Vital pulp capping is the dressing of an exposed pulp with the aim of maintaining pulp vitality. Throughout the life of a tooth, vital pulp tissue contributes to the production of secondary dentin, peritubular dentin (sclerosis) and reparative dentin in response to biologic and pathologic stimuli. The pulp tissue — with its circulation extending into the tubular dentin — keeps the dentin moist, which in turn ensures that the dentin maintains its resilience and toughness. These characteristics ensure that the teeth can successfully resist the forces of mastication.

Although several studies have shown that endodontic procedures have an effect on the tooth’s dentin, others have suggested that it is the cumulative loss of dentin and the loss of the pressoreceptive mechanism and not the endodontic procedures that affect clinical performance. Whatever the reason, several studies have reported the higher failure rate of restored endodontically treated teeth. Since a non-vital tooth requires 2.5 times more of a load than a vital tooth to register a proprioceptive response, the natural protection against an overload is reduced and the probability of fracture increases.

Also, because posts do not reinforce teeth but may weaken them, restorative procedures that help preserve pulpal vitality and eliminate the need for posts are desirable. However, if endodontic therapy is unavoidable, conservation of the remaining tooth structure is most important.

Major advances in the practice of vital pulp capping have been made, and the emphasis has shifted from the “doomed organ” concept of an exposed pulp to one of hope and recovery. Long-term assessments of vital pulp caps with calcium hydroxide have shown very high success rates. Other studies have demonstrated that the exposed pulp possesses an inherent capacity for healing through cell reorganization and bridge formation when a proper biologic seal is provided and maintained against leakage of oral contaminants. Direct pulp capping should be used only on a vital pulp that has been accidentally injured and shows no other symptoms. Direct pulp capping should not be performed on a pulp that has been exposed as a result of penetrating caries. A successful pulp cap has a vital pulp and a dentin bridge within 75 to 90 days.

The major causes of post-operative inflammation and pulp necrosis are non-sterile procedures and bacterial micro-infiltration of the pulp via dentinal tubules. These may result from contamination of an exposed pulp prior to or during cavity preparation, or as a result of improper sealing of the entire dentin substrate interface when placing the restoration. To decrease the chances of contamination the rubber dam either must be in place from the start of the restorative procedure or be placed once a pulp exposure has been recognized.

VITAL PULP CAPPING TECHNIQUES

Two techniques have demonstrated success with vital pulp capping — the calcium hydroxide technique and the total etch technique (Fig. 1).
For vital pulp capping to be successful, the tooth should be asymptomatic or have minimal symptoms and the bleeding must be controlled. This control may be achieved by washing the area with sterile saline and drying it with either paper points or cotton pellets, by using cotton pellets soaked with hydrogen peroxide or 5.25% sodium hypochlorite, or, if necessary, by using a hemostatic agent such as Hemodent (Premier Dental Products, Norristown, Pa.). If bleeding fails to stop after two or three attempts, then endodontic therapy should be considered. Several studies have indicated that the size of the perforation is less important than obtaining hemostasis.

Following hemostasis, a disinfectant (e.g., Cavity Cleanser, Bisco Dental Products, Itasco, Ill., or Consepsis, Ultradent Products Inc., South Jordan, Utah) should be placed on the cavity floor. The area is then air dried, and calcium hydroxide in a formula such as Dycal (Dentsply Canada Ltd., Woodbridge, Ont.), Life (Kerr Manufacturing, Orange, Calif.) or Ultradent Calcium Hydroxide (Ultradent Products Inc., South Jordan, Utah) is placed directly in contact with pulp tissue. This step is very important, for the better the contact of the calcium hydroxide dressing with the pulpal wound, the better the healing. The calcium hydroxide should then be covered with a resin-modified glass ionomer extended onto dentin. Subsequently, a permanent restoration can be placed, with a dentin bonding system used to seal the margins of the restoration. An alternative is to place a zinc oxide–eugenol (IRM, L.D. Caulk, Dentsply Ltd., Woodbridge, Ont.) restoration over the calcium hydroxide cap. Zinc oxide–eugenol provides an excellent seal and, with its anti-microbial properties, makes for a very good temporary restoration. After three months, assuming pulp vitality and no symptoms, the zinc oxide–eugenol can be removed and a more permanent sealed restoration placed.

For the total etch procedure, as with calcium hydroxide, hemostasis must be obtained. The exposure site is then covered with a non-setting calcium hydroxide paste (e.g., Pulpdent, Pulpdent Corp. of America, Brookline, Mass.) and the cavity preparation completed. Following disinfection of the cavity, the enamel and dentin are etched with 32% phosphoric acid for 15 seconds. The acid and calcium hydroxide are rinsed off and the preparation is lightly dried. The entire preparation— including enamel, dentin and pulpal tissue—is treated with a dentin bonding system (a fourth-generation system with a separate primer and adhesive is recommended, as little research has been published to date on the fifth-generation dentin bonding systems). Following placement of several layers of the hydrophilic primer, a thin layer of the adhesive resin is painted onto the enamel, dentin and pulpal tissue and light cured. A second layer of unfilled resin is applied, and a thin layer of resin-modified glass ionomer is also applied over and around the exposure site to mechanically protect the perforation from intrusion of the restorative material during packing or condensation. These layers are also light cured. The restoration is subsequently completed in conventional fashion.

**DISCUSSION**

The opponents of calcium hydroxide claim that it does not exclusively stimulate sclerotic dentin formation, dentinogenesis, reparative dentin formation or dentin bridge formation. They also claim that it may dissolve after one year, that acids will degrade the interface during etching, and that calcium hydroxide does not adhere to dentin and will not adhere to bonding resin composite systems. One study found that calcium hydroxide bases under resin composite restorations tended to pull away from the cavity surface during resin polymerization, leaving a gap between the calcium hydroxide and dentin. Cox and others found a high rate of multiple tunnel defects (89%) in dentin bridges under calcium hydroxide. This high rate of defects, they suggest, places the long-term therapeutic effect of calcium hydroxide in serious doubt. They also suggest that calcium hydroxide disintegrates and is lost over a period of time.

It has been suggested that a very small exposure, and certainly a near exposure, cannot be treated with calcium hydroxide, as it is essential that the calcium hydroxide dressing make contact with living pulp tissue. In addition, Pashley states that there may be little difference between a vital pulp cap and a situation where the remaining dentin thickness is less than 1 mm. He attributes this similarity to the high permeability of the dentin near the pulp. In a recent study, opponents of the total etch technique found a 40% loss of pulp vitality over a period of 75 days with three bonding systems on exposed primate pulps. Of the remaining surviving pulps, only 53% even attempted bridge formation. Proponents of this technique point out that germ-free studies have shown that pulp heals rapidly even when bonding agents are placed directly on pulpal tissue.

The healing of pulp exposures may depend on the capacity of the capping material to prevent bacterial microleakage. Pashley states that to minimize the pulpal response, restorative
materials must seal the cavity margins, prevent microleakage and block bacterial substrates from penetrating through dental tubules to the pulp. However, if microleakage around various restorations could be measured in vivo, it is likely that all would exhibit some degree of leakage.38 If these teeth remain asymptomatic, it is probably because the rate at which exogenous materials permeate across dentin to the pulp is balanced with the rate of removal of these materials by pulpal circulation, thus ensuring pulpal vitality.40 Therefore, it is desirable to maximize the barrier effect of dentin to provide the best pulpal protection.38 Each situation must be assessed to determine which method is most likely to achieve a maximal barrier effect.

A dentist’s inability to perform proper pulp cap procedures can lead to microbial contamination, leftover dental debris in the wound and a lack of a dentin seal. Poor operator performance, therefore, rather than the inadequacies of the medicament, may be the cause of pulp-cap failure.15 In the case of recurrent pulpitis, therefore, one must distinguish between pulp-cap failure and failure of the restoration subsequently placed over the pulp-capping agent.43

CONCLUSION

Mechanical exposures are more likely than various exposures to be successfully capped. If the operator properly selects the case, obtains hemostasis, disinfects the exposure and the cavity preparation, and adequately seals the exposure and the cavity preparation, success can be obtained with either the calcium hydroxide technique or the total etch technique. Although both techniques can achieve successful vital pulp caps, the calcium hydroxide technique has demonstrated its success over a longer period of time. Which technique offers the better prognosis awaits the results of many more long-term studies.

For unknown reasons, the pulp-capping agent used, and not the procedure itself, has been the subject of controversy among researchers. ♦

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