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Pulpotomies Utilizing Povidone-Iodine in Primary Teeth

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PULPOTOMIES UTILIZING POVIDONE-IODINE IN PRIMARY TEETH

by

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VITA

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INTRODUCTION

Pulpotomy may be defined as the complete removal of the coronal portion of the dental pulp, followed by placement of a suitable dressing or medicament that will stimulate repair, fixation, or mummification of the remaining vital radicular pulp. Although this has become an accepted dental procedure under certain conditions, the treatment of the exposed pulp stumps remains controversial.

Attempting to organize the literature, Hoffman[26] divided the development of vital pulp amputations into three eras. She referred to the period before 1874 as the "Empirical Era." From 1874 to 1921 the "Antiseptic Era" and from 1921 to 1937 the "Aseptic Era." Bergman[2] would like to think of the present as the "Experimental or Biological Era."

During the early or empirical era, Pfaff attempted to cap exposed pulps with small pieces of gold foil. In 1826 Kocker cauterized the exposed pulp with a hot iron wire and then placed silver or lead caps over the exposures[6]. Later, in 1899 Biro recommended that all diseased pulps be treated with arsenic and then covered. Only personal, subjective evaluations were offered for

pulpal treatments in those days[6].

Several medicaments were recommended during the second era. King (1881-1886), who used zinc oxide mixed with 2 per cent carbonic acid in creosote as a pulp capping agent, wrote "for capping to be made an invariable success must ever remain an impossibility"[6].

As early as 1874 Witzel[80] introduced the deliberate amputation of the infected portion of the pulp as a therapeutic measure. He believed that the entire coronal portion of the pulp should be removed and the remaining radicular stumps covered with antiseptics. He preferred weak phenol solutions to destroy bacteria and prevent decomposition. Witzel[46] stressed that the essence of antiseptic treatment rests upon the principle of cleanliness, a principle which is the keynote of successful wound healing today.

During the next five decades, other medicaments were advocated. Thomes[73] described in detail the histological structure of a tooth that has healed by forming a calcified bridge under a large pulp exposure. He observed many dentin splinters within the new calcified bridge. Thomes believed that treatment should be aimed to stimulate the formation of secondary dentin so that an exposure may be recovered. Therefore, it was improper to use any medication that was too strong or caustic because it would destroy the formative cells and thus interfere with the healing process. Thomes' work provided one of

the earliest histologic descriptions of a pulpotomy treatment.

Since the beginning of this century, zinc oxide and eugenol has been used to promote a protective layer of secondary dentin. Reports by Szabo[59], Hense[24] and others[10] using this material as direct pulp capping agents were not convincing. Reports by Howe (1919) and Glass & Zander[22] showed the inability of zinc oxide and eugenol to promote healing of the dental pulp.

Hoffman[26] capped forty-seven clinically healthy teeth with autogenous dentin splinters and alien, sterilized dentin powder. She claimed 82 percent success. The only teeth that did not form secondary dentin were infected. Among the teeth that were successful upon treatment she noticed the presence of a necrobiotic calcific layer before secondary dentin formation.

Materials which are physiologically inert have also been used to cover the exposed pulp. These included substances such as asbestos fibres, charcoal, silver plates, silver foil, gold foil, paraffin, gelatin sponge and methycellulose. Differences in opinion existed as to the effects of inert materials on the exposed pulp.

Castagnola[10] cited Hermann[25], who in a dissertation at Wurburg recommended a preparation containing calcium hydroxide for healing of the exposed pulp. Hermann produced a compound of $Ca(OH)_2$ in combination with other

salts. The product was known as Calxyl. The drug was used by Zander[82], who reported a pH of $Ca(OH)_2$ of 12.4. Studying the reaction of the pulp to calcium hydroxide he found a zone of amorphous calcification with cell enclosures. He referred to it as a zone of structureless dentin. "On the outside of the dentin bridge there is a dark, amorphous, structureless layer showing at some places cell enclosures or empty spaces." Teuscher and Zander[72], were the first to suggest the use of calcium hydroxide over exposed pulp and Zander supported its use with several clinical and histologic studies.[82]

The histologic investigation of Glass and Zander[22] was probably the first to analyze the process of pulpal healing as a dynamic process. They showed early changes that take place during pulpal healing and found that, after twenty-four hours, the portion of the pulp in contact with the calcium hydroxide was necrotic. The necrotic zone was separated from healthy pulp tissue by a zone of deeply staining basophilic material which they called calcium proteinate. They believed that this layer occurred at the depth of penetration of the calcium hydroxide. After fourteen days, the necrotic zone and the basophilic zone of calcium proteinate zones were still apparent, but against the latter, a new era of coarse fibrous tissue was formed which was partially calcified.

Cells resembling new odontoblasts were lined up along the pulpal surface of the coarse fibrous tissue. After twenty-eight days, a well defined zone of new dentin, with adherent odontoblasts, was seen deposited against the fibrillar zone. The necrotic zone was no longer apparent. After eight weeks, there was no significant difference except that the dentin barrier was thicker. They reported that the healing process was rapid and relatively free of inflammation. Some of the findings of these investigators are still acknowledged today.

Critical analysis of calcium hydroxide pulpotomies on primary teeth showed discouraging results. Via[78] indicated a pulpotomy success rate of 35 to 50 percent in primary teeth. Intraradicular and internal resorptions were leading causes of failure. However, some criticisms of this study are:

a) Pulp hemorrhage was controlled with phenylephrine 1% or epinephrine (1:500) when pressure with cotton pellets was not able to stop the bleeding. This introduces another variable into the technique.

b) Results were based on radiographic findings only, using as main parameters for success or failure the presence of a radiographically observable dentin-bridge, or the presence of internal root resorption. Absence of radiographic dentin-bridge findings was considered a failure.

Law[32] showed a 49% success rate with $Ca(OH)_2$ vital pulpotomies. The indication for pulpotomies was that of carious teeth with exposures larger than 1mm in diameter. Law and Lewis' study[33] was commonly cited to support the ineffectiveness of $Ca(OH)_2$ as a pulp capping agent in pulpotomies of primary teeth. However, there is only a comment about pulp reaction to $Ca(OH)_2$. It was not used in the study.

The unreliability of calcium hydroxide led dentists in the 1950's to search for another pulpotomy medicament. The technique advocated by Sweet[70], using Buckley's formocresol[8], gained popularity rapidly. Today formocresol remains the most frequently employed agent for pulpotomy procedures in the primary dentition. However, the future status of formocresol as a vital pulp agent is questionable. Conflicting evidence exists as to whether fixation is limited to the coronal half of the radicular pulp or whether it extends throughout the radicular tissue in harmful amounts.[39,50] Harmful effects may result with formaldehyde as the possibility has been reported that when pulp tissue is treated with formaldehyde the damaged tissue components may be rendered antigenic and induce antibody formation[41,42,43].

Berk[4] and Berk & Cohen[5] found that calcium hydroxide in a methylcellulose base promoted earlier bridging and less inflammation than calcium hydroxide in

water. This was probably due to the lower pH and less caustic action of the methylcellulose preparation. Doyle et al.[14] used the same preparation and found only 50% success after 6 months using clinical, radiographic and histologic assessments. Failure was considered in those cases with incomplete bridging and presence of inflammation. Internal resorption was reported in 20% of the cases. A periapical radiograph used as an example of internal root resorption shows a partial pulpotomy (faulty technique).

Phaneuf et al.[48] tested the pulp reaction to three different preparations of calcium hydroxide. They used primary cuspids in 25 children. The three preparations were: Dycal*, Hydrex** and Pulpdent***. Pulp reaction to Dycal was favorable with some variation in time required for bridge formation. Pulpdent showed consistently positive results with bridge formation after 28 days. Reaction to Hydrex was inconsistent and, in general, was unacceptable.

Koslov and Massler[31] used drugs attempting to accelerate or retard pulpal healing. They reported a good reparative dentin response under calcium hydroxide with metacresylacetate (Cresatin). Weiss and Brojvatn[79] investigated methods of pulp protection of young teeth in

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*
** Dycal - L.D. Caulk Comp., Milford, Del.
***Hydrex - Kerr Manufacturing Comp.
Pulpodent - Pulpdent Corp.

monkeys. They reported that Cresatin mixed with calcium hydroxide used as a pulp capping material produced initial bridging after one week with only a small necrobiotic zone separating the medicament and the vital pulp. After thirty days a well defined bridge covered the pulp exposure.

Sandler, Frankl and Ruben[60] studied the action of cresatin on the dental pulp. The investigation developed from the use of metacresylacetate as an analgesic dressing before and during pulpectomy procedures. They placed a cotton pellet saturated with cresatin in contact with the pulpal tissue and sealed it with Cavit and amalgam. They reported inflammation in 50 percent, secondary dentin formation in 40 percent and internal root resorption in 20 percent of the cases. The presence and amount of vital and necrotic pulp tissue in the root canal was inconsistent. Low success rate when using $Ca(OH)_2$ as a pulp capping agent was shown when the study samples could already have compromised pulp circulation and vitality, due to advanced carious lesions.

Schroeder[56] noted a correlation between the inflammatory response and the barrier formation: "The slighter the inflammation, the higher the frequency of bridging." Furthermore, barrier formation was reported to occur in close contact with two cements, without a visible intermediate necrotic layer under the light microscope.

Some investigations with cariously exposed pulp have not confirmed any positive effect of calcium hydroxide[45,68]. Internal dentin resorption of primary teeth does not seem to depend on the effect of calcium hydroxide, but on chronically inflamed pulp, i.e., infiltration of mononuclear inflammatory cells that are present at the time of treatment or induced by improper wound treatment (such as leaving a blood clot between the wound surface and the calcium hydroxide).

Citron[11] studied the clinical and histological response of human pulps to two medicaments, metacresylacetate (Cresatin) and calcium hydroxide in a methylcellulose base (Pulpdent), acting synergistically in a vital pulpotomy procedure. He placed a cotton pellet moistened with cresatin in the pulp chamber for five minutes. After removing the cotton pellet, he placed calcium hydroxide compound on the pulp stumps. The dental pulp formed an osteodentin bridge and the pulp adjoining the bridge remained histologically viable and free of inflammation. A necrobiotic zone also divided the dressing apart from the dentinal bridge. Gardner et al.[17] and Eggers et al.[14] have reported that vancomycin hydrochloride in combination with calcium hydroxide and in combination with hyaluronidase stimulates regular reparative dentin bridges.

Case reports throughout the literature frequently

mention that success of vital pulpotomies with $Ca(OH)_2$ in primary teeth is enhanced using Cresatin. Both medicaments are being reported as causing a burn on the pulpal surface which is referred to as the necrobiotic zone. The suggestion is made that the zone acts as an organizer for the pulp tissue to form an osteodentin bridge[10,30,54,72].

In 1959, Shroff[64] wrote, "It is true, also, that the best method available today involves the use of calcium hydroxide preparations which are essentially necrotizing in their action. These materials produce a dystrophic calcification which substitutes for normal repair of the exposed surface but, which certainly cannot be regarded as the ideal."

The search continues for medications that could serve as what Hess calls, "biologic wound dressings." Phaneuf[48] and Masterton[37] are among the authors who agree that the pulp has good recuperative powers for recovery after an injury and that usually it heals in a favorable environment. This means that a healthy pulp will recuperate from a wound if the existing irritants, mainly bacterial, are removed. After reviewing the literature, we can conclude that a pulp dressing should be inert to the pulp, mildly or non-irritating, to the exposed tissue and its application must be such that conditions for treatment are capable of maintaining an aseptic field.

Sealing the cavity from the oral environment is another very important factor, which can be accomplished by the dressing material or by the final restoration.

Since the latter part of the nineteenth century iodine has been extolled as an antiseptic. This is partly because it is highly functional. Iodine has been used in various forms: as an antiseptic for the skin, wounds and mucous surfaces of the body, for sterilization of the air and other things such as catgut and surgical instruments; as a prophylactic and therapeutic agent in diseases caused by bacteria, viruses, fungi, yeasts and protozoa, and for other disinfecting purposes.

Iodine [25,27,33] is a highly reactive element and because it is so reactive it is a good germicide. When iodine preparations act as disinfectants, free iodine generally is the effective agent. McCulloch believes[38] that iodine destroyed microorganisms by the formation of salts with proteins by direct halogenation.

Iodine is essentially bactericidal over a wide pH against a broad spectrum of pathogens, including the already mentioned bacteria, viruses, protozoa and spores of bacteria and fungi. Iodine eliminates them immediately rather than by a prolonged period of stasis. There do not seem to be microorganisms capable of forming any type of resistance to this agent. Iodine penetrates the cell wall of microorganisms and yeast cells and unbroken skin and leather.[20]

Iodine has not been broadly used as an antiseptic due to its toxicity over the tissues, its lack of stability and its insolubility in water. The combination of iodine and a solubilizing agent or carrier diluted in water is called an iodophor. These complexes contain and slowly release free iodine. I_2 in presence of salivary or neutrophilic peroxidase plus H_2O_2 yields oxidative form of iodine. They maintain the bactericidal action of iodine but its odor, vapor pressure and toxicity are highly reduced.

Polivinylpirrolidone (PVP)[38,46,63], also known as Povidone, is similar in configuration to the breakdown products of hemin. It also resembles the proteins of natural plasma in its capacity for binding water and absorbing other substances.

PVP displays a retarding and potentiating effect on a large number of parenterally administered drugs. This characteristic has been used in intramuscular antibiotic preparations.

PVP also has the ability of detoxifying in vivo certain dyes and toxins.

In view of the detoxifying properties of PVP, Shelanski[57] and Cantor & Shelanski[9] studied its effects upon inorganic toxic materials. One of the first of such materials studied was iodine in Lugol's solution and as a tincture. Lugol's solution and tincture of iodine in

combination with PVP were shown to be less toxic than either solution alone. Altered physical properties were produced by the presence of PVP. The color was altered and the vapor pressure reduced.

They prepared an aqueous solution of PVP-1 without the aid of usual solvents, showing that PVP itself acts as a carrier of iodine.

Less than 1 percent of iodine is converted directly into inorganic iodide and about 30 percent of iodine is converted into organic iodide. The remaining 69 percent of the iodine exists as a free elemental iodine which is made soluble by the organic iodide.

Further investigations demonstrated that the iodine in PVP-1 was available to perform the same functions as iodine in tincture or Lugol's solution namely, its bactericidal, viricidal and protocidal capabilities have not been decreased. In the same study, Shelanski[57] reported that PVP-1 solution is 1/4 to 1/9 as toxic as Lugol's solution, or 4 to 9 times safer than Lugol's solution.

PVP has no antibacterial effect. It acts only as a carrier and detoxifying agent. The combination of the polymer with iodine has a germicidal effect similar to that of tincture of iodine. Garnes[18] mentions that solubilized povidone-iodine has enhanced antisepsis as compared to an equivalent concentration of ordinary iodine

because of a lesser amount of I_3.

With the use of PVP-I the germicidal action of the iodine is prolonged and the danger of cutaneous irritation, sensitization reactions and burns are minimized.

Animal studies, in which paired scarred lesions were employed, demonstrated that neither povidone-iodine interfered with wound healing.[81]

Further studies have been done on humans[17,20,81]. No systematic reactions and extremely low incidence of sensitivity reactions to topical application of PVP-1 have been reported. It has been successfully used as a vaginal microbicide, as a trichomonicide, as a sporicide, as an antiseptic on skin and mucous membranes, as a protective antimicrobial agent in treatment of first, second and third degree burns and abraded skin.

The following study was designed to evaluate the reaction of the pulp to Polivinylpirrolidone-Iodine, when used as a capping agent in pulpotomized primary teeth in monkeys. It was also intended to compare the reaction of the pulp to I-PVP and to a $Ca(OH)_2$ compound (Dycal) when used in the same manner.

The hypothesis for this study was that PVP-I would promote healing of the vital pulp tissue when applied as a dressing agent to pulpotomized sites.

CHAPTER II

METHODS AND MATERIALS

The study was performed at the Research Biological Laboratory from the University of Illinois at Chicago, where animals, housing and general anesthetic were provided, and at Loyola University of Chicago, Dental School, where the preparation of teeth, slides for histologic evaluation and evaluation of results were performed.

Eight Papio-Anubis monkeys were used for the study. Seven monkeys were 14 months of age and one was 30 months of age at the beginning of the study. All monkeys had full primary dentition and the apexes of all teeth were completely formed as confirmed radiographically.

The study consisted of 32 first primary molars, which were randomly assigned to one of three groups:

All procedures were performed under general anesthesia. The following outlines protocol.

1. Premedication with Ketamine HCL 5-10 mg/kg 1M.
2. Induction with Thiopentahal sodium (Surital) 2.5% IV, to effect (=10-15 mg/kg).
3. Intubation=Orotracheal intubation.
4. Maintenance with 0_2 (2 l./min), N_20 (1 l./min and Halothane 0.8% using a "Narcovet" gas anesthesia machine.

5. An IV catheter was used to maintain a patent IV line throughout surgery, with normal saline drip.

6. After extubation animals were returned to their regular baboon-cages, following uneventful recovery.

a) Control group: Four first primary molars were used as the control group. No treatment was performed.

b) Sham control group: Twelve first primary molars were treated with $Ca(OH)_2$ compound (Dycal) at the pulpotomized site.

c) Experimental group: Sixteen first primary molars were treated with PVP-I (Betadine) at the pulpotomy site. When performing the procedure, an oropharyngeal pack was placed in the monkeys at the outset to avoid inhalation of foreign particles. All pulpotomies were performed under rubber dam isolation.

The access to the cameral pulp was gained through the occlusal surfaces using a #330 SS bur at high-speed in a hand piece with water irrigation. The cameral pulp was removed using a #4 SS bur at slow speed. The cavity was irrigated with sterile water, and sterile cotton pellets were used to control bleeding from the pulp stumps. The pulp tissue was covered with either $Ca(OH)_2$ paste (Dycal) or a thick paste of PVP-I (Betadine) which was made by mixing the commercial ointment presentation and a powder provided by Purdue-Fredericks Labs. (PFL), and which is

unavailable commercially. The capping bases were then covered with IRM zinc-oxide base, lining varnish (Copalite) and amalgam, using the procedures common for dental restorations. Subsequently the teeth were extracted as follows:

a) seven days post operatively, four experimental group molars and four sham-control group molars were extracted;

b) twenty-eight days post operatively, two control group molars, six sham-control group molars and eight experimental povidine-group molars were extracted;

c) seventy-six days post operatively, two control, two sham-control group and four experimental group molars were extracted.

Immediately following the extractions the apical third of the roots of the teeth were removed with wire-cutting pliers to allow better fixation of the pulpal tissue. Specimens were fixed in a 10% formalin solution for 48 to 100 hours. Then, the teeth were placed in a decalcifying solution consisting of:

Solution #1 - 450cc of 45% Formic Acid solution and 550cc of distilled water = 1 liter.

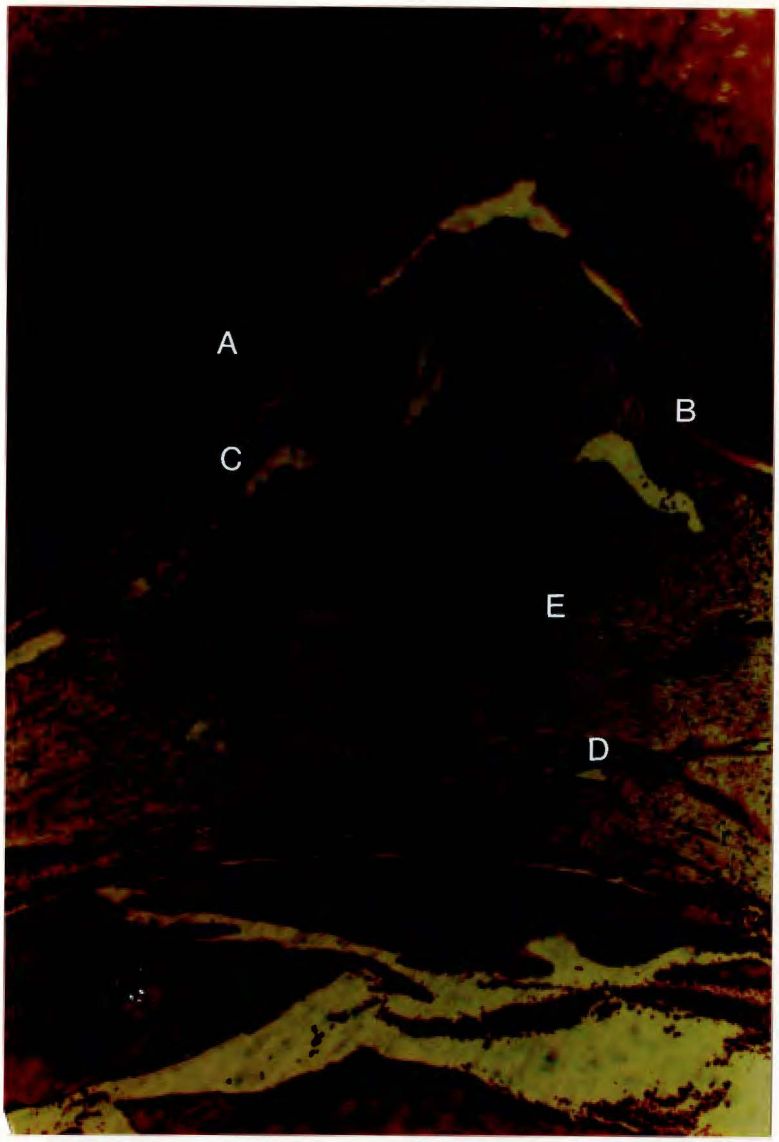
Solution #2 - 200cc of 2% sodium citrate solution and 800cc of distilled water = 1 liter.

Equal parts of solutions #1 and #2 were mixed for

use as a decalcifying solution. The volume of decalcifying solution was twenty times higher than the volume of the specimen to be decalcified.

The specimens were placed in a decalcifying agent for 25 days, with the solution being changed every three days. Complete decalcification was assessed by radiographic means as loss of radiopacity.

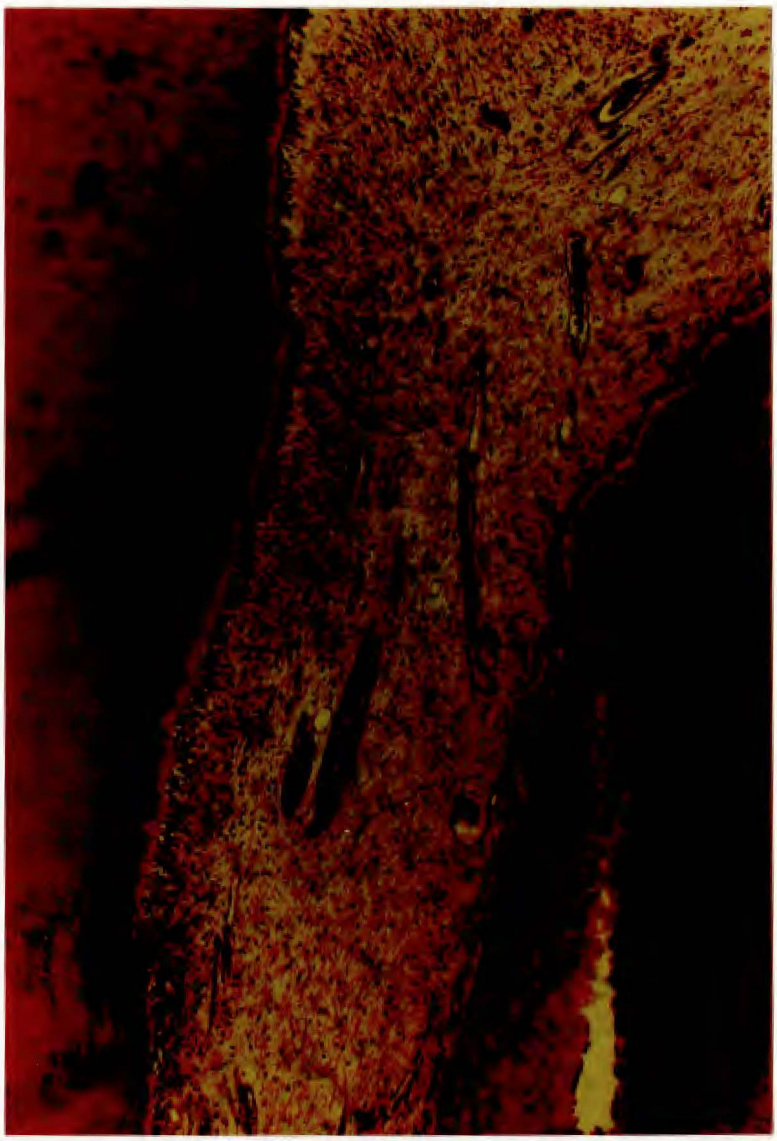
The teeth were then dehydrated and embedded in paraffin. Sections 3 μ thick were cut with AO Microtome 820 and stained with H&E.



Appearance of normal pulpal tissue in a

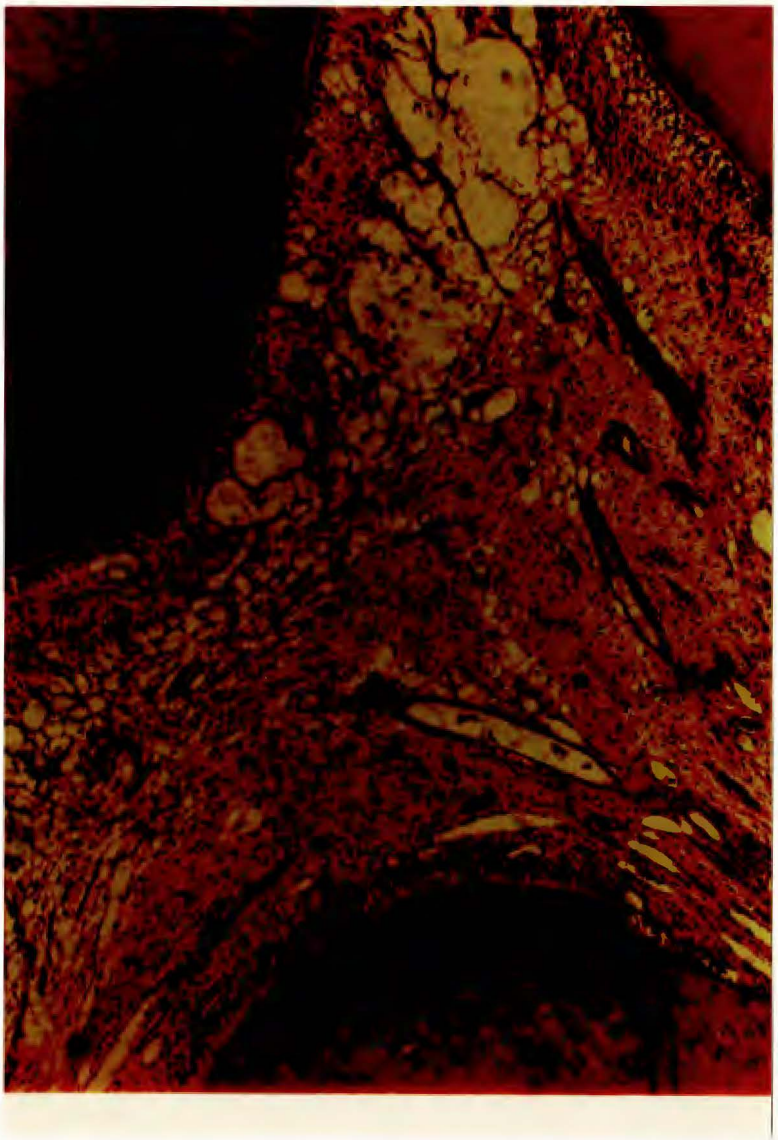
Note:
A) Dentin
B) Predentin
C) Odontoblastic layer
D) Blood Vessels
E) Nerve Bundles

N.B. Relatively mild concentration of fibroblasts.
No infiltration of inflammatory cells.



28 days control group specimen (5102 UL)

Well organized vital pulp tissue



Specimen 5093 UL - Control Group - 76 days period

CHAPTER III

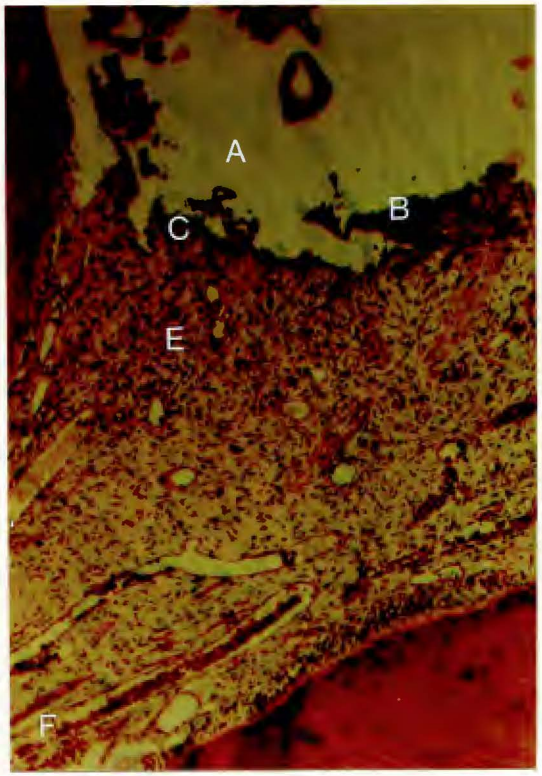
RESULTS

$Ca(OH)_2$ - 7 Days

Of the three teeth extracted seven days post pulpotomy, two were vital. Both showed proliferation of fibroblasts, collagen fibers and blood vessels, as well as local infiltration of inflammatory cells which were present in coronal portion of the radicular pulp near the amputation.

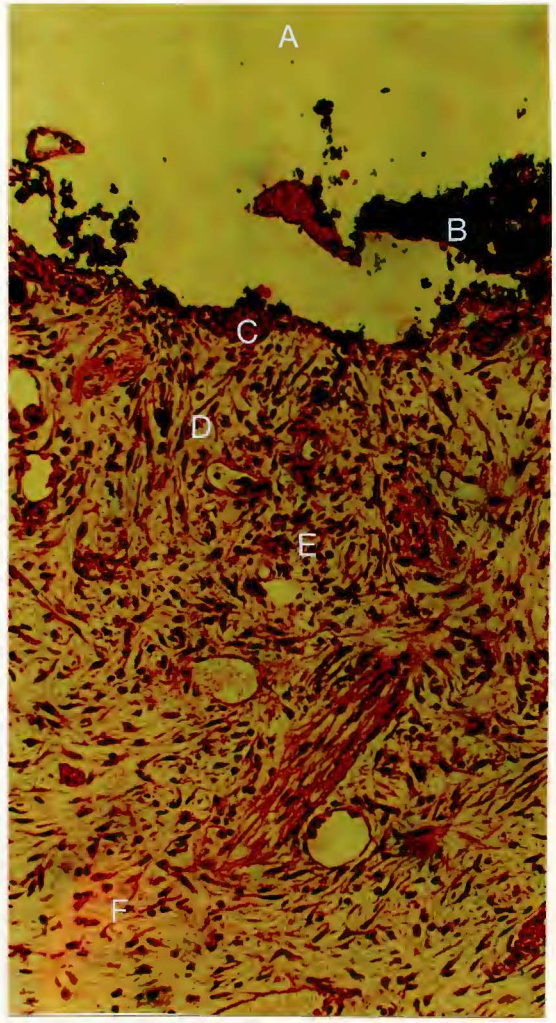
The pulpal layer in contact with the $Ca(OH)_2$ was a mixed layer running across the canal. In some areas the layer was made up of a necrotic row of cells under which fibroblasts ran parallel to the outer surface. In other areas fibroblasts were parallel to the pulp surface and in contact with the medicament. Sometimes new blood vessels formed part of the superficial or outer layer. Fibroblasts beneath the superficial layer had a fairly perpendicular arrangement towards the outer surface. Collagen fibers were abundant and had no specific orientation. The increased number of fibroblasts, collagen fibers and proliferation of blood vessels was accentuated only at the most coronal portion of the pulp tissue.

Of the other tooth two roots were available for the study. One root was necrotic and the other was considered



Specimen 5094 UR seven days after pulpotomy with $Ca(OH)_2$.
Note:

A) Amputation site
B) $Ca(OH)_2$ capping agent
C) Proliferation of blood vessels close to amputation site
D) Proliferation of fibroblasts
E) Mild acute local inflammation
F) Vital pulp tissue



vital. In the non vital root an increased amount of secondary dentin was evident on the lateral walls of the canals. The reaction was limited to the area close to the amputation site.

The vital root in this specimen presented generalized intrapulpar hemorrhage, poor cellular detail, disorganized odontoblastic layer and extrapulpal hemorrhage. Also evident were remnants from the cameral pulp which was not completely removed. The tooth was considered to be degenerating and with a poor prognosis.

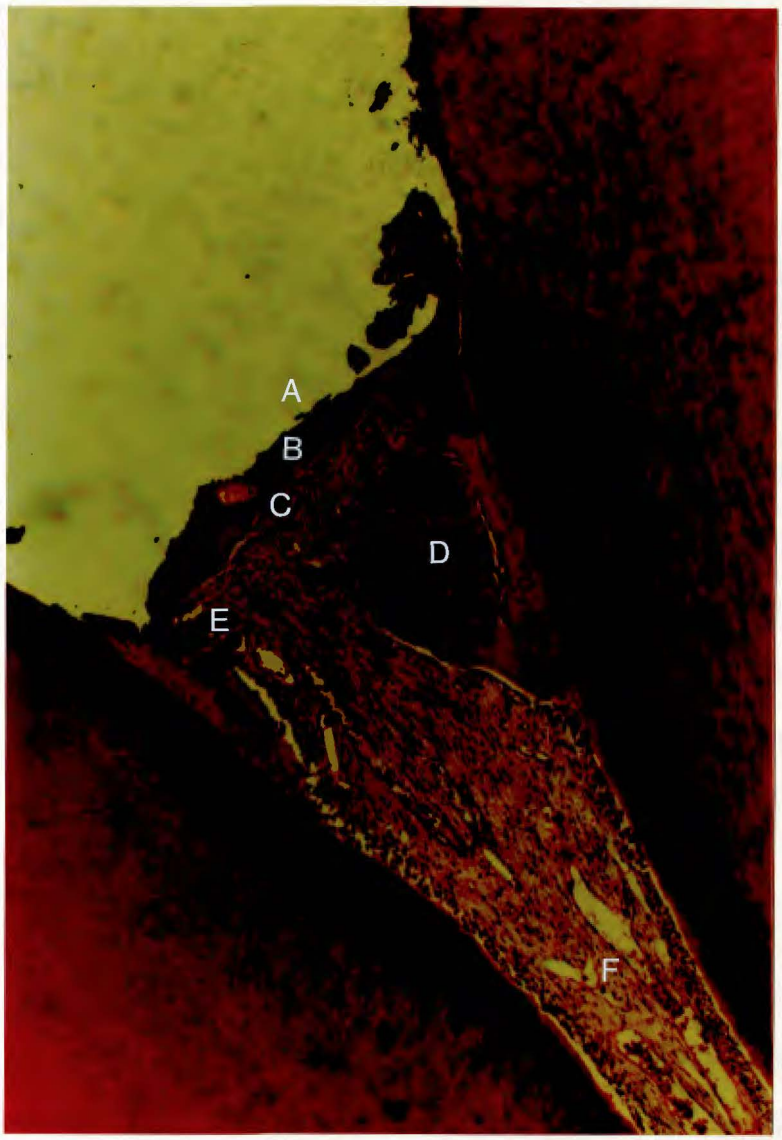
No internal dentin resorption was observed in any specimen.

$Ca(OH)_2$ - 28 Days

All six teeth in this group presented vital pulp tissue. After twenty-eight days, proliferation of fibroblasts, collagen fibers and blood vessels were evident. Inflammation was very mild or absent; a few inflammatory cells were scattered close to the reparative process area.

Five of the specimens had an incomplete dentin-bridge in one or more of the canals. One tooth presented complete bridging over all the pulp tissue canals.

The appearance of the tissue forming the bridge was similar to osseous tissue. It was homogeneous, lacking the tubular formations typically seen in dentin. There



Specimen 4979 RL 28 days after pulpotomy with $Ca(OH)_2$.
Note:

A) Amputation site
B) $Ca(OH)_2$ capping agent
C) Dentin bridge (complete)
D) Amorphous calcified formations with inclusion of dentin-shavings
E) Proliferation of blood vessels and fibroblasts at the area of repair
F) Apically, well organized vital tissue

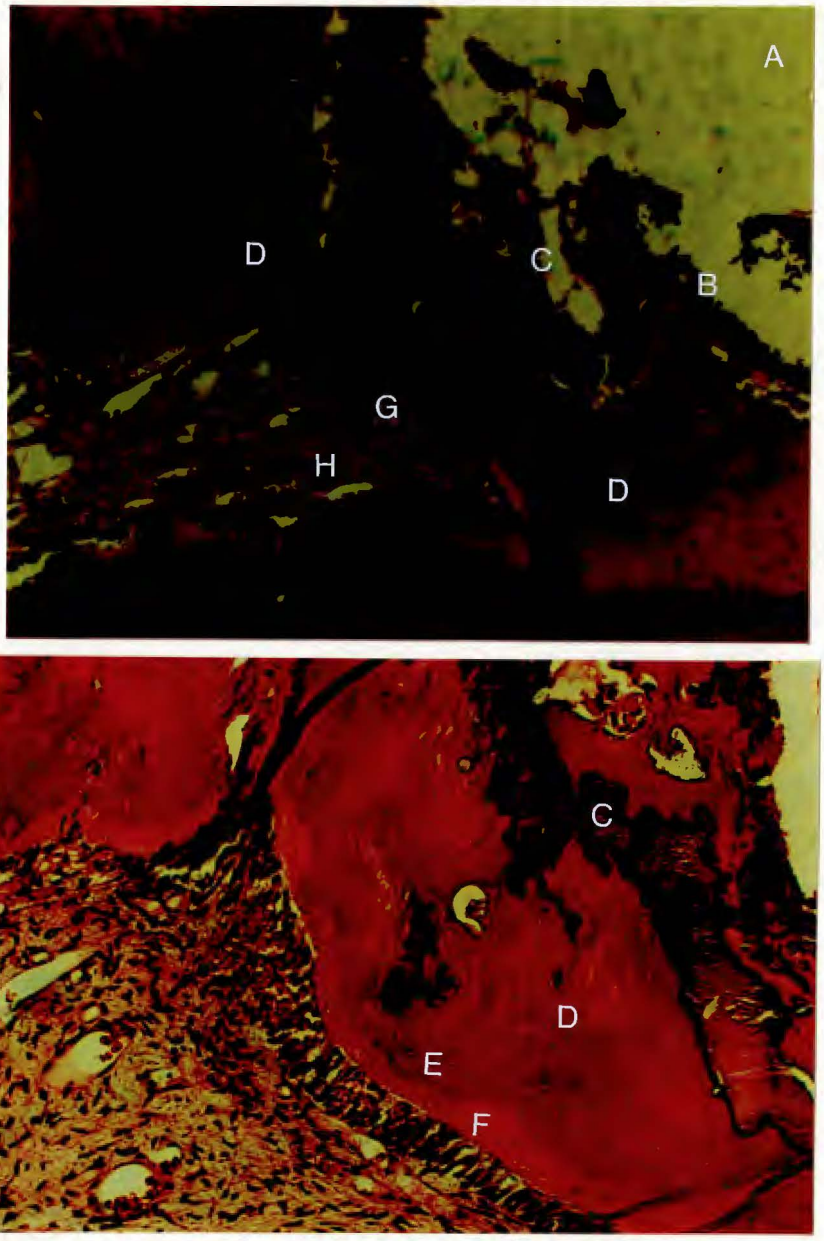
was some inclusion of cells. The tissue was stained in an eosinophilic manner. This tissue was found to be in contact with the capping agent.

In some instances an ingrowth of osteodentin layer protruded towards the center of the canal. The protrusion was amorphous and consisted of the surrounding eosinophilic bone-like layer containing a basophilic irregular material. The basophilic material was formed in part by dentin chips, or dentin shavings. In other cases, isolated dentin shavings were found to be surrounded by the same newly formed osteodentin material. Some of these specimens also showed formation of the same osteodentin on the lateral walls of the canals, which were protruding medially. The reaction was limited to an area close to the amputation site.

In the tissue between the secondary dentin from the lateral walls, some canals showed isolated formation of dentin. Some had dentin shaving inclusions, others did not. It appeared as if the collagen fibers started to form the predentin matrix, containing cell inclusions.

$Ca(OH)_2$ - 76 Days

From the two specimens extracted after a 76 day interval, one tooth was vital and presented a complete osteodentin bridge. Two roots were available from this specimen. From the other specimen only one root was



Specimen 5093 LR 76 days after pulpotomy with $Ca(OH)_2$.
Note:

A) Amputation site
B) $Ca(OH)_2$ capping agent
C) Amorphous dentin layer
D) Complete dentin bridge
E) Tubular dentin
F) Predentin
G) Odontoblastic layer
H) Well organized vital pulp tissue

available for the study. This root presented necrotic pulpal tissue with indications of extrapulpal hemorrhage at the pulpotomy site. The osteodentin bridge in the vital roots covered the healthy pulp tissue, and no indication of inflammation was observed.

The osteodentin bridge at this stage had advanced so that five different layers could be identified. These were:

a) an osteo-dentin which closely resembled bone. It was the first layer formed at the amputation site.

b) osteodentin with increasing number of dentin tubules.

c) osteodentin which resembled normal dentin.

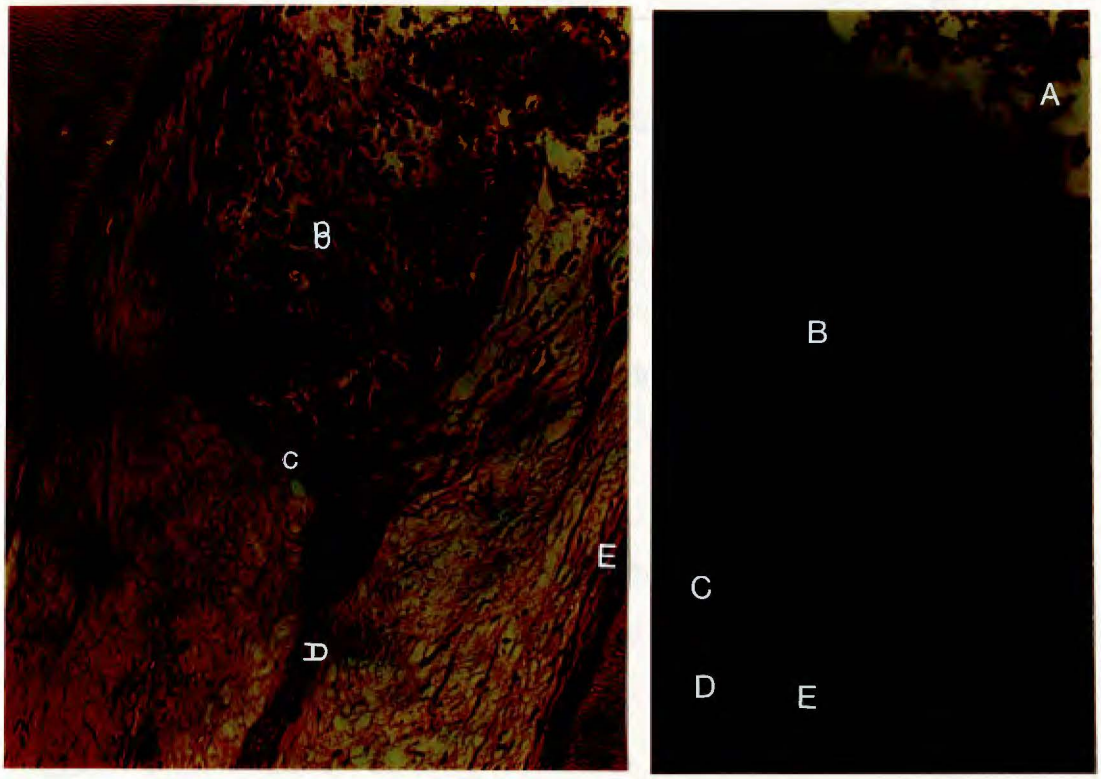
d) a predentin type of layer, with tubules.

e) a well developed dentinoblastic (odonto-blastic) layer.

PVP I - 7 days

There were four teeth in this group. One had a vital pulp and three had necrotic pulps.

The tooth with vital tissue in the canals showed reduced vascularity, intra and extrapulpal hemorrhage, some necrotic tissue and lack of pulpal structure. No indication of reparative reaction was seen at the amputation site or further down the canal. There was some specific arrangement of collagen fibers tending to make a fibrous barrier. This was found between the medication



Specimen 5094 LL seven days after pulpotomy with PVP-I

Note:

A) Amputation site
B) Disorganized and necrotic tissue
C) Collagen barrier
D) Nerve bundle
E) Flattened odontoblastic layer

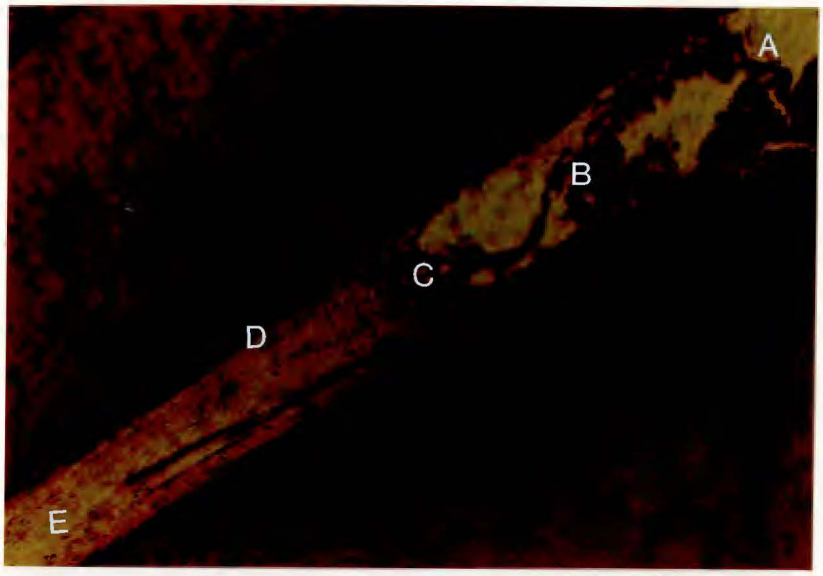
and necrotic tissue. Inflammatory cells were present throughout the canal.

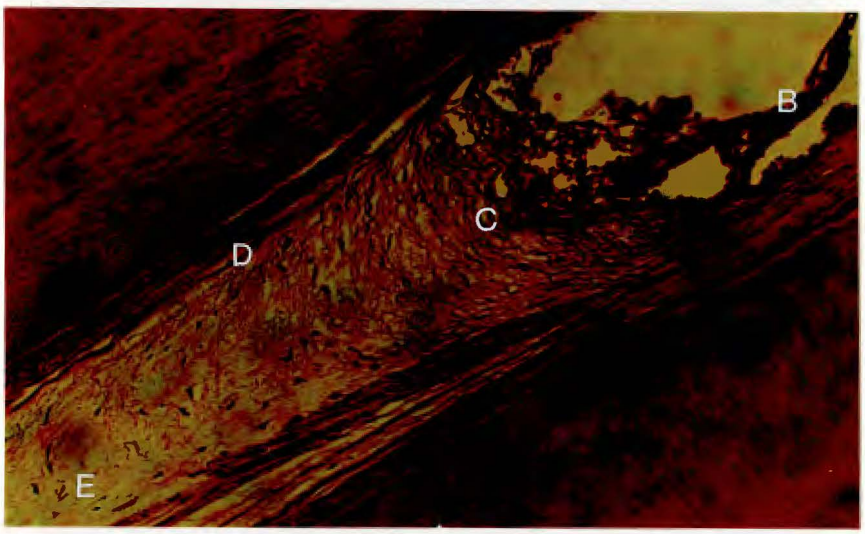
A degenerative process was the main finding in the pulps of these teeth. Some of the tissues started to form this fibrous-like arrangement of collagen fibers separating the PVP-I and the pulpal tissue, but the tissue did not resolve the wound at the amputation site and degeneration of the whole pulp tissue took place. In other cases there was no formation of collagen fibers in the canal. The tissue also did not show cellular infiltration. The pulps were necrotic.

PVP I - 28 days

There were eight specimens in this group. Two teeth showed healthy-looking vital pulp tissue in the two canals available for histologic study. Three molars showed one root with vital tissue and one with necrotic tissue. The roots of the other three specimens showed necrotic pulp tissues only.

The general pattern in the canals with vital pulp tissue is that of necrosis at the amputation site, poor cellular detail, disorganized odontoblastic layer and infiltration of inflammatory cells. At some distance from the amputation surface, the fibrous-like barrier, which was formed by the collagen fibers, separated the coronal portion of the tissue from the more apical vital tissue.

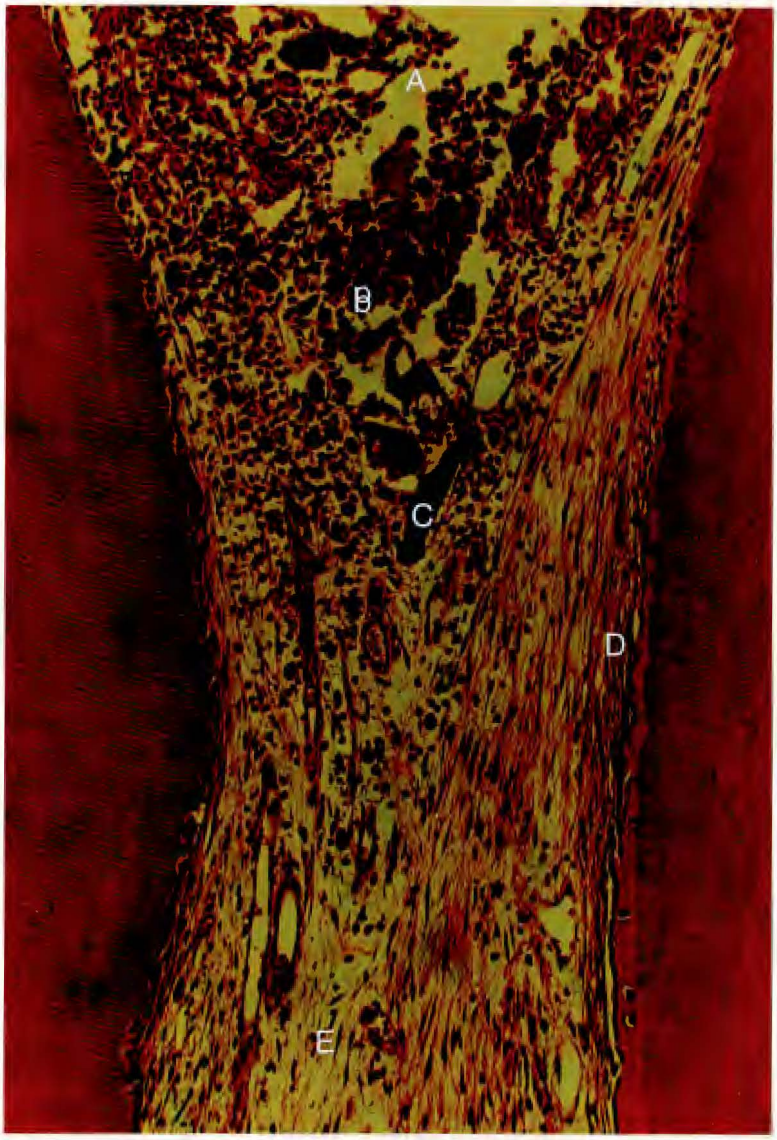




Specimen 5100 LL 28 days after pulptomy with PVP-I

Note:

A) Amputation site
B) Necrotic disorganized pulpal tissue
C) Collagen barrier-like formation
D) Flattened odontoblastic layer
E) Absence of blood vessels



Specimen 5100 UR 28 days after pulpotomy with PVP-I
Note:

A) Amputation site
B) Area of chronic inflammation
C) Dentin-shavings
D) Flattened odontoblastic layer
E) Vital pulp tissue

N.B. Chronic inflammatory cells are highly concentrated at the amputation site, but they extend throughout the canal.

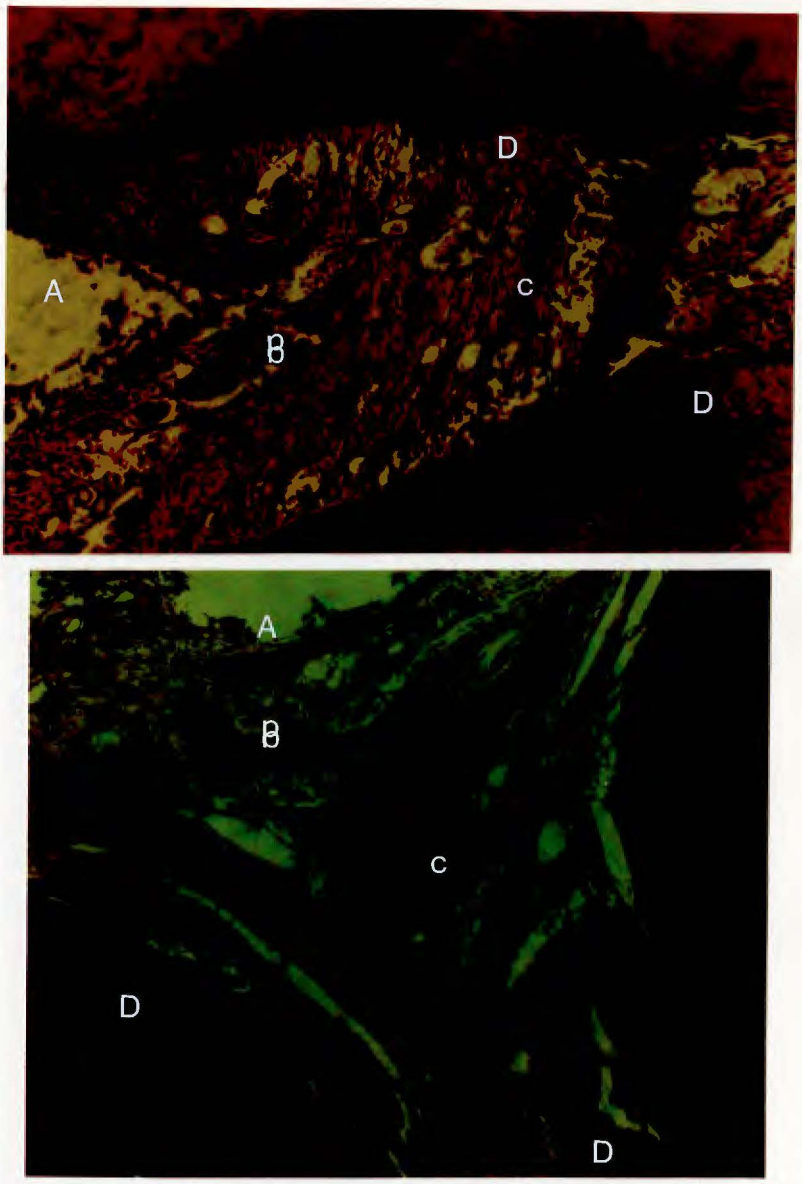
The fibroblasts in the band of collagen fibers were parallel to the collagen fibers and to the surface of necrotic tissue that they encapsulated. The vital tissue beneath this layer appeared to be normal healthy pulp or, in some instances, it contained fewer blood vessels and erythrocites lighter in color. Canals with necrotic tissues showed residues of the collagen fiber barrier and the necrotic cell left by the degenerated pulp.

Variation was the rule otherwise. Six of the vital canals showed some indication of secondary or reparative dentin formation one third to one half down the canal. Three of them were more advanced showing short projections of reparative dentin from the lateral walls towards the center of the pulp.

Considering all specimens, in the study only one root showed an indication of internal root resorption. The tooth, which was in this group, had vital tissue, infiltration of inflammatory cells, chronic inflammation with presence of plasma cells, and no indication of dentin bridge formation. The small area of resorption was located in the middle third of the root.

PVP I - 76 days

The PVP-I, 76 days contained four teeth. Two showed canals with vital tissue. One had canals with necrotic tissue. One specimen contained a necrotic canal and a



Specimens 5093 UR and 5098 LR 76 days after pulpotomy with PVP-I.

Note:
- A) Amputation site
- B) Disorganized tissue with necrotic cells and chronic inflammation
- C) Parallel organization of fibroblasts and collagen fibers in relation to the amputation site
- D) Secondary-reparative dentin

Specimen 5098 UR 76 days after pulpotomy with PVP-I

Necrosis. Degenerated pulpal tissue.

canal with a degenerating process. The roots with necrotic tissues had residues of pulpal cells, no blood vessels, poor cellular detail and rests of the fibrous-like collagen band.

The roots with vital tissues showed infiltration of inflammatory cells. These cells were mainly at the amputation sites. Separating the PVP-I and necrotic tissue from the healthy tissue, was an encapsulating band of collagen fibers and fibroblasts. Apically to the collagen capsule an incomplete dentin bridge was observed. This was formed by elongated projections of irregular dentin from the lateral walls of the canals. These projections were always found at some distance from the PVP-I and necrotic tissue.

Dentin chips left during the operative procedure were found to be surrounded by irregular dentin. In all instances, a wider layer of secondary dentin was observed throughout the canals.

The root undergoing degeneration also revealed projections tending to form a bridge. The tissue showed infiltration of inflammatory cells, poor organization of the pulpal structure, fewer blood vessels and a disrupted odontoblastic layer.

Table 1

COMPARISON OF EFFICACY OF Ca(OH)2 AND PVP-I AS PULP CAPPING AGENTS OVER 7, 28 AND 76 DAYS, USING VARIOUS CRITERIA

	7 Days						28 Days						76 Days						All CASES > 7 Days						
DRUG	Ca(OH)2			PVP			Ca(OH)2			PVP			Ca(OH)2			PVP			Ca(OH)2			PVP			Prob-
NUMBER OF TEETH	3			4			6			8			2			4			11			16			ability
SCORES	0	1	2	0	1	2	0	1	2	0	1	2	0	1	2	0	1	2	0	1	2	0	1	2	
VITAL ROOTS PER TOOTH	0	0	3	3	0	1	0	0	6	3	3	2	0	2	0	1	1	2	0	2	9	7	4	5	<.05
1=1 2=2 OR 3 PROPORT'N	0.0	0.0	1.0	0.8	0.0	0.3	0.0	0.0	1.0	0.4	0.4	0.3	0.0	1.0	0.0	0.3	0.3	0.5	0.0	0.2	0.8	0.4	0.3	0.3	
NECROTIC ROOTS /TOOTH	2	1	0	1	1	2	6	0	0	0	6	2	1	1	0	2	1	1	9	2	0	3	8	5	<.01
PROPORTION	0.7	0.3	0.0	0.3	0.3	0.5	1.0	0.0	0.0	0.0	0.8	0.3	0.5	0.5	0.0	0.5	0.3	0.3	0.8	0.2	0.0	0.2	0.5	0.3	
PMN	0	1	2	3	1	0	0	4	2	2	4	2	1	1	0	0	2	2	1	6	4	5	7	4	NS
1=few 2=many PROP'N	0.0	0.3	0.7	0.8	0.3	0.0	0.0	0.7	0.3	0.3	0.5	0.3	0.5	0.5	0.0	0.0	0.5	0.5	0.1	0.5	0.4	0.3	0.4	0.3	
MACROPHAGES	0	1	2	3	1	0	0	3	3	2	4	2	1	1	0	1	1	2	1	5	5	6	6	4	NS
1=few 2=many PROP'N	0.0	0.3	0.7	0.8	0.3	0.0	0.0	0.5	0.5	0.3	0.5	0.3	0.5	0.5	0.0	0.3	0.3	0.5	0.1	0.5	0.5	0.4	0.4	0.3	
PLASMA CELLS	2	1	0	4	0	0	4	2	0	7	1	0	1	1	0	4	0	0	7	4	0	15	1	0	NS
1=few 2=many PROP'N	0.7	0.3	0.0	1.0	0.0	0.0	0.7	0.3	0.0	0.9	0.0	0.0	0.5	0.5	0.0	1.0	0.0	0.0	0.6	0.4	0.0	0.9	0.1	0.0	
FIBROUS TIS PER TOOTH	1	2	0	2	2	0	2	4	0	5	0	3	2	0	0	0	1	3	5	6	0	7	3	6	<.05
1=few 2=many PROP'N	0.3	0.7	0.0	0.5	0.5	0.0	0.3	0.7	0.0	0.6	0.0	0.4	1.0	0.0	0.0	0.0	0.3	0.8	0.5	0.5	0.0	0.4	0.2	0.4	
DENT BRIDGE EXTENT	3	0	0	4	0	0	0	5	1	8	0	0	0	1	1	1	3	0	3	6	2	13	3	0	<.05
1=par 2=comp PROP'N	1.0	0.0	0.0	1.0	0.0	0.0	0.0	0.8	0.2	1.0	0.0	0.0	0.0	0.5	0.5	0.3	0.8	0.0	0.3	0.5	0.2	0.8	0.2	0.0	
DENT FORMAT'N INCR'SE	2	1	0	4	0	0	0	4	2	5	2	1	0	2	0	0	1	3	2	7	2	9	3	4	<.05
1=min 2=major PROP'N	0.7	0.3	0.0	1.0	0.0	0.0	0.0	0.7	0.3	0.6	0.3	0.1	0.0	1.0	0.0	0.0	0.3	0.8	0.2	0.6	0.2	0.6	0.2	0.3	
PULP STONES PER TOOTH	3	0	0	3	1	0	6	0	0	8	0	0	2	0	0	4	0	0	11	0	0	15	1	0	NS
1=min 2=major PROP'N	1.0	0.0	0.0	0.8	0.3	0.0	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0	0.9	0.1	0.0	
ROOT RESORP PER TOOTH	3	0	0	4	0	0	6	0	0	7	1	0	2	0	0	4	0	0	11	0	0	15	1	0	NS
1=min 2=major PROP'N	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0	0.9	0.1	0.0	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0	0.9	0.1	0.0	
LYMPOCYTES PER TOOTH	0	2	1	3	1	0	2	2	2	1	5	2	1	1	0	1	0	3	3	5	3	5	6	5	NS
1=few 2=lots PROP'N	0.0	0.7	0.3	0.8	0.3	0.0	0.3	0.3	0.3	0.1	0.6	0.3	0.5	0.5	0.0	0.3	0.0	0.8	0.3	0.5	0.3	0.3	0.4	0.3	

Pulpal Tissue Reaction to $Ca(OH)_2$ and PVP-I

Table 2

MEDICAMENT USED	PERIOD	# OF TEETH	# OF ROOTS	# OF NECROTIC ROOTS	# OF VITAL ROOTS	NO BRIDGE	INCOMPLETE BRIDGE	COMPLETE BRIDGE
CaOH	7 days	3	7	1	6	7	0	0
PVP1	7 days	4	7	5	2	7	0	0
CaOH	28 days	6	13	0	13	0	9	4
PVP1	28 days	8	17	10	7	17	0	0
CaOH	76 days	2	3	1	2	1	1	1
PVP1	76 days	4	9	3	6	3	6	0

CHAPTER IV

DISCUSSION

The present study was based on the hypothesis that PVP-I would promote healing of vital pulp tissue when applied as a dressing agent to pulpotomized sites. Key to the hypothesis is the definition of healing. Glass and Zander[22] proposed the following definition of dental pulp healing:

"In general, healing may be defined as the restoration of a tissue to its normal structure and function. The dental pulp is normally encapsulated by dentin and an adherent odontoblastic layer. Therefore, in applying this definition to the pulp, healing means the presence of healthy tissue capable of carrying on the function of the pulp, a continuous odontoblastic layer, and the erection of a dentin barrier walling off the exposure." Factors cited as important to promote healing of the remaining pulp stumps in pulpotomy sites are:

a) healthy pulp tissue with minimal or no inflammation present.

b) adequate pulpotomy technique

c) a non-toxic, biologically compatible pulp dressing material.

Factors that have been reported as interfering with pulpal healing are:

a) infection[28,61]

b) chronic inflammation[31,61,73]

c) toxic dressing materials[31,37]

d) faulty pulpotomy techniques[34,37]

In consideration of these factors, the study was designed so that only healthy primary first molars in young monkeys were used. No carious teeth were used and artificial inflammation of the pulp was not promoted prior to the pulpotomy procedures. Procedures were performed under aseptic conditions. Following this design prevents the introduction of contaminating variables in the study. As this eliminates the possibilities of infection and previous chronic pulp inflammation, results could only be to the surgically induced inflammation, the dressing materials and the technique employed.

Considering the parameters of Glass and Zander, $Ca(OH)_2$ containing dressing materials have produced the best pulpal healing reactions.[4,21,55,56,57,68,82] Dycal* was chosen as the dressing agent for the control group because it is a commercial product that has yielded consistently good results.[1,55,60,68,75]

The results in the present investigation are in agreement with previous reports.[48,55,56] The samples treated with Dycal showed positive healing reactions. That is, the pulpal tissue remained vital, virtually non-inflamed, reorganized itself, and formed an

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*Dycal - L.D. Caulk Comp., Milford, Del.

odontoblastic layer and a dentinal bridge. The findings confirm the adequacy of the pulpotomy procedures, the health status of the teeth and the healing potential of the pulps.

The initial tissue reaction could not be evaluated for either of the two dressing agents. The initial reaction as reported by Schroeder[55,56] and Glass et al.[21] is described as the formation of a superficial layer of necrosis, with a zone of coagulation necrosis demarcating the vital tissue. They reported migration of inflammatory cells into the wound area, about six hours after the procedure. After a few days, migration and proliferation of pulpal cells, most probably mesenchymal and endothelial cells, are expected to approach the necrotic zone. Unfortunately, the first post treatment samples were obtained at one week and these changes were observed much earlier in the postoperative period. Indication of these events, nonetheless, could be observed in the present study at the seven day interval. Concomitantly, proliferation of collagen fibers and blood vessels was evident.

Histological observations of the seven day $Ca(OH)_2$ samples also showed an indication of vital tissues undergoing repair. The finding is similar to a report by Schroeder and Granath.[56] Some of the lining cells were also found in contact with the medicament, without the

presence of a necrotic zone. This also has been observed by previous investigators.[56] Proliferation of fibroblasts, collagen fibers and blood vessels are indications of tissue that is undergoing normal wound repair.

The necrotic layer found in conjunction with the $Ca(OH)_2$ treatments may have importance. Veis[77] proposed that this layer offers a biologically receptive surface for the pulp cells to attach and polarize prior to expressing their odontoblastic potential. The proposal is derived from the theory that, as the dental papillae develops, the epithelial cells elaborate a basal lamina to which a layer of mesenchymal cells is attached. Veis considered that treatment of an exposed pulp must be directed to the creation of a surface to which those pulp cells partially committed to an odontoblastic program, will attach and polarize.

We could not confirm this theory as in many cases no necrotic layer could be observed between the $Ca(OH)_2$ dressing and the newly formed dentin layer. Nonetheless, what was very obvious is that the $Ca(OH)_2$ dressing did promote pulpal wound healing. It was beyond the scope of this study to determine what is the specific action of Dycal^R on the pulp. However, serum and fibroblasts furnish fibronectin and new blood vessels laminin and fibronectin. These are substrate adhesion molecules of

extracellular matrix.[53]

The $Ca(OH)_2$ mechanism to promote healing and bridge formation with reorganization of the odontoblastic layer is unknown. Sciaky and Pisanti[58] used a paste of $Ca(OH)_2$ containing radioactive calcium (Ca^{45}) over amputated pulps in dogs, to determine if the calcium ions of the dressing agent entered into the formation of the new dentin bridge. They showed that the calcium taking part in the mineralization of the barrier came from the tissue and not from the calcium hydroxide medicament. However, the opposite conclusion was reached by Holland et al.[27] They used calcium, barium and strontium hydroxides in histochemical studies of dog pulp following pulpotomy. Similar results were obtained with the three pulp-capping agents, i.e., a necrotic zone followed by a layer of large von Kossa-positive granulations. The large granulations consisted of calcium, barium and strontium carbonates, which indirectly showed that the calcium was derived from the calcium hydroxide and not from the pulp tissue.

After the initial dentin layer was formed, the osteodentin bridge continued reorganizing itself towards the creation of a dentin barrier. Eventually the barrier should wall off the pulpal exposure in a healing process, as advocated by Glass and Zander.[22]

Seventy-six days following the pulpotomy procedures, those pulp stumps that were covered with calcium hydroxide

showed vital tissue and increased bridge formation. It was interesting to observe this tissue reorganizing itself initially, forming a "structureless" layer of dentin, continuing to lay down a dentin with bone-like appearance and turning itself into well-structured dentin in the latest layers formed.

The pulpal reaction to PVP-I did not form a cell-polarizing surface. There was lack of organization of the pulpal tissue underlying the medicament. The tissue actually became inflamed and without displaying signs of recovery. There was always a layer of necrotic tissue in contact with the PVP-I. The cells in deeper areas of the canal reacted forming a fibrous-like barrier that isolated the healthy tissue from the toxic effect of the medicament and/or the necrotic tissue in the superficial layers. The collagenous barrier also failed to act as the polarizing surface, as the odontoblasts did not reorganize into their common layer along the coronal surface of the pulp tissue. The dentin barrier was only formed by the reparative dentin coming from the lateral walls.

This is an indication of a negative stimulus by the medicament. No dentinal bridge was found near the site where the dressing material was applied. Inflammatory cells were always present in the specimens of these groups. Abundant infiltration of PMN's and lymphocytes

was present in the tissue surrounding the medicament, whereas progressively less inflammatory cells were observed towards the apical areas.

In summary, the appearance of the tissues reacting positively when the PVP-I was used as the dressing agent, showed the dressing agent in contact with necrotic tissue, followed by a wide area of disorganization and poor cellular detail with significant infiltration of inflammatory cells. This wide layer appeared to form a cushion, after which a fibrous layer was being formed and organized along with some fibroblasts in a barrier-like disposition, apparently a temporary barrier against the effect of the superficial layers on the more apical healthy tissue.

Whenever a dentin bridge was in the formative stage, it was always found in an area apical to the fibrous-like barrier. The dentin bridge was formed by protruding elongations of secondary dentin coming from the lateral walls towards the center of the canal. The formation of the collagen barrier was an indication of a foreign material effect or toxic stimulus affecting the pulp. The initial formation of the barrier was inadequate for pulpal healing, as some of the specimens underwent tissue necrosis after the initial organization of the barrier. The prevalance of inflammation throughout the entire study was another indication of the toxicity that the PVP-I

presented to the dental pulp.

The preparation of iodine with povidone does not affect the toxicity that the iodine, as an allogen that it is, has to the soft tissues. It was also possible to confirm that necrotic tissue is an irritant to the remaining vital pulp tissue.

We could observe two different reactions to the irritation produced by necrotic tissue. With calcium hydroxide there was a reorganization of the paralellized bodies of the odontoblasts. With PVP-I and necrotic-cells layer there was an encapsulating reaction of the pulp. This appears to be a common barrier reaction of the connective tissue towards irritating stimuli.

It was interesting to observe in the PVP-I groups the healing potential of the dental pulp and its ability, like other connective tissues, to encapsulate foreign body material, or organize a collagen fiber barrier to isolate the toxic material from the healthy tissue.

Another important aspect differentiating the reactions of the pulp to both medicaments was the pattern of healing. Whenever a secondary dentin bridge was being formed in the PVP-I specimens, the matrix was laid down more rapidly at the lateral walls of the canals. In the $Ca(OH)_2$ specimens, the initial osteodentin was laid down at the wound innerface without always being a continuation

from the tissue being formed at the lateral walls. Isolated masses of osteodentin were laid down along the path where the bridge was to be formed.

The few cases which presented necrotic pulps in the $Ca(OH)_2$ groups were accompanied by extrapulpal hemorrhage. This was an indication of failure in controlling pulpal bleeding before applying the dressing agent. The hemorrhage that induces inflammation apparently interfered with the healing properties of the $Ca(OH)_2$.

There were no findings of internal root resorptions in the $Ca(OH)_2$ groups. Inflammation was not present as an indication of deeply damaged tissue, but as a normal local factor present at the repair site of a clean wound.

Dentin shavings impacted in the pulp stumps during mechanical removal of the cameral pulp tissue were found to be surrounded by new amorphous, atubular dentin. It has been referred to in the literature as a "seeding" effect, because the isolated shavings of dentin are encapsulated by the reparative dentin which apparently enlarges with time. The finding, although more pronounced in the $Ca(OH)_2$ specimens, was evident in the PVP-I groups as well. Dentin is also known as an ostegenic inducer of calcifiable martrix in muscle.[76,81]

CHAPTER V

CONCLUSIONS

Based on the conditions of the present investigation, the following was concluded:

1. PVP-I is not an innocuous medicament when used as a pulp dressing agent.

2. PVP-I does not appear to assist the pulpal tissue in reorganizing itself after being wounded.

3. PVP-I does not promote healing of the vital pulp tissue when used as a capping agent in pulpotomies on primary molars.

4. The reaction of the pulpal tissue to PVP-I is variable and unreliable.

5. PVP-I may induce chronic pulpal inflammation.

6. The study supports previous reports in reaffirming the healing potential of pulpal tissue when a $Ca(OH)_2$ medicament is used.

7. Commercially supplied $Ca(OH)_2$ (Dycal) promotes pulpal healing when used to cover non-inflamed pulpotomized pulps.

8. $Ca(OH)_2$ (Dycal) does not promote pulpal inflammation under the conditions used in the present study.

9. $Ca(OH)_2$ (Dycal) promotes pulpal tissue repair and dentin bridge formation.

10. Controlling pulpal bleeding prior to the placement of a pulpal medicant is an important factor for achieving healing in pulpotomized dental pulps.

11. The dental pulp has different protective and reparative mechanisms. Some of the different reactions include: (a) dentinal bridging under the dressing material, (b) encapsulation or isolation of necrotic and toxic material by a collagen barrier, and (c) formation of secondary or reparative dentin at the lateral walls of the canals.

12. Pulp tissue forms osteodentin bridge arising from the lateral walls of the canal, as well as bridge formed by the union of calcifying isles that appear in the pulpal tissue itself.

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

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