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Electrosurgical pulpotomy in primates—a comparison with formocresol pulpotomy

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Abstract

The purpose of this study was to contrast histologically the pulp response to formocresol with the response to electrosurgical pulpotomy in a primate model. Both the primary and permanent incisors and molars were treated. Coronal pulp amputation was accomplished mechanically. The remaining pulp stumps were treated by either formocresol or momentary electrosurgery exposure. Results were evaluated at one hour, one week, and two months postoperatively. Results with the conventional formocresol treatment were consistent with other studies showing favorable pulp response. The electrosurgical approach produced no furcation or periapical involvement and exhibited no evidence of necrosis in the apical two-thirds of the root. Histologically, in this study electrosurgery pulpotomy had as favorable a tissue response as the conventional formocresol treatment for the time studied.

The formocresol pulpotomy recently has come under close scrutiny due to possible systemic distribution of formocresol with attendant potential toxic effects.[1-11] Buckley's original concept to detoxify the intermediate and end products of pulpal infection and inflammation was based upon a nonbiologic empirical theory.[1-3, 8, 12] The technique has been used with varying degrees of clinical success for years.[1-3, 5, 6, 13-15] Surveys suggest that the formocresol pulpotomy in primary teeth is considered the treatment of choice in a majority of pedodontic departments in North America.[1] The degree of clinical success has ranged widely.[14] Success, however, must incorporate the possible systemic effects of formocresol and potential involvement of permanent succedaneous teeth. Several studies have shown that formocresol or one of its metabolites appears outside the tooth both locally and systemically.[4-11] Systemic manifestations must be considered a possibility since formocresol is a material toxic to cells and tissue.[1-7, 9, 16] Enamel surface defects on permanent successors are an additional potential problem.[5]

Alleged biocompatible alternatives have been advocated including the use of calcium hydroxide[17, 18] glutaraldehyde,[16] and electrosurgery.[19-21]

Various texts and papers have suggested electrosurgical pulpotomy for years.[19-21] There is a paucity of histological data concerning pulpal tissue response to electrosurgery. The only available information presents conflicting results and these appear to be technique-dependent.[18, 22]

The electrosurgical pulpotomy has merit. It is much quicker than the formocresol approach. It is self-limiting—pulpal penetration is only a few cell layers deep. There is hemostasis and good visualization without chemical coagulation and no systemic involvement.

The purpose of this study was to evaluate electrosurgical pulpotomy at the histologic level. The results of the standard formocresol technique were contrasted to that of electrosurgical pulpotomy in primary and young permanent teeth in pigtail monkeys.

Methods and Materials

Three *Macaca nemestrina* monkeys (two male, one female) designated M_1, M_2, M_3[a], age approximately two years, were selected because of incomplete development of permanent teeth and reduced amount of resorption of primary teeth.

The animals were tranquilized with ketamine hydrochloride (10 mg) prior to treatment and then given 25 mg/cc thiamyial sodium[b] intravenously until fully sedated. Preoperative radiographs were made. Rubber dam was applied using quadrant isolation and the field was swabbed with alcohol sponges. All equipment was sterilized prior to use.

All teeth were prepared with a 330 bur in a high-speed handpiece until the enamel was penetrated and the pulp chamber exposed. A #2 round bur was used to amputate

[a] Primates from the Regional Primate Research Center, University of Washington, supported by NIH Grant #RR00166.
[b] Surital, Parke-Davis, Division of Warner Lambert Co., Morris Plains, N.J.

the coronal pulp in conjunction with a sharp spoon excavator. Hemorrhage was controlled with a dry sterile cotton pellet. Any remaining debris was removed by irrigation with normal saline and spoon excavator as required. Molars were then treated with formocresol alternating side and arch, either first permanent or second primary. A pellet of cotton was moistened with Buckley's formula[c] formocresol and thoroughly blotted by squeezing it twice between a sterile gauze square. The pellet was placed in apposition to the amputated pulp stumps for five minutes. Upon its removal a paste of zinc oxide and eugenol (ZOE) was applied to the pulp stump with as little pressure as possible. An amalgam restoration was then placed with minimum pressure and carved.

The alternate molar in each quandrant was treated electrosurgically. The pulp exposure and amputation was identical to the formocresol approach. Once hemostasis was accomplished with dry cotton pellets, the pulp stumps were touched by the U-shaped electrode. Fully rectified unfiltered current was used, intensity setting of three, which caused neither sparking nor tearing of tissue (this indicated an appropriate setting). The electrical unit[d] was used because it allowed current selection. The pulp stumps were touched only momentarily by the electrode in a brushing type of stroke. When additional electrocoagulation was necessary, we allowed 10 seconds to elapse before readministration to decrease possible lateral heat accumulation. ZOE and amalgam were then placed after the pulp treatment as previously described.

The maxillary anteriors (centrals and laterals) all were treated electrosurgically to obtain a larger number of experimentally treated teeth. Eight teeth in each animal were treated with electrosurgery and four teeth with formocresol.

Monkey M_1 was sacrificed one hour after the pulpotomy treatment; M_2 one week after treatment; and M_3 two months after treatment. All were sacrificed with formalin perfusion to enhance fixation. Following sacrifice, the mandibles and maxillas were removed in bloc and then sectioned into quadrants which were placed immediately in 10% formalin. After fixation for one week, the quadrants were x rayed and placed in 8 N formic acid in sodium formate for demineralization. Subsequently, the quadrants were grossly bisected longitudinally. Semiserial paraffin sections, five microns thick, were prepared to include the pulp, the apex, and surrounding tissue. In all quadrants the first primary molars were untreated and served as controls. The sections were stained with hematoxylin and eosin.

The sections were distributed randomly and the treatment modality for each case was not revealed to the examining oral pathologist. A histological comparison between the formocresol- and electrosurgically treated teeth was then made by the oral pathologist. The degree of inflammation, necrosis, and repair was described and classified. Inflammation was classified as acute or chronic. Necrosis was classified as localized or diffuse and any involvement of the periapical areas was noted (as was any attempt at repair).

[c] Buckley Pharm. Co., Burbank, Calif. (10% formaldehyde, 35% cresol dissolved in glycerine and water).

[d] Ellman 90 FFP Dento-Surg., Ellman International, 1135 Railroad Ave., Hawlett, N.Y.

Results

Post-treatment—One Hour

The histological evaluation of the formocresol-treated teeth demonstrated comparable results with previously published data for all time periods.[23] At one hour the pulpal tissue exhibited a proteinaceous fluid at the pulp chamber-pulp interface. Proceeding apically, there was localized necrosis with minimal chronic inflammation progressing to an acellular zone. An area of pulpal edema preceded the normal pulpal tissue, observed from the coronal one-third of the canal to the apex. No furcal or periapical involvement was observed.

The electrosurgery results demonstrated cellular and nuclear debris and localized coagulation necrosis at the pulp chamber-pulp interface. Below this, a layer of increased fibrosis preceded normal pulpal tissue. Only the coronal one-fourth of the canal was affected. No periapical or furcal involvement was observed.

Post-treatment—One Week

Formocresol-treated teeth demonstrated a layer of fixed tissue followed by a cell-free zone. Directly below this zone, necrotic cellular debris was accompanied by a minimal acute and chronic inflammatory infiltrate. An area of increased fibroblastic proliferation gradually preceded normal tissue. Only the coronal one-third was affected with no periapical or furcal involvement.

The electrosurgically treated teeth revealed a layer of coagulation necrosis 2–3 cell layers thick followed by a cell-free zone and edema. A cellular layer of proliferating fibroblasts and extravasated red blood cells was present above the normal pulpal tissue which comprised approximately four-fifths of the canal. There was no periapical or furcal involvement.

Post-treatment—Two Months

The formocresol pulpotomies revealed an eosinophilic zone at the pulp chamber-pulp interface, followed by localized necrosis with mild acute and chronic inflammation apically (Figures 1 and 2). Fibroblastic proliferation was accompanied by an increase in intercellular collagen fibers. Secondary dentin often was deposited on the canal walls directly below a fibrous bridge. Normal pulpal tissue was present at the apical half. No periapical or furcal involvement was observed.

The electrosurgical pulpotomy demonstrated minimal necrotic debris at the pulp chamber-pulp interface with

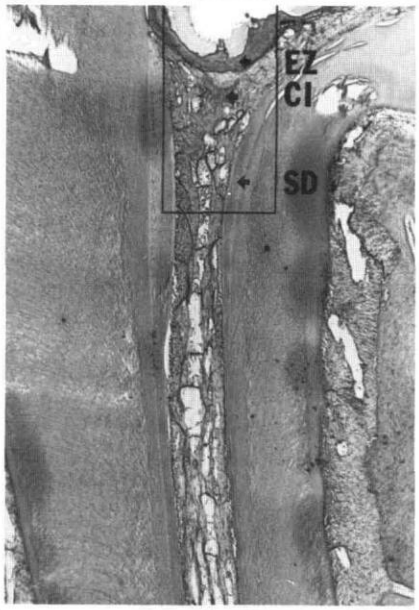

Figure 1. (left) Two months postformocresol pulpotomy; an eosinophilic zone is seen at interface (EZ). Viable pulp with minimal chronic inflammation is seen below the interface (CI). No horizontal fibers are present. Secondary dentin is present (SD) 60×.

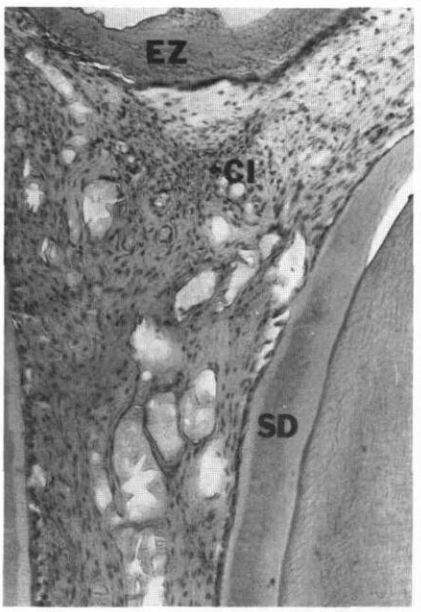

Figure 2. (right) Same features at higher power (100×).

...nd chronic inflammatory response (Figures 3 and 4). Directly below the inflammatory response, a reorganization of the fibroblasts into parallel bands perpendicular to the long axis of the pulp was observed (Figures 3 and 4). Apical to this area, secondary dentin was deposited characteristically on the lateral walls. In some cases the dentin deposition almost formed transpulpal bridges. The remaining two-thirds of the root revealed a normal to slightly fibrotic pulp. No furcal or periapical involvement was observed. One tooth revealed foreign body-type reaction with internal resorption just above normal pulpal tissue.

None of the experimental or control teeth exhibited total pulpal necrosis or abscess formation.

Discussion

The empiricism supporting the use of formocresol as a pulpotomy agent has been questioned for many years. Because of systemic dissemination and potential toxicity, alternative approaches to the maintainance of partial pulpal vitality have been sought. Even though the electrosurgical pulpotomy technique has been advocated for years, there was very little histological data to support or refute its use prior to this study. An appropriate animal model needed to be studied prior to any clinical use. The primate model used in this study (*Macaca nemistrina*) proved to be excellent. The formocresol results very closely duplicated results previously established on humans.[24]

The age of these monkeys allowed both primary and permanent teeth to be evaluated. Both anterior and posterior teeth were treated to duplicate clinical situations. The times of post-treatment evaluation consisted of an immediate response; a response of an acute nature at one week; and a delayed response of eight weeks after a period of recovery from mechanical instrumentation.

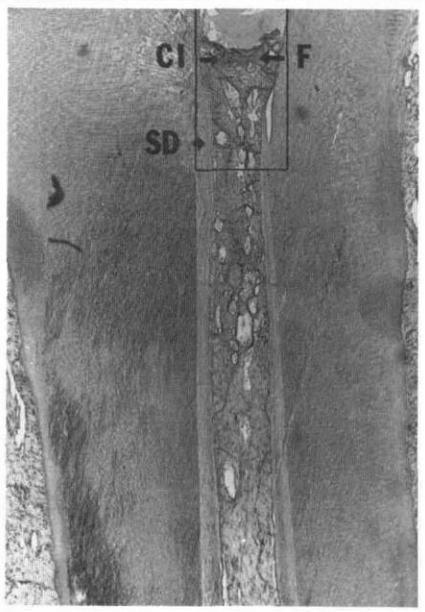

Figure 3. (left) Two months postelectrosurgical pulpotomy; viable pulp tissue with minimal chronic inflammation below interface (CI). The coronal connective tissue fibers are horizontal (F). Secondary dentin is present (SD) 60×.

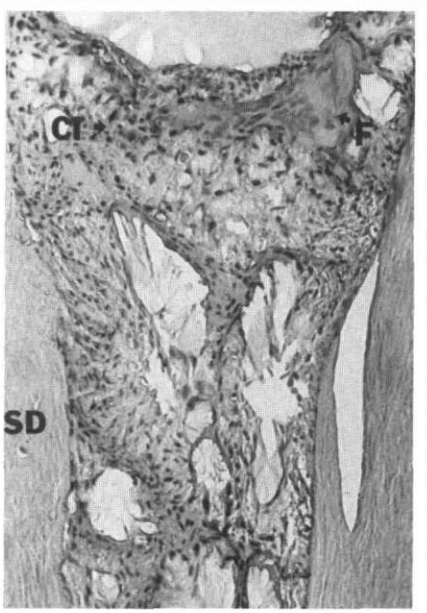

Figure 4. (right) Same features at higher power (100×).

The discrepancy in size between primate and human teeth creates a potential area of concern and deserves discussion. The smaller pulps and body size of the monkey may be an important factor in the amount of lateral heat accumulation and the correct amount of current used. Differences in type of current and duration of application are as significant for electrosurgery as concentration and duration of application are in the use of formocresol. Local anatomy and differences in status of pulp prior to treatment also must be considered. In this study no difference was noted between primary and permanent teeth. Only healthy pulp was treated. A follow-up study treating inflamed or infected pulp would be of value.

ZOE was used as a base over the pulpal stumps not because of its biologic properties but because it is the treatment traditionally prescribed. Alternative biocompatible bases might provide better results, eliminating the inevitable tissue response seen with ZOE. It is difficult to differentiate pulp response to pulpotomy agent vs. the response to ZOE. On the other hand, it is possible that the electrosurgery might provide a coagulation layer that would block the effects of ZOE.

The use of electrosurgery in an opened pulp probably does not accumulate as much lateral heat as an enclosed pulp.[26] In a study with negative electrosurgical results[22] the coronal pulps were partially removed with the electrosurgery. This might have been from an excess amount of lateral heat which can accumulate, causing resorption and necrosis. Therefore, it is imperative that electrosurgery be limited to touching the surface of the root pulp stump only, with 10-second delays between applications.

The question arises as to whether or not electrosurgery decreases the chance of an immune reaction. Formocresol can alter postpulp tissue and render it antigenically active.[1, 27] A similar study should be performed testing antigenicity of electrosurgically treated pulps.

The lack of periapical, furcal, or necrotic involvement suggests electrosurgery to be a viable pulpotomy procedure without the dissemination and toxic ramifications of formocresol. Evidence of secondary dentin formation in the teeth treated with electrosurgery would suggest an effort by a healthy, vital pulp to wall off or heal the area of insult. Since this is a relatively short postoperative evaluation (8 weeks) further studies with longer follow-up need to examine the effect of electrosurgery on both infected pulps and human teeth.

Conclusions

This histological study examined formocresol and electrosurgical pulpotomy techniques. Both systems maintained vitality for up to eight weeks in this primate model. No evidence of periapical, furcal involvement, or total pulp necrosis was observed.

The views expressed herein are those of the authors and do not necessarily reflect the views of the United States Air Force or the Department of Defense.

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Quotable Quotes

Huntingdon's disease, characterized by involuntary movement and progressive dementia, is transmitted as an autosomal, dominant trait whose effect is not seen until the third or fourth decade of life. The tragedy of the disorder lies not only in the effects of the disease itself, but in the absence of any genetic marker to diagnose those at risk; hence transmission of the disease to offspring commonly occurs before symptoms develop. Any inquiry into the cause of the disease must tackle this central issue of how a gene mutation can permit normal development, integration and function of neuronal systems for some thirty to forty years and yet then bring about the relentless, regionally selective, premature death of neurones. . .

Perhaps the answer will ultimately come from the new techniques of molecular genetics. The Huntington's disease gene is considered to have a penetrance of 100% and the mutation rate is exceedingly low; indeed, no proven cases of sporadic mutation have ever been described which satisfy the criteria of proven parentage, with parents surviving to an old age without symptoms, pathologically proven Huntington's disease in the new case, and subsequent transmission to a child. The low mutation rate makes it possible and practicable to search for a DNA polymorphism which might be useful as a genetic linkage marker. . . In the long term, it should be possible to survey each chromosome in the human genome sequentially, first for a polymorphism to provide a genetic linkage and ultimately to localize the gene itself. There will then be a real possibility of identifying the metabolic defect.

From: Martin, Joseph B. Huntington's disease: genetically programmed cell death in the human central nervous system. Nature 299:205, September, 1982.