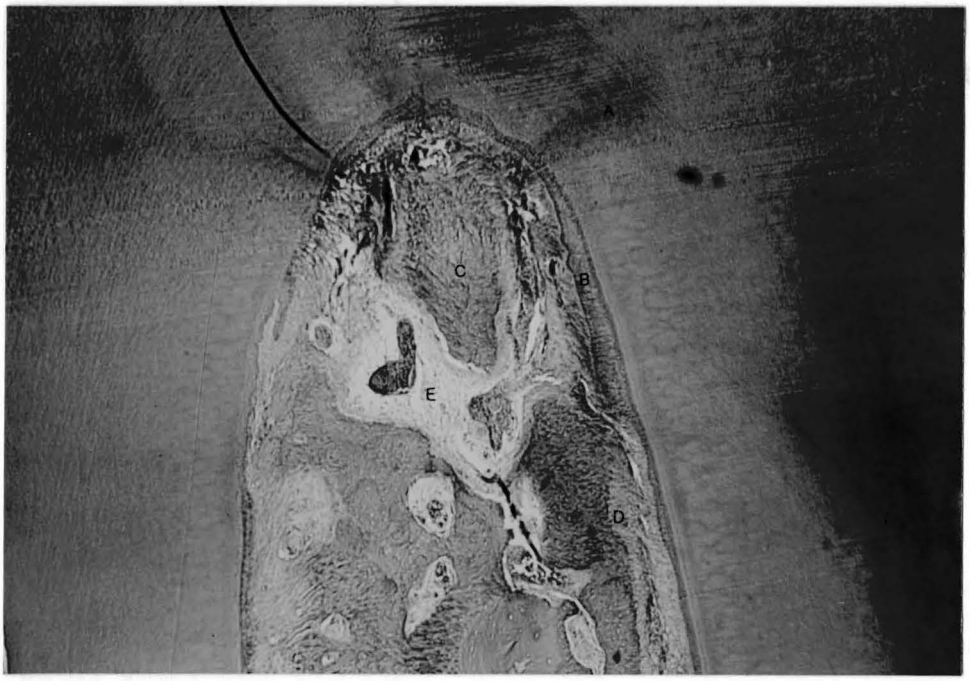
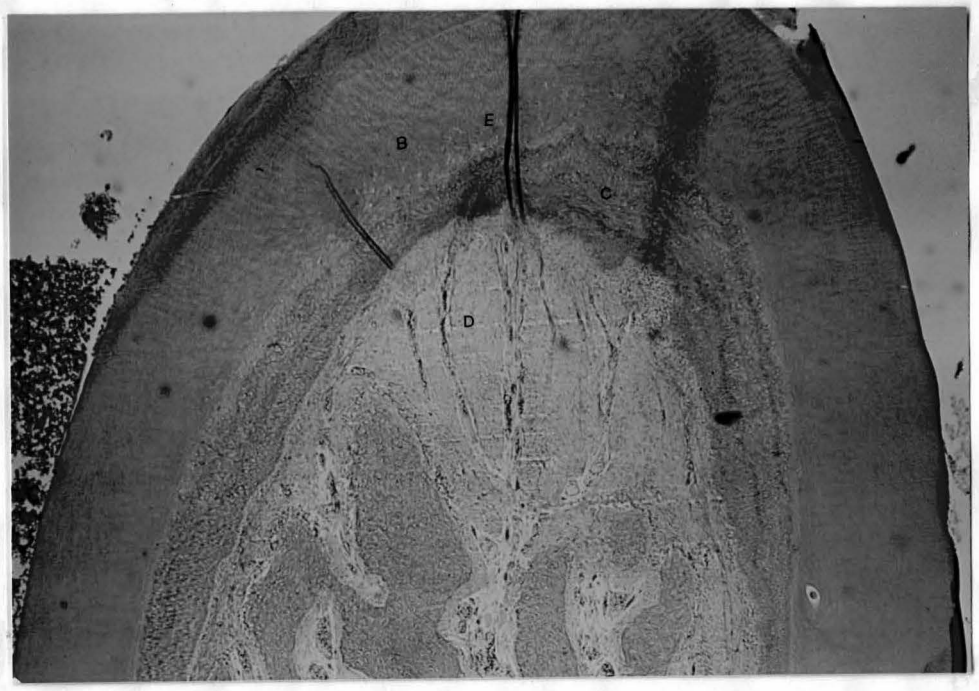

A
B
C
D
E

Figure 20: Furcation area of the mandibular left fourth premolar (treated with saline) - 150 day animal. Root canal space with necrotic debris (A), dentin (B) cellular cementum (C) dentin (D), artifact (E). (25X, H&E stain).

Figure 21: Furcation of maxillary right second premolar (control) of the 90 day animal. Crestal bone (A), periodontal ligament (B), cellular cementum (C), dentin (D), artifact (E). (25X, H&E stain).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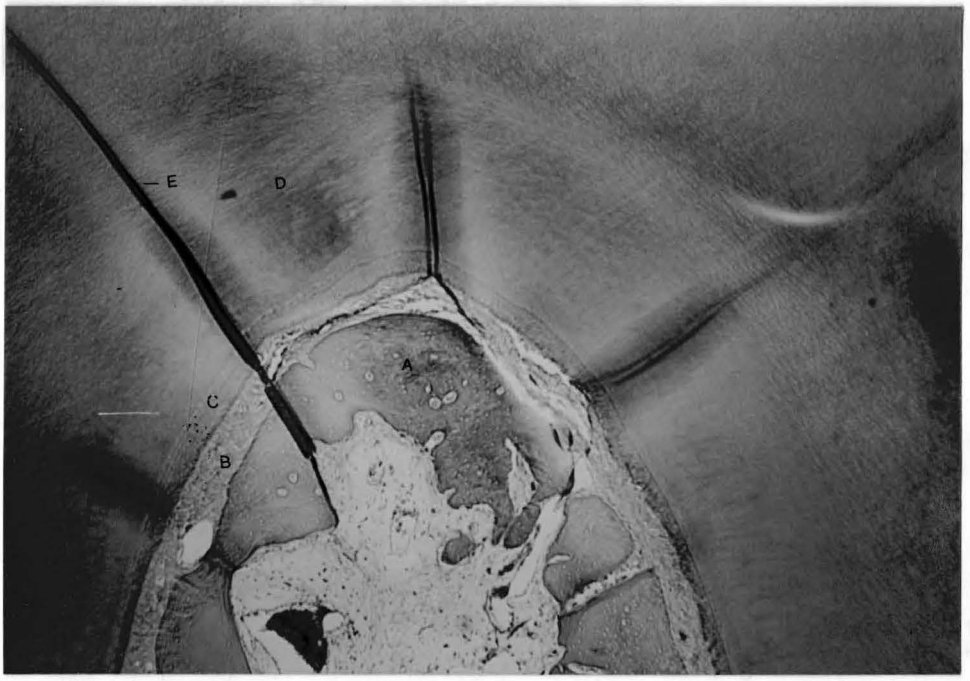
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D
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APPROVAL SHEET

The thesis submitted by Richard A. Munaretto, B.S., 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 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.

4/15/82
Date

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