**Vital pulp therapy**

Vital pulp therapy is broadly defined as treatment initiated to preserve and maintain pulp tissue in a healthy state, tissue that has been compromised by caries, trauma, or restorative procedures. The objective is to stimulate the formation of reparative dentin to retain the tooth as a functional unit

The focus is directed toward the preservation of the pulpally involved permanent tooth based on the premise that pulp tissue has an innate capacity for repair in the absence of microbial contamination.

The pulp status must be determined carefully, establishing a differential diagnosis using multiple tests. According to the American Academy of Pediatric Dentistry, ‘‘Teeth exhibiting provoked pain of short duration, that is relieved, upon the removal of the stimulus, with analgesics, or by brushing, without signs and symptoms of irreversible pulpitis, have a diagnosis of reversible pulpitis and are candidates for vital pulp therapy.’’

And proper case selection cannot be overemphasized. The pulp status must be determined carefully, establishing a differential diagnosis using multiple tests.

According to the American Academy of Pediatric Dentistry, ‘‘Teeth exhibiting provoked pain of short duration, that is relieved, upon the removal of the stimulus, with analgesics, or by brushing, without signs and symptoms of irreversible pulpitis, have a clinical diagnosis of reversible pulpitis and are candidates for vital pulp therapy.’’

The assessment of a definitive pulpal status prior to treatment is often difficult to establish; however, a diagnosis of reversible pulpitis increases the probability of a favorable outcome.

A negative patient report, that is subjective and variable, does not always indicate that the pulp capping or pulpotomy procedure cannot be successful. Moreover, pain associated with cold testing prior to treatment or a pulp exposure during caries excavation does not necessarily mandate a poor prognosis for the involved tooth.

**Indications for Vital Pulp Therapy**

Vital pulp therapy is indicated whenever the remaining pulp exhibits reversible pulpitis and can be selectively induced to produce a reparative barrier that protects the tissue from microbial challenges.

**The Vital Dental Pulp**

The pulp is a highly vascular tissue that has the unique distinction of being encased within a rigid chamber composed of dentin, cementum, and enamel.

The tissue performs several important functions, including dentinogenesis, immune cell defense, and nutrition and proprioreceptor cognizance.

Circulating immunocompetent cells limit microbial challenges, and functioning proprioceptors and pressoreceptors guard against excessive occlusal loading. By contrast, structurally compromised teeth that have been endodontically treated and restored with various post and core systems are more susceptible to fracture and failure owing to the loss of protective mechanisms.

The dental pulp is composed of four distinct zones: a cell-rich zone, a core composed of major vessels and nerves, a cell-free zone, and the odontoblastic layer at the periphery. The major cell populations found in the pulp include fibroblasts, undifferentiated mesenchymal cells, odontoblasts, macrophages, and other immuno- competent cells.

After an injury, the tissue adjacent to the exposure is characterized by inflammatory cells, extravasated erythrocytes, and potentially necrotic tissue. An acute response is mounted, dominated by neutrophils in the presence of exudated fibrinogen and blood coagulation. Vascular permeability is altered when proinflammatory cytokines are released by immunocompetent cells in response to both trauma and bacterial by-products.

Chemotactic signals prompt adhesion molecule interactions between leukocytes and endothelium, enabling transmigration of inflammatory cells. These cell inter actions form an adhesion cascade mediated by chemoattractant/activator molecules interacting with sets of cell adhesion molecules.

Competent immune defense mechanisms, it is desirable to preserve the vitality of an exposed pulp since its retention is crucial to the tooth’s long-term survival. If the hard casing of the tooth is compromised and the pulp is subjected to microbial ingression, inflammatory changes can lead to pulp necrosis and further pathologic changes, including infection and its consequences.

Circulating immunocompetent cells limit microbial challenges, and functioning proprioceptors and pressoreceptors guard against excessive occlusal loading. By contrast, structurally compromised teeth that have been endodontically treated and restored with various post and core systems are more susceptible to fracture and failure owing to the loss of protective mechanisms. Although studies show that the loss of moisture from dentin after endodontic therapy is minimal, cumulative loss of tooth structure is implicated in the failure of root-treated teeth.

The dental pulp is composed of four distinct zones: a cell rich zone, a core composed of major vessels and nerves, a cell-free zone, and the odontoblastic layer at the periphery. The major cell populations found in the pulp include fibroblasts, undifferentiated mesenchymal cells, odontoblasts, macrophages, and other immuno- competent cells. The odontoblasts have the distinction of forming a single layer lining the periphery of the pulp and feature odontoblastic processes extending into the dentin, sometimes to the dentoenamel junction.

When the dental pulp is injured by trauma or carious exposure, the mechanism of healing is similar to that observed in normal connective tissue

Wound healing is a continuous process, and a sequence of four phases of healing overlap, including hemostasis, inflammation, proliferation, and remodeling.

After an injury, the tissue adjacent to the expo sure is characterized by inflammatory cells, extravasated erythrocytes, and potentially necrotic tissue. An acute response is mounted, dominated by neutrophils in the presence of exudated fibrinogen and blood coagulation. Vascular permeability is altered when proinflammatory cytokines are released by immunocompetent cells in response to both trauma and bacterial by-products. Chemotactic signals prompt adhesion molecule inter- actions between leukocytes and endothelium, enabling transmigration of inflammatory cells. These cell inter- actions form an adhesion cascade mediated by che- moattractant/activator molecules interacting with sets of cell adhesion molecules.

**Reparative Dentin Formation**

The reformation of a protective dentinal bridge by tertiary dentinogenesis is a primary goal of vital pulp therapy. The repair of pulpodentinal defects is orchestrated by the migration of granulation tissue to the site from the cell-rich and deep pulp subodontoblastic layers that differentiate into new odontoblast-like cells.

Although these progenitor cells are most likely derived from undifferentiated mesenchymal cells, other cell populations migrating via the bloodstream, such as bone marrow stem cells and perivascular cells, have been proposed as possible precursors.

Calcium hydroxide–pulp interface, a continuous in- flux of newly differentiating odontoblast-type cells with initial matrix formation was observed as early as day.

Labeled odontoblast-like cells showed differences in cell types and grain counts between zones, indicating that at least two deoxyribonucleic acid (DNA) replications had occurred between initial treatment and differentiation. Studies have suggested that the mineralization of dentin bridges is more dependent on the extracellular matrix than the pulp capping or pulpotomy material.

After the disappearance of the vesicular membrane, a calcified front formed as crystals and aggregate accumulated. The presence of calcium and phosphate ions within the crystals suggested that they were produced during the calcification process, similar to the calcification in other biologically normal or diseased tissues. Dentin bridge formation can be seen after 1 month at the site of the surgical wound, although pulp healing defects can be associated with different pulp capping agents and include tunnel defects, operative debris, pulpal inflammatory cell activity, and bacterial microleakage.

**Techniques for Generating Reparative Dentin**

* Direct pulp capping
* Indirect pulp capping
* Pulpotomy partial pulpotomy

**Pulp capping materials**

* Calcium hydroxide
* Resin modified glass inomer
* MTA

**Direct pulp capping**

Direct pulp capping is defined as the ‘‘treatment of an exposed vital pulp by sealing the pulpal wound with a dental material placed directly on a mechanical or traumatic exposure to facilitate the formation of reparative dentin and maintenance of the vital pulp.

Indication: exposures as a result of caries removal, tooth preparation, or trauma.

**INDIRECT PULP CAPPING**

Indirect pulp capping is defined as ‘‘a procedure in which a material is placed on a thin partition of remaining carious dentin that, if removed, might expose the pulp in immature permanent teeth.

**PULPOTOMY**

Pulpotomy is a more extensive procedure defined as

‘‘the surgical removal of the coronal portion of a vital pulp as a means of preserving the vitality of the remaining radicular portion.’’

After the complete removal of the coronal pulp, a material is placed over the canal orifices. A variety of dressing materials, with varying toxicity, have been used for this purpose. They include phenol, creosote, ferric sulfate, polycar- boxylate cement, glutaraldehyde, ZOE, Ca(OH) and formaldehyde, which mummifies the remaining tissue.

**PARTIAL PULPOTOMY**

Partial pulpotomy (Cvek pulpotomy) is defined as

‘‘the surgical removal of a small portion of the coronal portion of a vital pulp as a means of preserving the remaining coronal and radicular pulp.’’

In this instance, inflamed tissue is removed to expose deeper, healthy coronal pulp tissue.

Direct pulp capping and partial pulpotomy are considered similar procedures and differ only in the amount of undestroyed tissue remaining after treatment.

**Vital Pulp Therapy Materials characteristics:**

•Stimulate reparative dentin formation

•Maintain pulpal vitality:

•Release fluoride to prevent secondary caries

•Bactericidal or bacteriostatic

•Adhere to dentin

•Adhere to restorative material

•Resist forces during restoration placement

•Must resist forces under restoration during lifetime of restoration

•Sterile

•Radiopaque

•Provide bacterial seal

**Calcium hydroxide**

Beneficial characteristics include a bactericidal component owing to its high alkaline pH and the irritation of pulp tissue that stimulates pulpal defense and repair.

Has been shown to be cytotoxic in cell cultures, does not exclusively stimulate reparative dentin formation, shows poor marginal adaptation to dentin, and induces pulp cell apoptosis.

Dentin bridges beneath Ca(OH) are associated with tunnel defects, and the material fails to provide a long-term seal against microleakage when used as a pulp capping agent.

The disintegration of Ca(OH) under restorations associated with defects in the dentinal bridge can provide microorganisms with a pathway for penetration into pulpal tissue and the subsequent stimulation of circulating immune cells, inducing pulpal irritation and potential pulpal calcification and canal obliteration.

**ADHESIVE RESINS AND RESIN-MODIFIED GLASS IONOMERS**

Vitrebond (3M EspeDental Products, St. Paul, MN) and Clearfil Liner Bond 2 (Kuraray Co., LTD, Osaka, Japan) resulted initially in a moderate to intense inflammatory response and did not stimulate reparative dentin formation This is an indication that these pulp capping materials do not allow for predict- able pulpal healing, nor do they provide a favorable environment for reparative dentin formation and the exclusion of microorganisms. Repair should proceed successfully beneath the material when bacterial microleakage is preclude.

**MINERAL TRIOXIDE AGGREGATE (MTA)**

* Introduced in 1990
* Was initially composed of tricalcium silicate, tricalcium aluminate, tricalcium oxide, silicate oxide, and other mineral oxides.
* Substitution of dicalcium silicate for tricalcium silicate and the addition of tertracalcium aluminoferrite, calcium sulfate dehydrate, and bismuth oxide; the latter was added to impart radiopacity
* Grey and white
* Set in the presence of blood and moisture
* Superior marginal adaptation and is nonabsorbabale, and when it cures in the presence of calcium ions and tissue fluids, it forms a reactionary layer at the dentin interface resembling hydroxyapatite in structure
* Sustained alkaline pH after curing, small particle size, and a slow release of calcium ions.
* The high alkalinity of MTA and its calcium release and sustained pH at 12.5 is most likely responsible for preventing any further microbial growth of residual microorganisms left after caries excavation

**Diagnostic Criteria for Successful Outcome**

* Young age.
* Minimum exposure
* Less microbial contamination (isolation)
* Pulpal diagnosis, healthy pulp and reversible pulpitis
* Presence and absence of pain
* Incomplete Root formation
* Clinical evaluation must also include assessment of mobility, periodontal probing, localized swelling, and the presence of sinus tracts
* Radiographs (both periapical views and bitewings) must be assessed for the absence of periapical pathosis, furcation radiolucencies, internal or external resorption defects, and pulp calcification owing to previous restorations or trauma.

**Caries removal**

RD isolation m caries detecting dye along with optical magnification.

Two carous layers

The development of caries staining using a propylene glycol solution of Acid Red 52 dye (a common food and cosmetic coloring dye) provides visible differentiation of the two carious layers and is a selective method to remove the necrotic and infected dentin

The retained cariesaffected layer of dentin, which contains banded, intact collagen, allows for the remineralization of the altered tissue by calcium phosphate secreted from the pulp via the dentinal tubules. In the dentinal tubules, calcium and phosphate ions induce the formation of whitlockite crystals, which block the tubules.

Pulpal preservation without injuring residual caries-affected dentin that is reparable and remineralizable when a bonded composite restoration is placed to prevent microbial leakage.

**Hemostasis**

ferric sulfate, disinfectants such as Concepsis (Ultradent Products Inc., South Jordan, UT) and Tubulicid (Global Dental Products,

North Bellmore, NY) epinephrine, and varying concentrations of hydrogen peroxide (H2O2) and NaOCl.

The most commonly accepted technique has been direct pressure at the exposure site with cotton pellets moistened in sterile water or saline.

Another emerging potential hemostatic agent is MTAD (Biopure, Tulsa/Dentsply), an irrigant and an antimicrobial agent introduced for removal of the smear layer during nonsurgical initial endodontic treatment and retreatment.

The solution is a mixture of tetracycline isomer (doxycycline), an acid (citric acid), and a detergent (Tween 80). The irrigant shows many desirable properties and may be a suitable replacement for ethylenediaminetetraacetic acid in conjunction with NaOCl.

**Pulp capping treatment recommendation: Read the one step – two step pulp capping procedure (page 1320-1322)**

**Final restoration**

In the absence of microleakage, the pulp will have the highest probability for wound repair and survival.

Restorative procedures for immature permanent teeth include full-coverage restorations, composite resins, and bonded or unbonded amalgam restorations. The more conservative the restorative treatment, preserving the remaining healthy tooth structure, the higher the probability of pulp survival.

**Postoperative Follow-Up**

In an investigation in which direct pulp capping was completed with Ca(OH)2, it was determined that 3 months was an adequate time period for a tentative diagnosis of survivability.

0ne to two years for the prognosis of long term survival

MTA is used in the two-visit protocol (described earlier in this chapter), the clinician has the opportunity to examine the treated tooth at 5 to 10 days. If the treatment appears successful at that appointment, the provider can confidently schedule the next follow-up at 6 weeks if possible and then at 6 and 12 months.

Following contralateral tooth development is an excellent way to observe the success of vital pulp therapy