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Review Article

Vital Pulp Therapy

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INTRODUCTION

Dental caries is one of the greatest challenges to the integrity of the developing tooth. It can result in irreversible pulpal damage, eventually causing necrosis of the pulpal tissues and arrested development of the tooth root. Ultimately, abnormal root development will impact the long-term prognosis of the tooth.[1,2,3,4] The aim of vital pulp therapy is to maintain pulp viability by preventing and eliminating bacteria from the dentin-pulp complex and to establish an environment in which apexogenesis can occur.

Exposure of dental pulps can happen as a result of caries, trauma or cavity preparation. A multitude of harmful elements, alone or in combination, may under clinical conditions cause adverse reactions in the dentalpulp.[5]

These effects are the result of inflammation and associated tissue destruction which can be devastating for the pulp. Infection and inflammation in the periapical tissue frequently follow such an event, termed pulpal necrosis. The morbidity associated with treating pulp exposures is consequential, often requiring either extraction or root canal therapy.[6] Both the loss of the tooth and its replacement, or endodontic treatment and tooth restoration, involve multiple appointments and considerable expense. An alternative procedure to extraction or endodontic therapy is pulp capping.

IMPORTANCE OF THE VITAL TOOTH AND PRACTICE OF ENDODONTICS

The disease unique to endodontics is apical periodontitis. Apical periodontitis is, as the name implies, inflammation of the periodontal ligament around the apical region of the tooth. [7] In order to prevent or eliminate a disease we need to know its cause. In simple terms apical periodontitis is caused by bacteria in the root canal system. Generally, bacteria will be present in necrotic tissue. Thus in clinical terms a necrotic infected pulp is required for apical periodontitis to be present. Conversely, if the pulp is vital there should be few or no bacteria present in the root pulp space and thus apical periodontitis should not be present. In this type of case vital pulp therapy is effective mode of treatment. Therefore, the purpose of this study is to preserve the vital pulp to prevent apical periodontits. During the last decade, exceptional progress was made in the field of vital pulp therapy. Materials used in the past were both biologic and non-biologic. [9] Since the concept of indirect pulp capping was first documented in the eighteenth century by Pierre Fauchard, clinicians have recognized the innate capacity of the pulp tissue to initiate repair after injury from trauma, caries, or mechanical exposure. [10] The first documented instance of vital pulp therapy is attributed to Philip Pfaff, who placed gold foil against an exposed pulp with the intention to promote pulpal healing. [11]

One of the most important issues in vital pulp therapy is the status of the pulp tissue. The traditional school of thought is that vital pulp therapy should only be carried out in teeth with signs and symptoms of reversible pulpitis. [12] The problem is how we can

accurately assess the status of the pulp. The clinical signs and symptoms such as sensibility and pain testing do not precisely reflect the pulp condition. [13,14,15] Furthermore, several studies have reported successful treatment outcome in vital teeth with cariously exposed pulp with signs and symptoms of irreversible pulpitis and periapical lesions. [16,17,18,19] The degree of pulpal bleeding may be a better indicator of pulpal inflammatory status. [16] Increased bleeding on exposure site that is difficult to stop, suggests that the inflammatory response extends deeper into the pulp tissue and the treatment procedure should be modified, for example by shifting from direct pulp capping to partial pulpotomy.

There is a long-held perception that pulp exposures in a carious field have an unfavourable prognosis and that more aggressive treatment, such as pulpotomy or pulpectomy, should be considered.[20,21] These strategies are based on traditional treatment protocols and materials that did not provide a consistently suitable environment for pulpal repair and reparative bridge formation. The challenge is to identify a reliable pulp capping or pulpotomy agent and a suitable delivery technique. Emphasis has shifted from the "doomed "organ concept of an exposed pulp to one of hope and recovery. The era of vital pulp therapy has been greatly enhanced with the introduction of various pulp capping materials [22] The introduction of mineral trioxide aggregate (MTA) in 1993 had opened a new frontier in vital pulp therapy and has changed the perception that pulp capping in cariously exposed teeth is unpredictable and therefore contraindicated. Today materials that are better than MTA have been developed. These include Biodentine and MTA Plus. Apart from these new materials, lasers today play an important role in vital pulp therapy.

The outcome of vital pulp therapy will depend on the age of the patient, the size of the pulp, bacterial contamination, the pulp capping material, and the quality of final restoration. The pulp status must be determined carefully, establishing a differential diagnosis using multiple tests.

HISTORICAL BACKGROUND OF PULP THERAPY

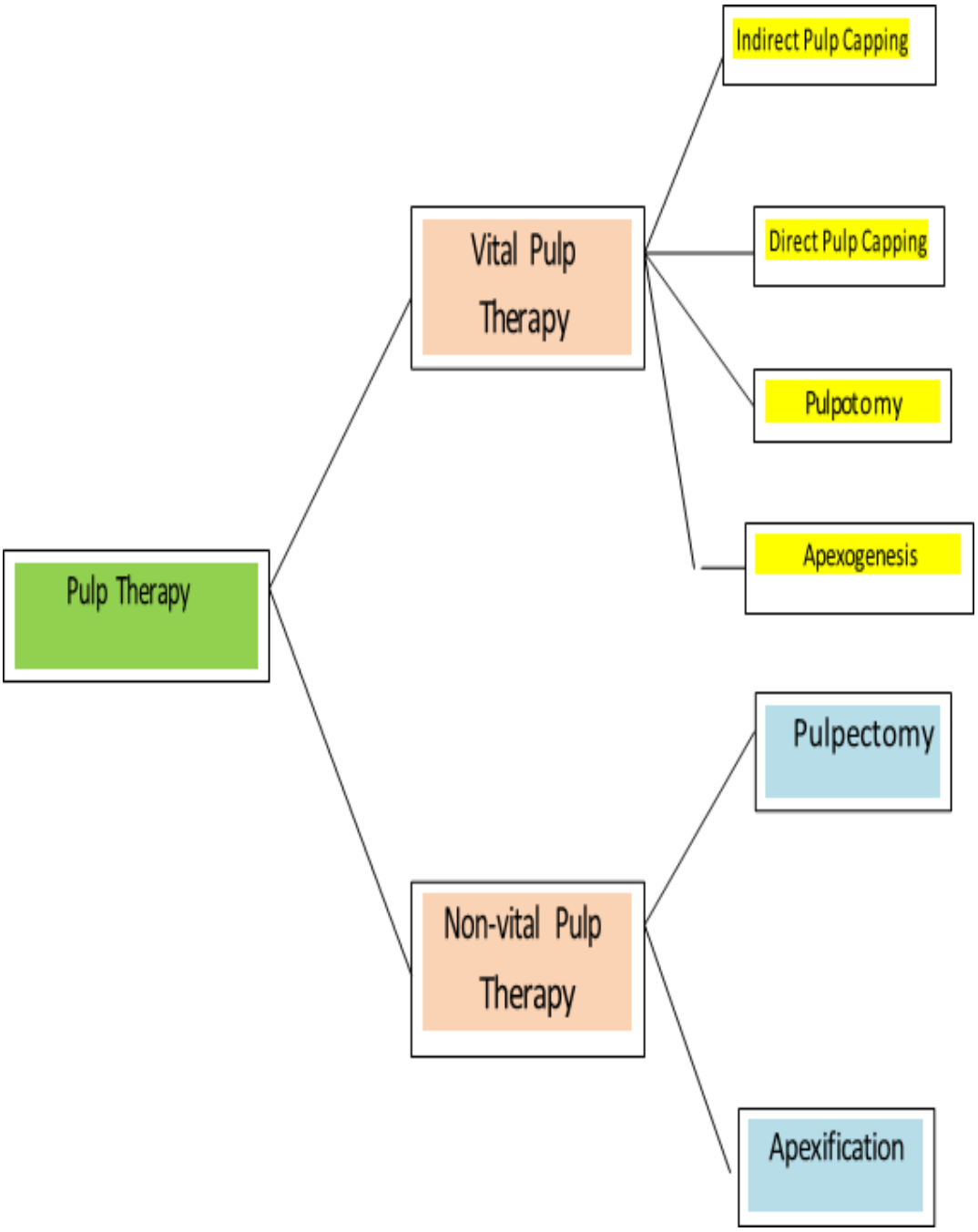
The first method of capping exposed pulps, using gold foils, was described by Pfaff in 1756. Thereafter, numerous agents for direct pulp capping have been recommended. Until the end of the 19th century, most materials were used empirically with the idea that the pulp tissue must be irritated by etching or cauterization to heal. Later, more attention was drawn to disinfecting agents, because it became obvious that microorganisms were the reason for pulp inflammation - but these agents were cytotoxic. Since insufficient or inappropriate diagnoses were made before treatment, even necrotic pulps were capped. The first scientific clinical study to compare different capping materials was made by Dätwyler in 1921, where upon zinc oxide-eugenol showed the best results. One year later, Rebel performed the first animal experiments with disastrous results, so he regarded the exposed pulp as a doomed organ[14]

In 1920 Hermann, introduced calcium hydroxide for root canal fillings. Between 1928 and 1930 he studied the reaction of vital pulp tissue to calcium hydroxide to prove that it was a biocompatible material. Since then, calcium hydroxide has been recommended by several authors for direct pulp capping, but it took until the middle of 20th century until it was regarded as the standard of care[13]

PRINCIPLES OF VITAL PULP THERAPY

The fundamental objective of vital pulp therapy is the maintenance of the treated tooth as a dental arch unit. The tooth acts as a natural space maintainer, fulfilling an esthetic and functional role. Whenever possible, vitality should be sustained. This is especially important in young teeth with incompletely formed apices because pulp vitality and the integrity of Hertwig's root sheath are responsible for continued apical development.[15]

CLASSIFICATION OF PULP THERAPY



PULP CAPPING MATERIALS

A plethora of materials have been used as potential pulp-capping materials. The healing of a pulpal exposure is not dependent on the effect of a particular type of material, but on the reaction to the capping agent and the surface sealing to prevent bacterial leakage. However, although pulpal repair is possible with a range of capping agents, an important proviso is that the environment should not only be free from infection, but the capping agent should, subsequently, become relatively innocuous. Therefore, the predictability and potential success of pulp capping is influenced by the capping agent chosen.[18]

Various Materials Used In Pulp Therapy Are as follows

1. Calcium Hydroxide.
2. Zinc Oxide Eugenol.
3. Corticosteroids and Antibiotics
4. Polycarboxylate Cements.
5. Inert materials
6. Collagen
7. Bonding Agents
8. Calcium Phosphates
9. Hydroxyapetite
10. Lasers
11. Glass Ionomer/Resin modified Glass Ionomer
12. Mineral Trioxide Aggregate

13. MTYA1-Ca
14. Growth factors
15. Bone Morphogenic protein
16. Recombinant Insulin Like Growth Factor
17. Other Growth Factors
18. Bone Sialoprotein
19. Biodentin
20. Enzymes
21. Simavastatin
22. Stem cells
23. Propolis (Russian Penicillin)
24. Novel Endodontic Cements
25. Emdogain
26. Odontogenic Ameloblast Associated Protein
27. Endo sequence root repair materials
28. Castor oil bean cement
29. Theracal.

CALCIUM HYDROXIDE

Calcium hydroxide was introduced to the dental profession in 1921 by Hermann and has been considered the 'gold standard' of direct pulp capping materials for several decades because of their property of stimulating sclerotic and reparative dentin formation and protecting the pulp against thermal stimuli and antibacterial action. Calcium hydroxide serves as protective barrier for pulpal tissues not only by blocking patent dentinal tubules but also by neutralizing the attack of inorganic acids and their leached products from certain cements and filling materials. [20]

ZINC OXIDE EUGENOL CEMENT

Tronstad and Mjö r stated that ZOE cement is more beneficial for inflamed and exposed pulp.Zinc oxide eugenol was introduced as a pulp capping agent as it was known toexhibit sedative and palliative effects.[24]
Gonzales- Lara et al, conducted a study wherein, pulpotomy was carried out using ZOE witha follow-up period of 24 months, it appeared that over the remnants of pulp, a layer of dentin like tissue had formed with no stained bacteria observed in any of the samples. However failed pulpotomy case was taken and a layer of immature cells probably primitive dentine-like cells were seen in that case too suggestive of reparative dentin.[25]

CORTICOSTEROIDS AND ANTIBIOTICS

Corticosteroids like hydrocortisone, Cleocin, cortisone, Ledermix (calcium hydroxide plus prednisolone), penicillin, neomycin and Keflin (cephalothin sodium) along with calcium hydroxide was used for pulp capping with the thought of reducing or preventing pulp inflammation. Gardner, et al., found that vancomycin, in combination with calcium hydroxide was somewhat more effective than calcium hydroxide used alone and stimulated a more regular reparative dentin bridge.
Watts and Paterson cautioned that anti-inflammatory compounds should not be used inpatients at risk from bacteremia[26]

POLYCARBOXYLATE CEMENT

When zinc polycarboxylate cement is used the bond occurs between carboxylic acid groups in the liquid polyacrylic acid and the calcium in the tooth structure. The powder of the cement is essentially zinc oxide .This cement continues to maintain some presence in the marketplace because it offers good biocompatibility with the pulp tissue.[27] However McWalter, G et al., found that it lacks an antibacterial effect and calcific bridge formation.

INERT MATERIALS

Bhaskar SH et al., and Heys DR et al., investigated isobutyl cyanoacrylate and tricalcium phosphate ceramic as direct pulp capping materials. Although pulpal response in the form of reduced inflammation and unpredictable dentin bridging were found, but none of these materials have been promoted to the dental profession as a viable technique.[28]

COLLAGEN

Collagen-calcium phosphate gel paste and dentin formation peripherally to pulp and at the tissue-paste interface, but no tissue infiltration of the space occupied by the paste. The dentin bridge formation and cellular proliferation coronal to the formed bridge took place as two independent processes at essentially the same time.
Dick HM and Carmichael DJ reported that collagen fibers are less irritating than Ca (OH)2 and promotes mineralisation but does not help in thick dentin bridge formation.

BONDING AGENTS

According to Miyakoshi et al., 4-META-MMA-TBB adhesives and hybridizing dentin bonding agents provide superior adhesion to peripheral hard tissues and effective seal against micro leakage. But they have poor outcome due to its cytotoxic effect and absenceof calcific bridge formation .[30]

CALCIUM PHOSPHATE

Calcium phosphate cement was suggested as viable alternative because of its good biocompatibility, superior compressive strength and its transformation into hydroxyapatite over time.
Yoshimine et al., demonstrated that in contrast to calcium hydroxide, tetracalcium phosphate cement induced bridge formation with no superficial tissue necrosis and significant absence of pulp inflammation.[31]

HYDROXYAPATITE

It is the most thermodynamically stable of the synthetic calcium phosphate ceramics. It has good biocompatibility with neutral pH -7.0. It can be used as scaffolding for the newly formed mineralized tissue.

LASERS

Melcer et al., suggested between the years 1985 and 1987 that the carbon dioxide (CO2) (1W) laser used for direct pulp capping. Yasuda Y, et al., did a study to examine the effect of CO2 laser irradiation on mineralization in dental pulp cells in rats and the results suggested that CO2 laser irradiation stimulated mineralization in dental pulp cells.

Neodymium-doped yttrium-aluminium-garnet laser emits an infrared beam at a wavelength of 1064nm can be of therapeutic benefit for direct pulp capping and pulpotomy in clinical practice.[32]

GLASS IONOMER/RESIN MODIFIED GLASS IONOMER

Glass ionomer also provides an excellent bacterial seal and good biocompatibility when used in close approximation but not in direct contact with the pulp. RMGIC as direct pulp capping agent exhibited chronic inflammation and lack of dentin bridge formation; whereas the calcium hydroxide control groups showed significantlybetter pulpal healing.[32]

MINERAL TRIOXIDE AGGREGATE (MTA)

MTA was introduced by Torabinejad in early 1900s.
Composition:-
Mixture of 3 powder ingredients:-
Portland Cement (75%)
Bismuth Oxide (20%)
Gypsum (5%) MTA includes:

- Tricalcium silicate.
- Tricalcium aluminate.
- Tricalcium oxide.
- Silicate oxide.
- Bismuth oxide.

It produces more dentinal bridging with superior structural integrity than Ca(OH)2 in a shorter time span with significantly lesser inflammation. It has superior ability to resist for further penetration of bacteria than calcium hydroxide. Has significant antimicrobial property on some of the facultative bacteria.

MTA is highly biocompatible with pulpal tissue and it is hydrophilic as it sets in presence of moisture. Set MTA has pH of 12.5 and may induce dentinogenesis. The presence of blood has little impact on degree of leakage of MTA.[33]

TYPES OF MTA

Two types of MTA: White and Gray MTA.

The handling characteristics of two paste formulation of MTA is different from calcium hydroxide which is easier for many operators.

MTA as a pulp capping material in certain clinical trials have shown to have comparable results to calcium hydroxide, however, certain short term studies with a clinical follow up period of two years suggested MTA to have superior clinical results than calcium hydroxide (Hilton et al, 2013).

MTYA1-Ca

Atsuko Niinuma developed resinous direct pulp capping agent containing calcium hydroxide. The powder composed of 89.0% microfiller, 10.0% calcium hydroxide and 1.0%benzoyl peroxide and was mixed with liquid (67.5% triethyleneglycol dimethacrylate, 30.0% glyceryl methacrylate, 1.0% o-methacryloyl tyrosine amide, 1.0% dimethyl amino ethyl methacrylate and 0.5% camphorquinone).

MTYA1-Ca developed dentine bridge formation without formation of a necrotic layer, revealed to have good physical properties, and was not inferior to Dycal histopathologically. Therefore, it is suggested that the newly developed material, MTYA1-Ca promises to be a good direct pulp capping material.[33]

GROWTH FACTORS

Growth factors regulate growth and development and induce wound healing and tissue regeneration.

1. Bone morphogenic Protein

BMP belongs to super family transforming growth factor beta (TGF-b). TGF b is apotent modulator of tissue repair in different situations. BMP-2, 4, and 7 plays a role in the differentiation of adult pulp cells into odontoblasts during pulpal healing. Lianjia et al., found that BMPs are responsible for dentinogenesis, inducing non differentiated mesenchymal cells from the pulp to form odontoblast-like cells, obtaining osteodentin and tubular dentin deposition, when used as direct protectors.[34]

2. Recombinant insulin Like Growth Factor

Lovschall H, et al., evaluated recombinant insulin like growth factor-I (rhIGF-I) in rat molars and concluded that dentin bridge formation was equal to dycal after 28 days .

3. Other Growth Factors

Hu et al., evaluated the various growth factors like epidermal growth factor, basic fibroblast growth factor, insulin-like growth factor II, platelet-derived growth factor-BB, TGF-b 1in rat molars and concluded that only TGF-b 1-enhances reparative dentin formation.[35]

BONESIALOPROTEIN

According to Goldberg M et al., Bone sialoprotein (BSP) was the most efficient bioactive molecule, which induced homogeneous and well mineralized reparative dentin. Both BSP and BMP-7 were superior to calcium hydroxide in their mineralization inducing properties

BIODENTINE

Biodentine is new bioactive cement with dentin like mechanical properties and can be used as dentin substitute. It has a positive effect on vital pulp cells and stimulates tertiary dentin formation .[39]

ENZYMES

Heme oxygenase-1(HO) is the rate limiting enzyme in heme catabolism. Odontoblasts and oxidatively

stressed dental pulp cells express HO-1, indicates that the pulp might respond to oxidative stress at the molecular level. HO-1 induction protects against hypoxic stress and nitric oxide- mediated cytotoxicity. It has been reported that HO-1 might play a cytoprotective role against pro inflammatory cytokines and nitric oxide in human pulp cells. In addition, bismuth oxide containing Portland cement (BPC) induced HO-1 expression in dental pulp cells plays a protective role against the cytotoxic effects of BPC .

SIMVASTATIN

It is a 3-hydroxy-3-methylglutaryl coenzyme, a reductase inhibitor and first line drug for hyperlipidemia. Statin improves the osteoblast function via the BMP-2 pathway and suppresses osteoclast function, resulting in enhanced bone formation. Therefore, statin might improve the function of odontoblasts, thus leading to improved dentin formation. Statin is known to induce angiogenesis and increase neuronal cells, indicating the possible effectiveness of statin in pulp regeneration along with dentin regeneration. It has an anti-inflammatory effect in various tissues, so it is considered as an ideal active ingredient in pulp capping material to accelerate reparative dentin formation .[40]

STEM CELLS

Dental pulp stem cells (DPSCs) and Stem cells from Human Exfoliated Deciduous Teeth (SHED) have been identified as a novel population of stem cells that have the capacity of self-renewel and multi lineage differentiation. Nakamura S et al., used mesenchymal stem cells for clinical application in tissue engineering and regenerative medicine. In this study, they compared the proliferation and stem cell marker of SHED, DSPCs and Bone Marrow Derived Mesenchymal Stem Cells (BMMSCs). In addition, gene expression profile of DSPCs and SHED were analyzed by using DNA microarray. They concluded that SHED has got significantly higher proliferation rate than that of DSPCs and BMMSCs and this could be a desirable option as a cell source for therapeuticapplications.[41]

PROPOLIS (RUSSIAN PENICILLIN)

It contains flavonoids, phenolics, iron, zinc and other various aromatic compounds . Parolia A, et al., compared propolis, MTA and Dycal histologically in human dental pulp and concluded that Propolis and MTA showed similar bridge formation when compared to Dycal.

NOVEL ENDODONTIC CEMENT (NEC)

NEC consists of calcium oxide, calcium phosphate, calcium carbonate, calcium silicate, calcium sulfate, and calcium chloride. Mohammad Hassan Zarrabi evaluated MTA and NEC histologically in human dental pulp and concluded that NEC induced a thicker dentinal bridge with less pulp inflammation.[43]

INDIRECT PULP CAPPING

Caries is the result of an ecologic shift within the dental biofilm to acidogenic and aciduric bacterial biofilm. So it seems that with reduction of bacterial number and activity, the ecologic and metabolic balance within the biofilm will be re-shifted, thus remineralization is promoted and the caries lesions is expected to be arrested.[44]

DEFINITION

"Indirect pulp capping (IPC) is defined as a procedure in which carious dentin closest to the pulp, is preserved to avoid pulp exposure and is covered with a biocompatible material."

It is a procedure wherein the deepest layer of the remaining affected carious dentin is covered with a layer of biocompatible material in order to prevent pulpal exposure and further trauma to the pulp.[49]

RATIONALE FOR INDIRECT PULP CAPPING

Indirect pulp capping is based on the knowledge that decalcification of the dentin precedes bacterial invasion within the dentin. This technique is predicated on removing the outer layers of the carious dentin, that contain the majority of the microorganisms, reducing the continued demineralization of the deeper dentin layers from bacterial toxins, and sealing the lesion to allow the pulp to generate reparative dentin. This treatment method is intended to protect primary odontoblasts and promote reactionary dentin formation at the pulp– dentine junction. However, some primary odontoblasts may be destroyed depending on the severity of carious involvement of the dentin-pulp complex and reparative dentin is formed in conjunction with reactionary dentin . An important function of the bioactive liningmaterial is to stimulate the odontoblasts to form reactionary and reparative dentin and promote remineralization of existing dentine, thus encouraging the dentin-pulp complex .

Fusayama and colleagues demonstrated that in **acute caries**, dentin discoloration occurred far in advance of the microorganisms, and as much as **2 mm of softened or discoloured dentin was not infected**. In a later study, Fusayama found that carious dentin actually consists of two distinct layers having different ultramicroscopic and chemical structures. The outer carious layer is irreversibly denatured, infected, and incapable of being remineralized and should be removed. The inner carious layer is **reversibly** denatured, **not infected**, and capable of being remineralized and should be preserved. The two layers canbe differentiated clinically by a solution of basic fuchsine.

STEPWISE CARIES EXCAVATION (SW)

Stepwise excavation and is done over two patient visits. The first excavation is intended to remove the

superficial necrotic, infected, and affected dentin by completely excavating the periphery of the lesion. This excavation does not excavate caries near the pulp to avoid a pulp exposure . A retentive temporary restoration is placed leaving soft, moist, discoloured dentin on the pulpal floor. The dentin is then covered with calcium hydroxide and a self-setting glass ionomer temporary filling . A resin-modified glass ionomer has also been advocated in place of the self-setting glass ionomer . SW is intended to allow remineralization of the affected dentin and formation of more tertiary dentin . The carious lesion is re-entered in 8–12 weeks by removing the temporary, and a final complete excavation is done leaving only central yellowish or greyish hard dentin in the pulpal floor . A final restoration is placed with the intention to seal the pulp from any microleakage . In two systematic reviews and meta-analysis studies , SW significantly reduced pulp exposures compared to complete excavation. [50]

PARTIAL (INCOMPLETE) CARIES EXCAVATION

The third type of caries excavation is termed partial or incomplete excavation involving one appointment removal of the peripheral decay but intentionally leaving the deepest caries in place to avoid a pulp exposure. An indirect pulp treatment is then completed . Partial excavation omits the re-entry and second excavation done in SW by sealing off the caries with a final restoration placed at the same appointment. Partial excavation attempts to shift the microbial balance within the affected dentin to promote dentin remineralization and arrest the carious lesion.

Maltz et al. published the results of one randomized controlled trial (RCT) comparing stepwise to partial excavation in 299 permanent molars in two time frames. The results of the 2- and 3-year follow-up showed significantly higher success rates of one appointment partial caries excavation compared with SW (96 % and 91 % compared with 81% and 61 % after 2 years and 3 years, respectively). Maltz et al. speculated on why stepwise excavation success was only 61 % after 3 years . The authors felt some SW patients did not return for the final excavation and restoration in 1–2 months, and the success rate in these patients was very low (13 %). Patients treated with SW that had a final excavation and permanent restoration had survival rates not statistically different from those of partial excavation (88 % with SW vs. 91 % partial excavation).[51]

Indirect pulp capping therapy can be justified by the following desirable results :[35]

1. Disinfection of residual affected dentin is more readily accomplished.
2. It eliminates the need for more difficult pulp therapy by arresting the carious process and allowing the pulp reparative process to occur.
3. Patient comfort is immediate.
4. Rampant dental decay is halted when all carious teeth are treated.

There have been many studies on IPT success. One RCT investigated the success of IPT in primary and permanent molars using a one- and two-visit approach versus complete excavation. This study involved 94 primary second molars and 60 immature permanent first molars with 50 one-visit IPTs, 49 with two-visit IPTs, and 55 done with complete excavation. The IPT base was calcium hydroxide, and the two-visit method used reinforced zinc oxide as a temporary filling over the calcium hydroxide. The primary teeth were restored with a compomer, and the permanent teeth with a bonded composite.[20,21] There were significantly less pulp exposures with both IPT methods (p = .008). The combined IPT groups after 1 year had a success of 91 out of 92 teeth (99 %) that was not statistically different than complete excavation success 41 out of 43 teeth (95 %). Another RCT evaluated IPT in primary molars without a base (bonded composite to the demineralized dentin) versus a calcium hydroxide IPT restored with a bonded composite. The 2-year follow- up of 31 teeth showed an overall success of 87 % with no statistical difference between groups. A third RCT report showed a 94 % success rate of a calcium hydroxide IPT in primary molars after 12–29 months. A longer follow-up period (>3 years) was reported in a retrospective study of 108 IPTs versus 118 formocresol pulpotomies . The IPTs had glass ionomer as a liner. The authors found that after 1 year, the 98 % IPT success was not statistically different from the 95 % pulpotomy success. After the 1–2-year time frame, IPT success was always statistically higher, and the >3-year follow-up showed that IPT success was 94 % while formocresol pulpotomy success was 70 %. All teeth were restored the dayof treatment with a steel crown, amalgam, glass ionomer, or composite. It appears from these studies and others that IPT success remains above 80 % no matter the follow-up time, the liner used, and whether it was a one-visit or a two-visit procedure.[61]

OBJECTIVES[62]

Edelman et al.(1965) stated the following objectives:

- Arresting the carious process.
- Promoting dentin sclerosis.
- Stimulating the formation of tertiary dentin.
- Remineralizing the carious dentin.

INDICATIONS

Indirect pulp capping procedure should be based on the following

- **History:**

a) Mild discomfort from chemical and thermal stimuli
b) Absence of spontaneous pain

- **Clinical examination:**

a) Large carious lesion.
b) Absence of lymphadenopathy.
c) Normal appearance of adjacent gingival.

d) Normal colour of tooth.

- **Radiographic examination**

a) Large carious lesion in close proximity to the pulp.
b) Normal lamina dura.
c) Normal periodontal ligament space
d) No periapical radiolucency.

CONTRAINDICATIONS

- **History:**

a) Sharp penetrating pain that persists after withdrawing stimulus.
b) Prolonged spontaneous pain, particularly at night.

- **Clinical examination:**

a) Excessive tooth mobility.
b) Parulis in the gingival approximating the roots of the tooth.
c) Tooth discoloration.
d) Non responsiveness to pulp testing techniques.

- **Radiographic examination**

a) Large carious lesion with apparent pulp exposure.
b) Interrupted or broken lamina dura.
c) Widened periodontal ligament space.
d) Radiolucency at the root apices or furcation areas.

- **Response to Treatment**

Sayegh found three distinct types of new dentin in response to **indirect pulp capping**:

1. cellular fibrillar dentin at 2 months post-treatment
2. presence of globular dentin during the first 3 months
3. tubular dentin in a more uniformly mineralized pattern

In the study of 30 primary and permanent teeth, Sayegh concluded that new dentin forms fastest in teeth with the thinnest dentin remaining after cavity preparation. He also found that the longer treatment times enhanced dentin formation.

Diagnosis of the type of caries influences the treatment planning for indirect pulp capping. In the **active** lesion, most of the caries-related organisms are found in the outer layers of decay, whereas the deeper decalcified layers are fairly free of bacteria. In the **arrested** lesion, the surface layers are not always contaminated, especially where the surface is hard and leathery. The deepest layers are quite sclerotic and free of microorganisms. Deep carious dentin is even more resistant to decomposition by acids and proteolysis than is normal dentin. This was especially true in arrested caries.

ONE- APPOINTMENT TECHNIQUE

The selection for one- appointment indirect treatment must be based on clinical judgment and experience with many cases in additional previously mentioned criteria.

Leung et al, and Faibourn and colleagues have been able to show a significant decrease of bacteria in deep caries lesions after being covered with calcium hydroxide or modified zinc oxide eugenol for period ranging from 1-15 months. These investigators suggests that re- entry to remove the residual minimal carious dentin after capping with calcium hydroxide may not be necessary if the final restoration maintains a seal and the tooth is asymptomatic.[47]

If clinician had to leave considerably more carious dentin owing to patient symptoms, re- entry, a more invasive vital pulp therapy techniques such as direct pulp capping or pulpotomy would be indicated.

A challenging aspect of indirect pulp capping is determining the exact boundary point where caries excavation is terminated. Therefore, the technique is based primarily on subjective criteria and the operator's skill. Further complicating the process is the presence of potential voids under the provisional restoration; during the mineralization process, these can permit dentin to lose volume during desiccation. Another drawback is the rapid reactivation of dormant lesions after restoration failure. However, in younger patients with management issues, indirect pulp capping has shown promising results for immature permanent teeth in which the apical foramina are large, the canal walls are thin, and pulp vascularization is pronounced. Indirect pulp capping has been shown to be Indirect pulp therapy an effective technique for caries and patient management in the primary dentition, but it remains controversial in permanent teeth[50].

The pulp of a tooth can be exposed due to several causes: caries, trauma or mechanical reasons, the latter typically due to a misadventure during tooth preparation. The direct pulp cap, in which a material is placed directly over the exposed pulp tissue, has been suggested as a way to promote pulp healing and generate reparative dentin. If successful, this procedure precludes the need for more invasive, more extensive and expensivetreatment. [53]

DEFINITION

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.

OBJECTIVES

- To seal the pulp against bacterial leakage.
- To maintain the vitality of underlying pulp.
- No post-treatment clinical signs or symptoms of sensitivity, pain, or swelling shouldbe evident.
- Pulp healing and reparative dentin formation should occur.
- No radiographic evidence of internal or external root resorption, periapicalradiolucency, abnormal calcification, or other pathologic changes should be evident.
- Teeth with immature roots should show continued root development and apexogenesis.

INDICATIONS

1. Exposures as a result of caries removal, tooth

preparation, trauma.
2. Pinpoint mechanical exposures surrounded with sound dentin.
3. Exposed pulp tissue should be bright red in color with slight haemorrhage that is easily controlled with dry cotton pellets applied with minimal pressure.
4. Absence of pain
5. No bleeding at the exposure site.

Frigoletto noted that **small exposures** and a **good blood supply** have the best healing potential. Although imprecise, the term ―pinpoint conveys the concept of smallness to the exposed tissue, which should have the lowest possibility of bacterial access. An empirical guideline has been to limit the technique to exposure diameters of **less than 1 mm.**[56] Stanley has determined, however, that the size of the exposure is less significant than the quality of the capping technique in avoiding contamination and mechanical trauma to the exposure site and careful application of the medicament to haemostatically controlled pulp tissue. Equally important is the quality of the temporary or permanent restoration to exclude microleakage.

CONTRAINDICATIONS

Before electing to perform a direct pulp capping, the following contraindications should be carefully considered:[57]

1. Pulp tissue, jeopardized by a long-standing exposure to oral micro-organisms and acute inflammation.
2. History of spontaneous and nocturnal toothaches.
3. Excessive tooth mobility.
4. Thickening of periodontal ligament.
5. Radiographic evidence of internal or external root resorption, furcal or periradicular degeneration.
6. Uncontrollable haemorrhage at the time of exposure.
7. Purulent or serous exudate from the exposure.

CASE SELECTION

Success with direct pulp capping is dependent on the radicular pulp being healthy and free from bacterial invasion.[60] It must rely on :

- Physical appearance of the exposed pulp tissue.
- Radiographic assessment.
- Diagnostic tests to determine pulpal status.

TECHNIQUE[66]

1. DEBRIDEMENT

It is important to remove peripheral masses of carious dentin before beginning the excavation where an exposure may occur. Because this necrotic and infected chips are invariably pushed into the exposed pulp during the last stage of removal and this debris can impede healing in the area, by causing further pulpal inflammation and encapsulation of the dentin chips. When exposure occurs, the area should be appropriately irrigated with non irritating solutions such as normal saline to keep the pulp moist.

2. HEMORRHAGE AND CLOTTING

Hemorrhage at the exposure site can be controlled with cotton pellet pressure. A blood clot must not be allowed to form after the cessation of hemorrhage from the exposure site, as it will impede pulpal healing. The capping material must directly contact pulp tissue to exert a reparative dentin bridge response. Hemolysis of erythrocytes results in an excess of hemosiderin and inflammatory cellular infiltrate, which prolongs pulpal healing.

3. CONTROL OF BLEEDING

A capping agent should never be placed against a bleeding pulp. If enough bleeding or oozing occurs under the dressing and permit the formation of a blood clot or a thick fibro- purulent membrane. This membrane will attract the element for granulation tissue replacement and will favor organization and differentiation of fibroblast and odontoblasts to create ectopic reparative dentin formation in the wrong places, such as in the cavity preparation rather than at the exposure site. While this process in taking place the clot or stagnated is subject to secondary infection. This can lead to complete loss of pulp vitality. Bleeding usually can be stopped just by washing the area with sterile saline solution and drying with paper point laid across the exposure site. In larger exposure sites cotton pellets are used. If bleeding is not arrested with sterile saline solution alone, then hydrogen peroxide may be applied.

A LARGER EXPOSURE

This type is encountered when a leathery dentin is removed which is often associated with a watery exudates or pus at the exposure site. These conditions are indicative of advanced degeneration and often of internal resorption in the pulp canal . A more rapid reduction in pulpal bleeding and attainment of hemostasis is observed when cotton pellets saturated with Hemodent. Wide opening of the pulp is especially important in cases of carious exposures with or without manifested symptoms of pulpitis. As in other wounds some pulpal bleeding is essential to wash away all debris such as food remnants, clumps of bacteria, pus from localized abscesses, necrotic tissue, and carious or non-carious dentinal fragments force into the pulp during the operations.

It was pointed out a pulp or a pulp stump that continues to bleed after 5 minutes may be indicative of pulpal disease of an irreversible degree. After hemorrhage has stopped, the paste can be very lightly condensed against the pulp stem large- diameter endodonticpluggers or a plastic instrument. The paste is condensed to a thickness of 2 to 3mm. Some techniques now use syringes.

4. EXPOSURE ENLARGEMENT

There have been recommendations that the exposures site be enlarged by a modification of the direct capping technique known as pulp curettage or partial

pulpotomy prior to the placement of capping material.
Enlarging this opening into the pulp itself serves three purposes:

1. It removes inflamed and / or infected tissue in the exposed area.
2. It facilitates removal of carious and non-carious debris particularly dentin chips.
3. It ensures intimate contact of the capping medicament with healthy pulp tissue below the exposure site.

The location of the exposure is important, as there should be no pulp tissue coronal to the exposure. Exposure in a cervical cavity would lead to reactionary dentine formation, which would restrict blood supply to tissue, more coronal to it, leading to necrosis and failure.
These teeth should therefore be root canal treated.

5. BACTERIAL CONTAMINATIONS

Watts and Paterson and Cox have both emphasized the fact that bacterial microleakage under various restorations causes pulpal damage in deep lesions, not the toxic properties of the cavity liners and/ or restorative materials. The success of pulp- capping procedures is dependent on prevention of microleakage by an adequate seal. Cox et al have shown that pulp healing is more dependent on the capacity of the capping material to prevent bacterial microleakage rather than the specific properties of the material itself.
Predominate belief is that pulp therapies appropriate for permanent teeth may not always be equally effective in treating similar pulpal conditions in immature permanent teeth.
Prognosis after any type of pulp therapy improves in the absence of contamination by pathogenic microorganism. Thus biocompatible neutralization of any existing pulpal contamination and prevention of future contamination (Ex. Microleakage) are worthily goals in vital pulp therapy. It is recognized that pulp tissue can recover from variety of insults spontaneously in a favourable environment.

FACTORS AFFECTING PROGNOSIS OF DIRECT PULP CAPPING

According to Seltzer and Bender carious pupal exposure is normally associated withinflammation and subsequent necrosis. Hence, mechanical exposure always have a better prognosis than a carious exposure. Mechanical exposures should be pulp-capped immediately.[66,67]

Care should be taken to ensure that the bleeding is controlled before pulp is capped. Other local factors would include:

1. Size of exposure
2. Bacterial contamination
3. Area of exposure
4. Microleakage
5. Treatment plan

The definition of success in direct pulp capping by the presence of dentine bridge is still controversial. Glass and Zander were among the first to sue this formation of reparative dentin as a criterion of success. It has been demonstrated in other studies; however that healthy pulp can exist beneath a direct capping, even without a dentin bridge.

INDIRECT VERSUS DIRECT PULP CAPPING: REACTIONARY VERSUS REPARATIVE DENTIN

Indirect pulp capping and the formation of reactionary dentin[70]

Facing a limited carious dentin, a certain number of layers are found. In this context, the most external part of the dentin constitutes a disorganized layer including a high number of bacteria tightly packed. They colonize and enlarge the dentino-enamel junction, wideningthe gap between the carious enamel and the soft carious dentin. Debris such as cell walls remnants remaining after by vegetal chewing, and fibers issued from muscle-like ‘meat’ may also be present. The soft carious dentin located beneath this zone of food debris is a mixture of bacteria and demineralized dentin that may be removed without drilling, using only manually sharp excavators. At the surface of this layer, the carious dentin is fully demineralized. The soft carious dentin displays enlarged tubules, containing bacteria acting within these reservoirs.
This layer is totally deprived of peritubular dentin. From the surface to the depth of the lesion, the dentin progressively is less demineralized. Apatitic crystals provide some consistency to this layer. Gradually peritubular dentin reappears. The next carious layer includes a mixture of infected tubules filled by cariogenic bacteria and bacteria-free tubules. A continuous ring of peritubular dentin surrounds the lumens of the canaliculi. Intertubular dentin gradually reach the original structure. Demineralization and reprecipitation occurs at the surface of apatitic crystals, increasing their size in three directions. In the sound dentin, crystals display the following main dimensions: 34 A˚ thick, 139A˚ wide and 250A˚ long. In the carious dentin, the crystals increase in thickness, and enlarge. Measurements indicate a 90A˚ (65-70A˚) thickness x 300A˚ in width, and a length of x 500 Crystals reach a final a diameter of 120 135A˚ according to Takuma et al.
In the sclerotic zone, the tubules are filled by intratubular precipitations. The needle- like crystals are thicker and longer than the crystals located within the sound intertubular dentin. In addition to hydroxyapatite crystals, whitlockite, octacalcium phosphate, and amorphous calcium phosphate (ACP) have been also identified. A calico-traumatic line separates the dentin already formed during the secondary dentinogenesis from the reactionary dentin. The newly formed dentin is either tubular, or formed by calco spheritic globules that have not merged. The reactionary dentin is in continuity with ‘normal’ tubular dentin. The dentin may be formed in reaction to the carious decay, or this material is structured as a

bonelike tissue, including cells into osteoplasts displaying a bone-like appearance to this structures.

Different types of dentin result from the speed of formation of the tertiary dentin. All these structures are elaborated by odontoblasts and eventually they may be replace by the differentiating cells of the Hö hl layer acting as substitute.

Bjorndal & Kidd have suggested that it is not necessary and even dangerous to eliminate the deepest zone of the soft carious dentin. There is a risk of pulp exposure. This layer should be removed either by hand excavators or by drilling up to the affected dentin. The deepest carious region, near the sclerotic zone should be kept in order to avoid a pulp exposure, and "re-mineralization" occurs within a few months. Then the carious tissue may be eliminate without taking any risk.

Calcium hydroxide or other bioactive agents contribute efficiently to the formation of reactionary dentin, a structure close to secondary dentin. These indirect capping through transdentinal stimulation of the odontoblasts and Hö hl cell layers stimulate reactionary dentinogenesis. Demonstration was done by Sognnaes and Wislocki that these layers containing acid muco polysaccharides, alkaline phosphatase, glycogen and carbohydrate groups are stained.

Anti- osteopontin, antibodies against the dentin phosphophoryn was present except in the mantle dentin, predentin and inner non-calcified layer of dentin. BSP, reduced antigenicity for type I collagen and proteoglycans was detected in the sclerotic dentin (Septier et al., 1998).

In dentin, DMP1 is normally cleaved, releasing N-terminal (N- ter) (37kDa) and a C- terminal (C-ter) (57kDa) fragments. Labeling was enhanced with the anti-DSPP. In the sclerotic dentin layer, DMP1 was intensely labeled in both the peritubular and intratubular dentin. This focus on some specificities of the carious dentin.

DIRECT PULP CAPPING AND THE FORMATION OF REPARATIVE DENTIN

The treatment of a pulp exposure involves direct capping. Pulp exposed after a deep carious lesion may be treated by bioactive molecules or by differentiated pulp cells. Since the discovery by Herman of the calcium hydroxide [Ca(OH)2] method, we get a better understanding of the mechanisms of reparative dentin formation which are involved.[70] The benefits of direct capping of the dental pulp results from the specific effects of each agent. A Zinc Oxide Eugenol (ZnO), coverage seems to be favorable and not cytotoxic. Glass Ionomer (GI), the resin-modified glass ionomer (RMGI), adhesive system, calcium-silicate based materials, have also been used, but all were displaying some cytotoxicity. The capping agents include Mineral Trioxide Aggregate (MTA), TheraCal a light-curable MTA- like material, Proroot MTA and other bioactive molecules of the SIBLING family. Their efficiency varies according to the inflammatory degree, and depending on the pulp degradation by bacteria releasing exogenous proteases, in close association with endogenous metallo proteinases. Many effects of these molecules acting as capping agents have been already reported. Bone sialoproteins, MEPE protein , amelogenin gene-splice products, pulp stem cells and Dentin Extracellular Matrix Molecules were evaluated for their efficiency as capping agents. Accidental pulp exposure is followed by a series of events.

Apical pulp cells (SCAP) slide along the pulp root, beneath the odontoblasts and Hö hl cell layer. They are issued from the apical part of permanent or exfoliated deciduous teeth (SHED)[71].

These stem cells may be in reduced number or totally absent. Stem cells may survive and arise within the inflamed pulp. They migrate, proliferate, and underwent differentiation into terminal cell lines. The lack of progenitors may be counterbalance by the Induced Pluripotent Stem Cells (iPS). The targeted addition of Growth Factor or Transcription Factors may provoke the de differentiation of adult cells and influence their commitment into cells displaying self renewal and multipotency. Retroviral introduction of *Oct3/4, Sox2, c-Myc and Klf4* induced the differentiation of pluripotent stem cells (iPSC). Positive markers contribute to the terminal differentiation. The positive markers include STRO-1, CD13, CD44, CD24, CD29, CD73, CD90, CD105, CD106, CD146, Oct4, Nanog and β2 integrin, wheras there are also negative markers such as CD14, CD34, CD45 and HLA-DR.

Once pulp cells are differentiated into odontoblasts/osteoblasts, they contribute to cell proliferation and differentiation. Beneath a scars layer due to the elevated pH of the alkaline capping agent, pulp cells accumulate, trans-differentiate and form a layer expressing positive markers of dentin. The cells accumulate in front of the pulp exposure.

They become odontoblast-like, and contribute to the formation of a dentin-likebridge (osteodentin). They mineralize and despite the presence of tunnels (tunnel-like structures) the dentinal bridge isolates the dental pulp from the oral cavity. Exposure to saliva contributes to the remineralization of this layer[72].

This osteodentin bridge displays barrier properties. The presence of reparative dentinocclude more or less the pulp exposure, and keep the tooth alive. Reparative dentin displays high type I labeling. Bundes of type III collagen are in close vicinity with fibronectin.

Transforming Growth Factor isoform is expressed in carious tissue, both in odontoblasts and this factor increases in tertiary dentin.[72,73]

- Reactionary and reparative dentins are related to indirect or direct pulp capping usedas therapeutic methods.Reactionary dentin results from the activation and stimulation of synthetic activities of odontoblasts and Hö hl cells. Beneath the calico-traumatic line, the newly formed dentin is constiute a reaction toward the carious lesion or

restorative materials.

- Reparative dentin is produced by the recruitment, proliferation and differentiation ofpulp stem cells becoming later odontoblast and/or osteoblast-like cells. They will further contribute to the formation of a bone-like structure.
- Altogether the two structures are implicated in the tooth healing and/or to pulp regeneration and mineralization. These reactions constitute a therapeutic aspect, whereas the other is related to pulp regeneration by using pulp stem cells.

PULPOTOMY

Treatment of severely decayed and pulpally involved young permanent teeth in the child or adolescent creates a dilemma. Preservation of the remaining pulp is critical. The treatment objective is to maximize the opportunity for apical development and closure, known as **apexogenesis**, and enhance continual root dentin formation. These objectives can occur only if the radicular pulp is maintained in a healthy state—the intent of the pulpotomy technique.[72]

DEFINITION

A pulpotomy is the removal of the coronal portion of the pulp and the treatment of the remaining radicular pulp in an attempt to maintain the tooth and its supporting structure in a state of health. – Kennedy

Surgical removal of entire coronal pulp presumed to be partially or totally inflamed andquite possibly infected, leaving intact the vital radical pulp in the canals. – Ingle

Complete removal of the coronal portion of the dental pulp, followed by placement of a suitable dressing or medicament that will promote healing and preserve the vitality of the tooth. – Finn

OBJECTIVES

1. Preservation of vitality of the radicular pulp through the surgical excision of the coronal pulp, leaving vital, uninfected pulpal tissue in the root canal.
2. Relief of pain in patients with acute pulpalgia and inflammatory changes in the tissue.
3. Ensuring the continuation of normal apexogenesis in immature permanent teeth by retaining the vitality of the radicular pulp.

RATIONALE

When the coronal pulp is exposed by trauma or operative procedures, or caries ingress of bacteria, it produces inflammatory changes in the tissue. Through the surgical excision of the infected and inflamed coronal pulp, the vital uninfected pulpal tissue can be left behind and preserved in the root canal. The removal of the inflamed portion of the pulp affords temporary, rapid relief of pulpalgia and further may undergo repair while completing apexogenesis that is root end development and apical closure.[74]

Materials used for this procedure either mummify or fix the tissue or promote healing by formation of a bridge.

CLASSIFICATION

Pulpotomy can be classified according to:

- **TREATMENT OBJECTIVES** given by Ranley et al:

1. Devitalization Pulpotomy – (Mummification, Cauterization)

a. Formocresol pulpotomy
b. Electrosurgical pulpotomy
c. Laser pulpotomy

2. Preservation (Minimal devitalization, Non – inductive)

a. Gluteraldehyde
b. Ferric sulfate

3. Regeneration (Inductive, Reparative)

a. Calcium Hydroxide
b. Bone morph genetic Protein
c. Mineral trioxide aggregate

- **THE NUMBER OF VISITS:**

1. Single visit pulpotomy.
2. Multiple visit pulpotomy.

- **TYPE OF MEDICAMENT**

1. Calcium hydroxide pulpotomy
2. MTA pulpotomy
3. Formocresol pulpotomy.

- **AMOUNT OF PULPAL TISSUE REMOVED**

1. Cervical pulpotomy
2. Partial pulpotomy

PERMANENT TOOTH PULPOTOMY INDICATIONS

Carious or mechanical exposure of vital primary & young permanent teeth, where inflammation is restricted to coronal pulp only. Pain if present is neither spontaneous nor persistent.

CONTRAINDICATIONS

1. History of spontaneous pain / pain at night.
2. Presence of swelling / fistula.
3. Tenderness on percussion.
4. Pathological mobility of the tooth.
5. External / internal resorption (seen on radiographs).
6. Pulp calcification.
7. Pus / serous exudates from exposure site.
8. Uncontrollable bleeding from amputated pulp stumps

APICAL CLOSURE PULPOTOMY [43]

This is the treatment of choice whenever an immature permanent tooth with an open apex and reversible pulpitis suffers a carious exposure of a large diameter (more than 1mm) traumatic pulpal exposure, of any duration, occurs.

DEFINITION

(Andreasen) It is defined as the removal of damaged and inflamed tissue to the level of a clinically healthy pulp, followed by a calcium hydroxide dressing.

It was described in 1938 by Teuscher and Zander. Kaiser in 1964 and Frank in 1966 popularized the technique. It became famous as *'Frank's technique'*.

Traditionally, pulpotomy implied the removal of the entire coronal pulp upto the cervical area.

Today the depth of tissue removal is based on the clinical judgement : only tissue with profuse bleeding, judged to be inflamed or infected, should be removed, as the dressing should be placed on healthy tissue. Depending on the size of exposure and time elapsed since injury, different levels are described:

1. *Partial pulpotomy*, also known as shallow, low-level or *Cvek's pulpotomy.*
2. *Cervical pulpotomy*, also known as deep, high-level, total or conventional pulpotomy. Neither exposure size nor time interval between injury and treatment are critical for healing when the pulp is reversibly inflamed.

Calcium hydroxide pulpotomy in a young permanent molar. The cavity is prepared, caries and the chamber roof are removed, and the pulp is amputated to the canal orifices. Following hemostasis, commercial calcium hydroxide is placed and protected with zinc oxide–eugenol and amalgam filling or a stainless crown.

PARTIAL PULPOTOMY

Also called as Cvek technique. It is the removal of only the outer layer of damaged and hyperemic tissue in exposed pulps and is considered to be a procedure staged between pulp capping and complete pulpotomy. Cvek reported a high success rate of 94-96% with this procedure, when carefully performed. He has shown that in exposures resulting from traumatic exposures, pulpal changes are characterized by a proliferative response, with inflammation extending only a few millimetres into pulp. When the hyperplastic tissue is removed, healthy pulp is found. Care should be taken to remove all the tissue coronal to amputation site to prevent continuation of bleeding, contamination and discolouration of the tooth.[74]

TECHNIQUE

1. Anesthetize and isolate.
2. A diamond bur, corresponding to the size of the exposure is used in a high speed contraangle handpiece is used with water spray to provide effective cooling.
3. Cutting is performed intermittently for brief periods and without undue pressure.
4. Level of amputation is 2 mm below the exposure site. This provides for removal of inflamed tissue and adequate cavity for dressing and sealing material. The cavity is made box-like with undercuts for retention.
5. Pulpal wound is rinsed with saline till bleeding ceases. Hemorrhage can be controlled with moist cotton pellets applied with light pressure.
6. Wound is covered with calcium hydroxide, which is adapted with light pressure. Surplus is removed with spoon excavator.
7. Cavity is sealed with IRM or GIC. Advantages of partial pulpotomy

a. Minor injury to the pulp and undisturbed physiologic apposition of dentin,especially in the critical cervical area of the tooth.
b. The limited loss of coronal pulp allows for vitality testing.
c. Limited loss of crown precludes need for post and core.
d. Allow the continuation of normal development of the tooth, including further root development and maturation. Apex formation and thickening of thin root walls may occur in young teeth.

Compared with pulp capping it implies:

a. Better wound control.
b. Better sealing against microleakage.

CERVICAL PULPOTOMY

Pulpotomy in mature teeth is performed only when irreversible pulpitis is diagnosed, and should be considered as an emergency treatment. In these teeth, root canal treatment will follow at next appointment. In immature permanent teeth, it is indicated when necrotictissue or obviously impaired circulation is present at the site of exposure. Here, pulp should be amputated to a level at which fresh bleeding tissue is found i.e. at cervical level. Bleeding should be controllable. It is performed in teeth in which it is assumed that healthy pulp tissue has a potential to produce a dentin bridge and complete the formation of the root.[74] Due to problems with adequate cooling of the diamond bur at high speed at that level, around carbide bur at low speed should be used. For, molars, a spoon excavator can be used to amputate pulp till the floor level.

When using calcium hydroxide powder, Webber recommends the addition of barium sulphate, in the ratio of 4 parts calcium hydroxide and 1 part barium sulphate. This increased radiopacity will enable radiographic confirmation of apposition of calcium hydroxide onto the orifices (pulp stumps), which is essential for the therapy to be effective.

PULPOTOMY WITH MTA

The procedure is similar to that described for calcium hydroxide. Here, MTA ismixed with saline in a ratio of 3:1 on a glass or paper slab and is placed against the pulp stump. It is lightly patted against the pulp and a moist cotton pellet is placed against it toenable setting, as it sets in the presence of moisture. The cavity is sealed with IRM or GIC. Alternatively, the entire cavity can be filled with MTA and protected with a wet piece of gauze for 3

to 4 hours. The coronal 3 to 4 mm of MTA is removed, and a final restoration is placed immediately. MTA sets in about four hours and is difficult to remove from the canal.Hence, it should not be used in teeth which may be indicated for pulpectomy in future.[74]

OTHER MATERIALS

Newer materials such as bone growth factors and bone morphogenetic proteins are under research and have shown to be successful.

Formocresol pulpotomy is only indicated for primary teeth. Pulpotomy is not considered a permanent option by some researchers. Thus the procedure must be used very selectively.

Clinical and radiographic follow-up of the teeth is essential to ensure that pulpal or periapical pathosis is not developing. It is of utmost importance to perform a permanent restoration as soon as possible to prevent bacterial leakage and ensure the success of the treatment.

FOLLOW UP

The tooth which has been treated with pulpotomy should be checked with radiographs and vitality tests every 3 months. In case of any sign of pain or death of pulp, endodontic treatment should be initiated if the apex is mature. If the apex is immature, then apexification should be done.

APEXOGENESIS

The root apex is of interest to endodontics because the stages of root development and the type of tissue present within the roots of teeth are significant to the practice of endodontics. [60] The development of the root begins after the enamel and the dentin formation has reached the future cemento-enamel junction. The completion of root development and closure of root apex occurs upto 3 years after tooth eruption. Irreversible injury to the dental pulp of an immature permanent tooth from either infection or dental trauma before complete root development poses a clinical challenge and depending on the vitality of the affected pulp calls for a specially tailored treatment plan.

DEFINITION

"Vital pulp therapy procedure performed to encourage continued physiologic development and formation of the root end." (American Association of Endodontists Glossary)[67]

"**Apexogenesis** is treatment designed to preserve vital pulp tissue in the apical part of a root canal in order to complete formation of the root apex ."

"**Maturogenesis** has been defined as physiologic root development not restricted to the apical segment. The continued deposition of dentin occurs throughout the length of the root, providing greater strength and resistance to fracture.

The current terminology is **vital pulp therapy** and is defined by **(Walton and Torabinejad)** as a treatment of vital pulp in an immature tooth to permit continued dentin formation and apical closure.

BIOLOGY OF THE ROOT APEX

The apical region of an immature permanent tooth is comprised of dental pulp, apical papilla and periodontal tissues, which have developed through a series of ectomesenchymal interactions. During the bell stage of tooth development, the dental papilla becomes partially enclosed by the envaginating epithelium, and the condensed ectomesenchyme surrounding the enamel organ and dental papilla forms the dental follicle (TenCate 1997). The dental pulp is soft tissue of ectomesenchymal and mesenchymal origin, which develops from the dental papilla. It is composed of water, ground substance, connective tissue, blood vessels, nerves, lymphatics, fibroblasts, immune cells and odontoblasts (Trowbridge 2003). The odontoblasts secrete dentin and are integral to the pulp–dentin complex. Primary dentin is formed until completed root development, following which dentin formation proceeds as secondary dentinogenesis and continues at a slower rate throughout the lifetime of the individual. As the root and pulp develop, the dental papilla is located apical to the developing pulp and is called the apical papilla. The dental follicle surrounding the developing tooth root contains progenitor cells for the developing periodontium: cementum, alveolar bone and PDL. Meanwhile, the inner and outer enamel epithelia fuse to form a structure known as Hertwig's epithelial root sheath (HERS). As HERS migrates apically, the ectomesenchymal tissues are divided into the dental papilla on one side and dental follicle on the other. HERS has a role in root development and shape, but the exact function of thecells is less certain. Once the first layer of mantle dentine has been laid down, the root sheath begins to disintegrate, allowing the attachment of cells from the dental follicle onto the exposed root dentine with the subsequent deposition of cementum (Handa et al. 2002). Individual cells from the root sheath migrate away from the root to the region of the future periodontal ligament to form the rests of Malassez. HERS is very sensitive to trauma and once destroyed, there is cessation in normal root development with no further odontoblast differentiation. In an immature permanent tooth, this leaves an open root apex, thin weak root walls and a discontinuous periodontal ligament.[70]

B, Success is evidenced by continued root development (length and wall thickness) and formation of a calcific barrier in response to the wound dressing.

OPEN APEX

- At the time of tooth eruption root development is only 62-80% i.e., 2/3rd of theroot is formed.
- If due to trauma or caries exposure, dentin formation ceases and root growth isarrested.
- The resultant immature root will have an open

apex which is also called as blunderbuss canal.

As root development takes almost 2-3 years after the tooth has erupted into the oral cavity, injury to the young developing tooth results in a short root with a wide canal that can be either divergent or parallel and is associated with flared or cylindrical root apex. The term "blunderbuss apex" is specifically used to indicate an open or flaring apex, resembling the barrel of a blunderbuss rifle.

PROBLEMS FACED WITH OPEN APEX

- Due to large apical diameter and smaller coronal canal diameter debridement isdifficult.
- Lack of apical stop makes obturation difficult.
- The thin root canal walls become prone to fracture. (fig.49)

RATIONALE FOR APEXOGENESIS

The canal obturation for incompletely formed roots and open apices presents unique problems with conventional endodontic techniques. The relatively thin dentinal walls of the large canals place the tooth at greater risk for tooth fracture over time. In these instances, the treatment objective is to maximize the opportunity for apical development closure, known as apexogenesis, and enhance continual root dentin formation[70]. These objectives can be fulfilled only if the radicular pulp is maintained in a healthy state – the intent of the pulpotomy technique.

GOALS OF APEXOGENESIS

1. Sustaining a viable Hertwig's epithelial sheath to allow continued development of root length for a favourable crown-to-root ratio.
2. Maintaining pulp vitality to help maturation ofroot.
3. Promoting root end closure to create a natural apical constriction.
4. Generating a dentinal bridge at the site of pulpotomy (a sign that the pulp hasmaintained its vitality).

INDICATIONS

1. Traumatized or pulpally involved vital permanent tooth when root apex isincompletely formed.
2. No history of spontaneous pain.
3. No sensitivity on percussion.
4. No haemorrhage.
5. Normal radiographic appearance.

CONTRAINDICATIONS

1. Purulent drainage.
2. Prolonged pain.
3. Necrotic debris in canal.
4. Periapical radiolucency evident on radiograph.
5. Avulsed and replanted or severely luxated tooth.
6. Severe crown-root fracture that requires intra radicular retention forrestoration.
7. Tooth with an unfavorable horizontal root fracture (i.e.close to the gingivalmargin)
8. Carious tooth that is unrestorable.

TREATMENT CONSIDERATIONS

A thorough clinical and radiographic examination must be performed prior to the initiation of treatment. This procedure should only be considered for incompletely developed teeth with vital pulps without evidence of peri radicular pathosis. An assessment of pulp vitality and periradicular health must be made. Pulp sensitivity testing (thermal stimulation, electric pulp testing) will indicate the presence or absence of pulp vitality in most clinical situations. Other clinical tests, such as percussion and palpation, will provide information regarding inflammatory changes in the peri radicular tissues. [73]The presence of a sinus tract, swelling, coronal tooth discoloration or increased mobility will also provide information on the status of the pulp, peri radicular tissues and attachment apparatus. Radiographic examination will provide specific in formation regarding the extent of the carious lesion, the development of the roots, the presence of peri radicular pathosis, root resorption and crown and/or root fractures.

TECHNIQUE

The clinical procedure is essentially a deep pulpotomy undertaken to preserve the formative capacity of the radicular pulp in immature teeth that have deep pulpal inflammation. Examples include carious exposures and some trauma cases in which treatment of the exposed pulp is delayed, and it becomes necessary to extend further into the canal to reach healthy tissue.[73] Many different materials have been used for pulpal wound dressing, however, the use of calcium hydroxide has been shown to be the most predictable with regard to long-term clinical success. Pure calcium hydroxide paste, which has a pH of about 12.5, causes a limited and shallow chemical injury to the vital pulp tissue. The response of the vital tissue is a self-limiting inflammatory reaction, followed by the proliferation of cells and new collagen. Mineralization of the newly formed collagen starts with dystrophic calcification, which is then followed by tubular dentin formation. An alternative gold standard, mineral trioxide aggregate (MTA), is available for use in VPT. MTA is shown to be able to induce hard tissue formation in pulpal tissues and in comparison with CH, MTA has demonstrated a greater ability to maintain the integrity of the pulpal tissues. Histologic evaluations showed that MTA produces a thicker dentinal bridge, less inflammation, less hyperemia, and less pulpal necrosis compared with CH.[76]

Calcium hydroxide is preferred over MTA because:

1. If procedure fails, Ca(OH)2 facilitates re-entry to the root canal to perform apexification or pulp regeneration.
2. If apexogenesis is successful and root-end

formation is complete, tooth could bere-entered if desired for conventional root canal treatment.

Calcium hydroxide powder has usually been preferred over hard-setting products, carried into the canal with an amalgam carrier or gun system employed for MTA application. Small increments of $Ca(OH)_2$ powder are carefully teased against the entire surface of the pulp stump with a rounded-end, plastic instrument, ideally with microscope control. Care must be taken not to pack the $Ca(OH)_2$ into the pulp tissue because this causes greater inflammation and increases the chances of failure. Even if pulpotomy is successful, there is an increased risk that the remaining pulp tissue will mineralize aroundimpacted particles of $Ca(OH)_2$.

Meligy and Avery (2006) conducted a study to compare mineral trioxide aggregate (MTA) with calcium hydroxide [$Ca(OH)_2$] clinically and radiographically as a pulpotomy agent in immature permanent teeth (apexogenesis). Fifteen children, each with at least 2 immature permanent teeth requiring pulpotomy (apexogenesis), were selected for this study. All selected teeth were evenly divided into 2 test groups. In group 1, the conventional $Ca(OH)_2$ pulpotomy (control) was performed, whereas in group 2, the MTA pulpotomy (experimental) was done. The children were recalled for clinical and radiographic evaluations after 3, 6, and 12 months.[70] The follow-up evaluations revealed failure due to pain and swelling detected at 6 and 12 months postoperative evaluations in only 2 teeth treated with $Ca(OH)_2$. The remaining 28 teeth appeared to be clinically and radiographically successful 12 months postoperatively. Calcific metamorphosis was a radiographic finding in 2 teeth treated with $Ca(OH)_2$ and 4 teeth treated with MTA. Based on this study it was concluded that, Mineral trioxide aggregate showed clinical and radiographic success as a pulpotomy agent in immature permanent teeth (apexogenesis) and seems to be a suitable alternative to calcium hydroxide.[78] Calcium enriched mixture (CEM) cement is a new endodontic cement with similar clinical applications as MTA but different chemical composition. CEM cement has antibacterial effect comparable to CH and superior to MTA and sealing ability similar to MTA.The biologic response of the pulpal tissue to MTA and CEM cement has been shown to be similar in dogs' teeth. [79]

RECALL SCHEDULE

The patient is usually scheduled for recall appointments at 3- to 6-month intervals to monitor pulp vitality and apical growth. The total follow-up time varies, depending on the initial degree of root maturation. If the root were in a very early stage of development, root formation could take 2 to 3 years. Recall appointments should then be done for atleast 4 years. Absence of symptoms does not indicate absence of disease. Monitoring of signs and symptoms, periodic pulp testing, and radiographs are required to determine pulp and periapical status. One advantage of pulp capping or shallow pulpotomy over conventional pulpotomy is of the ability to test the pulp.[7]

CLINICAL EVALUATION OF APEXOGENESIS

1. No clinical symptoms
2. No radiographic changes in pulp or peri apex
3. Continued root development
4. Radiographically observed hard tissue barrier at the site of procedure
5. Sensitivity to vitality testing.

FAILURES OF APEXOGENESIS

1. Cessation of root growth
2. Development of signs and symptoms or peri apical lesions
3. Calcific metamorphosis – common occurrence after conventional pulpotomy.

One problem with calcific metamorphosis is that if the pulp becomes necrotic and root canal treatment is necessary, canals may not be negotiable, and surgerymay be necessary.

CASE REPORT

1. Apexogenesis with $Ca(OH)_2$

An 8-year-old girl presented with complicated crown fractures in all the mandibular incisors as a result of trauma. The examination was performed 10 hours after the car accident that caused the trauma. A medical and dental history was taken. Clinical and radiographic examination was performed and this ruled out any root fractures. Examination revealed that all mandibular incisors had immature apices and exhibited pulp exposures (Fig. 51 A). Under local anaesthesia, cervical pulpotomies were performed in all the mandibular incisor teeth using a diamond bur. The pulps were covered with pure calcium hydroxide powder that was mixed with physiologic saline to a very dry thick mix and which was condensed with a light, vertical pressure to a thickness of 2–3 mm. The teeth were restored with glass ionomer cement. After 1 week, part of the glass ionomer was removed and the teeth and their crowns were restored with light-cured composite resin (Fig. B). Six months later the patient was followed up for clinical and radiographic evaluation. Radiographic images showed root development (Fig. C). Follow-up examination after 11 years showed that the patient was symptom-free and comfortable. A radiographic image (Fig. D) showed that all of the teeth had favourably developed and there was no evidence of either resorption (internal or external) or periradicular pathologicchanges.[79]

1. APEXOGENESIS WITH MTA

INITIAL PRESENTATION

A nine-year-old female with a non-contributory medical history presented in with traumatic pulp exposures of the maxillary right and left permanent central incisors. Both teeth (11 and 21) exhibited

mesial-incisal fractures, sustained after the patient fell off her bicycle.

CLINICAL FINDINGS & DIAGNOSIS

On examination, extra-oral findings were within normal limits. Intra-oral soft tissues were within normal limits as well. Teeth 11 and 21 showed moderate tenderness to percussion relative to the surrounding dentition, but both teeth tested within normal limits to cold. Tooth 11 had grade I mobility. Radiographically (Fig. 52 A), pulp exposures were apparent and the roots were confirmed to be incompletely developed.

TREATMENT PROCEDURE

The recommended treatment was MTA pulpotomy to facilitate apexogenesis. Ifsuccessful, this would enable long-term retention of the teeth as well as continued root development. Following rubber dam isolation, an access opening was made, first in tooth 11, then 21. In both cases, the pulp was gradually removed until bleeding could be controlled by using pressure from the wide end of a paper point. This occurred slightly apical to the cement-enamel junction. Irrigation was performed with two percent chlorhexidine in order to disinfect the pulp with minimal irritation. When haemostasis was confirmed, white coloured MTA was placed over pulp stump. The MTA was covered by a moist cotton pellet and the access opening was sealed with Cavit (Fig. B)

APEXOGENESIS WITH CEM

An 8-year-old boy presented with a history of prior impact trauma 4 weeks before his initial visit. The patient's chief complaint was sensitivity to cold beverage and pain in chewing. No spontaneous pain was reported by the patient. Clinical examination showed a complicated crown fracture with pulpal exposure on the left maxillary central incisor. The tooth was sensitive in percussion test but not in palpation. Radiographic examination showed that fractured tooth had immature apex (Fig.53 A). The patient's medical history was non-contributory. Under local anaesthesia and rubber dam isolation, access cavitywas prepared with a diamond fissure bur followed by cervical pulpotomy by using a long shank round diamond bur, with high speed and copious water spray to prevent heat damage to subjacent pulp. The area was rinsed with normal saline solution. Haemostasis was achieved by gentle placement of a moistened sterile cotton pellet over the amputated pulp. Following the manufacturer's instructions, CEM cement powder and liquid were mixed to achieve a creamy consistency. An approximately 2-mm-thick layer of CEM was placed over the exposed clot-free pulpal wound by using an amalgam carrier and was gently adapted to the dentinal walls of the access cavity with a dry cotton pellet. Thecavity was filled with normal saline, and a moistened cotton pellet was placed gently over it. Then the tooth was temporarily filled with Cavite (Fig. B). A day later, temporary restoration was removed to confirm the setting of the capping material and the tooth was permanently restored (Fig. C). Patient was recalled 6 and 12 months after the treatment for follow up. The tooth was functional and asymptomatic, with no evident clinical signs. [77] Root development, formation of root apex, and formation of a calcified bridge beneath CEM cement were radiographically evident at the follow-up sessions (Fig. D andE).

CONCLUSION

Vitality of dental pulp is essential for long-term tooth survival. The aim of vital pulp therapy is to maintain healthy pulp tissue by eliminating bacteria from the dentin pulp complex.

The discipline in Vital Pulp Therapy is continuously evolving and has a positive stand. The current understanding of caries and remineralization has shifted the treatment philosophy to more conservative surgical management of deep carious lesions. Further clinical research with larger sample sizes is encouraged.

There are several different treatment options for vital pulp therapy in extensively decayed or traumatized teeth. Pulp capping or pulpotomy procedures rely upon an accurate assessment of the pulp status, and careful management of the remaining pulp tissue.

The development of biomaterials, new biological pulp capping agents and strategies of regeneration therapies are giving a new insight to pulpal repair.

The candidates of vital pulp therapy should be selected wisely.

Root canal treatment is not always the treatment of choice in deep carious lesions approaching pulp. The success rate of vital pulp therapy procedures gathered through previous studies comes to average of 84.76% . [80] Maintaining vitality of pulp is important for a longer survival of the teeth hence is should be promoted and performed wherever necessary.

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