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Vital Pulp Therapy

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Department of Conservative in Partial Fulfillment for the

Bachelor of Dental Surgery

By

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Certification of the Supervisor

I certify that this project entitled “Vital Pulp Therapy” was prepared by the fifth-year student Abdulla Hassan Abdulla under my supervision at the College of Dentistry/University of Baghdad in partial fulfillment of the graduation requirements for the Bachelor Degree in Dentistry.

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Dedication

I humbly dedicate this piece of work to the ones who've always been there for me and gave me support and Inspiration along my studying years.

To my grandmother Iqbal

To my uncle Jaber

To My parents Dr. Hassan, Enas Khalil

To my uncle Dr. Haider and his wife Dr, Duaa

To my uncle Dr. Anees

To my aunt Zainab

To my brothers Yousif and Anees

and my friend Qamar

To my friend Dr. Aeshah Zeyad

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List of abbreviation

Abbr.	Acronyms
Csc	calcium silicate cement
AAE	American association of endodontics
Dpc	direct pulp capping
VPT	vital pulp therapy
Ch	calcium hydroxide
Naocl	sodium hypochlorite
AAPD	American association for pediatric dentistry
Chx	chlorohexidine
RMGI	resin modified glass ionomer
MTA	mineral trioxide aggregate

Introduction

Introduction

A nerve: any of the filamentous bands of nervous tissue that connect parts of the nervous system with the other organs, conduct nerve impulses, and are made up of axons and dendrites together with protective and supportive structures.

Dental pulp: The soft tissue forming the inner structure of a tooth and containing nerves and blood vessels. Tooth pulp is the most vital part of the tooth. From above we know that pulp houses the source of pain. As a human the pain was always a big problem, unpleasant sensation, it is make you struggle and cannot focus when you do anything.

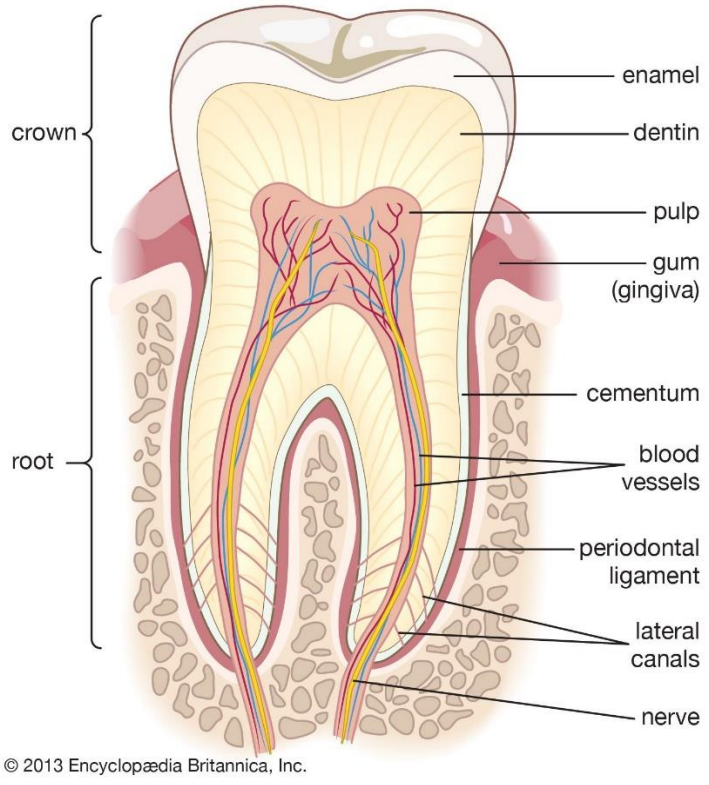

Figure 1 anatomy of the pulp

Functions of Tooth Pulp

The pulp has several important functions, including:

Sensory function: Pain from trauma to the dentin and/or pulp, differences in temperature, and pressure are caused by stimulation of the pulp.

Formation of dentin: The pulp is responsible for the formation of dentin. In response to trauma, the pulp forms secondary dentin, also known as reparative dentin.

Nourishment: The pulp contains blood vessels that keep blood flowing to help to prevent the tooth from becoming brittle by keeping it moisturized and nourished.

The Role of Dentin

The most important job of pulp is to produce dentin, which is a calcified tissue that serves as the second layer of the tooth, supporting the enamel above it.

when dentin becomes exposed. This usually causes pain, especially when you eat or drink something that's hot or cold because the dentin stimulates your tooth pulp or nerve. So the pulp is the most important part of the tooth, we have to focus on keep it vital.

Pulpitis

Painful inflammation of the pulp results in a condition known as pulpitis. Tooth decay is the number one cause of pulpitis, followed by injury. If the inflammation is mild, it may be reversible, but if it's severe, the pulp can die. Pulpitis can also cause an infection at the root of your tooth, known as an abscess. If you have pulpitis, you'll know it because it's extremely painful.

Vital Pulp Therapy (VPT)

They are techniques are means of preserving the vitality and function of the dental pulp after injury resulting from trauma, caries, or restorative procedures. VPT

procedures have traditionally included indirect or direct pulp capping, and partial or complete pulpotomy. (which could be symptomatic or asymptomatic).

Traditionally the designation of a pulpal diagnosis is based upon the clinician's consideration of a patient's pain history, and appropriate clinical testing to assess the status of the pulp including the application of cold stimulus and electric pulp testing. In addition to such pulp sensibility testing, percussion tests may infer pulpal conditions from the presence of symptomatic apical periodontitis; with the presence of percussion pain,

The viewpoint that VPT is an option only for cases where testing results were consistent with "reversible pulpitis" has recently been challenged. Based on clinical, biological and theoretical considerations, the irreversibility of the pulpal disease has come into question. Histologic evidence of the progression of pulpitis suggests that there is no discrete boundary that would render a pulp beyond repair. Rather, pulpitis may be interpreted as a temporally and spatially graded disease, with some suggesting the following terms for gradation: "initial", "mild", "moderate" and "severe pulpitis.

Complete caries removal is essential to eliminate infected tissues and visualize pulp tissue conditions under magnification when pulpal exposures occur. The primary goal of VPT procedures is the creation of optimal conditions for pulp tissue repair and preservation. The amount of pulp tissue removed or retained is dependent on tissue viability assessments based on access for visualization to evaluate hemorrhage control and clinical tissue appearance.

Until this time a lot of people think that extraction is best option for get rid of the pain. Especially in our country cause a lot of people don't know anything about dentistry.

VPT is the future of dentistry, it is important derived from its effort to be conservative as much as possible.it is very important to talk more about new

techniques and their advantage, so all people reach the point that extraction is the last solution, nothing will compensate normal tooth.

Chapter One

Literature Review

1.1 The living pulp tissue

Vital pulp tissue comprises cells including fibroblasts, undifferentiated mesenchymal cells, odontoblasts, macrophages, dendritic cells, and other immunocompetent cells. The cells of the subodontoblastic layer and odontoblasts form a thin border between the dentin and the periphery of the pulp known as the Höhl cell layer **(Dammaschke, 2010)**.

The odontoblasts are elongated columnar-shaped cells separated from the mineralized dentin by predentin and characterized by processes that extend into the dentin and possibly to the dentinoenamel junction **(Grötz et al., 1998).**

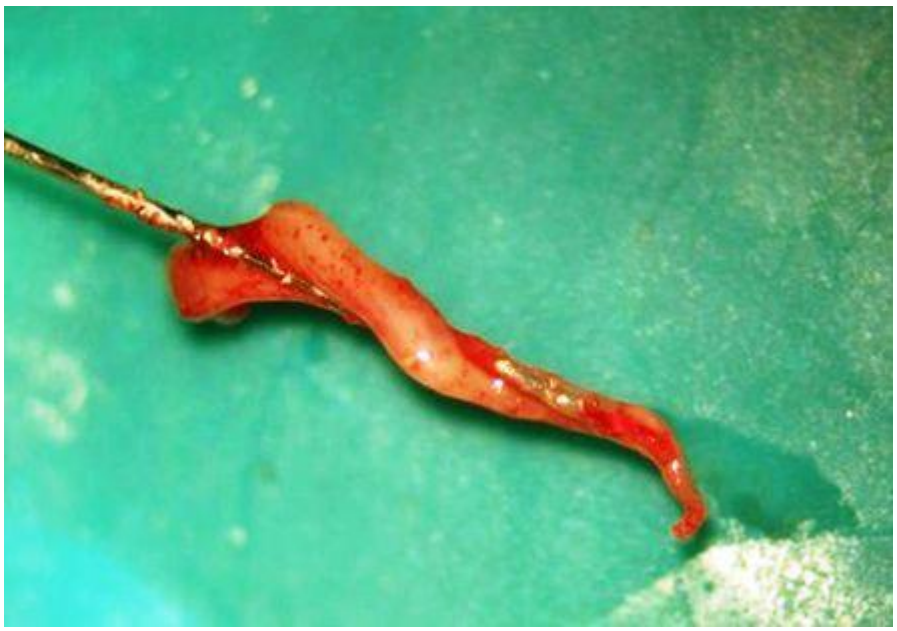

Figure 2 tooth pulp

Odontoblasts form the mineralized predentin-dentin matrix, which includes phosphoproteins, glycoproteins, proteoglycans, and sialoproteins **(Kim et al., 2008).**

Repair mechanisms in the pulp are similar to those in normal connective tissue injured by trauma. When the enamel and dentin are challenged and the pulp is exposed to advancing microorganisms, inflammatory changes can induce pulp necrosis, which precedes problems including infection and its complications **(Bjørndal et al., 1998).**

Young dental pulps show greater cell and tissue differentiation, proliferation, and development of the lymphatic, hematologic, and immune systems compared to older pulps where the apoptosis pathway is highly expressed **(Tranasi et al., 2009).**

In summary, the main functions of the dental pulp include dentin formation during development and life of the tooth, the transmission of stimuli via proprio- and pain receptors, and immune responses. The pulp also produces reparative dentin as a defense mechanism against external stimuli, and tissue during the formation and closure of the root apex **(Berman and Hargreaves, 2020).**

1.2 Inflammatory reaction of the pulp

Dental caries is a localized, destructive, and progressive infection of dentin, which, if left unchecked, can result in pulp necrosis and potential tooth loss. Both bacterial by-products and products from the dissolution of the organic and inorganic constituents of dentin mediate the effects of dental caries on the pulp. Three basic reactions tend to protect the pulp against caries: (1) decreases in dentin permeability due to dentin sclerosis, (2) tertiary dentin formation, and (3) inflammatory and immune reactions in the pulp **(Kim et al., 1998).**

The microbial advance in carious lesions is the primary cause of pulpal inflammation and potential necrosis. Acidogenic gram-positive bacteria, predominately oral streptococci and lactobacilli, produce lactic acid as the main metabolic by-product in active lesions that demineralizes enamel and dentin. Immune responses and inflammation occur when the caries front is within 1.5 mm of the pulp and bacterial antigens and metabolites diffuse through dentinal tubules **(Farges et al., 2011).**

If bacterial challenge continues, immune cell responses lead to increased inflammation and edema, initially characterized clinically by pulpal pain. In the low-compliance environment of the pulp space, inflammation eventually leads to pulp disintegration and apical pathosis. The pioneer microbes first encounter a positive outward flow of dentinal fluid, characterized by the deposition of immunoglobulins and serum proteins that slow the diffusion of antigens **(Hahn and Liewehr, 2007b).**

Potent microbial metabolites, such as lipoteichoic acid and lipopolysaccharide, also activate the innate immune system. The by-products stimulate signaling by Toll-like receptors in odontoblasts when these cells first encounter the carious front. They also stimulate pro-inflammatory cytokines, including interleukin-1, interleukin-8, interleukin-12, tumor necrosis factor alpha, vascular endothelial growth factor (VEGF), and transforming growth factor beta (TGF-β) **(Farges et al., 2011).**

VEGF promotes vascular permeability and angiogenesis. Dentin mineralization and matrix metalloproteinase secretion are also induced by the increased expression of TGF-β. Cariogenic bacteria also activate complement pathways and induce the proinflammatory cytokine interferon γ, responsible for killing phagocytosed bacteria by activated macrophages **(Hessle et al., 2005).**

As pulpitis progresses, vasoactive neuropeptides contribute to increased vascular permeability and intrapulpal blood flow. Increases in neuropeptide concentration and nerve sprouting characterize neurogenic inflammation, which can cause a transient increase in interstitial tissue pressure and contribute to painful pulpitis **(Byers et al., 1990).**

The primary effector cells in innate responses include natural killer cells, neutrophils, monocytes, and macrophages. Immature dendritic cells and T cells contribute to immunosurveillance during the progression of caries. Macrophages eliminate both pathogens and senescent cells while contributing to tissue homeostasis by repairing and remodeling tissue after inflammation. Cytokines are small, cell-signaling proteins secreted by innate immune cells that induce phagocyte extravasation during inflammation. Chemokines secreted by odontoblasts, fibroblasts, immature dendritic cells, and macrophages stimulate leukocyte recruitment by directing monocyte and neutrophil migration extravascularly to sites of infection **(Hahn and Liewehr, 2007a).**

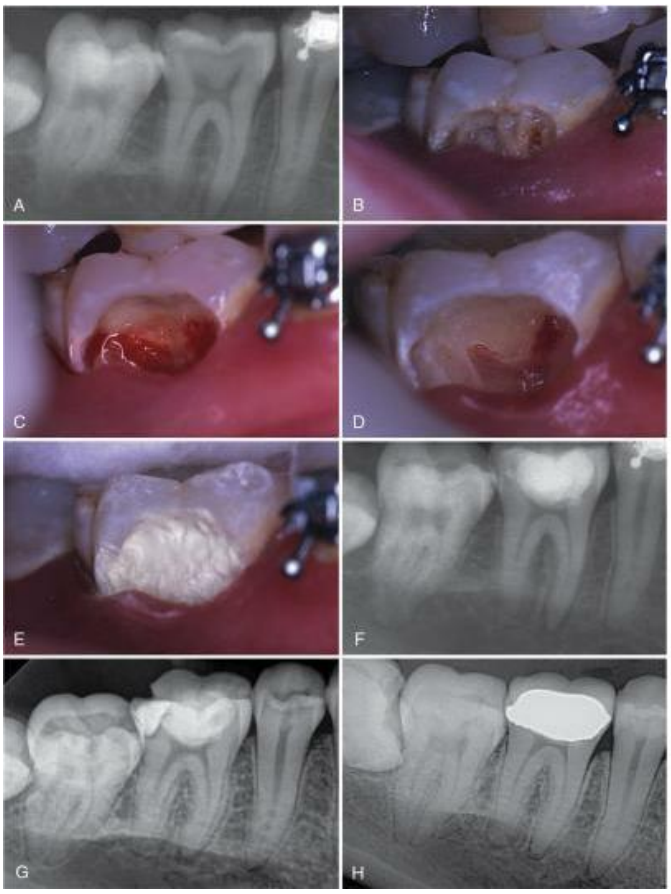

Figure 3 A 15-year-old female patient presented with full orthodontic banding and deep cervical caries of the mandibular right first molar (tooth #30). The tooth was sensitive to cold and biting pressure. A, Periapical radiograph revealing large carious lesion and normal periapical structures. B, Clinical photograph showing advanced cervical caries extending below crestal gingiva. C, View of initial pulp exposure during caries excavation guided by caries detector dye. D, Photograph after complete caries removal, large pulp exposure and 6% sodium hypochlorite hemostasis. E, Clinical photograph after placement of thick white mineral trioxide aggregate aliquot over entire pulp and surrounding dentin. F, Radiograph showing pulp capped molar with temporary restoration (Photocore). G, Four-year radiographic control showing recurrent caries and fractured distal marginal ridge tooth #30. H, Five-year radiographic review showing no detectable apical pathosis and recently cemented

full coverage porcelain fused to metal crown. The molar was asymptomatic and responded normally to sensibility testing **(Berman and Hargreaves, 2020).**

A combination of an increased deposition of intratubular dentin and the direct deposition of mineral crystals into the narrowed dentin tubules to decrease dentin permeability is the first defense to caries and is called dentin sclerosis. The combination of increased deposition of intratubular dentin and tubule occlusion by precipitated crystals results in an effective decrease in dentin permeability underneath the advancing carious lesion **(Pashley et al., 1991).**

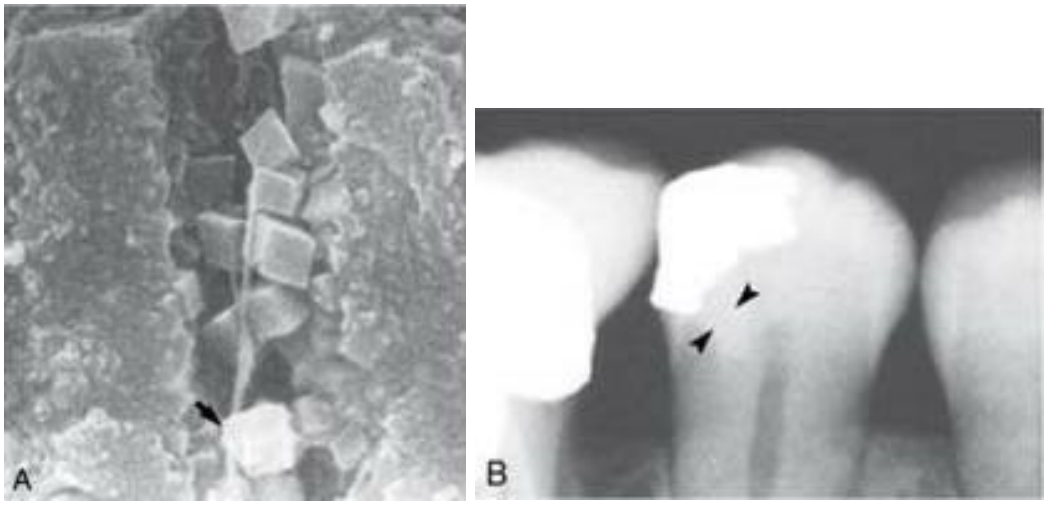

Figure 4 A, Whitlockite crystals occlude the dentinal tubules in sclerotic dentin. B, Dentinal sclerosis is radiographically apparent beneath a deep class II lesion **(Linde and dentinogenesis, 1984).**

Pulp exposure in primary and immature permanent teeth can lead to a proliferative response or hyperplastic pulpitis. Exuberant inflammatory tissue proliferates through the exposure and forms a “pulp polyp”. It is presumed that a rich blood supply facilitates this proliferative response. Conventional root canal therapy or therapeutic pulpotomy would be indicated in these cases.

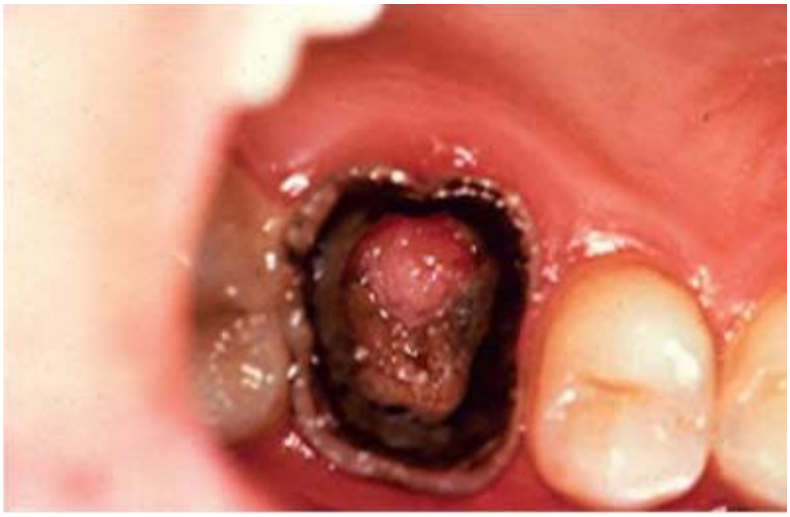

Figure 5 A proliferative response to caries in a young tooth, typically referred to as proliferative pulpitis, hyperplastic pulpitis, or pulp polyp **(Courtesy Dr. Howard Strassler at the University of Maryland, with permission).**

The importance of inflammation has been underestimated in pulpal healing, and in the past, it has been considered only as an undesirable effect. Associated with moderate inflammation, necrosis includes pyroptosis, apoptosis, and nemosis. There are now evidences that inflammation is a prerequisite for pulp healing, with series of events ahead of regeneration. Immunocompetent cells are recruited in the apical part. They slide along the root and migrate toward the crown. Due to the high alkalinity of the capping agent, pulp cells display mild inflammation, proliferate, and increase in number and size and initiate mineralization. Pulp fibroblasts become odontoblast-like cells producing type I collagen, alkaline phosphatase **(Goldberg et al., 2015).**

1.3 Diagnostic tests of the pulp condition

A differential diagnosis based on clinical symptoms and radiographic findings is the aim in assessing pulp vitality. However, an accurate determination of the pulpal condition before treatment initiation is more challenging in younger patients **(Takita et al., 2006).**

Establishing a diagnosis of reversible versus irreversible pulpitis in immature teeth can be complicated by subjective symptoms and testing responses that may not accurately reflect pulp histopathology **(Camp and Fuks, 2006).**

However, efforts should be directed toward the ultimate goal of pulpal preservation and continued apexogenesis in immature permanent teeth **(Dammaschke et al., 2019).**

A diagnosis of irreversible pulpitis, based on signs and symptoms, along with clinical testing procedures, does not preclude vital pulp therapy options. Regardless of the treatment choice of pulp capping or partial or complete pulpotomy, preservation of the radicular pulp and apical papilla allows for root maturation in cases of trauma or deep caries **(Fuks et al., 1993).**

Diagnostic quality intraoral radiographs of the involved tooth must be taken to evaluate accurately the extent of root formation and periradicular or furcation changes associated with the periodontal ligament and supporting bone **(McDonald et al., 2010).**

Because the faciolingual dimension of most immature roots is greater than the mesiodistal dimension, apical closure may be difficult to determine radiographically **(Camp, 2002).**

Before arriving at treatment decisions, the clinician should carefully assess all available information; the medical history, patient report, radiographic evidence, clinical evaluation, and vitality (cold) testing are recommended. Periodontal probing, mobility assessment, and the presence of any localized swelling or sinus tracts should be recorded during the evaluation. Radiographs, including bitewings and periapical views, should be evaluated for periapical and furcation pathosis, resorptive defects, and pulpal calcification resulting from trauma or previous restorations **(Berman and Hargreaves, 2020).**

Patients with deep carious lesions often experience sensitivity to cold, heat, or sweet or acidic foods, and cold tests may evoke a short lingering response of 1 to 2 seconds. This may not be a definitive indicator that the pulp is irreversibly damaged. Determination of the pulpal condition with the aid of contemporary testing methods can be challenging, even for experienced clinicians, because of possible excessive responses to pulp percussion and palpation testing in children **(Katge and Patil, 2017).**

Clinical evidence indicates that cold testing with carbon dioxide ice is a more reliable prognosticator of pulp status in immature permanent teeth than electronic testing devices **(Bogen and Chandler, 2010).**

Clinically, the difference between reversible and irreversible pulpitis is often determined on the basis of the duration and intensity of pain. 147 Unprovoked, spontaneous pain of long duration or unrelenting symptoms forcing sleep deprivation are consistent with irreversible pulp inflammation or an acute periapical abscess **(Bjørndal et al., 1998).**

It has been proposed that the terms initial, mild, moderate, and severe pulpitis be used to replace current terminology for reversible and irreversible pulpitis, better reflecting conditions encountered in the clinical setting. The recategorization of stages of pulpal inflammation attempts to better direct minimally invasive treatment selections using CSCs and is designed to improve outcomes for pulp tissue preservation. However, these assessments can be more complicated than previously proposed, as diagnosed pulp conditions are not always consistent with clinical findings during treatment **(Ricucci et al., 2014b).**

Teeth that experience luxation-type injuries can discolor and may not respond to cold testing for up to 4 months before they recover normal color and vitality. Also, biologically or pharmacokinetically immunosuppressed patients may not respond to conventional treatments because of abnormal function of related repair mechanisms **(Mahmoud et al., 2010).**

Most clinical investigations clearly indicate that successful outcomes for vital pulp therapy decrease as the patient's age increases. Although aging of the pulp diminishes pulpal volume, vascularity, and host immune responses, functional repair mechanisms can still provide favorable treatment outcomes in older patients **(Mjör, 2001).**

The initial pulpal diagnosis can be confirmed after visualization of the exposed pulp and assessed during tissue hemostasis. If no hemorrhaging is seen, this area of the tissue is most likely necrotic and must be removed with a high-speed round diamond bur until bleeding is evident . After hemostasis with NaOCl, a large bulk of CSC can be placed directly against the remaining tissue. Alternatively, if hemorrhage control cannot be achieved after 5 to 10 minutes of direct contact with

3% to 6% NaOCl, the pulp is likely to be irreversibly involved, and a full pulpotomy or pulpectomy is recommended **(Berman and Hargreaves, 2020).**

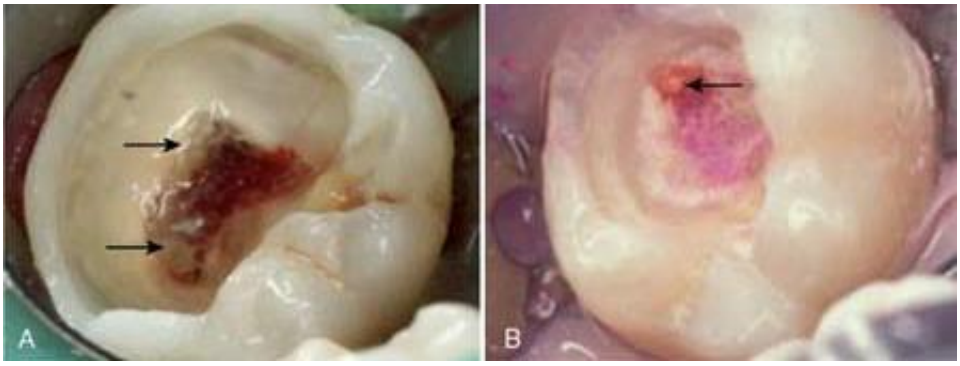

Figure 6: Clinical examples of diseased pulp tissues after sodium hypochlorite hemostasis. A, Photograph of exposed pulp tissue of a mandibular right first molar in a 13-year-old patient. Note necrotic pulp tissue (arrows) that was subsequently removed with the remaining coronal pulp during a complete pulpotomy procedure. B, Clinical presentation of mandibular right molar in a 7-year-old patient after pulp exposure during caries excavation using a caries detector dye **(Berman and Hargreaves, 2020)(Dr. George Bogen).**

Teeth that have a history of trauma or previous restorations or that display pulpal calcification have a poorer prognosis than teeth showing only initial caries. In the selection of a specific vital pulp treatment, it is important to consider the remaining tooth structure and future restorative plan. In patients with uncontrolled caries or extensive loss of coronal structure in which full coverage is indicated, pulpotomy rather than pulp capping is recommended **(Eugenio Brambilla et al., 2000).**

The foregoing clinical evidence indicates that when pain is severe, or when mild to moderate pain is present with a previous history of pain in the aching tooth,

with or without periapical radiolucency, the tooth is in the IRPP category. Treatment dictates endodontic therapy or extraction. On the other hand, when clinical evidence indicates that the pain is mild or moderate with no previous history of pain, normal pulp vitality, and there is no positive percussion sign, the pulp is in the RPP category. Treatment dictates indirect or direct pulp capping in teeth with or without periapical radiolucency. The success rate favours teeth with no periapical radiolucency, 98%; in teeth with periapical radiolucency the success rate is less favorable, 43% **(Bender, 2000).**

1.4 Deep seated carious lesion

A method for the clinical management of deep carious lesions in which the advancing carious front has approached or even reached an otherwise vital pulp exhibiting no significant untoward reactions. When a favorable history is obtained and the carious dentine is tough, non-traumatic operative procedures should be applied in the careful removal of all carious decalcified dentine except for a small amount which is left to protect the pulp from exposure. If the prognosis is favorable, restoration is completed immediately; if not, the temporary filling may be allowed to remain for as long as 6 months before final restoration **(Canby and Burnett, 1963).**

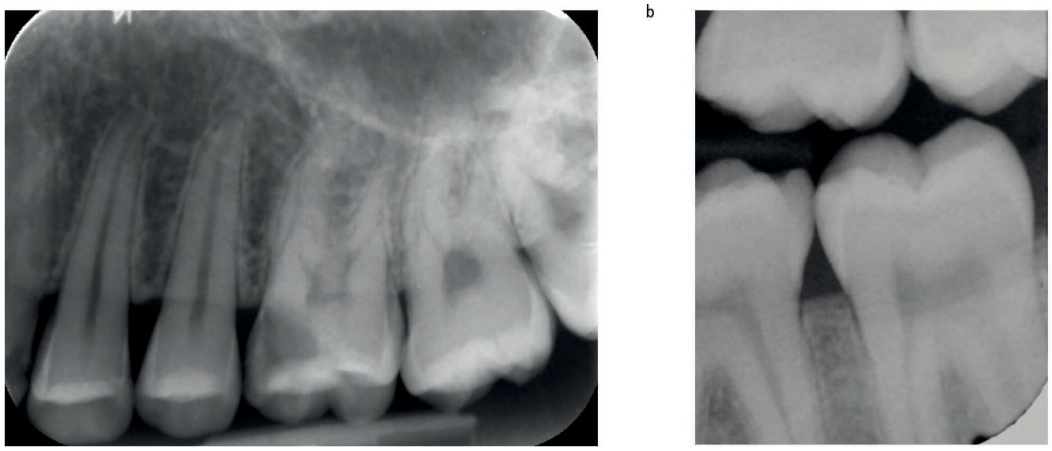

Figure 7 (A) Radiograph of a maxillary left first permanent molar with a deep carious lesion extending to the inner (pulpal) one third of dentine where preservation of pulp health should be prioritised during operative intervention; and (B) a shallow carious lesion in the mandibular left second molar (confined to the inner third of the dentine) where the tooth-restoration complex longevity might have more significance when deciding on the minimally invasive operative management options **(Banerjee et al., 2017).**

The International Caries Consensus Collaboration (ICCC) recommended the level of hardness (soft, leathery, firm, and hard dentine) as the criterion for determining the clinical consequences of the disease and defined new strategies for carious tissue removal: 1) Selective removal of carious tissue – including selective removal to soft dentine and selective removal to firm dentine; 2) stepwise removal – including stage 1, selective removal to soft dentine, and stage 2, selective removal to firm dentine 6 to 12 months later; and 3) non-selective removal to hard dentine – formerly known as complete caries removal (a traditional approach no longer recommended). Adoption of these terms will facilitate improved understanding and communication among researchers, within dental educators and the wider clinical dentistry community. Controlling the disease in cavitated carious lesions should be

attempted using methods which are aimed at biofilm removal or control first. Only when cavitated carious dentine lesions are either non-cleansable or can no longer be sealed, are restorative interventions indicated. Carious tissue is removed purely to create conditions for long-lasting restorations. Bacterially contaminated or demineralised tissues close to the pulp do not need to be removed. The evidence and, therefore these recommendations, supports minimally invasive carious lesion management, delaying entry to, and slowing down, the destructive restorative cycle by preserving tooth tissue, maintaining pulp sensibility and retaining the functional tooth-restoration complex long-term **(Banerjee et al., 2017).**

1.5 Indications for vital pulp therapy

The inflammatory response extends deeper into the pulp tissue when carious dentin is present during exposure, with possible bacterial penetration, compared to superficial inflammation when the pulp is just mechanically exposed **(Langeland, 1981).**

Pulps with profuse and lingering hemorrhaging had a significantly poorer outcome than those with modest bleeding or bleeding of short duration **(Matsuo et al., 1996).**

Clinically, bleeding should be controlled within 5 to 10 minutes.According to current recommendations in cases of irreversible pulpitis, the healing of the pulp tissue cannot be achieved predictably after removal of the triggering stimulus (e.g., caries). The diagnosis “irreversible pulpitis” often leads to the initiation of pulpectomy and root canal treatment **(Ricucci et al., 2014b).**

Maintaining pulp vitality can only be successful if bacterial infection of the tissue during and after the therapy is eliminated. Vital pulp treatments should

generally be performed on teeth that do not show pronounced and unprovoked pain symptoms or severe pain on percussion. Vital pulp therapy should not be performed without a response to the sensibility test (in which the pulp status must be verified after pulp exposure) alongside radiographic evidence of periapical disease. In cases diagnosed with irreversible pulpitis, advanced treatment options such as CSC pulpotomy should be considered, especially in younger patients. All vital pulp treatment procedures require a quality bacteria-tight coronal restoration to preclude infection, with dental dam placement, the use of sterile instruments and complete excavation of caries. If not, root canal treatment should be given preference or extraction for teeth deemed unrestorable **(Dammaschke T, 2016).**

The introduction of new bioactive materials, along with modified protocols, make more teeth with deep caries, traumatic injuries, and mechanical exposures viable candidates for innovative pulp therapies designed to maintain pulp survival. Outcomes depend on case selection, hemostatic agents, choice of pulp capping material, and the integrity of the sealed permanent restoration. The purpose in vital pulp therapy is to avoid or delay root canal treatment and advanced restorative care because these procedures, together, reduce long-term tooth survival compared to teeth with vital pulps **(Caplan et al., 2005).**

1.6 Procedures generating mineralized tissue barriers

1.6.1 Indirect pulp capping

Indirect pulp capping is defined by the American Association for Pediatric Dentistry (AAPD) as “a procedure performed in a tooth with a deep carious lesion approximating the pulp but without signs or symptoms of pulp degeneration. Indirect pulp treatment is indicated in a permanent tooth diagnosed with a normal pulp with

no signs or symptoms of pulpitis or with a diagnosis of reversible pulpitis."**(Dent, 2009).**

Hence indirect pulp capping is the permanent capping of a thin, pulp-nigh, cariously altered dentin layer, without complete excavation of the caries **(Babbush et al., 2007).**

Clinically, this situation usually arises in the excavation of an extensive carious lesion, so that indirect capping is also called treatment of deep carious lesions. A more general term would be "treatment of the pulp-nigh dentin," since indirect pulp capping may be necessary even in caries-free teeth, for example, after dental trauma **(Staehle and Pioch, 1988).**

Because only a minimal dentin layer remains above the pulp tissue, there is a risk of irreversible inflammation of the pulp via the dentinal tubules **(Murray et al., 2003).**

This can result from remaining bacteria or microorganisms actively entering the tissue and by cytotoxic components from restorative materials diffusing across thin residual dentin. Pulp-nigh dentin should be disinfected and bacteria-sealed with a capping material, stimulating the formation of tertiary dentin **(Ricucci et al., 2014a).**

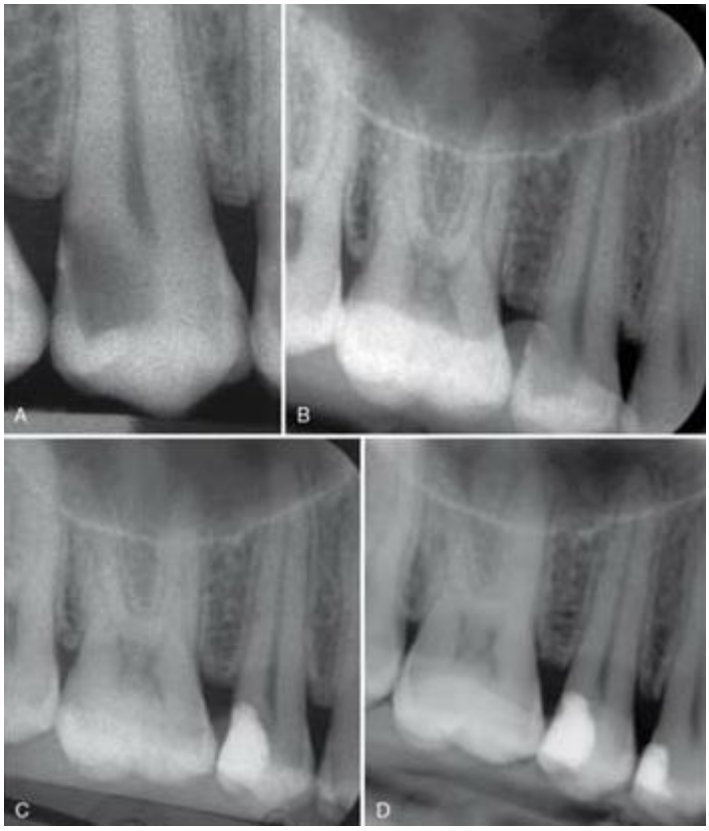

Figure 8 A 24-year-old male patient presented with a deep distal carious lesion extending to the inner pulpal quarter in a maxillary right second premolar. The tooth was symptomatic but responded normally to sensibility testing. A, Magnified bitewing radiograph showing extension of advanced caries close to coronal pulp. B, Preoperative radiograph with period indentical device for precise comparison. C, Eighteen-month radiographic review after indirect pulp capping completed with calcium hydroxide. D, Five-year radiographic control. Pulp sensibility test response was positive with a well-defined lamina dura demonstrated on the periapical radiograph **(Berman and Hargreaves, 2020)(Reprinted with permission, © Lars Bjorndal 2019.).**

Advancing microorganisms and their by-products during the carious process pose a threat to the pulp. 322 Therefore, during caries excavation, the number of microorganisms in the cavity and near the pulp should be reduced, with indirect

capping completed under dental dam isolation. To prevent spread of microorganisms, it is recommended to disinfect the clinical crown with NaOCl (1% to 5%) or chlorhexidine digluconate (CHX, 2%) prior to excavation. After caries excavation, the cavity should be cleaned with NaOCl or CHX and water spray **(Cao et al., 2016).**

It remains unclear how much altered dentin can be left while allowing the pulp to heal **(Bjørndal et al., 1998).**

Hence indirect capping materials should eliminate potential residual microorganisms, neutralize any acidic (due to the carious defect) tissues, remineralize dentin, and stimulate the pulp to form tertiary dentin **(Akhlaghi and Khademi, 2015).**

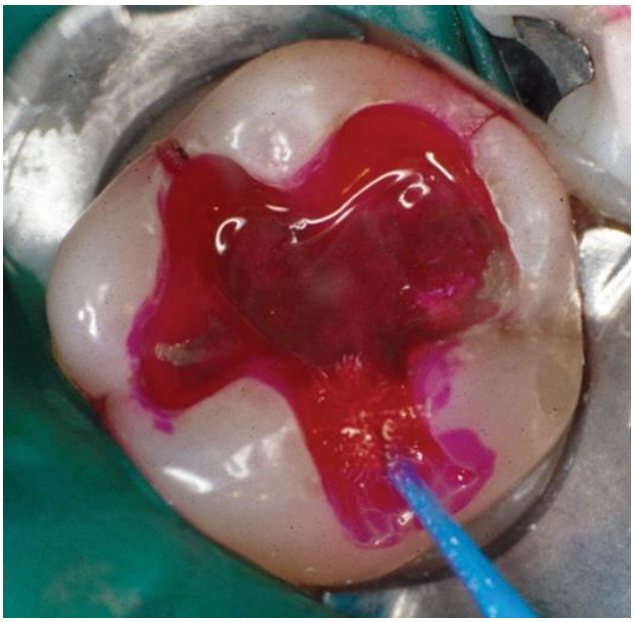

Figure 9 Caries detecting solution placed and allowed to dwell for at least 10 seconds.

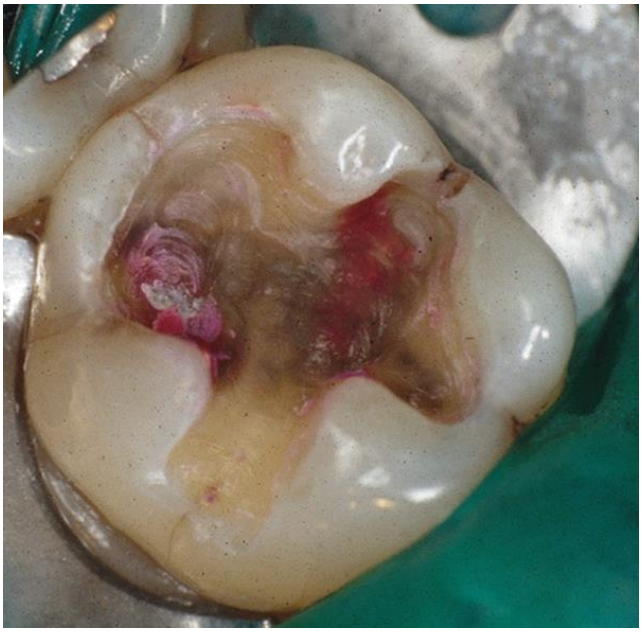

Figure 10 Tooth shown in Fig 1 after thorough rinsing and drying. Theoretically, only the caries-infected dentin picks up the stain.

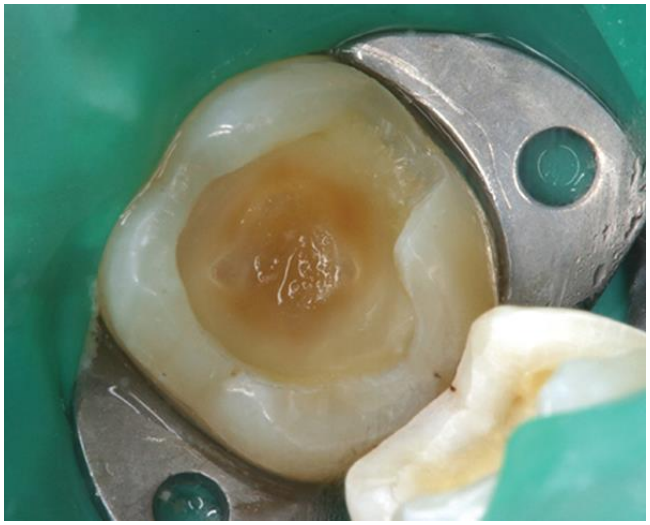

Figure 11 Deep cavity preparation showing what is most likely caries-affected dentin remaining. The preparation is cleaned and disinfected with a pumice/2% chlorhexidine mixture, rinsed, and briefly air-dried.

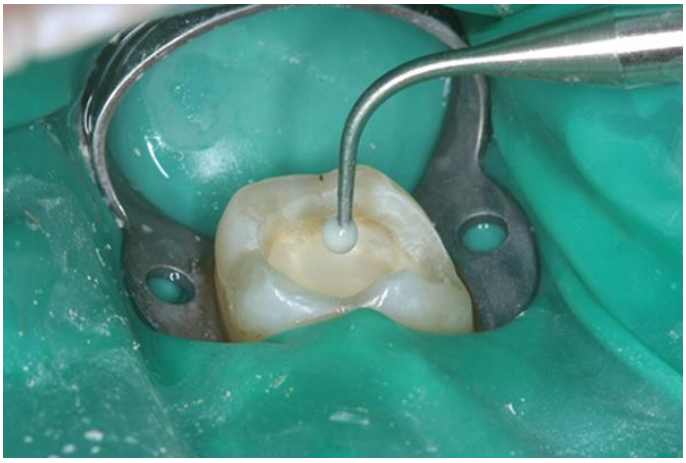

Figure 12 A thin (<1 mm) layer of a RMGI liner is placed on the floor and run up the walls of the preparation with a Dycal applicator.

Figure 13 RMGI liner is light-polymerized for at least 20 seconds.

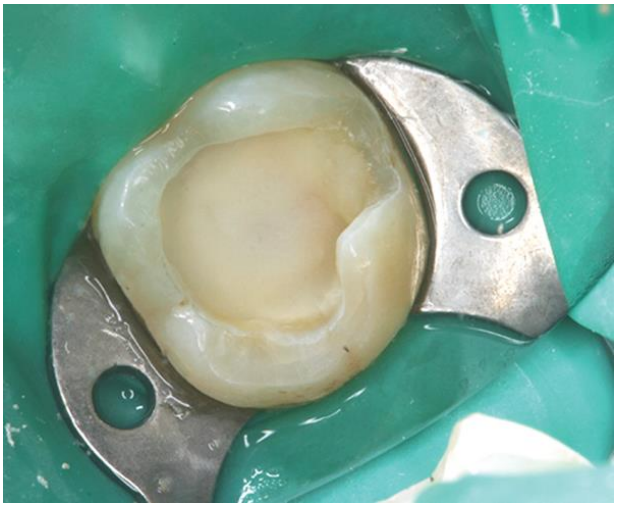

Figure 14 Finished indirect pulp cap with RMGI liner. **(Alex, 2018)**

Contraindication of indirect pulp capping is wide spread inflammation or evidence of periapical pathosis.

1.6.2 Direct pulp capping

Direct pulp capping is defined as "placing a dental material directly on a mechanical or traumatic vital pulp exposure" and "sealing the pulpal wound to facilitate the formation of reparative dentin and maintenance of a vital pulp." The procedure is indicated for pulp exposures incurred as a result of caries removal, trauma, or tooth preparation. When mechanical exposures occur during tooth preparation, the exposed tissue is generally not inflamed. However, in cases of trauma or carious exposure, the degree of inflammation is the key predetermining prognostic factor. According to the American Association of Endodontists (AAE), "In a carious pulp exposure, underlying pulp is inflamed to a varying or unknown extent." **(McClanahan et al., 2020).**

Direct capping is therefore geared towards the healing of the pulp and the preservation of the tooth. If the pulp survives this treatment (remains vital), this can be considered a clinical success. Direct capping is a noninvasive, relatively simple

and inexpensive treatment technique that, unlike root canal treatment, often does not require extensive restorative care **(Dammaschke et al., 2010).**

Outcomes of DPC in a carious field show significant variation and the procedure remains controversial.Some recent publications therefore recommend selective excavation and leaving carious tissue in the tooth, suggesting improved performance over direct capping. For a direct comparison of the two treatments, there are currently only three clinical studies available, two comparing 1- and 5-year data and the third showing 1- year data. In the second study, the prognosis of the two-stage excavation was compared with complete caries excavation treated by DPC or partial pulpotomy. 51 Five years after two-step caries excavation, the pulp was vital in 60% of the cases, whereas in the same period the success rates after direct capping were only 6% and after partial pulpotomy 11% **(Bjørndal et al., 2017).**

The reason for lower success rates may include the absence of disinfection after pulp exposures, the choice of Dycal (Dentsply, Konstanz, Germany) as capping material, and the use of long-term provisional restorations.However, in the third study, where no pulp exposures were detected after complete caries removal, indirect pulp capping resulted in a 100% success rate at 12 months. The results indicate that CSCs may be preferable in indirect pulp capping procedures and that hard setting CH cements do not provide long- or shortterm pulpal protection **(Harms et al., 2019).**

Contraindication for Direct Pulp Capping is cariously exposed deciduous teeth,swelling,fistula,tendernees on percussion,root reasorption,pulp calcification,and profuse hemorrhage from the exposed site.

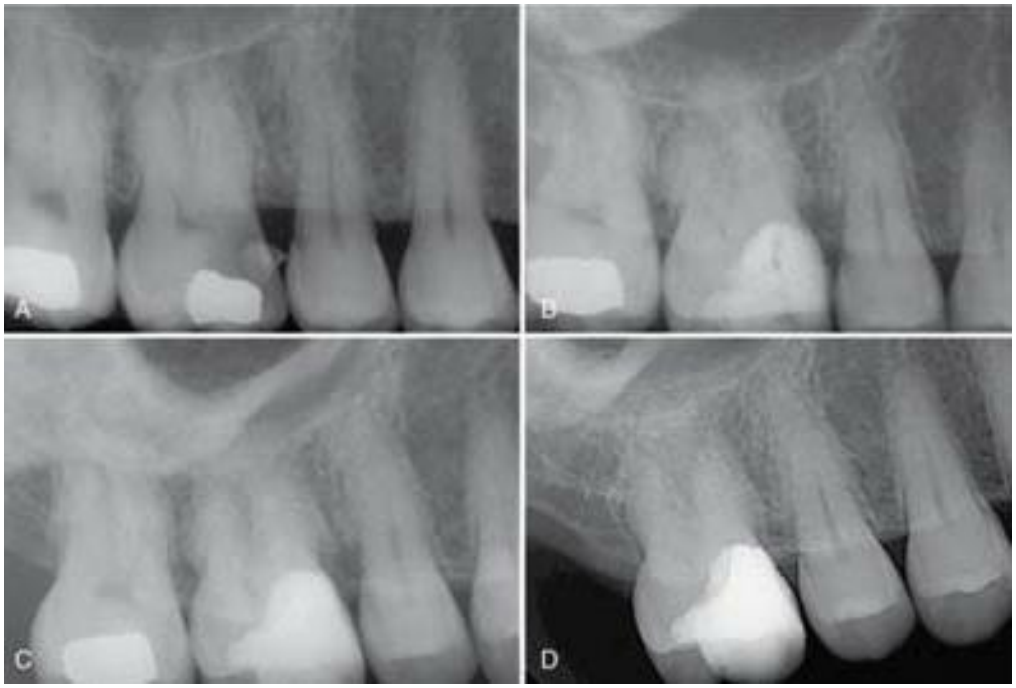

Figure 15 A 51-year-old patient who presented with a deeply carious but asymptomatic maxillary right first molar. A, Preoperative radiograph reveals extensive mesial caries and an occlusal amalgam. B, Postoperative radiograph after 1.5 mm pulpal exposure, sodium hypochlorite hemostasis, mineral trioxide aggregate (ProRoot MTA, Tulsa/Dentsply, Tulsa, OK) direct pulp cap, wet cotton pellet placement, and Photocore provisionalization. C, Radiograph 1 week after pulp capping and placement of a permanent bonded composite restoration. The patient was asymptomatic and showed a positive response to cold testing. D, Six-year radiographic recall; cold testing revealed normal vitality **(Berman and Hargreaves, 2020)(© Dr. George Bogen.).**

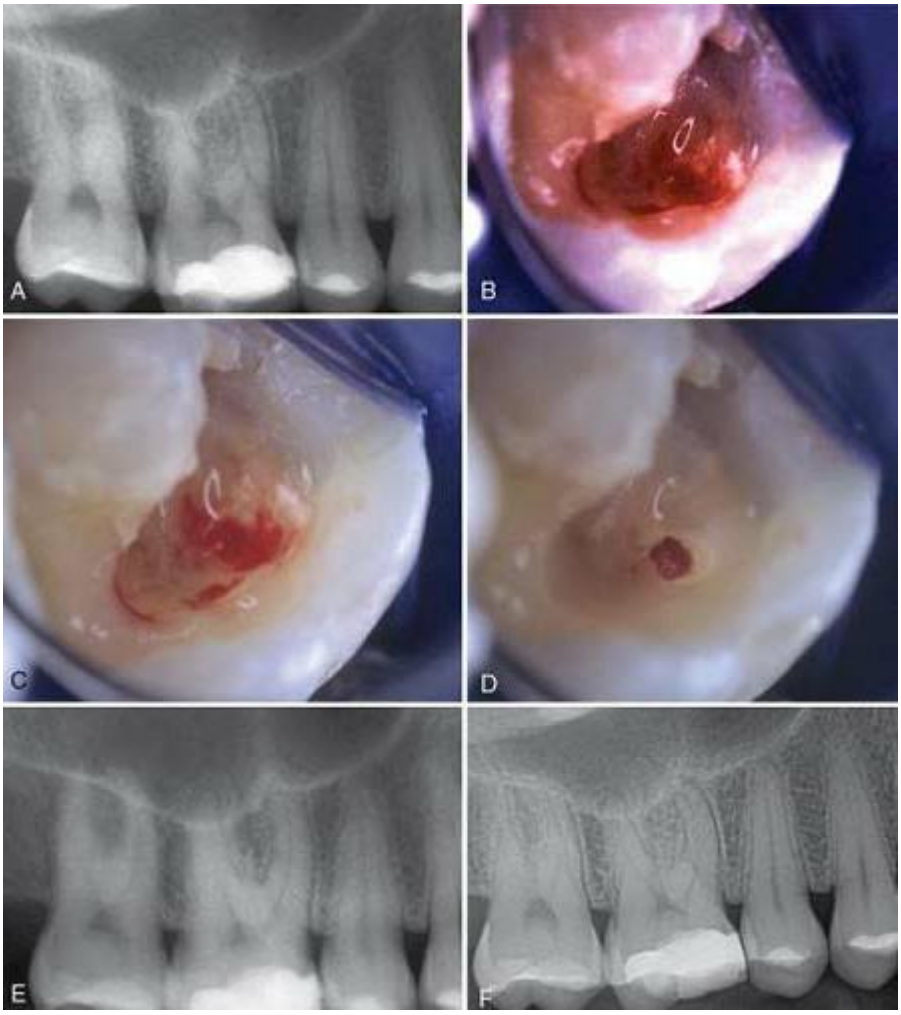

Figure 16 Thirty-four-year-old female patient presented for root canal treatment with temporary restoration 1 week after caries excavation and pulp exposure of the maxillary right first molar (#3). The patient had mild discomfort and responded with a short lingering pain to cold testing. A, Periapical radiograph showing temporary restoration in tooth #3 with normal apical structures. B, Photograph of pulpal hemorrhage after temporary removal. C, Clinical photograph showing diminished bleeding after 5-minute exposure to 5.25% sodium hypochlorite. D, Photograph showing hemorrhage controlled in 10 minutes and 2 mm pulp exposure after continued caries excavation. The exposure and peripheral dentin received bulk OrthoMTA (BioMTA, Seoul, Korea) pulp capping and was covered with a thin wet

cotton pellet and provisionalized with Photocore. The tooth was restored permanently with composite resin 1 week later. E, Two-year 6-month radiographic follow-up; tooth responded positive to sensibility testing. F, Control radiograph at 4 years. Patient exhibited normal pulp testing response and was asymptomatic with normal mobility and probings **(Berman and Hargreaves, 2020) (© Dr. George Bogen.).**

1.6.3 Partial pulpotomy

Partial pulpotomy (shallow or Cvek pulpotomy) is defined as the removal of a small portion of the vital coronal pulp as a means of preserving the remaining coronal and radicular pulp tissues **(McClanahan et al., 2020).**

During clinical treatment, the decision to remove a small or large portion of the coronal pulp is based on visual inspection of the tissue after pulp exposure during either caries excavation or exposure as a result of trauma (partial or shallow pulpotomy) **(Fuks et al., 1987).**

The coronal pulp is reduced from the exposed site by approximately 2 to 3 mm in order to remove necrotic or potentially inflamed and irreversibly damaged tissue, keeping the remaining pulp vital **(Bimstein and Rotstein, 2016).**

Partial pulpotomy is preferably performed with a high-speed handpiece and a small round diamond bur with copious water spray or continuous rinsing with physiological saline solution **(journal, 2006).**

After partial tissue removal, if bleeding cannot be controlled after 5 to 10 minutes of direct exposure to NaOCl, complete removal of the coronal pulp to the pulp floor is the preferred option **(Kang et al., 2017).**

NaOCl is a valuable diagnostic as well as clinical tool to differentiate irreversible from reversible pulpitis in the coronal pulp in order to help determine whether to proceed with partial pulpotomy, complete pulpotomy, or pulpectomy. This decision can be of paramount importance in young permanent teeth with open

apices, in which removal of tissue contaminated by microorganisms can reverse symptoms and stabilize inflamed tissue **(Eghbal et al., 2009).**

The removal of 2 to 3 mm of exposed coronal tissue or radicular tissue to access the deeper, healthy tissue, in cases of trauma, carious exposures or complete pulptomies can ensure pulp survival **(Cvek, 1978).**

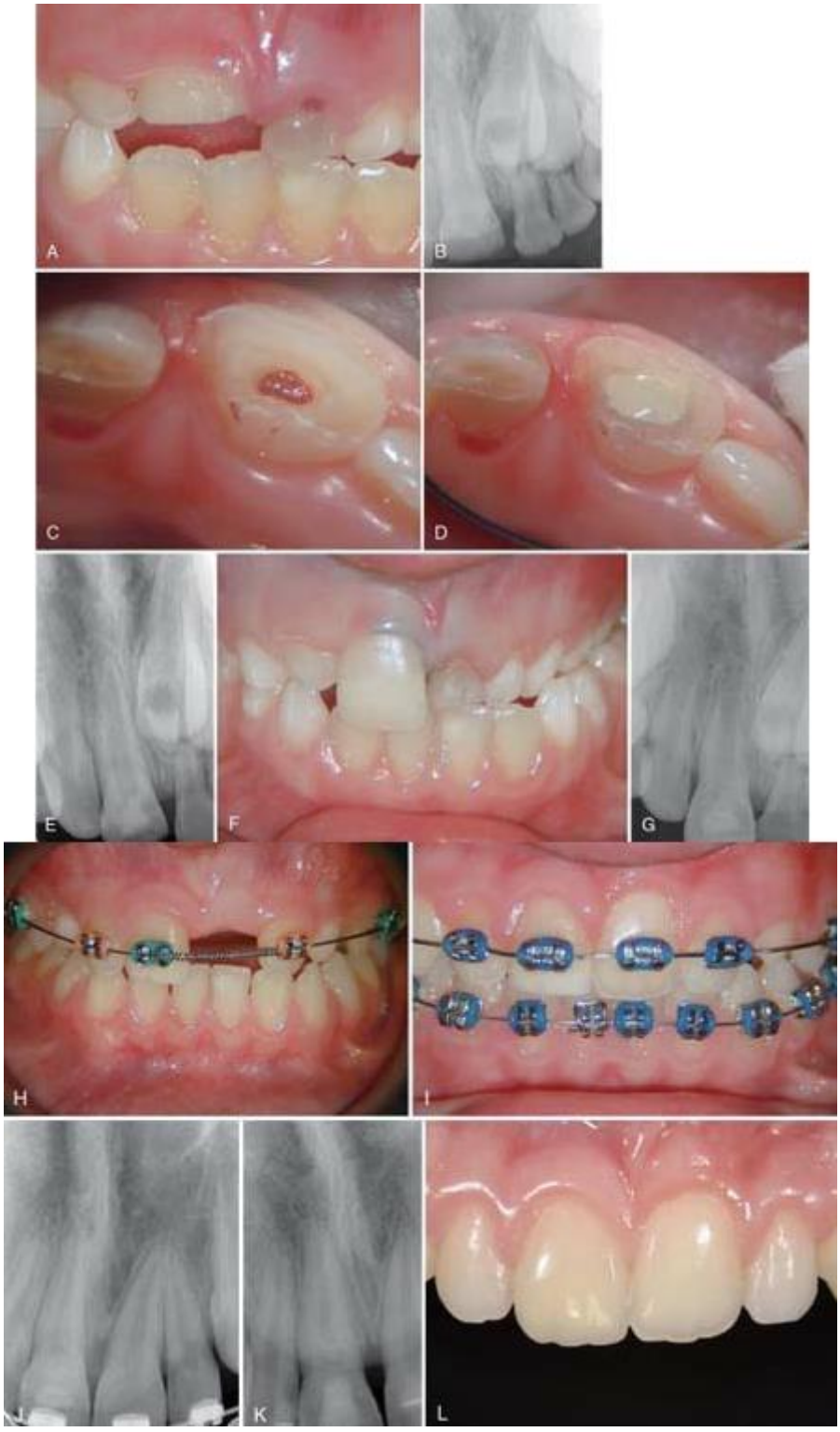

Figure 17 A 7-year-old male patient referred after trauma displaying a horizontal coronal fracture of his maxillary right central incisor #8 with a pulp exposure

covered with a glass ionomer cements by the general dentist and a retained deciduous left central incisor (F). A, Clinical photograph showing horizontally fractured right maxillary incisor and retained deciduous left central incisor. B, Periapical radiograph reveals open apex #8 and unerupted maxillary left permanent incisor #9. In addition, a mesiodens is visible. C, Photograph after shallow Cvek coronal pulpotomy. D, View of access cavity after sodium hypochlorite hemostasis and placement of 3 to 4 mm Biodentine (Septodont, SaintMaur-des-Fossés, France) plug. E, Radiograph after pulpotomy and temporary restoration placement. F, Clinical photograph 3 days after verified calcium silicate cements setting and reattachment of the fractured coronal segment after bonding. G, Radiograph of #8 with bonded crown segment reattachment showing immature open apex. H, Clinical photograph of full mouth banding after extraction of (F) and the mesiodens to permit normal eruption of tooth #9. I, Four-year clinical photograph with full orthodontic banding and complete eruption of #9 into the occlusal plane. Tooth #8 responded normally to cold sensibility testing at 1-, 2-, and 3-year recalls. J. Four-year periapical radiograph showing root-end closure of tooth #8 with normal periapical appearance. K, Five-year radiographic review of tooth #8 demonstrating complete radicular maturation. The incisor responded positive to cold testing. L, Clinical photograph at 5 years after finishing of a resin composite veneer created with a layering technique on tooth #8 **(Berman and Hargreaves, 2020)(Courtesy Dr. Marga H. Ree with restoration by Dr. Caroline Werkhoven, Amsterdam, Netherlands.).**

A current study examining MTA versus CH pulpotomy has shown superior performance by MTA in mature permanent teeth compared with a hard-setting CH (Dycal). Since the capping material is used in larger quantities in the partial pulpotomy than in the DPC, the risk of tooth staining is considered higher when using a bismuth oxide containing CSC **(Krastl and Weiger, 2014).**

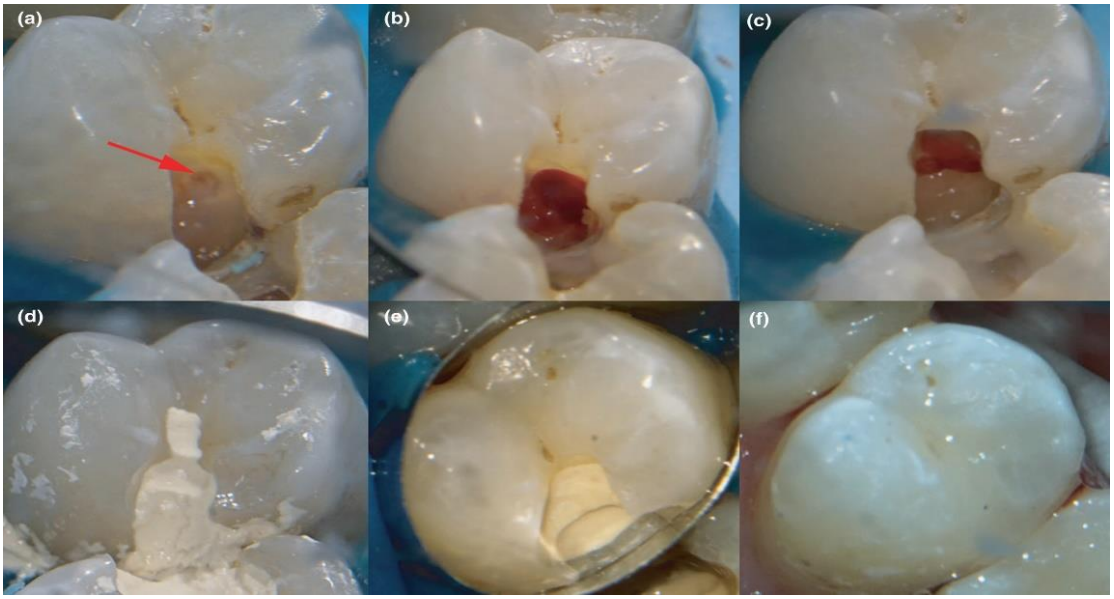

Figure 18 Partial pulpotomy clinical protocol. (a) Pulp exposure after non-selective caries removal (red arrow). (b) Superficial pulp tissue removed (to depth of 2–3 mm). Pulpal haemorrhage evident. (c) Haemostasis obtained after several minutes by placing cotton pellet soaked in 2.5% NaOCl, pressed against the exposed pulp tissue. (d) Biodentine™ used to restore the entire cavity. (e) Superficial removal of Biodentine™ at the second visit 1 week later. At least 3 mm of material is left above the exposure site before restoration. (f) Tooth permanently restored with resin-based composite material **(Careddu and Duncan, 2021).**

Contraindications of Pulpotomy is Tenderness on percussion, Root resorption more than 1/3rd of root length, Medical contradictions like heart disease, immunocompromised patient, Swelling or fistula, root resorption, Pathological mobility, Calcification of pulp.

Thirty-one studies have been included in a review on the outcomes of pulpotomy. Success rate varied from 42%–100% in cohort studies and clinical trials. The outcome criteria for pulpotomy in permanent teeth differed among studies **(Zanini et al., 2016).**

1.6.4 Complete pulpotomy

Complete pulpotomy, or complete pulp amputation, is a more intrusive procedure defined by the AAE as "the removal of the coronal portion of the vital pulp as a means of preserving the vitality of the remaining radicular portion: may be performed as emergency procedure for temporary relief of symptoms or therapeutic measure, as in the instance of a Cvek pulpotomy." **(Seo et al., 2012).**

The entire coronal pulp is removed and the vital pulp tissue capped at the entrance level of the root canal orifice. After successful hemostasis, it is treated analogously to DPC **(Krastl and Weiger, 2014).**

Figure 19 A 10-year-old female was referred with a mandibular right first molar pulp exposure generated during caries excavation and temporized by the general dentist. A, Radiograph showing open apices with no evident apical pathosis. The tooth was

diagnosed with reversible pulpitis. The patient received a new resin composite buildup to provide better isolation before a complete pulpotomy was performed. B, Clinical photograph of access opening through the buildup showing a sterile cotton pellet on the pulpal floor saturated with 5% sodium hypochlorite (NaOCl) for hemostasis. C, Photograph of radicular pulp in orifices after complete pulpotomy and 5% NaOCl hemostasis D, View through access opening after 5 mm thickness white mineral trioxide aggregate (MTA) placement onto radicular pulp tissue and pulpal floor. E, Postoperative radiograph showing white MTA with wet cotton pellet and provisional restoration. One month later, MTA setting was verified and a permanent composite restoration provided. F, Radiographic review at 6 years shows apical maturation and thickening of the root walls. The patient remained asymptomatic and the tooth responded positively to electric pulp testing **(Berman and Hargreaves, 2020)(Reprinted courtesy Dr. Marga H. Ree, Purmerend, Netherlands, with permission.).**

The assessment, selection, and amount of tissue removal are dependent on observer experience and completed on a case-by-case basis using magnification. Generally, after necrotic coronal tissue is completely removed, the inflamed pulp can be partially or completely amputated to the pulp floor or cervical area (pulpotomy) in the case of molars and some premolars **(Fuks, 2000).**

Complete pulpotomy has traditionally been recommended for primary and immature permanent teeth for which short-term outcomes are generally favorable **(Zias and Numeroff, 1987).**

Since the introduction of CSCs, there has been a renewed interest in pulpotomy as an alternative option to conventional orthograde root canal treatment, particularly in mature adult permanent teeth. Ongoing clinical investigations strongly support the use of CSCs for pulpotomy procedures in these teeth and show

acceptable outcomes in pain reduction with continued normal function **(Uesrichai et al., 2019).**

1.6.5 Pulpectomy

A pulpectomy is defined as a root canal procedure for pulp tissue that is irreversibly infected or necrotic as a result of caries or traumatic injury**.(Convissar, 2015)**

In pulpectomy, a resorbable material such as nonreinforced zinc/oxide eugenol (ZOE), a combination paste of iodoform and calcium hydroxide (Vitapex, Metapex) or a combination paste of zinc oxide and eugenol, iodoform and calcium hydroxide (Endoflas) are used to fill the canals **(Subramaniam and Gilhotra, 2011).**

It is indicated for any primary tooth with absence of its permanent successor,severe pulp necrosis.

It is contraindicated for patient with systemic illness, excessive tooth mobility,internal root reasorption in the apical third.

1.7 Materials for vital pulp therapy

1.7.1 Aqueous calcium hydroxide suspensions

CH has long been considered the universal standard material for vital pulp therapy. Although the material demonstrates many advantageous properties, long-term study outcomes in vital pulp therapy have been inconsiste.nt **(Hørsted et al., 1985)**

Desirable characteristics of CH include an initial high alkaline pH, which is responsible for stimulating fibroblasts and enzyme systems. It neutralizes the low pH of acids, shows antibacterial properties, and promotes pulp tissue defense mechanisms and repair. The drawbacks of CH include weak marginal adaptation to dentin, degradation and dissolution over time, and resorption in primary teeth.

Histologically, CH demonstrates cytotoxicity in cell cultures and has been shown to induce pulp cell apoptosis **(Poulsen et al., 2001).**

However, the primary difference between the two agents is that CH products are absorbable over time and dimensionally unstable. The slow disintegration of the CH after mineralized tissue formation can allow microleakage, permitting a slow ingress of microorganisms through defects. This can induce subsequent pulpal degeneration and lead to potential dystrophic calcification and pulp necrosis. Over extended periods, this problematic outcome can complicate any necessary nonsurgical root canal treatment at a later date **(Mohammadi and Dummer, 2011).**

A study observed 1075 teeth directly pulp capped with a CH-based agent; these teeth had either healthy pulps or showed signs of reversible pulpitis **(Neelakantan et al., 2012).**

Inclusion criteria limited pulp chamber roof exposures to no larger than 2 mm in diameter. Successful outcomes were 80.1% after 1 year and 68% after 5 years; this diminished to 58.7% after a 9-year observation period **(Willershausen et al., 2011).**

CH clearly has many favorable characteristics, but it can no longer be considered the preferred agent in vital pulp therapy.

1.7.2 Light-curing liners and cements

light-curing liners and cements with CH additives were developed, . These materials do not have sufficient mechanical strength despite their composite content, and specific CH effects are minimal.Moreover, the pH of these products is significantly lower compared to other CH products **(Subramaniam et al., 2006).**

The cytotoxicity of these products, which is due to the composite component, has been clearly demonstrated **(Poggio et al., 2014).**

After 1 day of direct contact with TheraCal LC, the fibroblast cell turnover rate decreased by 31.5% and after 1 week to 45.9%. 184 After 72 hours of contact

with TheraCal LC there was a marked decrease in the percentage survival of the cells **(Poggio et al., 2015).**

It is currently inadvisable to use photopolymerizable CH or calcium silicate-containing materials for indirect or DPC.

1.7.3 Composite resins, dentin adhesives, and resin-modified glass ionomer cements

Investigations that have examined responses to resin-based materials in human teeth have demonstrated unfavorable histologic reactions when the material is placed directly or in close proximity to pulp tissue. Histological material typically demonstrates inflammatory cell infiltrates consistent with pulp cell cytotoxicity, subclinical adhesive failures at the pulp interface, and a profound absence of biocompatibility **(Ranly and Garcia-Godoy, 2000).**

The application of dentin adhesive to a thin layer of dentin (0.5 mm) leads to an expansion of the blood vessels and chronic inflammation of the pulp tissue **(Hebling et al., 1999).**

In clinical studies the use of dentin adhesives in deep cavities results in relatively frequent pulp necrosis **(Schuurs et al., 2000).**

Dentin adhesives and composites are essentially not biocompatible and should not be used for indirect or DPC **(de Souza Costa et al., 2000).**

However, hydrophilic resins and RMGI cements provide excellent seals when they are combined with light-cured composites as permanent restorations and placed directly over pulp capping materials such as MTA and other CSCs **(Eid et al., 2012).**

1.7.4 Mineral trioxide aggregate

MTA was introduced as a pulp capping agent by Torabinejad and associates in the mid-1990s **(Faraco Junior and Holland, 2004).**

The material exhibits favorable physicochemical characteristics that stimulate reparative dentinogenesis by recruitment and activation of hard tissue–forming cells, contributing to matrix formation and mineralization **(Okiji and Yoshiba, 2009).**

MTA demonstrates superior marginal adaptation to dentin compared to CH-based agents and forms a dense bond to dentin. Mineral components from the cement penetrate into the tubules, producing adhesion to dentin comparable to GICs **(Kaup et al., 2015).**

MTA activates the migration of progenitor cells (fibroblasts) from the central pulp to the injury site and promotes their proliferation and differentiation into odontoblast-like cells without inducing pulp cell apoptosis **(Okiji and Yoshiba, 2009).**

The biocompatibility of set MTA upregulates the expression of transcription factors, angiogenic factors, and gene products, such as dentin sialoprotein, osteocalcin, and alkaline phosphatase **(Paranjpe et al., 2011).**

Overall, the data indicate that MTA promotes a biocompatible, noncytotoxic, antibacterial environment and surface morphology that is favorable for reparative calcific bridge formation. MTA stimulates the release of the dentin matrix components necessary for hard tissue repair and regeneration in mechanically exposed healthy and partially inflamed pulps **(Nair et al., 2008).**

One disadvantage of MTA is that it can lead to discoloration of the hard tooth tissue. This may be problematic, particularly in anterior teeth during trauma management **(Możyńska et al., 2017).**

It is due to heavy metals contained in MTA, such as bismuth oxide used for radiopacity or iron. The discoloration is mainly induced by the oxidation of these metals after contact with NaOCl or the uptake of blood components **(Shokouhinejad et al., 2016).**

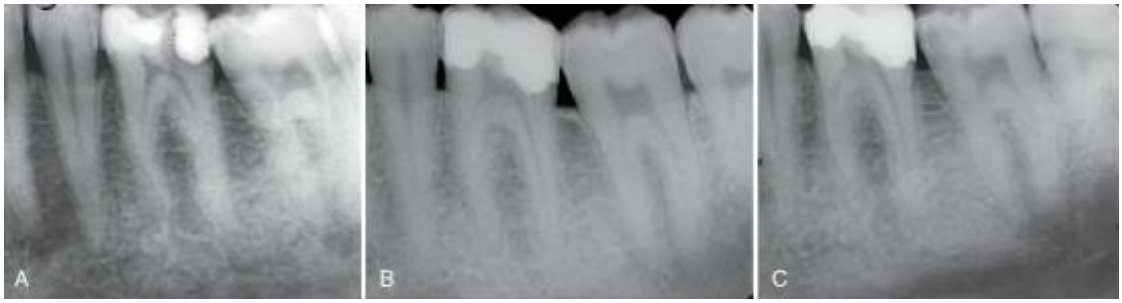

Figure 20 Symptomatic mandibular left first molar in a 32-year-old male patient with temporary restoration placed over deep caries on the distal aspect and referred for root canal treatment. Sensibility testing elicited a normal pulpal response. A, Periapical film showing deep caries extending towards the distal pulp horn. B, Radiograph of direct mineral trioxide aggregate pulp capping after 5.25% sodium hypochlorite hemostasis and placement of a three-surface bonded composite restoration. C, Nine-year 6-month radiographic control. The tooth was positive to cold testing and without symptoms. Note presence of mesial carious lesion in the second molar **(Berman and Hargreaves, 2020)(© Dr. George Bogen.).**

1.7.5 Calcium silicate cements

Preliminary investigations have demonstrated physicochemical and bioinductive properties comparable to MTA, indicating promise for their application in vital pulp therapy **(Dreger et al., 2012).**

Some of these tricalcium-based materials include BioAggregate , Biodentine, MTA-Angelus, MTA Bio, and MTA Branco **(Gonçalves et al., 2010).**

The main components of MTA and the new CSCs are tricalcium silicate and dicalcium silicate, the major components of Portland cement. Hydraulic tricalcium silicates promote reparative barrier formation by the upregulation of transcription factors after gaining immediate strength on hydration. The cements also encourage hydroxyapatite crystal formation on the cement surface when in contact with calcium- and phosphate-containing fluids **(Han and Okiji, 2011).**

Moreover, the release of CH from CSCs during hydration has a positive effect on cell regeneration. Osteoblasts, cementoblasts, periodontal ligament cells, and pulp cells are deposited directly on the CSC surface, as the material is recognized as "non-foreign," which affirms the high biocompatibility of these cements **(Torabinejad and Parirokh, 2010).**

BioAggregate is a bioinductive tricalcium cement that can induce mineralization in osteoblast cells by increasing levels of osteocalcin, collagen type 1, and osteopontin gene expression **(Yuan et al., 2010).**

The material also shows a greater resistance to dislodgement in an acidic environment compared to MTA, and higher fracture resistance when used as a filling material **(Tuna et al., 2011).**

Biodentine consists mainly of pure tricalcium silicate (about 80%) with calcium carbonate as filler (about 15%) **(Dammaschke et al., 2005).**

Zirconium oxide is added as radiopacifier (about 5%). In contrast to other CSCs and MTA, Biodentine does not contain dicalcium silicate or metal oxides except the radiopacifier **(Camilleri et al., 2013).**

It is considered a biocompatible dentin replacement material for use under restorative materials as a base **(Laurent et al., 2008).**

The material stimulates biomineralization and encourages hard tissue formation when used as a capping material **(Shayegan et al., 2012).**

Histological evaluation in humans revealed complete mineralized tissue formation 6 weeks after DPC with mild to absent pulp inflammatory reactions **(Nowicka et al., 2015).**

Success rates of human DPC with Biodentine are between 82.6% and 86% after 1.5 to 2.3 years and thus in the range given for ProRoot MTA **(Mente et al., 2010).**

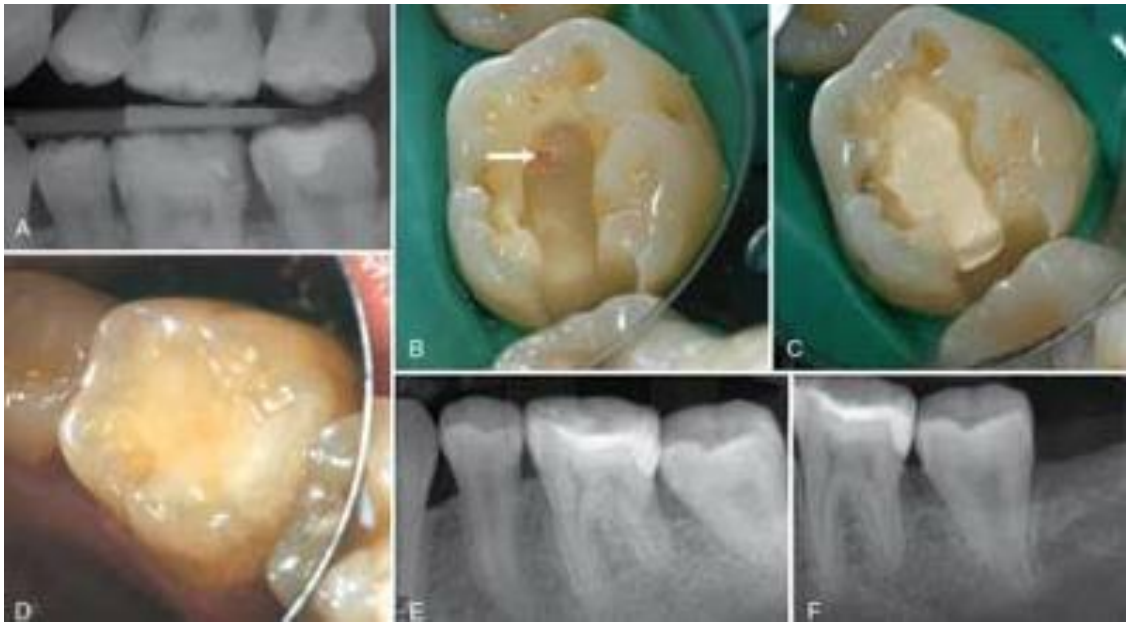

Figure 21: A, Bitewing radiograph of quadrants 2 and 3 showing defective restoration in a mandibular left first molar (#19) of a 27-yearold female patient. B, Clinical photograph showing pulp exposure (white arrow) after cavity preparation and complete caries excavation. C, Pulp capping and restorative base completed under slight pressure using Biodentine. D, Clinical photograph of final two-surface composite restoration placed after a setting time of 15 minutes. The restoration was placed after acid etching and bonded with a two-bottle dentin adhesive system. E, One-year periapical radiograph showing a normal periapical region and Biodentine radiopacity similar to that of surrounding dentin. F, Three-year radiographic review shows no evident pathological changes apically. The tooth registered normal responses to sensibility testing at both recall periods **(Berman and Hargreaves, 2020)(Reprinted with permission, Dr. Till Dammaschke and © Quintessence Deutschland.).**

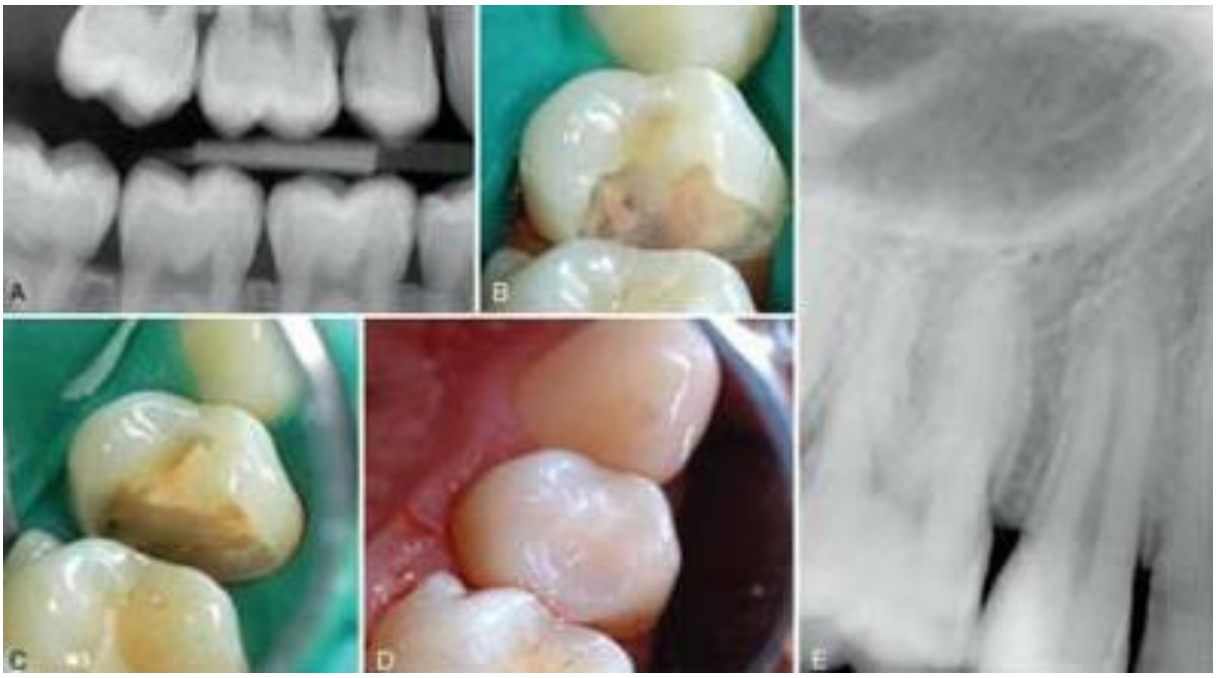

Figure 22 A, Bitewing radiograph of quadrants 1 and 4 of an 18-yearold patient displaying a distal carious lesion in the maxillary right second premolar. The patient did not present at the scheduled appointment for restorative care. B, The patient returned 4 years later for treatment. Clinical photograph showing iatrogenic pulp exposure at two sites during complete caries excavation. C, Photograph of direct capping and base placement with Biodentine applied to the cavity with cement pluggers using light pressure. D, Clinical view of cavity restored with bonded composite resin using a two-bottle dentin adhesive. E, Radiographic control recorded 4 years after direct capping showing normal periapical tissues. Tooth was asymptomatic with normal response to sensibility testing **(Berman and Hargreaves, 2020)(Reprinted with permission, Dr. Till).**

Chapter Two

Conclusion

Conclusion

Vital pulp therapy (VPT) They are techniques are means of preserving the vitality and function of the dental pulp after injury resulting from trauma, caries, or restorative procedures. VPT procedures have traditionally included indirect or direct pulp capping, and partial or complete pulpotomy.

The primary goal of VPT procedures is the creation of optimal conditions for pulp tissue repair and preservation. The amount of pulp tissue removed or retained is dependent on tissue viability assessments based on access for visualization to evaluate hemorrhage control and clinical tissue appearance.

Indirect pulp capping is defined by the American Association for Pediatric Dentistry (AAPD) as “a procedure performed in a tooth with a deep carious lesion approximating the pulp but without signs or symptoms of pulp degeneration.

Direct pulp capping is defined as “placing a dental material directly on a mechanical or traumatic vital pulp exposure” and “sealing the pulpal wound to facilitate the formation of reparative dentin and maintenance of a vital pulp.”

Partial pulpotomy (shallow or Cvek pulpotomy) is defined as the removal of a small portion of the vital coronal pulp as a means of preserving the remaining coronal and radicular pulp tissues.

Complete pulpotomy, or complete pulp amputation, is a more intrusive procedure defined by the AAE as “the removal of the coronal portion of the vital pulp as a means of preserving the vitality of the remaining radicular portion: may be performed as emergency procedure for temporary relief of symptoms or therapeutic measure, as in the instance of a Cvek pulpotomy.”

A pulpectomy is defined as a root canal procedure for pulp tissue that is irreversibly infected or necrotic as a result of caries or traumatic injury.

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