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Direct pulp capping with mineral trioxide aggregate

An observational study

George Bogen, DDS; Jay S. Kim, PhD; Leif K. Bakland, DDS

Preservation and maintenance of pulpal vitality is one objective in endodontics. Historically, the placement of a medication or material against a direct pulpal exposure during caries excavation has been considered controversial, and instead conventional endodontic therapy has been recommended.¹⁻⁵ The reluctance to place a direct pulp cap on an exposure in a carious field is based on unpredictable outcomes using traditional materials and treatment protocols. Moreover, when bacterial by-products induce pulpal inflammation, compromise immune responses and impede cellular differentiation and recruitment, normal pulpal repair mechanisms may not function properly. To date, researchers have been unable to identify a reliable nonabsorbable bioactive pulp-capping material that consistently stimulates cellular repair mechanisms, seals the dentin and promotes formation of a biologically stable reparative dentin bridge.

Clinicians have used many materials and techniques for direct pulp capping, including calcium hydroxide, hydrophilic resins, resin-modified glass ionomer cements, tricalcium phosphates and, more recently, mineral trioxide aggregate (MTA). Other innovative technical advances to halt the carious process and initiate the repair of potentially

ABSTRACT

Background. Pulp capping in carious teeth has been considered unpredictable and therefore contraindicated. A recently developed material, mineral trioxide aggregate (MTA), resists bacterial leakage and may provide protection for the pulp, allowing repair and continued pulp vitality in teeth when used in combination with a sealed restoration.

Methods. Forty patients aged 7 to 45 years accepted pulp-capping treatment when they received a diagnosis no more severe than reversible pulpitis after undergoing cold testing and radiographic examination. The primary author removed caries using a caries detector dye and sodium hypochlorite solution for hemostasis and placed MTA over the exposures and all surrounding dentin. The operator then restored the teeth provisionally with unbonded Clearfil Photocore (Kuraray Medical, Okayama, Japan). During a second visit, the operator restored the teeth with bonded composite after sensibility testing and confirmed MTA curing. At recall appointments, patients were evaluated for reparative dentin formation, pulpal calcification, continued normal root development and evidence of pathosis.

Results. Over an observation period of nine years, the authors followed 49 of 53 teeth and found that 97.96 percent had favorable outcomes on the basis of radiographic appearance, subjective symptoms and cold testing. All teeth in younger patients (15/15) that initially had had open apices showed completed root formation (apexogenesis).

Conclusions. MTA can be a reliable pulp-capping material on direct carious exposures in permanent teeth when a two-visit treatment protocol is observed.

Practice Implications. Vital pulp therapy using MTA is a treatment option for teeth diagnosed with a condition no more severe than reversible pulpitis.

Key Words. Reversible pulpitis; direct pulp capping; mineral trioxide aggregate; sodium hypochlorite.

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damaged tissue include the use of lasers, ozone technology and bioactive agents that induce and stimulate pulpal defenses.⁶⁻⁸ Success rates with direct pulp capping in a carious field have varied depending on the technique and materials. In humans, success rates range from 30 to 85 percent in two- to 10-year retrospective studies.^{4,9-13} Recent advances in our understanding of pulpal physiology, caries progression, inflammatory mediators and pulpal defense mechanisms have changed the clinical approach to caries removal and protocols for direct pulp capping.¹⁴

Direct pulpal exposures can be a challenging problem during excavation in a carious field. A diagnosis of reversible pulpitis before treatment is necessary for a successful outcome, but a definitive pulpal diagnosis often is difficult to establish.⁹ In 1966, Fusayama and colleagues¹⁵ made significant advances in the field of caries research and pulpal protection. They were the first to show that the upper layer of two distinctive carious layers could be stained selectively and that subsequent objective caries removal would allow for pulpal preservation and repair when teeth were sealed with bonded resin-based composites.¹⁶⁻¹⁸

In 1996, Matsuo and colleagues¹³ assessed factors that affect the success of direct pulp capping. They placed direct pulp caps on teeth in a carious field using a caries detector, 10 percent sodium hypochlorite (NaOCl) for hemostasis and fast-set calcium hydroxide. The patient's age, exposure size, responses to percussion or thermal stimulation, and type of tooth and location had no reflection on the success rate. When adequate hemostasis using 10 percent NaOCl could be attained, success rates increased. Thus, uncontrolled hemorrhaging may be directly proportional to the concentration of inflammatory mediators and the degree of intrapulpal pressure, which may affect the probability that hemostasis can be achieved. Nakanishi and colleagues¹⁹ reported evidence that inflammatory mediators such as immunoglobulin G, immunoglobulin A, immunoglobulin M, elastase and prostaglandin E₂ were present in higher quantities in clinically inflamed pulps.¹⁹

Calcium hydroxide—once considered the standard for pulp-capping materials—provides an option for reparative dentin formation, but long-term studies have shown results to be variable and somewhat unpredictable.⁹⁻¹² The material does not provide close adaptation to dentin, does not promote consistent odontoblast differentiation

and has been shown to be cytotoxic in cell cultures; the resultant reparative dentin formation can be characterized by tunnel defects.²⁰⁻²² Tunnel defects within dentin bridges may provide a pathway for the penetration of microorganisms to activate circulating immune cells, induce pulpal irritation and produce subsequent dystrophic calcification. Investigators using hydrophilic resins and resin-modified glass ionomer cements as direct pulp-capping agents have reported promising results in nonhuman primates^{23,24} but have not determined predictable outcomes when these agents are used in humans.²⁵⁻²⁸ Researchers using inventive modalities to test alternative pulp-capping agents also have been unsuccessful in stimulating pulpal repair and healing in cases of direct carious exposures.^{29,30}

MTA is a bioactive silicate cement that has been shown to be an effective pulp-capping material in canine models and in nonhuman primates.³¹⁻³³ The material is successful because of its small particle size, sealing ability, alkaline pH when set and slow release of calcium ions.³⁴ Investigators have reported that MTA induces pulpal cell proliferation,^{35,36} cytokine release,³⁷ hard tissue formation²² and the synthesis of an interface with dentin that resembles hydroxyapatite in composition.³⁴ The material is nonabsorbable, sets in the presence of moisture, has a relatively high compressive strength and has a sustained high alkaline pH.³⁸ Recent studies examining partial pulpotomies or direct pulp capping using MTA in humans have shown favorable short-term results.³⁹⁻⁴³ There are no clinical studies that have combined and evaluated the use of a caries detector dye, NaOCl hemostasis, direct MTA pulp capping and bonded composite placement in a two-visit protocol. The aim of our study was to monitor the long-term success rate of surgically repaired pulpal exposures in teeth with deep carious lesions using MTA and current adhesion technology.

METHODS AND MATERIALS

All patients in our study had been referred for endodontic treatment to a private office or the Children's Dental Health Clinic, Long Beach, Calif., and had completed periapical radiographs,

ABBREVIATION KEY. **MTA:** Mineral trioxide aggregate. **NaOCl:** Sodium hypochlorite. **PDL:** Periodontal ligament.

periodontal probing, percussion testing and vitality assessment with cold testing. The primary author (G.B.) selected 53 teeth for treatment over an eight-year period that included 51 molars, one maxillary premolar and one maxillary incisor. All teeth exhibited initial deep caries and no prior restorations. Both female and male patients had variable health histories and symptoms and ranged in age from 7 to 45 years. All radiographs showed evidence of deep caries in close proximity to the pulpal chamber with no evidence of thickened periodontal ligament (PDL), furcation radiolucencies, internal resorption or periradicular pathosis. Clinically, all teeth had mobility and periodontal probings within normal range with no evidence of sinus tracts or swelling. No teeth treated in this study exhibited pain on percussion. Fifteen younger patients had radiographically evident immature apices before treatment. We did not use control groups since the aim of the study was to monitor pulpal survival time using one specific protocol, and we planned to measure the outcomes against survival periods from previous studies that used calcium hydroxide.⁹⁻¹³ We obtained written consent from all study participants or their legal guardians. The Loma Linda University Institutional Review Board (Loma Linda, Calif.) reviewed and accepted the protocol.

One operator (G.B.) completed all direct pulp caps. After patients obtained profound local anesthesia, the operator placed a dental dam and used an oral sealant to prevent saliva leakage where necessary. The operator used a caries detector dye and performed caries excavation under $\times 3.5$ to $\times 18.0$ magnification using handpieces with high-speed diamond or carbide burs and no. 6-2 slow-speed carbide round burs. He continued caries excavation even after pulpal exposures occurred and stopped the excavation when either little or no dye staining was evident over the pulpal roof or axial wall.

The operator used either 5.25 percent or 6.00 percent NaOCl as a direct solution or on a soaked cotton pellet to achieve hemostasis. He established hemorrhage control within one to 10 minutes in all cases. The operator excluded one patient from this study when hemorrhaging continued beyond this time. After the operator achieved hemostasis, he sprayed the exposure site and dentin with water from a two-way syringe and then air-dried the area. The dental assistant mixed gray MTA or white MTA

according to the manufacturer's instructions (Pro-Root MTA, Dentsply Tulsa Dental, Tulsa, Okla.) and the operator placed a 1.5- to 3.0-millimeter thick layer of the cement directly over the exposure site and surrounding dentin, leaving 1 to 2 mm of dentin and enamel available circumferentially for the future bonded restoration. After placing the MTA, the operator laid a flat, water-moistened cotton pellet directly over the material and provisionally restored the tooth with unbonded Clearfil Photocore (Kuraray Medical, Okayama, Japan) that was photopolymerized for 60 seconds.

The operator instructed patients or their parents to call the office immediately if pain or discomfort occurred after treatment. Patients then returned to our office or the referring general dentists' offices for placement of the final restoration within five to 10 days. Two patients chose not to return for continued treatment. All patients in the study were asymptomatic and had a normal response to cold testing before placement of the final restoration. After patients again obtained profound anesthesia, the operator placed a dental dam and removed the Clearfil Photocore provisional restoration by using a carbide bur in a high-speed handpiece. He then assessed the MTA for hardness and removed the embedded cotton fibers with spoon excavators. Next, the operator placed Class I or II composite restorations incrementally and bonded them using a self-etching primer and resin with Clearfil LB2 Bond and Clearfil AP-X Composite (Kuraray Medical). The referring general dentists restored six teeth in five patients with bonded composites. Whenever possible, we recalled patients at six weeks, six months and on a yearly basis thereafter. Recall appointments consisted of obtaining self-reports from patients, taking periapical radiographs and testing the pulp-capped teeth with a cold stimulus. Two of us (G.B. and L.K.B.) evaluated radiographs for reparative dentin formation, pulpal calcification, continued normal root development and the absence of pathosis. We also evaluated the final restorations for marginal integrity. Three patients had to attend recall appointments out of state, either at a private endodontic office or a dental school facility.

Statistical analysis. Figure 1 shows the results of the study using the life table analysis and the Kaplan-Meier method to estimate the probability of pulpal survival and healing.

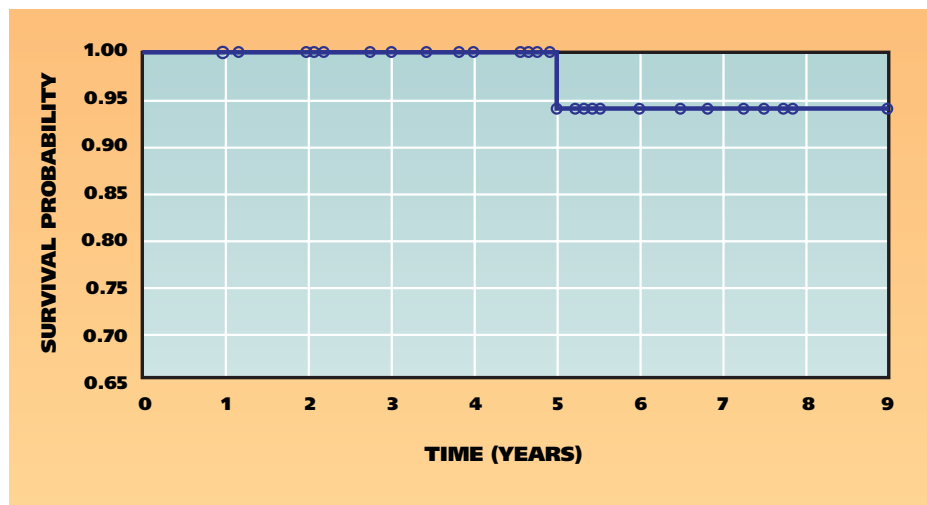


Figure 1. Kaplan-Meier survival probability estimate of pulpal survival and healing.

RESULTS

We observed 49 of 53 teeth in 37 of the patients between one and nine years (mean, 3.94 years) with a recall rate of 92.5 percent (n = 49). Three patients did not return for recall appointments and evaluations. The patients varied in age from 7 to 45 years, the mean age being 16.6 years. There were 22 females and 15 males. Three patients subjectively complained of moderate-to-severe pain before treatment; cold testing revealed a normal (nonlingering) response in 48 of 50 teeth. Pulpal exposure resulted in some degree of hemorrhage in all cases. All teeth in this study incurred one to four direct exposures, which varied from approximately 0.25 to not more than 2.50 mm in diameter. The operator placed direct pulp caps on 49 teeth using gray MTA and four with white MTA. Two molar pulp caps in the same patient on the second visit had MTA that had not set; the operator subsequently removed the material and placed new MTA that was determined to be properly cured on the patient's third visit. The treating endodontist (G.B.) restored 88 percent of the pulp caps, while the referring general dentists restored 12 percent. Three teeth did not receive a final restoration because two patients did not return for continued treatment. The overall pulpal survival (as established by radiographs taken at recall appointments, subjective symptomatology and cold testing) was 97.96 percent. Tables 1 and 2 show the interval outcomes and life table analysis of the survival data. The probability of the pulp's surviving at least five years (60

months) was 94.87 percent. Table 3 (page 310) shows the individual survival data.

DISCUSSION

Radiographic assessment of teeth that had open apices showed that 100 percent (15/15) had progressed to complete root formation and apex closure. We noted radiographic evidence of dentin bridge formation in 82 percent of cases (40/49) and found normal PDL thickness in all observed teeth. We also noted calcifications or pulpal stone formation in 10.2 percent

(5/49) of the evaluated teeth. Root canal width and pulpal volume remained approximately the same in 89.8 percent (44/49) of the cases. Class I restorations exhibited no restorative failures, whereas three Class II restorations that developed marginal ridge fractures required repair after five or six years of function. At no time did it appear that the failure of the restoration affected the long-term vitality of the previously treated pulp. Only one patient exhibited recurrent caries at a recall appointment. Some teeth with gray MTA pulp caps showed minor discoloration. All teeth with pulp caps continued to have normal responses to cold testing up to the nine-year follow-up examination with no radiographic evidence of pathosis (Figure 2, page 311).

Although there were only a small number of cases, the results of this study in which we used MTA as a direct pulp-capping agent when following the protocol described for a two-visit sequence show that this procedure can achieve a long-term favorable outcome. The important controlled variables included complete caries removal, visible hemostasis, verified MTA setting and placement of a bonded fifth-generation hydrophilic resin and composite. Since our study did not have a control group, the results provide, within the limitations of the study, a relatively low level of scientific evidence. However, the outcomes suggest that MTA is a more predictable pulp-capping material than calcium hydroxide. The data offer an alternative treatment option for certain patients who are not diagnosed with irreversible pulpitis.

The physical characteristics and bioactive

properties of MTA were a critical contributing factor to the success of this study.³⁴⁻³⁸ The cement is hygroscopic, and its ability to set is not affected by the presence of blood or serum fluids.⁴⁴ The high alkalinity of MTA, its calcium release and sustained pH of 12.5 most likely prevented any further microbial growth of residual microorganisms that were left after caries excavation. The high pH also extracts growth factors from adjacent dentin that are thought to be responsible for promoting dentinal bridging.⁴⁵ Furthermore, the release of calcium ions by MTA generates a reactionary interfacial layer of hydroxyapatite on its surface when it comes in contact with tissue fluids, and their presence also may contribute to reparative dentin formation.³⁴ The close physiochemical seal of dentin by MTA, determined to be 0 micrometers in resin replica models,⁴⁶ provides a more insoluble barrier against microleakage than does calcium hydroxide, which can show gaps at the dentin interface of 7 to 15 μm when placed under composite restorations.⁴⁷ Once set, it is nonabsorbable, whereas set calcium hydroxide is unstable and can degrade and dissolve under restorations, allowing potential ingress of microorganisms and subsequent bacterial contamination through tunnel defects in the dentin bridge. These events can induce continued pulpal irritation, dystrophic calcification and potential degenerative changes in the pulp.^{21,48}

Traditionally, dentists place pulp-capping agents over the exposure site and a small area of surrounding dentin. In our study, the operator placed MTA over the exposure site and the entire floor or wall of the restoration preparation to allow a 1.5- to 3.0-mm thickness of the material.

TABLE 1

Interval failure/success rate of survival data.					
INTERVAL (YEARS)	NO. OF TEETH AT BEGINNING OF INTERVAL	NO. OF TEETH FAILED DURING INTERVAL	NO. OF TEETH LOST TO FOLLOW-UP	INTERVAL FAILURE RATE (%)	INTERVAL SUCCESS RATE (%)
0, 1	49	0	5	0	100
1, 2	44	0	3	0	100
2, 3	41	0	9	0	100
3, 4	32	0	9	0	100
4, 5	23	1	7	4.35	95.65
5, 6	16	0	8	0	100
6, 7	8	0	2	0	100
7, 8	6	0	5	0	100
8, 9	1	0	1	0	100

TABLE 2

Life table analysis of survival data.					
INTERVAL (YEARS)	NO. OF TEETH AT BEGINNING OF INTERVAL	EFFECTIVE SAMPLE SIZE	PROBABILITY OF FAILURE	PROBABILITY OF SURVIVING (%)	INTERVAL SUCCESS RATE (%)
0, 1	49	46.5	0	100	100
1, 2	44	42.5	0	100	100
2, 3	41	36.5	0	100	100
3, 4	32	27.5	0	100	100
4, 5	23	19.5	5.13	94.87	94.87
5, 6	16	12	0	100	94.87
6, 7	8	7	0	100	94.87
7, 8	6	3.5	0	100	94.87
8, 9	1	0.5	0	100	94.87

If the exposure occurred on the axial wall in a Class II preparation, the operator placed MTA on the base of the gingival-cavosurface to the mesial and distal restoration extensions in a 1.5- to 2.0-mm thickness (Figure 3, page 312). After applying MTA, the operator trimmed and shaped the material with spoon excavators to expose 1.0 to 2.0 mm of peripheral dentin and enamel and then used a small moist cotton pellet to clean it to ensure an adequate surface area for bonding on the second visit. The operator placed direct pulp caps on only four teeth with white MTA since it was not commercially available until late into this study. We found no difference in short-term outcomes between the gray and the white MTA.

Optical magnification and careful caries removal aided by a caries detector dye also were

TABLE 3

Distribution of 49 carious teeth with direct pulp caps with mineral trioxide aggregate.

PATIENT	TOOTH NO.	AGE (YEARS)	SEX	DENTIN BRIDGE FORMATION	PULP VITALITY SURVIVAL TIME AT LAST RECALL
1	3	15	F	Undetermined	7 years, 9 months
2	14	8	F	Yes	7 years, 6 months
3	30 19	7 11	M	Yes Yes	7 years, 6 months 2 years, 9 months
4	3 14	10	F	Yes Yes	3 years, 10 months 3 years, 10 months
5	19	29	F	Undetermined	7 years, 10 months
6	2	17	F	Yes	9 years
7	30	7	F	Yes	4 years, 7 months
8	30	7	F	Yes	4 years
9	19	19	M	Yes	2 years, 2 months
10	30	7	F	Yes	7 years, 3 months
11	30	18	F	Undetermined	6 years, 6 months
12	15	13	M	Yes	4 years, 9 months
13	18 31	14	M	Yes Yes	2 years, 1 month 2 years, 1 month
14	15	26	F	Yes	3 years, 5 months
15	19	10	M	Undetermined	5 years (failure)
16	30	30	M	Yes	2 years
17	14	13	F	Yes	6 years, 10 months
18	2	18	F	Yes	4 years
19	14	29	F	Yes	5 years
20	3 14 19 30	7	M	Yes Yes Yes Yes	5 years, 4 months 5 years, 4 months 5 years, 3 months 5 years, 3 months
21	18	10	M	Yes	4 years, 5 months
22	19 30	9	M	Yes Yes	6 years 6 years
23	19	16	F	Yes	5 years, 5 months
24	13 18	17	F	Yes Undetermined	4 years, 11 months 4 years, 8 months
25	30	9	F	Yes	5 years, 6 months
26	13 14	27	M	Undetermined Yes	3 years 3 years
27	2	45	F	Undetermined	3 years, 5 months
28	15	28	F	Yes	2 years
29	31	33	M	Yes	3 years, 10 months
30	3	16	F	Yes	1 year
31	19 30	8	F	Yes Yes	2 years, 2 months 2 years, 2 months
32	3 14	12	M	Yes Yes	1 year 1 year
33	30	9	F	Yes	1 year
34	30	12	M	Undetermined	1 year
35	19	28	F	Yes	1 year, 2 months
36	9 19	11	M	Undetermined Yes	4 years 4 years
37	15	14	M	Yes	3 years

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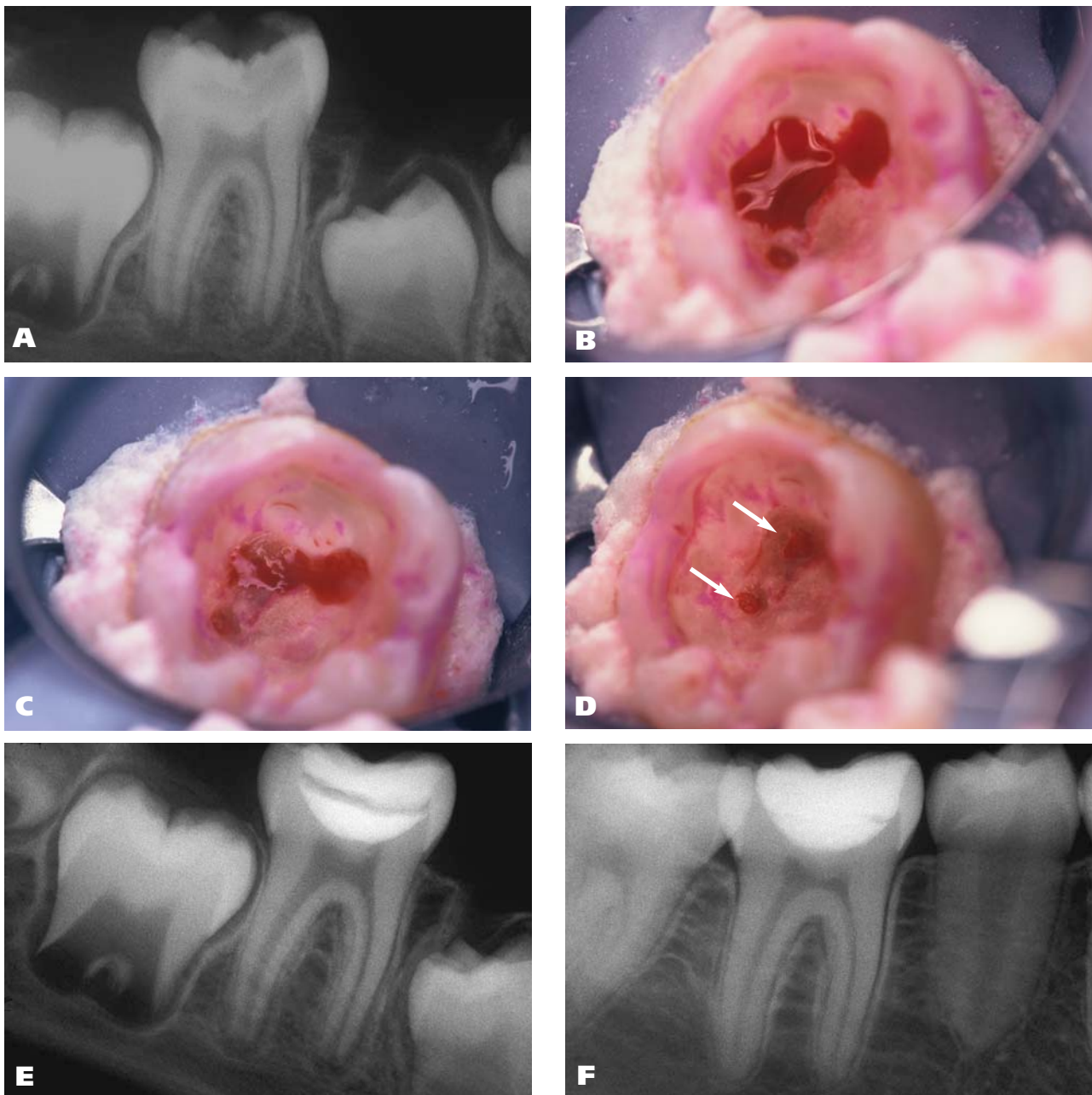


Figure 2. Radiographic and clinical sequence of mineral trioxide aggregate (MTA) direct pulp capping of a mandibular right molar in a 9-year-old female patient. **A.** Pretreatment radiograph showing initial deep caries and immature apices. **B,C,D.** Five-minute time of 5.25 percent sodium hypochlorite hemostasis, on two 1.5- to 2.0-millimeter exposures (arrows). **E.** Radiograph of molar with MTA, water-moistened cotton pellet and unbonded Clearfil Photocore (Kuraray Medical, Okayama, Japan) provisional restoration after initial visit. **F.** Radiograph taken at the 5.5-year recall appointment showing permanent restoration and evidence of complete root formation. The tooth exhibited a normal response to cold testing.

important components of this study. The study outcome shows that the human pulp has an innate healing capacity that can be enhanced using objective and conservative caries removal, a bioactive pulp-capping material and a sealed restoration. The high occurrence of pulpal repair and pulp-capping success appears to be more favor-

able in teeth of younger patients; success can be attributed to the presence of larger apical foramina and greater vascularization of the pulp, in which active immune cell surveillance may increase chances for repair and intensify vital pulpal maintenance.¹² Pulpal survival decreases with age and cumulative restorative trauma.^{49,50}



Figure 3. Posttreatment radiograph of mineral trioxide aggregate (MTA), moist cotton pellet and unbonded Clearfil Photocore (Kuraray Medical, Okayama, Japan) interim restoration placed against the axial wall of a maxillary left second premolar in a 17-year-old girl.

We used NaOCl as a hemostatic agent in this study on all pulpal exposures. Concentrations varied from 5.25 to 6.00 percent, since the concentration of commercially available solutions changed during the study. Sodium hypochlorite was first advocated for pulpal hemostasis in the late 1950s.⁵¹ The solution has several advantageous properties besides being an excellent hemostatic agent.⁵¹ Specifically, it can disinfect microbially contaminated dentinal chips and operative debris, inhibit fibrin clot formation and disinfect the adjacent perforated dentin interface. Although studies have shown that use of NaOCl at concentrations higher than 0.025 percent is detrimental to wound healing in human tissue when it is used as a fluid dressing for patients with burns,⁵² pulpal reactions appear to be remarkably favorable.^{53,54} No pulpal exposures in our study were affected adversely by direct contact with 5.25 to 6.00 percent NaOCl for periods of five to 10 minutes.

In no case of successful pulp capping did radiographs show teeth with furcation pathosis. All recall examinations showed complete apexogenesis of previously immature apices (Figures 2F and 4F), consistent with a case study by Patel and Cohenca.⁵⁵ Apexogenesis may indicate the continued normal physiological activity of cementoblasts and odontoblasts in the absence of irritants. Apical closure allows for more predictable endodontic treatment if teeth require pulpectomy at a later stage.

Investigators²¹ have reported calcification of the pulpal chamber and canal lumen in conjunction with calcium hydroxide pulp capping and the presence of tunnel defects in the reparative

dentin bridge. In our study, 89.8 percent (44/49) of pulp caps showed no significant evidence of calcific changes in either the pulp or canal spaces when a direct pulp cap had been placed with MTA. A total of 10.2 percent (5/49) cases showed some form of calcification or pulpal stone formation. Although we noted this posttreatment complication, we determined that all five of these teeth tested within normal limits to a cold stimulus. Teeth with larger multiple pulpal exposures (≥ 2.0 mm) tended to exhibit more aggressive reparative dentin formation and calcification of the pulp and canal lumens. Continued radiographic evaluation of these five cases did not reveal decreased lumen size in canals nor progressive pulpal calcification after 18 to 24 months (Figure 4A-4D).

We noted reparative dentin formation in most patients examined at the one-year recall appointment. Dentin bridge thicknesses varied from 0.5 to 1.5 mm at the recall appointment, although radiographic angulations may have prevented precise recognition of bridging. In two teeth, the operator inadvertently forced MTA into the pulpal chamber, yet the pulpal reaction and outcome were favorable (Figure 5, page 314). The presence of MTA at the interface of pulpal tissue, which may be affected initially by the caries process, most likely stimulates the release of growth factors necessary for pulpal cells to recruit and organize odontoblasts to lay down reparative dentin.⁵⁶ Odontoblastlike cells have the potential to produce reparative dentin or regenerate pulpal tissue when they differentiate into odontoblasts during reparative dentinogenesis.⁷ The injured pulpal tissue below the MTA interface should exhibit tissue healing characterized by angiogenesis and neovascularization, and cell proliferation of functional cuboidal cells in proximity to the damaged area.⁵⁷ During dentin formation, these cells are characterized by synthesis and secretion of multiple noncollagenous proteins on an extracellular collagen matrix that eventually mineralizes in the absence of bacterial microleakage.

In clinical practice, dentists are required to make a diagnosis for patients at the initial examination, but too often that diagnosis is based exclusively on patient report and radiographic interpretation. Sometimes clinicians initiate endodontic therapy without performing adequate pulpal testing in patients who have deep caries and a favorable prognosis for pulpal repair. When

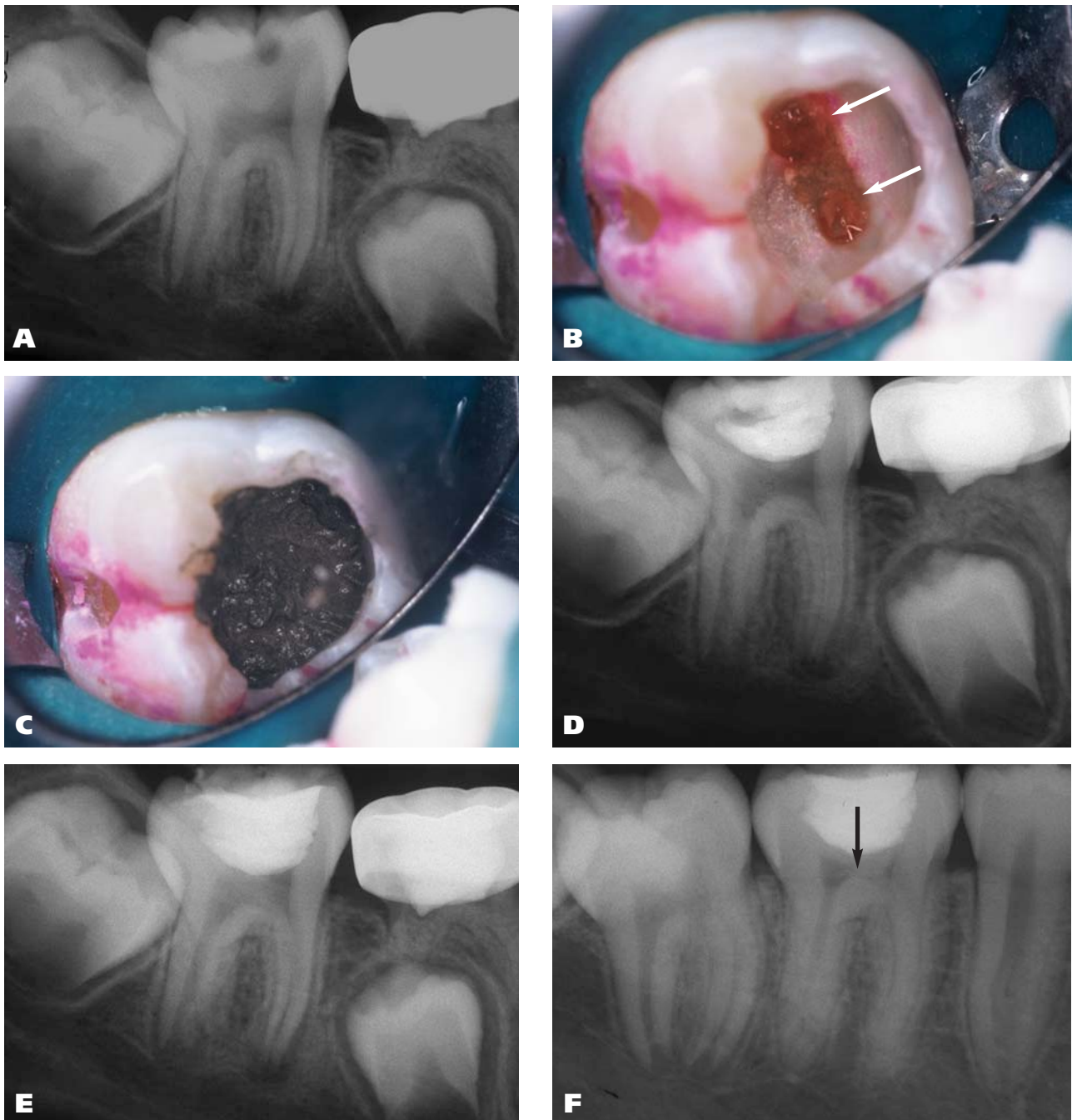


Figure 4. Treatment sequence of molar that was symptomatic on presentation in a 7-year-old boy. **A.** Pretreatment radiograph showing open apices and deep caries. **B.** Arrows showing two large 2.0- to 2.5-millimeter pulp exposures after caries excavation and hemostasis. **C.** Gray mineral trioxide aggregate placed over the entire pulpal roof. **D.** Radiograph after provisionalization. **E.** Molar after permanent restoration placement. **F.** Radiograph taken at the four-year, nine-month recall appointment showing extensive reparative bridge formation (arrow). Cold stimulus testing revealed a normal pulpal response.

sensibility testing, radiographic evaluations and patient self-reports indicate a normal pulp or a diagnosis no more severe than reversible pulpitis, then the clinician should consider vital pulp therapy to be a viable alternative to conventional endodontic treatment.

CONCLUSION

The use of MTA, enhanced magnification, caries removal using a caries detector dye, NaOCl hemostasis/disinfection, and a fifth-generation hydrophilic resin and composite is part of a reli-

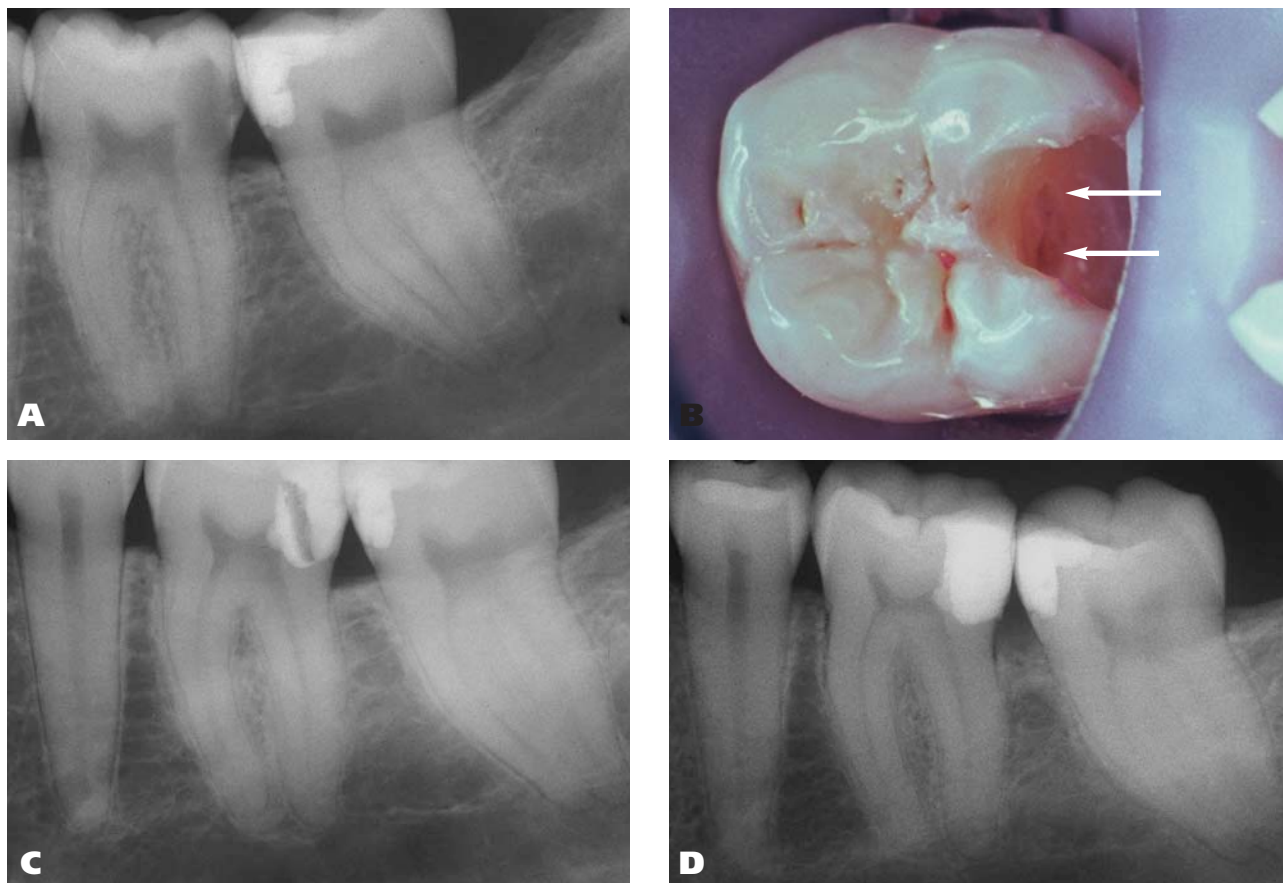


Figure 5. Treatment of molar with deep axial wall caries in 29-year-old female exhibiting a normal pulpal response to cold testing. **A.** Radiograph showing deep caries with no evidence of periradicular or furcation pathosis. **B.** Two exposures 1.0- to 1.5-millimeters to distal axial wall after excavation using caries detector and 5.25 percent sodium hypochlorite hemostasis (arrows). **C.** Posttreatment radiograph showing provisional restoration with mineral trioxide aggregate extending into the pulp chamber. **D.** Radiograph taken at the seven-year, 10-month recall appointment showing normal periodontal ligament and anatomical structures with no visual evidence of dentin bridge formation. The molar responded within normal limits to cold testing.

able two-visit pulp-capping protocol for direct carious exposures when the pulpal diagnosis is no more severe than irreversible pulpitis. Clinicians should be aware that the treatment described was performed by one operator; we cannot claim that every clinician will obtain similar results. Careful attention to diagnostic criteria and treatment procedures, however, should result in many successful outcomes. ■

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1. Tronstad L, Mjör IA. Capping of the inflamed pulp. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1972;34(3):477-485.
2. Langeland K. Management of the inflamed pulp associated with deep carious lesion. *J Endod* 1981;7(4):169-181.
3. Bergenholtz G. Advances since the paper by Zander and Glass

(1949) on the pursuit of healing methods for pulpal exposures: historical perspectives. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2005;100(2 suppl):S102-S108.

4. Al-Hiyasat AS, Barrieshi-Nusair KM, Al-Omari MA. The radiographic outcomes of direct pulp-capping procedures performed by dental students: a retrospective study. *JADA* 2006;137(12):1699-1705.
5. Ward J. Vital pulp therapy in cariously exposed permanent teeth and its limitations. *Aust Endod J* 2002;28(1):29-37.
6. Moritz A, Schoop U, Goharkhay K, Sperr W. The CO₂ laser as an aid in direct pulp capping. *J Endod* 1998;24(4):248-51.
7. Goldberg M, Six N, Decup F, et al. Bioactive molecules and the future of pulp therapy. *Am J Dent* 2003;16(1):66-76.
8. Dähnhardt JE, Jaeggi T, Lussi A. Treating open carious lesions in anxious children with ozone: a prospective controlled clinical study. *Am J Dent* 2006;19(5):267-70.
9. Hørsted P, Søndergaard B, Thylstrup A, El Attar K, Fejerskov O. A retrospective study of direct pulp capping with calcium hydroxide compounds. *Endod Dent Traumatol* 1985;1(1):29-34.
10. Baume LJ, Holz J. Long term clinical assessment of direct pulp capping. *Int Dent J* 1981;31(4):251-260.
11. Barthel CR, Rosenkranz B, Leuenberg A, Roulet JF. Pulp capping of carious exposures: treatment outcome after 5 and 10 years—a retrospective study. *J Endod* 2000;26(9):525-8.
12. Ausschil TM, Arweiler NB, Hellwig E, Zamani-Alaei A, Sculean A. Success rate of direct pulp capping with calcium hydroxide [in German]. *Schweiz Monatsschr Zahnmed* 2003;113(9):946-952.
13. Matsuo T, Nakanishi T, Shimizu H, Ebisu S. A clinical study of direct pulp capping applied to carious-exposed pulps. *J Endod*

- 1996;22(10):551-556.
14. Hahn CL, Liewehr FR. Relationships between caries bacteria, host responses, and clinical signs and symptoms of pulpitis. *J Endod* 2007;33(3):213-219.
 15. Fusayama T, Okuse K, Hosoda H. Relationship between hardness, discoloration, and microbial invasion in carious dentin. *J Dent Res* 1966;45(4):1033-1046.
 16. Fusayama T, Kurosaki N. Structure and removal of carious dentin. *Int Dent J* 1972;22(3):401-411.
 17. Sato Y, Fusayama T. Removal of dentin guided by fuchsin staining. *J Dent Res* 1976;55(4):678-683.
 18. Fusayama T. A Simple Pain-Free Adhesive Restorative System by Minimal Reduction and Total Etching. St. Louis: Ishiyaku EuroAmerica; 1993:6-72.
 19. Nakanishi T, Matsuo T, Ebisu S. Quantitative analysis of immunoglobulins and inflammatory factors in human pulpal blood from exposed pulps. *J Endod* 1995;21(3):131-136.
 20. Schröder U. Effect of calcium hydroxide-containing pulp-capping agents on pulp cell migration, proliferation, and differentiation. *J Dent Res* 1985;64(special number):541-548.
 21. Cox CF, Sübay RK, Ostro E, Suzuki S, Suzuki SH. Tunnel defects in dentin bridges: their formation following direct pulp capping. *Oper Dent* 1996;21(1):4-11.
 22. Andelin WE, Shabahang S, Wright K, Torabinejad M. Identification of hard tissue after experimental pulp capping using dentin sialoprotein (DSP) as a marker. *J Endod* 2003;29(10):646-650.
 23. Tarmn B, Hafez AA, Cox CF. Pulpal response to a resin-modified glass-ionomer material on nonexposed and exposed monkey pulps. *Quintessence Int* 1998;29(8):535-542.
 24. Cox CF, Hafez AA, Akimoto N, Otsuki M, Suzuki S, Tarim B. Biocompatibility of primer, adhesive and resin composite systems on non-exposed and exposed pulps of non-human primate teeth. *Am J Dent* 1998;11(special number):S55-S63.
 25. Hebling J, Giro EM, Costa CA. Biocompatibility of an adhesive system applied to exposed human dental pulp. *J Endod* 1999;25(10):676-682.
 26. do Nascimento AB, Fontana UF, Teixeira HM, Costa CA. Biocompatibility of a resin-modified glass-ionomer cement applied as pulp capping in human teeth. *Am J Dent* 2000;13(1):28-34.
 27. Mjör IA. Pulp-dentin biology in restorative dentistry, part 7: the exposed pulp. *Quintessence Int* 2002;33(2):113-135.
 28. Accorinte Mde L, Loguericio AD, Reis A, Muench A, de Araújo VC. Adverse effects of human pulps after direct pulp capping with different components from a total-etch, three-step adhesive system. *Dent Mater* 2005;21(7):599-607.
 29. Olsson H, Petersson K, Rohlin M. Formation of a hard tissue barrier after pulp capping in humans: a systematic review. *Int Endod J* 2006;39(6):429-442.
 30. Murray PE, Hafez AA, Smith AJ, Cox CF. Hierarchy of pulp capping and repair activities responsible for dentin bridge formation. *Am J Dent* 2002;15(4):236-243.
 31. Camilleri J, Pitt Ford TR. Mineral trioxide aggregate: a review of the constituents and biological properties of the material. *Int Endod J* 2006;39(10):747-754.
 32. Ford TR, Torabinejad M, Abedi HR, Bakland LK, Kariyawasam SP. Using mineral trioxide aggregate as a pulp-capping material. *JADA* 1996;127(10):1491-1494.
 33. Junn DJ. Quantitative assessment of dentin bridge formation following pulp-capping with mineral trioxide aggregate (master's thesis). Loma Linda, Calif.: Loma Linda University; 2000.
 34. Sarkar NK, Caicedo R, Ritwik P, Moiseyeva R, Kawashima I. Physicochemical basis of the biologic properties of mineral trioxide aggregate. *J Endod* 2005;31(2):97-100.
 35. Moghaddame-Jafari S, Mantellini MG, Botero TM, McDonald NJ, Nör JE. Effect of ProRoot MTA on pulp cell apoptosis and proliferation in vitro. *J Endod* 2005;31(5):387-391.
 36. Takita T, Hayashi M, Takeichi O, et al. Effect of mineral trioxide aggregate on proliferation of cultured human dental pulp cells. *Int Endod J* 2006;39(5):415-422.
 37. Koh ET, Pitt Ford TR, Torabinejad M, McDonald F. Mineral trioxide aggregate stimulates a biological response in human osteoblasts. *J Biomed Mater Res* 1997;37(3):432-439.
 38. Torabinejad M, Hong CU, McDonald F, Pitt Ford TR. Physical and chemical properties of a new root-end filling material. *J Endod* 1995;21(7):349-353.
 39. Aeinehchi M, Eslami B, Ghanbari M, Saffar AS. Mineral trioxide aggregate (MTA) and calcium hydroxide as pulp-capping agents in human teeth: a preliminary report. *Int Endod J* 2003;36(3):225-231.
 40. Witherspoon DE, Small JC, Harris GZ. Mineral trioxide aggregate pulpotomies: a case series outcomes assessment. *JADA* 2006;137(5):610-8.
 41. Barrieshi-Nusair KM, Qudeimat MA. A prospective clinical study of mineral trioxide aggregate for partial pulpotomy in cariously exposed permanent teeth. *J Endod* 2006;32(8):731-735.
 42. Iwamoto CE, Adachi E, Pameijer CH, Barnes D, Romberg EE, Jefferies S. Clinical and histological evaluation of white ProRoot MTA in direct pulp capping. *Am J Dent* 2006;19(2):85-90.
 43. Farsi N, Alamoudi N, Balto K, Al Mushayt A. Clinical assessment of mineral trioxide aggregate (MTA) as direct pulp capping in young permanent teeth. *J Clin Pediatr Dent* 2006;31(2):72-76.
 44. Torabinejad M, Higa RK, McKendry DJ, Pitt Ford TR. Dye leakage of four root end filling materials: effects of blood contamination. *J Endod* 1994;20(4):159-163.
 45. Tomson PL, Grover LM, Lumley PJ, Sloan AJ, Smith AJ, Cooper PR. Dissolution of bio-active dentine matrix components by mineral trioxide aggregate. *J Dent* 2007;35(8):636-642.
 46. Torabinejad M, Smith PW, Kettering JD, Pitt Ford TR. Comparative investigation of marginal adaptation of mineral trioxide aggregate and other commonly used root-end filling materials. *J Endod* 1995;21(6):295-299.
 47. Goracci G, Mori G. Scanning electron microscopic evaluation of resin-dentin and calcium hydroxide-dentin interface with resin composite restorations. *Quintessence Int* 1996;27(2):129-135.
 48. Cox CF, Keall CL, Keall HJ, Ostro E, Bergenholtz G. Biocompatibility of surface-sealed dental materials against exposed pulps. *J Prosthet Dent* 1987;57(1):1-8.
 49. Bernick S, Nedelman C. Effect of aging on the human pulp. *J Endod* 1975;1(3):88-94.
 50. Abou-Rass M. The stressed pulp condition: an endodontic-restorative diagnostic concept. *J Prosthet Dent* 1982;48(3):264-267.
 51. Hafez AA, Cox CF, Tarim B, Otsuki M, Akimoto N. An in vivo evaluation of hemorrhage control using sodium hypochlorite and direct capping with a one- or two-component adhesive system in exposed non-human primate pulps. *Quintessence Int* 2002;33(4):261-272.
 52. Heggers JP, Sazy JA, Stenberg BD, et al. Bactericidal and wound-healing properties of sodium hypochlorite solutions: the 1991 Lindberg Award. *J Burn Care Rehabil* 1991;12(5):420-424.
 53. Garcia-Godoy F, Murray PE. Systemic evaluation of various haemostatic agents following local application prior to direct pulp capping. *Braz J Oral Sci* 2005;4(14):791-797.
 54. Demir T, Cehreli ZC. Clinical and radiographic evaluation of adhesive pulp capping in primary molars following hemostasis with 1.25% sodium hypochlorite: 2-year results. *Am J Dent* 2007;20(3):182-188.
 55. Patel R, Cohenca N. Maturogenesis of a cariously exposed immature permanent tooth using MTA for direct pulp capping: a case report. *Dent Traumatol* 2006;22(6):328-333.
 56. Guven G, Cehreli ZC, Ural A, Serdar MA, Basak F. Effect of mineral trioxide aggregate cements on transforming growth factor β_1 and bone morphogenetic protein production by human fibroblasts in vitro. *J Endod* 2007;33(4):447-450.
 57. Durand SH, Romeas A, Couble ML, et al. Expression of the TGF- β /BMP inhibitor EVI1 in human dental pulp cells. *Arch Oral Biol* 2007;52(8):712-719.