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Dental Pulp Capping: A Literature Review

Said Dhaimy[1], Asmaa Hoummadi[2*] and Sellama Nadifi[2]

[1]Department of Odontology Restorative Endodontics, Faculty of Dentistry, Hassan II University of Casablanca, Casablanca, Morocco

[2]Genetics Laboratory, Faculty of Medicine and Pharmacy, Hassan II University of Casablanca, Casablanca, Morocco

[*]**Corresponding author:** Asmaa Hoummadi, Genetics Laboratory, Faculty of Medicine and Pharmacy, Hassan II University of Casablanca, Casablanca, Morocco, Tel: +212640635922; E-mail: asmaa.hoummadi@gmail.com

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Abstract

Pulpal capping, as it has been used for more than 200 years, helps to preserve the vital pulp and the dentinogenic potential of the tooth. Maintaining its vitality allows it to continue to warn the patient of a possible carious recovery. First made with different materials, it was a resounding success. The evolution of materials, techniques and knowledge of the dentin-pulp complex has gradually improved its results. Nowadays, there are many direct and indirect pulp capping materials. Some, present for more than 50 years, like calcium hydroxide or ZOE, have proven their worth. Others, such as Biodentine or MTA, which are very recent, show promising results. The aim of this paper is to accomplish a literature review concerning this issue.

Keywords: Dental pulp capping; MTA; Biodentin; Endodontic therapy; Pulp; Dentine; Direct capping; Indirect capping

Introduction

The vitality of the pulp is extremely important for the viability of the tooth, because it provides nutrition and acts as a bio-indicator of pathogenic elements. In dental clinics, most dental.

pulp infections are irreversible due to their anatomical positio n and organization. It is difficult for the body to eliminate the inf ection, which persists and worsens later.

Endodontic therapy, also known as root canal treatment, is one of the most widely used techniques in oral care. Endodontics is a procedure that consists of removing contaminated or injured pulp tissue and replacing it with a synthetic bio-compatible material to prevent future contamination. Thanks to advances in dental bio-materials and endodontic technology, the success rate of endodontics has increased considerably over the past decade [1].

The results of some cases that were previously considered complex or whose results were uncertain, such as re-treatment of root canal treatments, are now achieving high levels of clinical success [2,3].

This article presents a review whose main objective is to provide an exhaustive synthesis of the literature on pulpal capping therapies, an operation that consists of covering the dentine and pulpal wound with protection. Its main purpose is to prevent the passage of bacteria and to promote the healing of dental pulp. Its success will depend mainly on the correct installation and the choice of material used by the dentist.

An electronic search was conducted on PUBMED, Cochrane, and "Journal of Endodontics" using the following keywords: Dental pulp capping, MTA, Biodentin. Then, and in order to identify items that would have escaped the electronic search, a manual search was performed from the list of pre-selected items.

In this article, we will first review the histopathological knowledge of the dentino-pulp complex and the etiologies of the aggressions of this complex. We will define the indirect pulp capping and then, the direct capping; it's functioning as well as its detailed indications. Finally, and after having described the direct and indirect pulp capping materials, we will compare the existing studies between the different pulp capping materials.

Physiological and pathological reminders of the dentino-pulp complex

The dentin-pulp complex is the subject of many studies to understand the response of the tooth to different care procedures; it consists of the elements detailed below.

Materials and Methods

Dentin

It is a hard tissue, radiopaque in appearance and permeable. Its main component is the mineral phase at 70% (hydroxypatite), 8% organic matrix, mainly collagen types I, V and III, matrix proteins (osteocalcin, octeonectin, octeopontin and dentin-matrix-protein), proteoglycans, albumin, growth factors (TGFB-1, IGFI, and PDGF) and metalloproteases, and 12% water [4].

The dentin is bordered by the enamel at the crown, and by the cement at the root. It circumscribes the pulp and reacts with it in a way related to the stimuli, hence the name "dentino-pulp

complex". Dentin has a particular structure as it is perforated by millions of dentine tubules or ducts, all of which converge in a centripetal way towards the pulp, and the number of these ducts varies according to the dentine zone (58000 ducts per mm^2, whereas it is now only 8000 ducts per mm^2 in contact with enamel).

Inside each dentinal canal, there is an extension of an odontoblast surrounded by its fluid. The composition of this dentine fluid is similar to that of blood plasma, but with a higher concentration of calcium ions.

In the presence of dentine exposure, overpressure causes the dentine fluid to leak into the oral cavity. This leakage of liquid to the outside limits, among other things, is the spread of bacteria to the pulp [4].

There are several varieties of dentin: Predentine, which is an unmineralized dentin organic matrix located between the odontoblastic layer and the mineralized dentin [5]; primary dentin (or orthodentine) which develops during the formation of the dental organ, until the tooth erupts into the oral cavity. It constitutes the major part of the dentin present in the tooth; secondary dentin, which corresponds to the continuous opposition of dentin at the floor and ceiling of the pulp cavity, thus reducing the shape and size of the pulp cavity throughout the life of the tooth, its formation begins when the tooth erupts on the arch; tertiary dentine (sclera or repair); this dentin and unlike the two previous ones, is secreted during a reaction process, for example in the presence of a cavity or occlusal abrasion, its main function is the protection of the pulp, its structure is very organized in relation to the primary and secondary dentin due to the speed of application of this dentin, secreted by the odontoblasts who survived the attack. The tertiary dentin thus forms a "shield" around the pulp, this dentin is easily recognized, it is translucent because completely uniform [6].

Inter-tubular dentin is located between the dentine ducts and is found everywhere in the dentin, unlike intra-tubular dentin which lines the inner part of the dentine ducts, it differs from orthodentine by a lower concentration of collagen and richer in proteoglycans and minerals. This composition explains the high hardness of intra-tubular dentin as well as its vulnerability to acid attack.

Dental pulp

The dental pulp has the particularity of being almost totally surrounded by dentin, and therefore confined in an almost closed space. At the end of the root remains an orifice, the apical foramen, through which the elements that ensure the vascularization and innervation of this tissue arrive [7].

The pulp is divided into two distinct areas: A wide area located in the dental crown, called the pulp chamber containing the so-called cameral pulp, and a narrower area located in the roots, called root canals containing the root pulp [8,9]. The dental pulp consists of several layers; an odontoblastic palisade, an acellular zone called the WEIL zone which is traversed by nerve fibres (RASHKOW plexus) and capillaries [10]; and the central area of the pulp which is composed of large vascular and nervous trunks, hence the presence of extremely fibrous connective tissue (collagen types II, IV, V, and VI); this area mainly contains fibroblasts [11,12], pulp stem cells (DPSC), immunocompetent cells next to the extracellular matrix [13].

Etiological factors of aggression: The oral cavity is colonized by many microorganisms of which oral streptococci are the major components. dental caries is considered to be the main process causing inflammation and pulp necrosis, which are caused either by direct contact of the bacteria with the pulp tissue, or indirectly through bacterial antigens travelling in the dentinal ducts, this bacterial attack is favoured in the case of structural abnormalities of the tooth such as invaginated, evaginated teeth, dentine wells and fractures that allow direct contact of the pulp tissue with bacteria.

Traumatic causes such as "accidental" fractures with or without pulp tissue exposure, cracks, but also malocclusion, abrasions, dental attritions, orthodontic or prosthetic appliances that are poorly adapted present a source of alteration of the dentino-pulp complex.

During the treatment procedures, different elements used by the dentist can cause pulp damage, such as the use of rotating instruments, air spray, dental materials such as composite resins or the aggressiveness of orthophosphoric acid used in preparation for bonding can create pulp inflammation, as can products used for "lightening", such as hydrogen peroxide, sodium perborate and carbamide peroxide.

Responses of the dentino-pulp complex to the different aggressions: As seen above, the dentin-pulp complex can be subjected to many stresses, caused by both tooth decay and restorative materials and their processing procedures.

The oral bacteria responsible for early carious infection are mainly Gram-positive (streptococcus, lactobacillus and actinomycesspp) [14]. When they penetrate dental tissues, they come into contact with the dental pulp, and trigger an inflammatory and immune response leading to its necrosis [15].

The decrease in dentinal permeability is the first of the basic reactions that tend to protect the pulp from caries, hence an increase in intra-tubular dentine deposition by the appearance of mineral crystal deposits, moderating the effects of a carious lesion, these deposits appear very rapidly, *In vitro*, it has been demonstrated that TGF beta 1 (TGF-Transforming Growth Factor) is at the origin of these deposits which are called sclera dentin.

Like other tissues in the human body, the dentin-pulpal complex develops inflammatory and immune reactions, it is a series of events that tends on the one hand to neutralize and eliminate aggression factors, and on the other hand to initiate tissue repair.

Initially, odontoblasts detect bacterial antigens through PPR (Molecular Model Recognition Receptors), which recognize PAMPs (Pathogen-Associated Molecular Patterns) [16]. These patterns are highly preserved and present only in microorganisms where they are essential for their survival. The antigen thus identified triggers the host's immune defense reactions, which begin with the activation of NF-κB (nuclear factor-kappa B).

On the surface of odontoblasts and dendritic cells, there is a variety of TollLikeReceptors (TLR), "TLR 3, 5, 9 and 2 for the odontoblast" which, once stimulated, activates the release of NF-KB within the cell **(Figure 1)**. This NF-kB activates the secretion of pro-inflammatory cytokines, chemokins and antimicrobial peptides. This is followed by recruitment and stimulation of chronic inflammation cells [5].

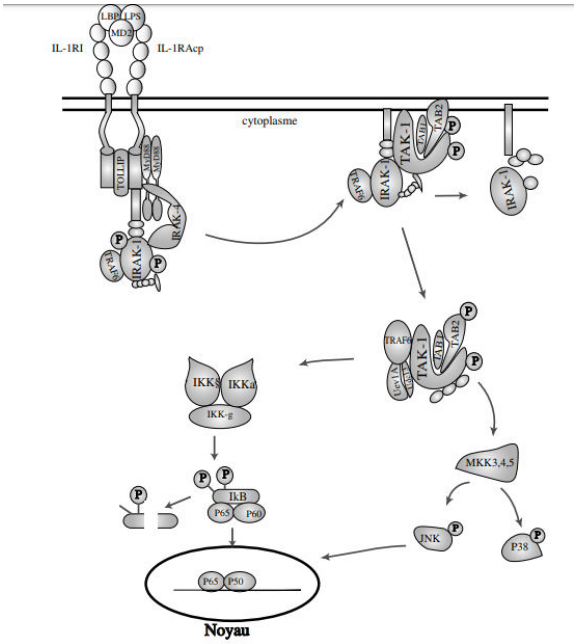

Figure 1: Presentation of IL1R/TLR Signalling Channels leading to the activation of NF-kB [17].

The fixation of the ligand on IL1R leads to the recruitment of MyD88 *via* a TIR-TIR interaction. Myd88 allows IRAK-4 to be matched to the receptor complex. Concurrently, the formation of the Tollip/IRAK-1 complex allows it to bind to MyD88 *via* its Death Domain (DD). Thanks to the IRAK-1/IRAK-4 interaction, IRAK-4 can phosphorylate IRAK-1, causing its activation. TRAF-6 will transiently interact with the complex through the activated form of IRAK-1.

The phospho-IRAK-1/TRAF-6 complex separates from the receptor complex and then interacts with the membrane complex of TAK1, TAB1 and TAB2, causing their phosphorylation and translocation in the cytosol. IRAK-1 remains at the membrane level where it will be degraded.

In the cytosol, the multimeric complex composed of TRAF-6/TAK1/TAB1/TAB2 will interact with the ubiquitin ligases Ubc13 and Uev 1A. This allows the ubiquitination ofTRAF-6 essential for the activation of TAK-1.

Once activated TAK-1 phosphorylates the IKK complex and some MAP kinase kinases (MKK) specific. IKK degrades the NF-jB I-jB inhibitor by phosphorylating it. NF-jB thus released, migrates into the nucleus where it will interact with specific promoters. The activated MKKs will in turn activate MAP-kinases of the JNK/P38 family leading to the activation of other transcriptional factors such as c-jun and c-fos.

The first-ever response to microfiltration is the presence of T lymphocytes in the extravascular space; this phase is painless for the patient, as long as these T lymphocytes do not release substances that modify the sensitivity of the surrounding nerve fibres. Then, a network of lymphocyte cells (B and T), macrophages, monocytes, and plasma cells is formed inside the pulp; these inflammatory infiltrates are dispersed in the pulp tissue.

As this inflammatory reaction progresses, many changes in pulp tissue are observed, in relation to the increase in leukocyte infiltration; there is a vasodilation of the pulp blood capillaries, and an increase in their number; a disorganization of the pulp tissue architecture may occur, accompanied by extravascular diffusion. This is called chronic inflammation.

Following this chronic inflammation phase, we are likely to have the appearance of acute inflammation foci, which are formed in the presence of certain bacterial toxins, thus damaging the pulp cells that secrete histamine, prostaglandins, and bradykinin.

During chronic inflammation, scarring of pulp tissue is observed, even in the presence of acute inflammation sites, if the etiological factors are removed.

Following these inflammation cycles, the pulp becomes less vascularized, less rich in cells, and more fibrous, it has a lower potential for repair during new attacks, it is reversible pulpitis.

If the inflammatory response is too severe, the tissue changes that have been made become irreversible, and this is called irreversible pulpitis. The pulp will then either "necrotize" over time in mute or necrotize quite quickly, causing severe pain in the patient.

It is very important to note that the age of the individual plays a very important role in the phenomenon of pulp healing; a young pulp will have more facility to heal unlike an older pulp which will have already suffered many episodes of inflammation.

Pulp capping

The pulp can be affected reversibly, irreversibly or necrosis due to multiple processes. As a reminder, the pulp is considered to be directly exposed when only 0.5 mm of residual dentine remains [4]. Several factors can cause dentine and pulp aggression.

Direct pulp capping

Direct pulp capping consists of the application of a biomaterial in direct contact with a pulp wound, in order to promote its healing and obliteration by a newly formed dentine bridge; the objective is then to induce the formation of a mineralization bridge at the pulp site exposed with a bioactive material, after eliminating all bacteria [18].

Before direct capping, the biological state of the pulp must first be determined, as it is only possible on a pulp that has been affected reversibly. It is then necessary to ensure that the treated tooth responds positively to vitality tests. The diagnosis of pulpal status should be a combination of tooth history, clinical

examination, and radiographs [19]. And to do this, the dentist has the choice between several tests, but the ideal test must be simple, fast, objective, painless, reproducible, and accurate [20].

Thermal tests (cold or hot tests) are the most commonly used, as they are easy to implement and reproducible, and allow the stimuli responsible for triggering, exacerbating or reducing pain to be easily produced. The intensity of the pain caused by the thermal tests indicates the condition of the pulp.

Among the tests used to study pulp vitality is pulsatile oximetry, which consists of measuring oxygen saturation in the blood; the machine consists of two electrodes placed on either side of the tooth. On the first electrode, a laser emits two wavelengths of light: infrared at 940 nm and red at 640 nm. Depending on the oxygen content and the haemoglobin encountered, either light is absorbed. These variations are recorded and analysed by the sensor on the other side of the tooth.

Doppler laser flow measurement is another technique widely used to determine the vitality of the pulp, it is based on measuring the blood flow within the tooth, and a probe is used to produce an infrared laser that is sent directly through the tooth. This laser interacts with both static elements (tissues) and dynamic elements (red blood cells); but only when in contact with the dynamic elements that radiation is emitted and captured by the probe, in accordance with the Doppler principle, can the state of blood flow and therefore the vitality of the pulp be determined.

For the factors that can influence pulp capping, we first find the causes of pulp exposure because they will determine the success rate of this procedure, and then we distinguish three main causes: Accidental exposure within a healthy dentin, it is produced during the preparation of a cavity or a pillar preparation of a crown, and has the highest success rate because the pulp is healthy and bacterial contamination is very limited. Exposure following a trauma, which also has a high success rate (97%) [21], even if there has been more or less long bacterial contamination. Pulpal exposure following a carious process, on the other hand, presents a strong and prolonged bacterial contamination; here the inflammation is much more important. In addition, during carious excavation, there is a risk of exacerbating the lesion by pushing infected dentin chips into the pulp. It is generally accepted that the chances of recovery are greater with small exposures at the end of the carious excavation [22-24].

The age of the pulp plays a crucial role in this pulp capping phenomenon, it is well known that young pulp has a potential for repair, and they have more stem cells and vessels, so it is better able to regulate inflammation than an older pulp that has undergone many repair phenomena, so pulp tissue will contain fewer cells and vessels and become much more fibrous [21].

The size and time of pulp exposure are among the parameters influencing direct pulp capping [25-28].

Before proceeding to direct pulp capping, the tooth must first be X-rayed to check its maturity and the size of the carious lesion, then sufficient anaesthesia must be given for the patient's comfort, then a dam must be placed to avoid any kind of bacterial contamination.

Afterwards, the cavity must be shaped, if pulp exposure takes place at the site of a cavity, the carious tissue and all restorations must first be removed with a turbine mounted diamond cutter; then the remaining small amount of dentine is placed on the pulp using a sterile tungsten cutter, mounted on a low speed contra-angle under fresh water spray [29]. If the pulp is already exposed, the exposed area should be enlarged with a sterile tungsten cutter mounted on a low-speed contra-angle handpiece and under irrigation of saline solution.

After removing the infected tissue, the debris area should be cleaned with saline solution. Sometimes, pulp exposure following a trauma is accompanied by the formation of a blood clot; it becomes necessary to remove this clot, which will prevent the success of styling, by interposing itself between the styling material and the pulp. In addition, a blood clot retains bacteria that threaten pulp vitality.

In the case of low bleeding, styling is recommended, but in the case of significant and inexhaustible bleeding, pulpotomy is the first option to consider, in which case all the tissue in the pulp chamber must be removed.

Bleeding control and disinfection are very important for successful pulp capping. It has been shown that the material placed in contact with a bloody pulp does not produce the formation of tertiary dentin, dentin bridge, and does not maintain the vitality of the tooth. To do this, a haemostatic material must be used in contact with the blood pulp. Physiological saline, or chlorhexidine [30,31], can be used.

Then it is necessary to prepare and apply the capping material properly, and there the dentist has the choice between a wide variety of pulp capping material; before proceeding to the last step which consists in a filling because these materials generally require to be covered by a thin layer of glass ionomer cement, which is in turn covered according to the aesthetic need and the physical constraints of the tooth involved (temporary crown, amalgam, composite resin) [32].

Following these steps, which usually take place in a single session; it will be necessary to carry out a long-term control of the vitality of the tooth as well as the appearance of a dentin bridge against the capping material [33-37]. The controls will consist of an X-ray, but also pulp vitality tests **(Figure 2)**.

Indirect pulp capping

Indirect capping of the dentino-pulp complex (Bonsack natural capping) is a therapy that consists of covering the dentin with a protective (and/or dentinogenic) material to promote dentino-pulp healing; and may involve a decayed or healthy dentin [4]. Indirect pulp capping consists of removing the aggression, creating a secondary dentin at the sites of pulp inflammation, suppressing the pulp inflammation, remineralizing the affected dentin and sealing the cavity to reduce the risk of new bacterial growth and proliferation **(Figure 2)**.

During indirect styling, the infected caries and dentine are removed, but before this operation, a local anaesthetic must be

performed. Carious tissue is removed using a tungsten carbide cutter mounted on a reducing counter-neck and a sharpened excavator before moving on to the removal of the infected dentine. This is difficult to identify, but there are vital dyes that help to highlight it (iodized alcohol, rhodamine, basic fuchsin).

After the removal of the cavity and the infected dentine under the dam, the cavity is sealed with a bioactive material that must be bactericidal and stimulate dentinogenesis.

The classic materials used for capping: The material used is generally CVI (glass ionomer cement) marketed under the name "gold standard", or zinc oxide eugenol (ZOE). Then, a second material is applied to the first layer that meets the following requirements: total absence of microfiltration by perfect sealing of the tooth/material interface; sufficient support of occlusal loads; and must be non-soluble.

Amalgam and composite resins are the most commonly used materials, but modified zinc oxide eugenols can be used. The purpose of this step is to trap all the remaining bacteria, isolating them from any nutrient intake. The material will help remineralize the collagen framework of the affected dentin and/or reduce pulp inflammation [4].

The material most commonly used in most capping is calicium hydroxide, is represented by the formula $Ca(OH)^2$. Its first application in dentistry dates back to 1920 by Hermann [38].

Since then, the field of action of calcium hydroxide has continued to develop and it remains, even today, a leading product in the dental surgeon's therapeutic arsenal [39].

It is obtained by hydrating quicklime (CaO). It is obtained by decarbonating a pure limestone at high temperature ($CaCO^3$).

The lack of adhesion of these materials and their porosity are secondary entry points for bacteria and their metabolites, which can lead to secondary inflammation, causing styling failure [40].

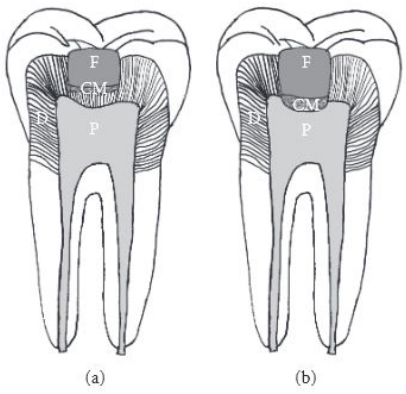

Figure 2: Diagram of pulp capping [37]; (a) Indirect pulp capping (b) Direct pulp capping. D: Dentin; P: Pulp; CM: Capping Material; F: Filler material.

The new materials: The limitations of conventional materials in pulp capping have led researchers to look for other materials to succeed in this treatment approach.

MTA:MTA or Mineral Trioxide Aggregate is a derivative of Portland cement, commonly used in building, it is composed of tri and dicalcium silicate (3CaO-SiO2, and 2CaO-SiO2), tri and tetracalcium aluminate (3CaO-Al2O3, 4CaO-Al2O3-Fe2O3) and bismuth oxide for 20% by weight, and gypsum for 5% by weight [41].

MTA has several biological properties; it is known for its anti-inflammatory action, as it does not affect the expression of inflammatory cytokines IL-10 and IL-12 (IL for inter leukine), nor does it affect phagocytosis of the marcophagus. This reaction can be explained by the gradual increase in the pH of MTA from 11 to 12.5 [42] between 0 and 4 hours.

MTA has antibacterial activity in aqueous media, thanks to its alkaline pH. It has no effect on strict anaerobic bacteria, but appears to be effective on some optional anaerobic bacteria such as *S. mitis, S. epidermis, L. species, S. salivarius, and S. mutans* [43]. It also has antifungal properties, particularly on *C. Albicans* [44].

Under MTA, the damaged pulp tissue heals by angiogenesis and neovascularization, as well as by proliferation of functional cubic cells in the vicinity of the damaged area. This angiogenesis would be stimulated by induction of the release of cytokines IL-8, cytokines involved in angogenesis [45]. Like calcium hydroxide, MTA causes coagulation necrosis upon contact with it [46]. This necrosis would be negligible in the case of MTA, unlike calcium hydroxide.

In vitro, MTA does not produce cellular apoptosis. Nor does it lead to the proliferation of odontoblast-like cells and undifferentiated pulp cells by increasing DNA synthesis within these cells.

Biodentine: Biodentine is a dentin replacement material based on calcium silicate; it is presented in the form of a capsule associated with a liquid monodose. After inserting the liquid monodose into the capsule, it must be vibrated using a three-dimensional vibrator.

Biodentine powder is mainly composed of tricalcium silicate (C3S) at 70%, dicalcium silicate (C2S) at 10%, zirconium oxide at 5%, calcium carbonate and oxides as filling material, and iron oxide in traces. For the liquid, it is composed of 100 ml of water, water-soluble polymers such as water reducing agents, and 15% calcium chloride as accelerator.

Biodentine differs from other traditional Portland cement-based materials (MTA, ProRootMTA) by the absence of aluminic components, and the presence of water-soluble polymers to ensure the balance between a low water concentration, necessary for curing the product, and a risk of porosity, followed by microfiltration and degradation of the material in the presence of too large an amount of water. It does not present any toxic risk. However, it has a lower cytotoxic risk than dycal (calcium hydroxide) and a biocompatibility similar to MTA [35].

Biodentine is a promoter of reactive dentine, mineralization and angiogenesis. Its use is therefore indicated for direct, indirect pulp capping, pulpotomies and perforation repairs [36].

MTA or biodentine: MTA is a Portland cement derivative whose first commercial form was distributed on the market as MTA pro rata in 1993, and has since been the subject of hundreds of publications [32]. Initially developed for endodontic surgery, its biological properties very quickly made it possible to develop its indications towards the treatment of perforations, the apexification of immature teeth in particular, its advantages are numerous, and this material really allowed endodontics to make a real evolution [33].

The main disadvantages of MTA are the difficulty of handling, the risk of dental discoloration due to Bismuth oxide [12,13], as well as its very high cost.

The active principle of this material is difficult to identify but calcium silicate is considered to be the most involved active principle [14].

Biodentine is a synthetic material, which is essentially composed of pure tricalcium silicate; some additives are added to modify its properties, and adapt them to clinical use.

In terms of scientific validation, the MTA is well ahead of schedule, other bioceramics appeared later, and scientific investigations are being carried out gradually.

In terms of clinical handling, MTA is not a coronary filling material. In the case of pulp capping, it is placed in first intention and, after disinfection of the cavity, it is placed in direct contact with the pulp, it is not used to protect it, ensure waterproofing, and induce the formation of a mineralized bridge; it must then necessarily be covered by a sandwich technique with a coronary obturation (direct or indirect).

Biendentin was initially developed as a coronary filling material; its biological properties (in particular bioactivity) were subsequently demonstrated.

In the case of pulp capping, the material is placed directly in contact with the pulp and then used to seal the entire coronary cavity; it allows for a capping restoration in the same session although it is advisable to allow 15 days between the capping session and the coronary reconstitution step.

Biodentine is prepared by mixing liquid powder in a capsule, and activated with the amalgamator, the ideal consistency is sometimes delicate to obtain, it is placed directly in the cavity, then it is advisable to shake it with a spatula in order to soften it and facilitate its spreading on the floor of the cavity.

In addition to pulpal (or pulpodentary) protection, these two materials have other indications for use, such as apexification, retro filling and perforation treatment.

Results and Discussion

In the literature, and as seen previously, many studies on the success of pulp capping, the tendency is to conclude that the success rate is above 70% on retrospective analyses of the activity of a professional population or even above 92% in finely controlled trials. However, we still have in mind the failures of pulpal styling and the extremely painful consequences for the patient very quickly after treatment, if these postoperative pains are an immediate sign of the failure of the procedure, we will notice that there is a form of failure that we call a long-term failure, which can be qualified by a control radiography several months apart by the placement of one or more apical lesions on the treated tooth; lesion that clearly signs pulpal necrosis and therefore, treatment failure.

These two forms of failure are ultimately linked to two completely different factors. In the presence of a deep carious lesion, it is the same one that will justify the styling procedure, we know that the pulp is systematically inflammatory; an inflammation however very circumscribed and not very widespread enough to justify the preservation of pulp vitality. But clinically how can we be sure that when we do pulp capping all the inflammatory tissue has been removed, the diagnostic tools are ultimately too unreliable and sensitive to distinguish between inflammatory tissue and healthy tissue, the most reliable test is still the control of hemorrhage after hemostasis, but how can we trust this test on a tooth that has just been anesthetized with a high concentration of vasoconstrictor, this difficulty in diagnosis explains the errors in diagnosis or rather in evaluating the inflammatory state of the pulp, and yet it is this pulpal and inflammatory state that determines the success of treatment in the short and long term. If the capping is applied on a still inflamed pulp, the procedure will accelerate the process, and from reversible pulpitis, the inflammatory status will change to irreversible status, and those in the hours following treatment, then these are immediate failures.

For long-term failures, the capping procedure consists in placing a material in direct contact with the pulp; this material will have a double effect. The first, an immediate effect of protecting the pulp by ensuring a tightness and thus preventing the passage of bacteria to the pulp parenchyma; the second expected role is to have a bioactive effect on the pulp in order to stimulate the installation of a mineralized barrier in direct contact with it; this barrier will eventually double this sealing.

Important lining because the tightness of coronal fillings decreases over time and bacteria that could infiltrate between the material and the tooth will very quickly reach this barrier, the nature and quality of the induced mineral barrier is closely dependent on the nature of the material used.

It is well known that the barrier obtained in contact with calcium hydroxide is porous, and especially that it does not adhere to adjacent dentine walls, unlike the barrier obtained with materials of the bioceramic family. MTA and Biodentine have an inductive effect on the pulp, which produces the formation of a very good quality barrier, and especially adheres to the walls, so that, even if the sealing provided by the coronary materials decreases, the passage of bacteria to the pulp is largely compromised or rather made more difficult. We have here one of the elements to explain why the long-term success of pulp capping is better when done with MTA, or biodentine, than when done with calcium hydroxide.

Conclusion

The evolution of our practices towards tissue preservation is leading us towards the conservation of pulp vitality. However,

many fear failure and prefer not to try to preserve rather than to do a systematic pulpectomy.

After many years of abandoning the concept of preserving pulp vitality once again redone face to face in dental offices, the development of materials from the family of bioceramics are largely responsible for this renewed interest as if we thought that it is thanks to these intelligent materials that pulp capping succeeds, is not completely false, but it is very far from being true.

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