Clinical Review

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Vital Pulp Therapy In Cariously Exposed Permanent Teeth And Its Limitations

Abstract

Vital pulp therapy for cariously exposed permanent teeth remains one of the most controversial areas in dentistry. Because a vital, functioning pulp is capable of initiating several defence mechanisms to protect the body from bacterial invasion, it is beneficial to preserve the vitality and health of an exposed pulp rather than replace it with a root filling material following pulp exposure. There is no consensus on the survival rate of formerly cariously exposed pulps. Observation time, judgement criteria, pulpotomy/pulp capping technique and, most importantly, pulpal status at the time of treatment, vary to a great extent amongst studies.

In mature teeth, a pulp exposed by caries is usually removed and the root canals are cleaned, shaped and filled. Amongst the methods for preservation of a cariously exposed pulp, partial pulpotomy has yielded a markedly high success rate in young teeth. Major limitations in the success of vital pulp therapy in cariously exposed permanent teeth exist. The lack of predictability and long-term success greatly influence decision-making. The decision-making itself is unreliable primarily due to the difficulty of accurately diagnosing the ability of the pulp to repair.

While there are indications for vital pulp therapy in young permanent molars, it must be remembered that ultimately, none of these procedures enjoy the long-term success of complete root canal therapy.

Introduction

The treatment of the cariously exposed permanent tooth should result in arresting the carious process and keeping the vital pulp free of inflammation (1). The aim of vital pulp therapy is to preserve the vitality and function of the coronal or remaining radicular pulp tissue (2). Carious lesions involving the pulp, if not treated, will lead to pulpal necrosis and often involvement of the periodontal tissues, with pain and discomfort for the patient (3). The treatment of the cariously exposed pulp is dependent upon the maturity of the tooth. In deciduous teeth, treatment is aimed at removing the infected, coronal pulp and fixing or mummifying the radicular pulp tissue, thereby preserving the tooth, not the pulp (4). In mature permanent teeth, pulpectomy and obturation of the root canal system is usually the treatment of choice.

In young permanent teeth, pulpotomy is classically undertaken to promote apexogenesis (5). Apexogenesis refers to a vital pulp therapy procedure performed to encourage physiological development and formation of the root end (6). The objective is to promote root development and apical closure. An open, divergent apex presents the challenge of producing an apical stop or constriction for placement of a hermetically sealed root canal filling (6). Because the pulp is necessary for the formation of dentine, the loss of vitality in young permanent teeth before completion of root formation leaves a thin, weak root that is prone to fracture and difficult to treat endodontically (7). The prognosis for permanent retention is limited when compared with fully developed teeth. Every effort must be made to maintain the vitality of the pulp in teeth with incomplete apices to achieve root development. Once root development is achieved and apices have closed, root canal treatment is completed. The need for root canal treatment was based on the premise that possible necrosis, continued calcification or internal resorption will occur following pulpotomy. Recent studies have proposed that as long as a hermetic seal is ensured, root canal treatment is not needed following pulpotomy (8).

Partial pulpotomy aimed at preserving pulpal vitality has been shown to be successful in traumatically exposed permanent teeth (9). This treatment regimen has been applied and showed some success in the treatment of cariously exposed young permanent molars (10, 11). The excellent blood supply of a young permanent tooth with incompletely formed roots may contribute towards this success (7). In contrast, the decreased resistance of the aged pulp may become significant when one considers that the vascular reaction to inflammation is considered a protective mechanism of the pulp against invading factors (12). From the clinical point of view, the vitality of the dental pulp of an aged person appears to be weaker than that of a young person (12).

Considerable literature emphasises the negative aspects of vital pulp therapy and discourages its practice. Many clinicians and researchers continue to condemn vital pulp therapy for the same reasons reported in the literature 80 years ago, despite the advances made in pulp biology (13). The immediate and long-term success of root canal therapy is well known, but clinicians are less certain of the success of vital pulp therapy. Cvek (9) reported a 96% success rate for partial pulpotomy in traumatically fractured teeth with an open apex that were healthy before trauma. Barthel et al (14), in comparison, found a success rate of only 13% ten years after capping cariously exposed, asymptomatic, vital pulps. Whilst observation time, pulpotomy technique and judgement criteria vary between studies,
the status of the pulp at the time of capping is the predominant factor in determining the healing of exposed pulps (14).

The long-term prognosis and the ability to restore a tooth are the overriding factors when assessing whether vital pulp therapy should be undertaken (15). The amount of crown destruction usually associated with a pulp exposure in an immature molar means that the tooth involved would require significant restorative maintenance in the long term.

Basic Concepts In Vital Pulp Therapy Of Cariously Exposed Permanent Teeth

The treatment objective following pulp exposure is to obtain healing of a pulp wound in order to preserve a vital tooth with a healthy pulp. Kakehashi et al (16), clearly showed that the key factor in pulpal healing after exposure is the absence of infection. Failures are caused by infection due to either remaining bacteria, or the exposure to new bacteria from leakage (17). Heys et al (18) demonstrated that exposed pulps heal even against an inert material such as Teflon, when bacterial contamination is eliminated.

Vital pulp therapy aims to treat reversible pulpal injury. There is a difference in the outcome between treating an inflamed and a non-inflamed pulp (17). The chance for successful outcome is markedly higher in the non-inflamed pulp exposed by trauma, than in the inflamed pulp exposed during caries excavation. Treatment will be successful only if the pulp, at the time of treatment, is sound or not seriously enough inflamed to result in a continuation of the inflammatory response (1).

Caries causes pulpal inflammation, even if the carious process has not reached the pulp tissue, because bacterial by-products will reach the pulp via the dentinal tubules. Consequently, with the majority of deep carious lesions, the dentine is often supported by an injured pulp that itself must undergo repair and remain clinically asymptomatic for vital pulp therapy to be considered a success (19). The presence of pain does not always indicate that a pulp injury is irreversible, although certain patterns and intensities of pain tend to suggest a greater likelihood of an irreversible change. The inflammatory status of the pulp during a pulpal exposure is a major factor in determining whether vital pulp therapy will be successful and is also extremely difficult to accurately diagnose.

For teeth in which the caries process is aggressive or in which the pulp may have a reduced reparative potential because of chronicologic or physiologic aging, there is generally a progressive inflammation and necrosis of tissue resulting in the loss of the pulp as a functioning organ (19). The inflammation in such cases is classified as irreversible. If the pulp can recover from the inflammation induced by a caries attack with appropriate treatment, the inflammation is referred to as reversible. Seltzer et al (20) showed that the correlation between diagnostic and actual histologic findings in the pulp is not strong and that this classification should be considered clinical rather than histopathologic. A low-grade, asymptomatic chronic inflammation leading to pulpal necrosis often exists long after caries had been removed and the tooth restored (20).

Reversible inflammation is more common in young permanent teeth than in mature permanent teeth (19). Growth, maturation and inflammation all cause the pulpal vasculature to undergo significant morphological changes. The superficial layer of the young pulp consists of a characteristic three-layer vascular network (12). After teeth grow into occlusion, the pulp cavity narrows and this vascular network undergoes reconstruction. Two of the three vascular layers disappear, leaving a coarse terminal capillary network (12). As these morphological changes progress with time, the protective vascular reaction to inflammation diminishes, influencing the pulp's ability to successfully protect against invading factors.

The success of vital pulp therapy techniques in cariously exposed permanent teeth is dependent upon the technique employed, the inflammatory status of the pulp tissue, the type of pulp therapy agent used, the period of observation and the criteria used to determine success (21). Accurate diagnosis of the pulp status prior to treatment, caries removal, leakage prevention and use of an aseptic technique, are key determinants in a successful treatment outcome.

Techniques

Vital pulp therapy techniques that are used for treatment of cariously exposed permanent teeth include:
- Indirect pulp capping
- Direct pulp capping
- Full coronal pulpotomy
- Partial pulpotomy

These procedures have been employed for various, mechanical, and traumatic exposures of the pulp. A high incidence of success has been reported, as judged radiographically and by the absence of clinical signs and symptoms (22). However, large variations in success rate between studies exist. Differences in case selection, length of study, and type of investigation are responsible for these variations (22).

Indirect pulp therapy is a technique for avoiding pulpal exposure in the treatment of teeth with deep carious lesions in which there exists no clinical evidence of pulpal degeneration or periapical disease (22). This technique is based on the theory that a zone of affected demineralized dentine exists between the outer infected layer of dentine and the pulp (23). When the infected layer is removed, the affected dentine can then remineralise and the odontoblasts form reparative dentine, avoiding a pulp exposure. Carious dentine left in the tooth undoubtedly contains some bacteria but calcium hydroxide and/or zinc oxide-eugenol cement can greatly diminish their numbers (24). The remaining soft dentine is covered and after two months the degree of remineralisation is tested and any residual softened dentine removed (24). If caries has reached the pulp, endodontic treatment is initiated. If there has been a favourable response and the dentine is hard, a permanent restoration can be placed. Some studies report repair in 74% to 99% of cases (13, 25). Other authors state that there is a low expectation for success and that failures increase with time (17). A difficulty with this technique is clinically determining what is infected or just affected dentine. To intentionally leave infected material is definitely not recommended as it leads to further pulpal inflammation and likely failure. From a practical standpoint, it is strongly advised to excavate carious dentine until the remaining dentine feels hard to a dental explorer and reflects light (17). The use of caries indicator dye may be of benefit also.

Direct pulp capping is a procedure in which an exposed dental pulp is covered with a protective dressing or cement that protects the pulp from additional injury and permits healing and repair (26). The outcome for direct pulp capping following a carious exposure is considered, at best, unpredictable (27, 28). During the direct capping procedure the capping agent is placed on tissue that has been exposed to microorganisms and where inflammation is present (17). Histologic examinations have shown chronic inflammation under many cariously exposed pulp caps and diminished success rates (22). Direct capping procedures should only be carried out on fresh, traumatic or mechanical exposures, before bacterial plaque has established on the exposure and the underlying pulp has become inflamed (17, 29).
Pulpotomy has become the treatment of choice for cariously exposed vital teeth (17). Pulpotomy is described as a procedure that involves the amputation of the coronal portion of the affected or infected dental pulp and treating the remaining vital pulp to preserve its vitality and function (30). Pulpotomy allows for the pulp wound to be placed in healthier tissue than at the site of exposure.

Full coronal pulpotomy involves the complete removal of the coronal pulp and the placement of a wound dressing at the canal orifice(s). Many different chemical compounds have been used as wound dressings. Wound dressings containing formaldehyde, phenol, creosotes and other highly toxic materials were advocated to “mummify” the remaining pulp tissue (17). The short-term clinical outcome for these materials is usually acceptable. On the other hand, with increasing time there is a decreasing success rate following pulpotomy with formaldehyde-containing materials (17). Due to the short lifespan of primary teeth, this form of pulpotomy is popular in paediatric dentistry.

The use of materials such as formocresol in the full coronal pulpotomy of permanent teeth with pulp exposures has been recommended (31). This goes against all modern endodontic principles as the remaining chemically necrotised tissue can serve as a substrate for bacteria as a result of leakage (17). In addition, Ruks et al (32) found a high incidence of internal resorption in formocresol-pulpotomised permanent monkey teeth and Rolling et al (33) found that the portion of canal apical to the fixed plug of tissue beneath formocresol pulpotomies in permanent teeth with pulp exposures, is often tortuous and extremely difficult to instrument when endodontic treatment is often needed years later. Formocresol pulpotomies are not recommended in teeth with mature root formation unless there are behavioural and/or socio-economic constraints to performing root canal therapy (15). Pulpotomy agents such as formocresol and glutaraldehyde are associated with systemic toxicity and carcinogenic potential, so their safety is questionable (34).

Partial pulpotomy has achieved very high success rates in the treatment of complicated crown fractures (95%) (32), and in young, posterior, symptom-free permanent teeth with carious exposures (91–93%) (10, 11, 35). Partial pulpotomy, as described by Cvek in 1978 (9), is the aseptic, surgical removal of the exposed pulp and dentine surrounding the exposure to a depth of 1.5–2.0 mm. This aims to surgically remove tissue that is irreversibly inflamed and exposed to microorganisms leaving a clean-cut wound surface on healthy tissue (17). The pulp wound is irrigated with sterile saline until physiologic haemostasis is achieved and then the pulp wound is covered with a paste of calcium hydroxide and water. An alternative irrigant is sodium hypochlorite (0.5–5.25%). This has been shown to be effective at haemorrhage control and surface disinfection and flushing, without any detrimental pulpal effects (5, 18). The wound dressing is then carefully dried and sealed with a suitable material. The direct apposition of the calcium hydroxide paste on the non-bleeding pulp tissue is essential, as an extra-pulpal blood clot will diminish the chances for hard barrier formation and long-term success (17). Partial pulpotomy creates a retentive cavity for the covering materials and the use of calcium hydroxide in combination with removal of blood clot gives predictable healing with a continuous hard tissue barrier (9).

With calcium hydroxide treatment, a dentine bridge will predictably form over healthy, remaining pulp tissue (13). In the absence of a bridge, the wounded pulp tissue is much closer to the surface and is more easily invaded by subsequent attacks of oral bacteria and their by-products (36). A dentine bridge allows more predictable repair by giving underlying pulp tissue something to attach to (13). Without a bridge, pulp tissue remains unattached, undergoes degeneration, atrophy and shrinkage away from dentine. Dentine bridges contain tunnels and porosity. The number and size of these tunnels reflects the degree of trauma to the pulp (13). Cellular inclusions and empty vascular spaces create porosity, and in combination with tunnels, contribute to the permeability of every dentine bridge. Dentine bridges have been condemned as ineffective protective barriers because of their permeability, although regular tubular dentine is itself, permeable (13). Even though the quality of a dentine bridge may be suspect as to its integrity, it nevertheless provides a physical barrier of some substance to protect the pulp. Dentine bridge formation is the best solution for healing of the underlying pulp (13).

Current Research

The success of vital pulp therapy in cariously exposed permanent teeth is the subject of much debate. The scepticism towards vital pulp therapy originates from an historical context, where treatment decisions were based on empirical reasoning (26). Strindberg in 1956 (37) reported a success rate for direct pulp capping with calcium hydroxide of 64%, compared to a success rate of 83% for root canal treatment. Since then, knowledge of pulpal physiology has broadened, leading to a better understanding of the conditions necessary to take advantage of the good potential of the pulp to heal. Recent clinical studies indicate that vital pulp therapy of cariously exposed young permanent teeth can produce success rates that compare favourably with the success rate of root canal treatment (2, 3, 10, 11, 12, 36).

Conflicting views have been expressed when relating pulpal changes to caries progression and clinical symptoms (20, 27, 39, 40). In 1968, Langeland and Langeland (41) showed that deep carious lesions might be found with relatively normal pulps or with only a slight increase in chronic inflammatory cells. Trowbridge (42) suggested that the pattern of pulpal inflammation is determined by the permeability of dentine and the proximity of the carious lesion to the pulp. Lin and Langeland (43) stated “whereas the repair capacity of pulp tissue following removal of all carious dentine without pulp exposure is excellent, after carious exposure it is questionable and unpredictable”. Seltzer et al (20) histologically considered up to 79% of permanent teeth with clinical pulp exposures to be in the non-treatable category.

It is generally accepted that in advanced stages of a carious lesion, just prior to or soon after pulp exposure, bacterial components cause only localised irreversible damage beneath the exposure, while the remaining pulp is infiltrated with inflammatory changes to a varying degree. The bacteria only seem to gain access to the pulp lumen after some part of the pulp has become necrotic (39, 40). These views seem to be confirmed by the studies of Zilberman et al (11) and Mejare and Cvek (10). The removal of the superficial layers of pulp tissue together with the contaminated dentine and the prevention of microleakage, sufficed to ensure healing in most of the treated teeth.

Seltzer et al (20) showed that under small carious lesions only an increase in reparative dentine formation is induced, whilst in moderately large cavities, macrophages and lymphocytes are observed under the involved dentinal tubules of the coronal pulp. In very deep lesions, chronic inflammatory exudate develops. When the pulp is exposed by caries, acute localised inflammation and liquefaction necrosis can be observed under the exposure site. Langeland (39) postulated that in order to preserve the remaining healthy pulp, it is necessary to remove this infected, necrotic and disintegrated pulpal tissue.
Farooq et al (30) showed that a carious primary tooth diagnosed with pain from reversible pulpsitis could be successfully treated with either an indirect pulp treatment or a formocresol pulpotomy. Success rates of 85% and 76% respectively were observed for these two techniques over a four-to-five year follow up. Gruythuysen and Weerheijm (44), in a similar study over a two-year period, achieved an 80% success rate with the placement of calcium hydroxide as a base after pulpotomy of carious primary teeth with signs of reversible pulpsitis. Both these studies (30, 44) showed a high failure rate of all vital pulp therapy when a preoperative diagnosis of irreversible pulpsitis or pulpal necrosis was formed. Careful history taking, together with symptoms and clinical/radiographic findings should help form the final diagnosis and determine the final treatment to be provided.

Formocresol pulpotomy in permanent teeth has shown good success rates. Hosseini (45) showed a 92% success rate after 10 years. Tepilisky (46) showed evidence of continued apical development following formocresol pulpotomy procedures on young permanent teeth with incompletely developed apices. Initially it was thought formocresol pulpotomy had more appeal than calcium hydroxide pulpotomy as calcification of the remaining pulp tissue was not seen (22). Contrary to these thoughts, calcification of canals with formocresol pulpotomy was shown. Full strength formocresol pulpotomy has been shown to produce obliteration of the root canal in up to 80% of teeth treated (47). Fuks and Bilstein (48) using a one-fifth dilution of full-strength formocresol found radiographic obliteration in only 29% of treated teeth. This obliteration by calcification may preclude future root canal treatment. Although this treatment has been reported to be partly successful, it cannot be routinely recommended until further research showing this technique to be successful has been completed (22).

As stated earlier, Cvek (9) reported a 96% success rate for partial pulpotomy in traumatically fractured teeth with an open apex that were healthy before trauma. Mejare and Cvek (10), using the partial pulpotomy technique adopted from Cvek (9), achieved a 93.5% success rate after carious pulp exposure of young permanent teeth exhibiting no clinical or radiographic symptoms. Zilberman et al (11) found 14 of 15 deeply carious permanent young molars exhibited a vital pulp response at 12–99 months following partial pulpotomy. Mass and Zilberman (35) achieved a success rate of 91.4% after a minimum of 12 months, using partial pulpotomy and careful inclusion criteria in the treatment of young permanent molars with carious pulp exposure. Barthel et al (14), in comparison, found a success rate of only 13% ten years after capping cariously exposed asymptomatic, vital pulps.

Matsuo et al (38) showed a success rate of 80% at >6 months following direct pulp capping of permanent carious teeth with some symptoms of irreversible pulpsitis. Unlike most other studies, teeth with lingering pain to thermal stimuli and with sensitivity to percussion were included in the study. Çalışan (2) showed that penapical involvement did not necessarily preclude cariously exposed permanent teeth from vital pulp therapy. Twenty-six permanent vital molars with carious pulp exposures and radiographic penapical involvement were treated using a full coronal pulpotomy, with calcium hydroxide placed directly on the radicular pulp tissue once bleeding had stopped. Of these, twenty-four teeth showed resolution of radiographic penapical involvement and dentine bridge formation after 16–72 months. The presence of radiographic penapical change accompanying reversible pulpsitis can best be rationalized as a sequel to reversible pulpal neurogenic inflammation, rather than as a direct response to an infected, dying pulp.

It was traditionally believed that the primary cause of pulp damage after direct pulp capping was direct toxic effects from dental materials. Animal studies however, have demonstrated that restorative materials do not cause pulp inflammation or necrosis when directly placed on the exposed pulp if bacteria are sealed off at the margins (49).

In recent years there have been many publications illustrating healthy pulps without dentine bridges in teeth treated with a total etch and bond technique. The concept is that these bonding techniques adequately seal the exposure sites from bacterial invasion and consequently a dentine bridge is not needed. Long-term success of this seal has not been established (13). Kitasako (49) found that because pulp tissue protruded into the cavity around the exposure site, direct resin pulp capping might permit oral bacteria to eventually access the pulp via microleakage. Exudation from the exposed pulp may also prevent the moisture sensitive resin materials from adhering to peripheral dentine (49). Hebling et al (50) showed a persistent inflammatory reaction and lack of complete pulp repair or dentine bridging 60 days after direct capping of non-inflamed pulpal exposures with an adhesive system, compared to pulp repair with complete dentine bridging with calcium hydroxide. At present, there is little long-term data available to support this technique.

The Future

Pulp therapy for cariously exposed teeth remains one of the most controversial areas in dentistry. No clear-cut diagnostic indication or therapeutic method exists. Current protocols are based on the empiricism of ages past. Much of this controversy is because scientific research has not provided us with the technology to overcome two great shortcomings in dentistry. Firstly we are unable to directly treat pulp infection and save the pulp. Secondly, we have no predictable inductive agent for stimulating reparative dentine (51). Also, we cannot predictably diagnose the inflammatory status of the affected pulp.

Pulp studies are very difficult. The anatomy of the pulp cannot be duplicated in vitro, pulp cells in culture quickly lose their phenotypes, animal teeth do not react like human teeth and clinical studies are often long and arduous (51). Molecular biology though, has made it possible to ask the questions that have never been asked. Odontoblast and pulp cell lines will now maintain their characteristics despite many passages in cell lines. Tooth specific proteins enable us to identify the differentiation state of cells and knowledge is increasing about dentine inducing Bone Morphogenetic Proteins (BMPs) and Transforming Growth Factor-Beta (TGF-β). Many of these bioactive molecules are expressed in the dental tissues during odontogenesis and systemically play a role in signalling cellular processes during health and disease (52).

Advances in material and biological sciences should offer at least two therapeutic approaches (5). Firstly, the development of dental adhesives for pulp capping procedures should offer greater success with pulpotomies. With diseased tissue removed, the response of the remaining pulp should be more favourable. Secondly are the recent advances in the field of BMPs. These growth factors have proven to be inductive for bone and dentine (5). The demonstration that reparative dentine can be induced by direct or indirect contact with a biologic agent, and its thickness determined by dose, elevates pulp therapy to a new level (5). Someday we might treat a pulpotomised permanent molar with growth factors and predictably induce sound dentine bridges, leaving the remaining pulpal tissue completely enclosed in healthy tissue, eliminating the need for root canal therapy.
Recombinant human BMP-2, -4 and -7 has been shown to induce reparative dentine formation in experimental models of large direct pulp exposures in permanent teeth (53–55). New reparative dentine replaces the stimulating agents applied directly to the partially amputated pulp. This new tissue forms contiguous with, and not at the expense of, the remaining vital pulp tissue. This permits the induction of a predetermined and controlled amount of reparative dentine.

These results provide experimental support for the potential of biologic agents to stimulate reactionary dentine formation (53–55). Further development of proper carrier vehicles that will optimise clinical handling characteristics and delivery will add to further possible uses (53). In the future, pulpotomies for primary and permanent teeth may be handled in exactly the same way (5).

Pulpal innervation has traditionally been regarded to serve a purely nociceptive function. This concept is being challenged by increasing evidence that sensory nerves also serve important effector functions (56). Rodd and Bioissonade (56) showed a canine-induced dynamic increase in neural density in both primary and young permanent teeth and speculated that this may be critical for the regulation of pulpal inflammation and healing.

In endodontics, it has been proposed that the laser has an application in direct pulp therapy as well as root-end preparation and canal disinfection (57). Montz et al (57) used a CO2 laser at 1 W to irradiate non-inflamed pulps for 0.1 sec pulses at 1 sec intervals until exposed pulps were completely sealed. A hard setting calcium hydroxide cement and a glass ionomer restoration were then placed over the pulp. In the control group, a hard setting calcium hydroxide cement and a glass ionomer restoration were placed directly on the pulp without prior laser treatment. At 12 months, laser Doppler flowmetry assessed 89% of the laser treated cases to be vital as compared to 68% of the non-laser treated cases.

The amount of data that has been accumulated over the years on the biology and the clinical behaviour of the dental pulp are impressive (58). Yet there are considerable gaps in our knowledge of the defence and repair mechanisms of the dentine-pulp complex that future research should address.

Limitations

Research data on vital pulp therapy can be misleading and confusing (13). In categorising success and failure of vital pulp therapy, many more factors need to be considered than just the presence or absence of microorganisms (13). In addition to the presence of microorganisms, degrees of inflammatory response and dentine bridges, one must differentiate between true tissue necrosis and mummification; recognise the potential for washout of microorganisms, leukocytes, and loss of extruded pulp tissue elements; the influence of impact of particles of pulp therapy agents and dentine chips; the control of haemorrhage and embolisation of particles of pulp therapy agents; the size of the pulp exposure; the final location of the dentine bridge; and the quality of its formation (13). A distinction between failure of the pulp therapy and failure of the overlying restorations must also be made.

While there are indications for vital pulp therapy in young permanent molars, it must be remembered that ultimately, none of these procedures ensure the success of complete root canal therapy (15). In the mature permanent tooth, pulpectomy and root filling are the preferred treatment (17). This is because firstly, pulpectomy with a subsequent root filling has a high success rate (90–96%), and secondly, with the wound placed apically, inflammatory responses to infection can radiographically be detected early. When vital pulp therapy has been performed, problems may develop later and periodic monitoring is essential (15).

Generally, partial pulpotomy of cariously exposed permanent teeth, most often with calcium hydroxide, serves to maintain the vitality of the remaining pulp tissue until apicogenesis is complete (5). Once the apex is closed, root canal treatment is performed. This practice is based on anecdotal evidence that calcium hydroxide will precipitate dystrophic calcification in the canals and prevent endodontic treatment later, if needed (5). This obliteration of the root canals results in diminished blood supply, which could lead to pulp necrosis (35). This relates to full coronal pulpotomy where too little pulp tissue is confined with too much calcium hydroxide. With partial pulpotomy, where the objective is to remove only infected tissue, dentine bridging is stimulated in the coronal area and canals are free of dystrophic calcification (5). Stanley (36) proposes that the negative side effects of inorganic calcium hydroxide, such as dystrophic calcification, have been eliminated in the lower pH, hard-setting, commercial calcium hydroxide preparations.

Success

A limit to determining success of vital pulp therapy in permanent teeth is the ability to recall patients regularly to determine success. Although histologic success cannot be determined, clinical success is judged by the absence of any clinical or radiographic signs of pathosis and the presence of continued root development in teeth with incompletely formed roots (22). Success rates decrease with time. Barthel et al (14) showed a success rate of 37% at 5 years, which dropped to 13% at 10 years. Canal calcification, internal resorption and/or pulpal necrosis are potential sequelae of vital pulp therapy and careful monitoring is needed to diagnose these potential problems as early as possible.

Age

The patient's age may be another limiting factor in the success of vital pulp therapy in cariously exposed permanent teeth. In older patients, the histological state of the pulp may affect its ability to overcome an insult. Pulpal vasculature constantly undergoes morphological changes incident to various conditions such as growth, maturation and inflammation. Over time, owing to the apposition of secondary and tertiary dentine, this leads to the pulp cavity becoming narrower and the vasculature within undergoes a reconstruction (12). The resistance of the pulp becomes reduced with age (12). This becomes significant when one considers that the vascular reaction to inflammation is considered a protective mechanism of the pulp against invading factors (12). Horssted et al (59) demonstrated a significantly lower tooth survival rate in the older age group (50–79 years of age) over longer observation periods. From the clinical point of view, the vitality of the dental pulp of an aged person appears to be weaker than that of a young person (12). Mass and Zilberman (35) achieved a 91.4% success rate in a sample limited to children, teenagers and young adults and suggested that vital pulp therapy of cariously exposed permanent teeth be limited to children or young adults. Chronological age does not always reflect physiological age, and neither one should be seen as an absolute contraindication for vital pulp therapy in permanent teeth (26).

Controversy exists as to whether the pulp should be re-entered after the completion of root development in the pulpotomised tooth (22). Root canal therapy is recommended because of the chance that calcification would render the canals non-negotiable at a future date. Recent research has shown that with good case selection, a gentle, aseptic technique and careful placement of calcium hydroxide onto the pulp, progressive calcification is an infrequent sequela of pulpotomy (22).
Size Of Exposure

The size of the carious pulpal exposure can influence the prognosis for vital pulp therapy. This is because generally the larger the exposure, the greater the bacterial penetration of the pulp (35). Also, the larger the exposure, the harder it is to seal. Long-term success is certainly less predictable with large carious exposures than with small carious exposures (60). It has been proposed that vital pulp therapy be limited to exposures less than 2 mm in diameter (35).

State Of The Pulp

The inflammatory status of the cariously exposed pulp is a major determining factor in the success of vital pulp therapy. The success of vital pulp therapy is dependent upon the ability of the pulp to recover from its inflammatory state (1). Ideally, the status of the pulp should be known before therapy is started (39). The diagnosis of pulpal inflammatory status is based on subjective signs and symptoms, which have been shown to be inconsistent, unreliable and do not correlate well with actual histologic data (19, 20). Mass and Zilberman (35) proposed that an ultimate pulp diagnosis be made immediately after removing 2-3 mm of the pulp during a partial pulpotomy procedure. A continuously bleeding pulp wound would contraindicate vital pulp therapy. A success rate of 91.4% was achieved at a minimum of 12 months following partial pulpotomy in cariously exposed young permanent molars using this criteria (35). Mass et al (29) proposed that due to the difficulty in accurately diagnosing pulp status, vital pulp therapy in cariously exposed young, vital permanent teeth should only be carried out when there are no symptoms at all. Because there is no single reliable technique of determining pulp status clinically, a careful interpretation of multiple tests is the most reliable method (26).

In general, clinicians agree that radiographically demonstrable periapical involvement is always associated with total pulp necrosis or irreversible pulpsitis (22). The first recommended treatment in irreversible pulpal pathological conditions is root canal therapy or extraction. Çalıkan (2) showed that this irreversible pulpsitis may be limited to the coronal pulp, because vitality of the radicular pulp and resolution of radiographic periapical involvement after full coronal pulpotomy with calcium hydroxide dressing was achieved. The dangers of dystrophic calcification of the root canals and prevention of later root canal treatment, if necessary, must be considered with use of this technique.

Extra-Pulpal Blood Clot

Stanley (36) concluded that the control of bleeding and the contact of calcium hydroxide with pulp tissue seems to have an influence on the success of the procedure. Matsuo et al (38) reported a significantly higher incidence of failures when the pulps showed heavy bleeding during the procedure, compared with moderate or poor bleeding. Excessive bleeding usually indicates a hyperaemic pulp with little chance of recovery (60). If clinical success is expected, bleeding from exposed pulp tissue should be minimal and stop soon after the exposure (60). Persistence of the coagulum-clot has been demonstrated as detrimental to pulp healing (61). The blood clot or its degradation products may interfere with healing and act as a bacterial substrate, as well as preventing the action of the capping material on the pulp (26). It must be removed before any definitive seal is placed. The presence of dentine chip fragments, dead cells, bacteria and restorative material particles in the wound site will also be detrimental to pulp healing (61). Five percent sodium hypochlorite solution will chemically amputate the blood coagulum at the exposure-pulp interface, arrest pulp haemorrhage and will remove most dentine chips, bacteria and damaged pulp cells (61). Most importantly, sodium hypochlorite will not cause damage to normal underlying tissues (61).

Choice Of Capping Material

The choice of a capping material can influence the success of vital pulp therapy (35). Calcium hydroxide has been the gold standard for direct pulp capping since the 1930s (61). This opinion has been based on the premise that calcium hydroxide was unique in its ability to stimulate dentine bridge formation. More recently it has been shown that many materials are biologically compatible against exposed pulps and permit an environment that is conducive to dentine bridge formation (16, 61, 62). The ideal medicament for pulp dressing after pulpotomy should be non-toxic, and possess anti-inflammatory activity and an anti-inflammatory potential to control pre-existing inflammatory states and surgically induced inflammation (34). Currently available agents possess two of these three requirements but none include all three. Cox and associates (62) investigated the biocompatibility of silicate cement, zinc phosphate cement, amalgam and composites on monkey pulps, with and without an additional surface seal of zinc oxide-eugenol. Their results indicate that the healing of dental pulp exposures is not dependent on the type of pulp capping material, but is related to the capacity of these materials to prevent bacterial leakage. Even inert materials such as Teflon®, show pulpal healing when used as a direct pulp capping material, as long as bacterial contamination is eliminated (18).

Mineral trioxide aggregate (MTA) has been shown to prevent dye and bacterial leakage and has a high level of biocompatibility (63, 64). Pitt Ford et al (65) used MTA as a pulp capping material on 12 mechanically exposed pulps of monkeys and all but one showed pulpal healing and dentine bridge formation adjacent to the pulp. Torabinejad and Chivian (63) speculate this is due to MTA’s sealing ability, biocompatibility, alkalinity, or other properties associated with this material. Based on these results, MTA has been considered as a suitable pulp capping material (63-65).

Anti-inflammatory agents have been examined but to date all are far too toxic or ineffective (34). Anti-inflammatory compounds used in general medicine, such as Tetrandrine, have been studied and whilst they have shown less inflammation than other anti-inflammatory agents such as corticosteroids, no long-term data exist (34). Corticosteroids have been tried and have shown reasonable short-term success, but again, no favourable long-term results exist (34).

Total etch techniques with resin bonding have shown mixed results (66-68). Short-term experiments have shown that modern, resin-based composite systems may be as effective as calcium hydroxide (67). Total cavity etching with 10% phosphoric acid seems to be safe for the exposed pulp, but 35% phosphoric acid may be disastrous (66-68). The cytotoxicity of the resin-based composites and the temperature rise during polymerisation seems not to be of concern, but microleakage, sensitisation and allergic reactions may pose problems (67). Pulp capping with resin-based composites is promising but long-term research is mandatory before the method can be recommended (66, 67).

Ledermix® (Lederle Pharmaceuticals, Cyanamid GmbH, Wolfbrathausen, Germany), a combination of an antibiotic and a corticosteroid, has been proposed as a pulp placement material (70). It was recommended that Ledermix be placed directly on the radicular pulp following full coronal pulpotomy of traumatically or cariously exposed vital teeth (70). This proposal was based on evidence that Ledermix will not induce a calcific bridge but will allow
dentinogenesis to continue around the radicular pulp. This is a temporary procedure to aid in apexogenesis before root canal treatment is begun. Ledermix cement\(^\text{\textregistered}\), a compound containing zinc oxide, eugenol and calcium hydroxide, as well as an antibiotic and corticosteroid, has shown some promise as a pulp capping material (71, 72). Concern has been expressed though, that the corticosteroid agent, triamcinolone, may suppress local repair as well as inflammation (73). Hume and Testa-Kenney (71) showed that 70% of the steroid is released in the first 24 hours after direct placement on the pulp and that during this period, steroid concentration is at a level that would be expected to reduce local inflammation and local repair. However, this inhibitory effect is likely to have little effect on long-term healing as the steroid concentration drops to below an effective inhibitory concentration after 24 hours (71).

Calcium hydroxide, which accelerates the deposition of a dentine bridge and possesses antibacterial properties, is the material of choice (74). Schröder (75) proposed that the firm, limiting necrosis caused by calcium hydroxide irritates the pulp, which stimulates the pulpal cells to defend and repair, and the firm necrosis may also induce minerals from the tissue fluid to calcify. Vesel (76) suggests that the necrotic layer presents a surface to which the pulp cells attach and polarise, and then begin to express their odontoblastic potential. Calcium hydroxide is generally used in one of three forms: as a powder, as a paste mixed with saline or aqueous methylcellulose, or incorporation into a setting cement. Kirk et al (77) showed that calcium hydroxide in paste form produces reparative dentine sooner than in cement form, but proposed that the cement form may be a more effective physical barrier to bacterial ingress than the softer, porous paste form.

Of the many calcium hydroxide cements, Dycal\(^\text{\textregistered}\) (L.D. Caulk, Dentsply Int. Inc., Milford, DE, USA), and Life\(^\text{\textregistered}\) (Kerr Manuf. Co., Romulus, MI, USA), have been shown to be satisfactory and associated with dentine bridge formation across pulp exposures (76, 78-80). Heys et al (74) showed that when bacterial microleakage occurred around an unlined amalgam restoration, the complex setting calcium hydroxide formulations such as Dycal and Life, were associated with a higher incidence of pulpal healing, when compared with the non-setting calcium hydroxide formulations. The non-setting, simple calcium hydroxide formulations were more consistent in promoting dentine bridge formation where bacterial microleakage was controlled (74). A dentine bridge generally forms directly against the capping material when calcium hydroxide cements such as Dycal and Life are used (79, 80), but forms at a distance from the capping material with a necrotic layer between the two, when the paste or powder formulations are used (78). This may be due to the increase in inflammation that is produced by the more alkaline paste and powder formulations. Stanley (36) proposes that the negative side effects of inorganic calcium hydroxide such as dystrophic calcification have been eliminated in the lower pH, hard-setting, commercial calcium hydroxide cements and recommends their use over the powder or paste preparations.

**Bacterial Contamination**

Bacterial contamination is the main factor that determines the prognosis of vital pulp therapy (26). Bacterial contamination of the pulp can occur directly through caries or through exposure to salivary contamination. Following caries removal and the aseptic removal of superficial pulp, it is important to control bacterial contamination of the pulp by microleakage at the tooth restoration interface (81). Cox et al (62) showed that three weeks after restorations were placed over a pulp exposure, pulp reorganisation and hard tissue repair occurred when a surface seal preventing marginal leakage was placed over the restoration. Without this surface seal, severe pulpal inflammation and necrosis were noted, with bacteria often found at the pulp-restoration interface. Barthel et al (14) found that placement of a definitive restoration within the first two days after carious pulp exposure contributed significantly to the survival rate of these teeth. Partial pulpotomy improves pulp sealing by creating a retentive cavity for the capping and restorative material (35).

The long-term prognosis and the ability to restore a tooth are the over-riding factors when assessing whether vital pulp therapy should be undertaken (15). The amount of crown destruction usually associated with a pulp exposure in an immature molar means that the tooth involved would require significant restorative maintenance in the long term. Extraction and orthodontic treatment may be preferable.

**Conclusion**

It is beneficial to preserve the vitality and health of an exposed pulp rather than replace it with a root filling material following pulp exposure. A vital, functioning pulp is capable of initiating several defensive mechanisms to protect the body from bacterial invasion (14). Pulp exposure without involvement of microorganisms can clearly be overcome by repair mechanisms, such as dentinal bridging. There is no consensus on the survival rate of formerly exposed pulps. Observation time, judgement criteria, pulpotomy/pulp capping technique and most importantly, pulpal status at the time of treatment, vary to a great extent among the studies. This is shown in the success rate of vital pulp therapy of cariously exposed permanent teeth varying between 13% and 93% (10, 14). Among the methods for preservation of a cariously exposed pulp, partial pulpotomy has yielded the highest success rate in young teeth (3, 10).

From the clinical point of view, the vitality of the dental pulp of an aged person appears to be weaker than that of a young person (12). In the future, more efficient wound dressings containing perhaps growth factors, together with improved local antimicrobial and anti-inflammatory materials will combine with the improved sealing ability of restorative materials, to preserve the vitality of the canally exposed pulp in permanent teeth.

Major limitations in the success of vital pulp therapy in cariously exposed permanent teeth exist. The lack of predictability and long-term success greatly influence decision-making. The decision-making itself is unreliable primarily due to the difficulty of accurately diagnosing the ability of the pulp to repair.

The success demonstrated in various clinical studies justifies recommending partial pulpotomy as a treatment option for asymptomatic, cariously exposed, young permanent molars (29). However, due to the lack of long-term success, these cases have to be monitored on a regular basis to avoid unnoticed necrosis of the pulp with invasion of bacteria into the root canal system.

**References**


