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JOURNAL TITLE: Operative dentistry
JOURNAL ARTICLE AUTHOR: Cox, F
JOURNAL ARTICLE TITLE: Tunnel defects in dentin bridges: their formation following direct pulp capping.
VOLUME: 21
ISSUE: 1
MONTH:
YEAR: 1996
PAGES: 4-11

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Tunnel Defects in Dentin Bridges: Their Formation Following Direct Pulp Capping

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S SUZUKI • S H SUZUKI

Clinical Relevance
This study reemphasizes the need to employ biologically relevant measures that will provide a long-term clinical seal against microleakage following direct pulp capping with Ca(OH)\textsubscript{2} medicaments alone.

SUMMARY

This study was conducted to observe the formation and nature of tunnel defects in dentin bridges, assess the nature of the associated soft tissue elements, and note the relationship of pulp inflammation and necrosis associated with these defects. A total of 235 teeth with class 5 cavity preparation exposures were randomly distributed throughout the dentitions of 14 adult rhesus monkeys. Each pulp was exposed and left open to the oral microflora at one of four time intervals, flushed with saline, debrided, capped with one of two hard-set calcium hydroxide medicaments [Ca(OH)\textsubscript{2} (Dycal or Life)] and restored with a dispersed-phase amalgam alloy. Observation times were 14 days, 5 weeks, and 1 and 2 years. A total of 192 dentin bridges formed against the Ca(OH)\textsubscript{2}, medicaments Life or Dycal in 235 pulp-capped teeth. Considering all four capping periods, 89% of all dentin bridges contained tunnel defects (172 of 192). Forty-one percent (78) of the 192 dentin bridges were associated with recurring pulp inflammation or necrosis and were always associated with the presence of inflammatory cells and stained bacterial profiles. This study demonstrates that a statistically significant number of dentin bridges contain multiple tunnel defects, most of which appear to remain patent. These patent tunnels fail to provide a hermetic seal to the underlying pulp against recurring infection due to microleakage. Most Ca(OH)\textsubscript{2} medicaments have been reported to disintegrate and wash out after 6 months, leaving a void underneath the restoration and thereby a pathway for bacterial infection. This study reemphasizes the need to employ biologically relevant measures that will provide a long-term clinical seal against microleakage following direct pulp capping with Ca(OH)\textsubscript{2} medicaments alone.

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INTRODUCTION

A comprehensive review of the pulp-capping literature (Baume & Holz, 1981) reported a 90% incidence of new dentin bridge formation in exposed pulps. Many authors maintain that regeneration of a new dentin bridge is a sign of successful pulp treatment. However, this only appears to be true when a long-term bacterial seal is effected and maintained. Studies (Hess, 1950; Nyborg, 1955, 1958; McWalter, el-Kafrawy & Mitchell, 1976; Ichikawa, 1976; Cvek, 1978; Haskell & others, 1978; Heys & others, 1980, 1981; Cox & others, 1982; Pitt Ford & Roberts, 1991) have reported that pulp healing and new dentin bridges form when exposed dental pulps are direct capped. Others (McWalter, el-Kafrawy & Mitchell, 1973; Watts & Paterson, 1979; Holland & others, 1982) have suggested that a dentin bridge will form against Ca(OH)$_2$ capping when exposed pulps are direct capped. Eda (1961) showed evidence of a bordering zone that developed at some distance from the level of pulp amputation, and Schröder (1972) speculated that Ca(OH)$_2$ provoked coagulation necrosis due to its high alkalinity. Clarke (1970) postulated that a complete ring of reparative dentin encircles the wound in a circumferential manner from the periphery of the exposure site, and that the dentin bridge fills in towards the center of the wound.

However, others have reported that dentin bridges are incomplete in their formation following direct pulp capping and healing. Zander (1939) reported inflammation, and Schröder (1972) reported coagulation necrosis under what appeared to be complete dentin bridge formation. However, neither reported tunnels within the new dentin bridges. Schröder and Granath (1972) reported that the first-formed hard tissue of any dentin bridge is irregular with tubular openings or canalicular lumina, which contain vessels and cells, but will further differentiate to form tubular dentin. Rowe (1967), Watts and Paterson (1979), and Cox and Bergenholtz (1984) reported vessel-filled porosity defects or voids within the substance of new dentin bridges. Langeland and others (1971b) questioned the nature of a complete dentin bridge by showing recurring spaces in serial sections adjacent and throughout the so-called complete dentin bridge. These perforation spaces were often filled with soft tissue inclusions and inflammatory cells. Russo, Holland, and Souza (1982) demonstrated that dentin bridges in human teeth were porous to dyes. Scanning electron microscopic studies by Ulmansky, Sela, and Sela (1972) and Goldberg, Masone, and Spielberg (1984) showed large multiple tunnel defects with cellular elements in dentin bridges of human premolars following pulp capping with Ca(OH)$_2$. A long-term capping study (Cox & others, 1985) showed recurring pulp inflammation and necrosis associated with tunnel defects in dentin bridges. Mjör (1972) cautioned that capping of young healthy pulps may lead to a poor prognosis and that the presence of a dentin bridge itself is not a proper criterion for successful long-term pulpal healing. He also reported that many of the hard tissue bridges contained strings of pulp tissue that traversed the hard tissue (Tronstad & Mjör, 1972). Walton and Langeland (1978) reported that seemingly unbroken dentin bridges often contain multiple perforations and suggested that the use of the term “dentin bridge” is a myth, due to the presence of imperfections in most bridges. The presence of imperfections is demonstrated in the data of Russo and others (1982).

This study of hard-tissue formation was completed to observe and evaluate the incidence of tunnel defects in new dentin bridges after pulp capping with two commercially available hard-set Ca(OH)$_2$ medicaments at various time intervals.

METHODS AND MATERIALS

This study of pulp capping and dentin bridge formation was carried out on 235 noncarious class 5 cavities that were distributed throughout the dentitions of 14 adult rhesus monkeys. Prior to operative procedures, each monkey was sedated with an intramuscular injection of Ketamine hydrochloride (Parke Davis, Rochester, MI 48307) and xylazine (15 mg/kg body weight). Each tooth was mechanically exposed using an ultra-high-speed air turbine (200-300,000 rev/min) with an air-water spray and a new inverted cone carborde bur (ISO size 010) on every fourth tooth. Following exposure to oral microflora for various time periods (Cox & others, 1982, 1985), each preparation was mechanically debrided with a curette, rinsed with sterile saline, and, following hemorrhage control, was capped with one of two hard-setting Ca(OH)$_2$ materials. The remainder of the preparation was sealed to the cavosurface margin with Dispersalloy amalgam. No other intermediate liners or therapeutic treatments were employed. Seven teeth were direct pulp capped and observed at 14 days, 135 teeth were direct pulp capped for 5 weeks, 42 teeth were direct pulp capped for 1 year, and 51 teeth were direct pulp capped for 2 years. Preparatory to sacrifice, each animal was sedated with sodium pentobarbital (25 mg/cc) and given approximately 1000 units of heparin sodium (Oraganon Inc, West Orange, NJ 07052) to prevent clotting and capillary obstruction during vital vascular flushing. A 10-minute left ventricular flush with 0.9% physiologic saline flush was completed, followed by a 15-minute vascular fixation using a glutaraldehyde phosphate buffered paraformaldehyde GTA-PBF, as per Cox and others (1982). Each tooth was cut from its surrounding alveolus, given a code number, and post-fixed in fresh
GTA-PBF at room temperature. After 24 hours, solutions were changed to 0.5M EDTA (pH 7.2) for demineralization. Following radiographic confirmation of endpoint demineralization, each tooth was rinsed through 20 changes of distilled water. dehydration was carried out through ascending grades of N-butyl alcohol. Each tooth was embedded in Paraplast Plus (Sherwood Medical, St Louis, MO 63103) and serial sectioned in a sagittal plane at 7μm. Slides were alternately stained with hematoxylin and eosin, Preece’s trichrome, and a bacterial stain (McKay, 1970). In order to observe the histological nature of each dentin bridge, complete mesiodistal serial sections were cut, beginning before the exposure and through the entire tissue of the dentin bridge and beyond into the bulk of normal secondary dentin. Histologic evaluation was carried out following previously defined criteria (Cox & others, 1982, 1985; Cox & Bergenholz, 1986). Each dentin bridge was observed via serial sections for the presence of single or multiple tunnel defects and whether they were present within the center or at the periphery of the dentin bridge interface.

RESULTS

This study histologically evaluated 235 mechanically exposed pulps that had been capped with various hard-set Ca(OH), medicaments. A total of 192 new hard-tissue dentin bridges were organized, of which 172 (90%) presented tunnel defects. Generally the healed pulps at 1 and 2 years following direct capping presented with stained dentin bridges. However, the pulp tissues subjacent to these dentin bridges were also associated with recurring pulp inflammation and necrosis as summarized in the table.

14-Day Hard-Tissue Repair

The 14-day exposed and pulp-capped teeth showed six of seven teeth with new hard-tissue dentin bridges adjacent to the medicament interface. All six dentin bridges showed multiple tunnel defects, which were seen as small lakes of pulp tissue, observed directly adjacent to the medicament interface (Figure 1). These tissue lakes were always associated with small blood vessels coursing from the wound site in continuity with the deeper vessels of the reorganizing granulation tissue of the pulp. The dentin bridges all presented normal patterns of tubular dentin, and were seen forming along the entire medicament interface, except at the lakes and at the periphery of the exposure. Normal pulp tissue was present below the dentin bridge, with odontoblastoid cell profiles along the dentin-pulpal interface. No inflammation or stained bacteria were seen in any teeth. No internal resorption was present in any of these pulps.

5-Week Hard-Tissue Repair

The 135 exposed and pulp-capped teeth observed at 5 weeks showed 103 dentin bridges. Ninety-four (91%) of these dentin bridges presented tunnel defects. Histologic sections through the center of each exposure site showed that each dentin bridge was composed of tubular dentin (Figure 2). However, further mesial or distal histological sections of the same dentin bridges showed several tunnel defects with vessels and cells coursing towards the axial edge of the exposure site (Figure 3). Often these tunnel defects formed a soft tissue lake between the medicament interface and a portion of the original axial floor. Forty-three dentin bridges, each with tunnel defects, showed stained bacterial profiles with both acute and chronic inflammatory cells in the subjacent pulp tissue. No internal resorption was seen in any of the pulps.

1-Year Hard-Tissue Repair

The 42 exposed and pulp-capped teeth at 1 year showed 34 dentin bridges with 29 (85%) multiple tunnel defects. Most dentin bridges were composed of tubular dentin with few cellular inclusions except in the area of the tunnel defect. Most of the dentin bridge defects ran as tunnels from the lake of debris below the medicament, along the axial edge of the exposure site of the dentin bridge to the deeper pulp (Figure 4). Cellular debris, inflammatory cells, and capillaries were present in the pulp tissue below the tunnel orifice at the pulp interface. Adjacent serial sections showed stained bacteria lying in debris below the medicament interface and the defect at the axial wall of the cavity preparation (Figure 5). Seventeen dentin bridges, each with tunnel defects, showed inflammatory cells and stained bacteria. No internal resorption was seen in any of the pulps. Ingested particles of the pulp-capping medicament

<p>| Number of Direct Capped Pulps with Dentin Bridges and Number of Tunnel Defects per Dentin Bridge per Time Period |
|-------------------------------------------------|------------------|-----------------|------------------|</p>
<table>
<thead>
<tr>
<th>Exposed &amp; Capped Teeth</th>
<th>Dentin Bridges</th>
<th>Tunnel Defects</th>
<th>Inflamed or Necrotic Pulps</th>
</tr>
</thead>
<tbody>
<tr>
<td>14-day capping</td>
<td>7</td>
<td>6</td>
<td>6 (100%)</td>
</tr>
<tr>
<td>5-week capping</td>
<td>135</td>
<td>103</td>
<td>94 (91%)</td>
</tr>
<tr>
<td>1-year capping</td>
<td>42</td>
<td>34</td>
<td>29 (85%)</td>
</tr>
<tr>
<td>2-year capping</td>
<td>51</td>
<td>49</td>
<td>43 (88%)</td>
</tr>
<tr>
<td>Total teeth</td>
<td>235</td>
<td>192</td>
<td>172 (89%)</td>
</tr>
</tbody>
</table>

(45%)
were often seen in fibroblast profiles within the subjacent pulp tissue underlying the various tunnels.

**2-Year Hard-Tissue Repair**

The 51 exposed and pulp-capped teeth at 2 years showed 49 dentin bridges, of which 43 contained multiple tunnel defects. Eighty-eight percent of the dentin-bridged exposures had tunnel defects at the 2-year observation period. Most dentin bridges were composed of tubular dentin except in the areas of the defect. Serial sections indicated that most dentin bridges showed multiple tunnel defects coursing from the medicament interface along the axial edge to the pulp. Each of the tunnels contained both fibroblasts and small capillaries (Figure 6). Eighteen dentin bridges showed inflammatory cells and stained bacteria lying in the lake area directly below the medicament in the tunnel defect. The capillaries and connective tissue below the remaining medicament communicated with the pulp through the tunnel defects and often showed various types of inflammatory cells. Certain of the fibroblasts presented with ingested particles of the pulp-capping medicament. No internal resorption was seen in any pulps.

**DISCUSSION**

Our data demonstrate that exposed and inflamed primate dental pulps often heal and form new dentin bridges directly adjacent to hard-set Ca(OH)$_2$ medicaments at the same level of success as reported by many other investigators (Zander, 1939; Nyborg, 1955, 1958; Rowe, 1967; Clarke, 1970; Langeland & others, 1971a; Tronstad & Mjör, 1972; Mjör, 1972; Ulmansky & others, 1972; Schröder & Granath, 1972; Walton & Langeland, 1978; McWalter & others, 1973, 1976; Watts & Paterson, 1979; Cvek, 1978; Haskell & others, 1978; Heys & others, 1980, 1981; Baume & Holz, 1981; Cox & others, 1982; Russo & others, 1982; Goldberg & others, 1984; Cox & Bergenholtz, 1984; Cox & others, 1985; Pitt Ford & Roberts, 1991). However, in addition to supporting that data, this study also shows that 89% of all dentin bridges contain multiple tunnel defects. In addition, each tunnel is patent and communicates with the underlying pulp from the medicament interface. However, our data do not agree with the suggestion of Ulmansky and others (1972), who stated that the quality of the bridge improves as the odontoblasts migrate apically with the deposition of dentin. These multiple tunnel defects present a morphological disruption of the dentin bridge barrier in that they not only fail to provide a permanent barrier, but they also fail to provide a long-term biological seal against bacterial infection. Consequently, the tunnels, via recurring microleakage, permit oral contaminants such as bacteria and their toxic factors to eventually gain access to the pulp tissue through the marginal gap. It is the presence of bacterial factors via microleakage that is responsible for pulp inflammation and necrosis and not the medicament per se. We agree with Langeland and others (1971a,b), Tronstad & Mjör (1972), Mjör (1972), and Woehrel (1977) when they cautioned that histologic demonstration of only one section through a dentin bridge following capping of an exposed pulp is not by itself a proper criterion for maintenance of long-term pulpal healing.

Presence of capping particles in the connective tissue of the pulp at each time interval indicates that the Ca(OH)$_2$ or other filler components of the medicament interface are in a progressive state of disintegration. This softening and disintegration phenomenon of Ca(OH)$_2$ cements has been reported by McComb (1983) and Hwas and Sandrik (1984). Another disadvantageous feature was reported in an in vitro study (Reinhardt & Chalkley, 1983), which reported that Ca(OH)$_2$ bases allow as much as more long-term softening of the adjacent composite resin interface than do other cements such as ZOE, IRM, or Cavitec. And Phillips and others (1984) reported a significant difference in resistance of certain hard-setting Ca(OH)$_2$ materials to attack and eventual dissolution by phosphoric acid-etching solutions. These observations pose an interesting question as to the long-term efficacy of using any commercially available Ca(OH)$_2$ base underneath a clinical composite procedure. Following placement and long-term dissolution of the commercially available Life or Dycal and subsequent migration of capping particles through the tunnels defects, the long-term therapeutically capacitive Ca(OH)$_2$ is now placed in serious doubt! Capping particles are often observed in both macrophages and giant cells of the subjacent pulp below the tunnels. In the deeper pulp tissue, these capping particles appear in long fibroblast-type cells (Figure 7). However, their presence does not seem to provoke a chronic inflammatory response. In addition, the presence of either the Ca(OH)$_2$ and other filler particles does not seem to initiate or to be involved in the internal resorption phenomenon as was reported in primary human molars (Via, 1955). However, it should be noted that Via's study was a radiographic interpretation, as no histological tissues were prepared for evaluation.

This study demonstrates a high rate of recurring infection from the bacterial and oral microcontaminants through dissolution of the Ca(OH)$_2$ at long-term intervals even though dentin bridges are present as indicators of initial healing.

**Clinical Considerations**

From a clinical standpoint, the ultimate failure of pulp capping is an inability of the medicament to
Figure 1. Pulp of a Life Ca(OH)$_2$, direct capped exposure after 14 days. Note the dark stained Life Ca(OH)$_2$ on the mid-left of the field (a). Tunnel defects (arrows) are seen across the medicament-pulp interface between the dentin bridge islands. Reorganizing granulation tissue is seen in the upper right of the field with small vessels coursing to the deeper pulp. Normal pulp tissue is seen in the lower left below the dentin bridge, with new odontoblastoid cells along the new dentin-pulpal interface. (Masson trichrome, original magnification X34.5)

Figure 2. Pulp of a Life Ca(OH)$_2$, direct capped exposure after 14 days. A new dentin bridge (arrow) is seen along portions of the medicament interface, except at the lower right periphery. Several vessels are present as clear spaces throughout the pulp. New reparative dentin matrix is seen around the periphery of the dentin chips (a) in the pulp and along the original dentin-pulpal wall interface, above and below. All reparative dentin zones show new odontoblastoid cells with red stained nuclei. (Masson trichrome, original magnification X32.5)

Figure 3. Pulp of a Life Ca(OH)$_2$, direct capped exposure after 21 days. A new dentin bridge (arrows) is seen along most of the medicament interface, except at the extreme right. The cleared vessels throughout the pulp are a consequence of vascular perfusion. New reparative dentin matrix (a) is seen along the original dentin-pulpal wall interface. Several histiocytes (b) are seen in the mid-central pulp with ingested particles of Life. (Masson trichrome, original magnification X34.5)

Figure 4. Pulp of a Dycal Ca(OH)$_2$, direct capped exposure after 13 months. A thick dentin bridge (a) is seen at the left of the exposure site composed of tubular dentin and reparative dentin matrix deposited around the dentin chips pushed into the pulp from the bur exposure. A portion of a tunnel filled with debris (arrow) is seen coursing towards the pulp through the middle portion of the dentin bridge. Several cleared vessels are seen throughout the pulp. (Masson trichrome, original magnification X34.5)

Figure 5. Peripheral edge of a dentin bridge of a Life direct capped exposure at 13 months. Note the dark stained Life Ca(OH)$_2$ (a) across the upper field. Yellow stained dentinal tubules (b) are seen on the upper right, with many purple-blue stained bacterial profiles (c) to the left and lower mid-field. (modified McKay bacterial stain, original magnification X162.5)

Figure 6. Periphery of a dentin bridge (a) of a Dycal direct capped exposure at 2 years. Dycal is seen on the left with a tunnel coursing from the upper left through the bridge to dental pulp. The tunnel shows red and light pink stained debris (b). Dark staining nuclei of chronic inflammatory cells are seen in the mid-pulp below the tubular dentin of the bridge and peripheral reparative dentin. Several small vessels are present in the connective tissue among the inflammatory cells. Some cells show ingested particles of the Dycal (c). (Masson trichrome, original magnification X41.4)

Figure 7. Pulp of a Dycal Ca(OH)$_2$, direct capped exposure after 13 months. Note the dark stained nuclei of the pulp fibroblasts within the connective tissue subjacent to the tunnels. Many of the cells show multiple aggregates of small dark stained Ca(OH)$_2$, capping particles (a) in fibroblasts, macrophages, and histiocytes throughout the field. (Masson trichrome, original magnification X34.5)
provide a long-term barrier to microleakage of bacteria and other irritants. At this point, direct pulp capping is considered by many clinicians to be a definitive or permanent procedure. However, it is apparent from this study that the dentin bridges that form under Ca(OH)₂ medicaments demonstrate a high rate of multiple tunnel defects (89%) and that the Ca(OH)₂ medicaments disintegrate and become lost over a period of time. This observation agrees with the clinical reports of Akester (1979), Barnes and Kidd (1979), and Lewin (1980). Consequently, these hard-set Ca(OH)₂ medicaments fail to provide an effective permanent barrier to microleakage of bacterial toxins through the cavosurface margin. This study and other data (Tronstad & Mjör, 1972) indicate that direct pulp capping as currently practiced with Ca(OH)₂ is not a permanent definitive procedure and that histological demonstration of only one section of a dentin bridge is no criterion of successful pulp healing. As discussed by others, only histological slides made in complete serial series should be considered to provide definitive information regarding the success of complete dentin bridge formation. Cvek (1978) suggested removal of the temporary restoration and capping material after 6 months as the best clinical procedure to identify a new dentin bridge. However, with our current knowledge of the importance of a bacterial seal of the cavity and the availability of fourth-generation dentin bonding materials, it should be possible to prevent the recontamination of Ca(OH)₂-treated exposures. Various studies have shown that other materials used in direct contact with the pulp will produce pulp healing if microleakage is prevented (Bergvall & Brännström, 1971; Brännström & Nyborg, 1977; Bengenholtz, Ahlstedt & Lindhe, 1977; Cox & Bengenholtz, 1984; Cox & others, 1987; Cox, 1987). For those practitioners continuing to use Ca(OH)₂ as a pulp-capping material, making certain that microleakage is prevented will assure the long-term success of the pulp-capping procedure.

CONCLUSIONS

This study demonstrates 172 tunnel defects in 192 dentin bridges at a level of 89%. Persistence of blood vessels near the medicament interface during wound healing and new dentin bridge formation appear to be associated with patency of the tunnel defects at all time periods. No internal dentin resorption is present in any teeth with or without tunnel defects. The irregular tunnel defects are associated with the rapid migration of oral contaminants and capping particles into the previously healed pulp, demonstrating the necessity for cavity sealing to prevent recontamination.

(Received 28 December 1994)

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