International Dental Journal (2005) **55**, 293–301

# Calcium hydroxide: a review

**Alireza Farhad and Zahed Mohammadi**
**Esfahan, Iran**

Calcium hydroxide is a multipurpose agent, and there have been an increasing number of indications for its use. Some of its indications include direct and indirect pulp capping, apexogenesis, apexification, treatment of; root resorption, iatrogenic root perforations, root fractures, replanted teeth and interappointment intracanal dressing. The purpose of this paper is to review the properties and various indications for the use of calcium hydroxide.

*Key words: Calcium hydroxide, properties, clinical applications*

Undoubtedly, the most important aim of endodontics is to preserve natural teeth. In order to do so, numerous medicaments have been introduced. One of these agents is calcium hydroxide, a multipurpose agent, with an increasing number of indications for its use. First introduced to dentistry by Hermann in Germany in 1920[1], it has since been used for number of procedures, such as direct and indirect pulp capping, apexogenesis, apexification, treatment of; root resorption, iatrogenic root perforations, root fractures, replanted teeth and interappointment intracanal dressing. Despite numerous investigations its mode of action has not yet been identified completely, although it is thought that the high pH is what gives it bactericidal properties and the ability to form dentine bridges. The main purpose of this paper is to review the various indications for calcium hydroxide.

## Chemical characteristics of calcium hydroxide

Calcium hydroxide is a white odourless powder with the formula $Ca(OH)_2$, and a molecular weight of 74.08. It has low solubility in water (about 1.2g/l at 25°C), which decreases as the temperature rises; it has a high pH (about 12.5–12.8) and is insoluble in alcohol. The low solubility is a good clinical characteristic as a long period is necessary before it becomes soluble in tissue fluids when in direct contact with vital tissues.

The material is chemically classified as a strong base[2], its main actions come from the ionic dissociation of $Ca^{2+}$ and $OH^-$ ions, and their effect on vital tissues, generating the induction of hard tissue deposition and being antibacterial[3]. According to Rehman *et al.*[4] calcium hydroxide dissociates into calcium and hydroxyl ions on contact with aqueous fluids. Hydroxyl ions are believed to be responsible for the highly alkaline nature of the calcium hydroxide, which is bactericidal. Calcium ions play an important role in the initiation of the remineralisation process and although hydroxyl ions play an important role in these effects, it is difficult to accept that, by producing an alkaline pH, they are the sole initiators of the healing process.

The diffusion of $Ca^{2+}$ and $OH^-$ ions has been investigated by many researchers. Wang and Hume[5] measured hydroxyl ion diffusion across the dentine between an occlusal cavity containing calcium hydroxide and a saline-filled pulp chamber at 16 days using a pH meter. By taking ground dentine (subsequently mixed with saline) from various depths, they demonstrated a gradient of pH values from the cavity layer decreasing to the middle and pulpal layers, indicating a slow movement of the hydroxyl ion through the dentine. Importantly, they showed in a series of experiments[5] that dentine has the capacity to buffer hydroxyl ions as these ions diffuse through it.

Tronstad *et al.*[6] examined histological sections of monkey teeth one month after placement of a calcium hydroxide root canal dressing and, using indicator solutions, found that there was a pH gradient with high values around the root canal and gradually decreasing toward the peripheral dentine. The pH of cementum remained unchanged, but in resorption areas where cementum was not present, the increased pH extended to the dentine surface. Fuss *et al.*[7] measured pH changes in distilled water surrounding teeth filled with calcium hydroxide and up to 10 days detected only very small changes in pH levels. Nerwich *et al.*[8] showed that hydroxyl ions derived from a calcium hydroxide dressing diffused through root dentine. They diffused faster and reached higher levels cervically than apically. Surface pH measurements showed that hydroxyl ions did not diffuse more than a minor distance through the intact root surface.

Diffusion of the hydroxyl ions through dentine can

© 2005 FDI/World Dental Press
0020-6539/05/05293-09

be explained by dentinal permeability as well as interactions between dentine and hydroxyl ions. The permeability of dentine is governed largely by tubular anatomy, their density, diameter, and length as well as features of solute such as size and charge[9]. Hydroxyl ions may also be affected by buffering, adsorption, and charge of the dentine[8]. It is useful to consider how diffusion mechanisms affect the passage of hydroxyl ions through dentine. Initially, as hydroxyl ions diffuse into the circumpulpal dentine, the permeability of dentine is the prime factor since there is insufficient bulk of dentine to significantly buffer or adsorb the ions. As the hydroxyl ions continue to traverse through dentine, the tubule diameter diminishes and buffering and related properties become more dominant as the dentine bulk increases. The hydroxyl ions must overcome these latter effects before diffusing further. Eventually, after 2 to 3 weeks, the whole thickness of dentine is saturated with hydroxyl ions as indicated by the detection of a raised pH at the outer dentine surface. It is then that the permeability of dentine dictates the final diffusion of the hydroxyl ion[8].

When $Ca^{2+}$ ions come into contact with carbon dioxide ($CO_2$) or carbonate ions ($CO_3^-$) in tissue, calcium carbonate ($CaCO_3$) is formed which alters the mineralisation process by the overall consumption of the $Ca^{2+}$ ions. Furthermore, calcium carbonate has neither biological nor antibacterial properties[10–12].

## Preparation of calcium hydroxide paste

When calcium hydroxide powder is mixed with a suitable vehicle, water being the easiest[2], an alkaline paste with a high pH is formed[13]. Used in endodontics, it is composed of the powder, a vehicle, a radiopacifier and possibly other substances added to improve physicochemical properties or the antibacterial action.

In an *in vitro* study, Estrela and Pesce[14] showed that the type of vehicle has a direct relationship with concentration and the velocity of ionic liberation as well as the antibacterial action when the paste is carried into a contaminated area. Barbosa *et al.*[15] recommended the addition of a tensioactive agent to saturated $Ca(OH)_2$ solution to improve its antiseptic and cleansing action, while Ozcelik *et al.*[16] compared its surface tension when mixed with different vehicles, concluding that this was lowest with anaesthetic solution. The highest values were obtained with saline and Ringer's solution, which were equivalent. Addition of $Ca(OH)_2$ to the vehicles resulted in an statistically significant increase in the vehicle's surface tension.

The selection of suitable vehicles is made rather empirically. Stamos *et al.*[17] concluded that the chemical reaction that occurs between $Ca(OH)_2$ and the vehicle could cause a decrease in pH, thereby changing the therapeutic properties, although they reported no significant difference in pH values when mixed with normal saline, lidocaine, or mepivacaine.

In general, three types of vehicles are used for preparing $Ca(OH)_2$: aqueous, viscous, and oily[2]. The first group is represented by water soluble substances including water, saline, dental anaesthetics with or without vasoconstrictor, and Ringer's solution. This type of vehicle promotes the high degree of solubility when the paste remains in direct contact with tissues and tissue fluids, causing it to be rapidly solubilised and resorbed by macrophages[2]. Some viscous vehicles are also water-soluble substances that release $Ca^{2+}$ and $OH^-$ ions more slowly for extended periods. They promote the lower solubility of the paste when compared with aqueous vehicles, probably because of their high molecular weights. Some examples of viscous vehicles are glycerine, polyethyleneglycol and propyleneglycol[2].Oily vehicles are non-water-soluble substances that promote the lowest solubility and diffusion of the paste within the tissues. Some examples of these vehicles are olive oil, silicone oil, camphor, and metacresyl acetate[2].

Clinical situations requiring a rapid ionic liberation at the beginning of treatment, need an aqueous vehicle-containing calcium hydroxide paste, while those which require a gradual, uniform ionic liberation need a viscous vehicle-containing paste. Pastes containing oily vehicles have restricted use and are only employed where very low ionic dissociation is needed.

## Calcium hydroxide as an intra-canal dressing

Microorganisms and their by-products are considered the major causes of pulpal and periapical pathosis. The aim of modern endodontic treatment of teeth with apical periodontitis is total elimination of intracanal microbes. Data show that relatively few root canals will be bacteria free following only mechanical instrumentation[18].

Calcium hydroxide is considered the most effective dressing currently in use. However, it fails to consistently produce sterile root canals[19,20]. Few microorganisms will survive when directly exposed to calcium hydroxide, but several factors may impair its antimicrobial potency in the root canal system where, for example, complex anatomy will make satisfactory packing difficult[21,22].

## Mechanisms of antimicrobial activity of calcium hydroxide

Most endodontic pathogens do not survive in $Ca(OH)_2$'s highly alkaline environment[23], as Bystrom *et al.*[24] showed by the elimination of several bacterial species commonly found in infected root canals after a short period in direct contact with the material. This is due to hydroxyl ions being highly oxidant free radicals that show extreme reactivity with several biomolecules[25]. This

reactivity is high and indiscriminate, so it rarely diffuses away from sites of generation. The lethal effects of hydroxyl ions on bacterial cells are probably due to the following mechanisms:

### Damage to the bacterial cytoplasmic membrane

The bacterial cytoplasmic membrane possesses important functions to the survival of the cell, such as selective permeability and transport of solutes; electron transport and oxidative phosphorilation in aerobic species; excretion of hydrolytic exoenzymes; bearing enzymes and carrier molecules that function in the biosynthesis of DNA, cell wall polymers and membrane lipids; and bearing the receptors and other proteins of the chemotactic and other sensory transduction systems[26].

Hydroxyl ions induce lipid peroxidation, resulting in the destruction of the phospholipid structural component of the cellular membrane. Hydroxyl ions remove hydrogen atoms from unsaturated fatty acids, generating a free lipidic radical. This free lipidic radical reacts with oxygen, resulting in the formation of a lipidic peroxide radical, which removes another hydrogen atom from a second fatty acid generating another lipidic peroxide. Thus, peroxides themselves act as free radicals, initiating an autocatalytic chain reaction, and resulting in further loss of unsaturated fatty acids and extensive membrane damage[25].

### Protein denaturation

Cellular metabolism is highly dependent on enzymatic activities. Enzymes have optimum activity and stability in a narrow range of pH, which turns around neutrality. The alkalinisation provided by calcium hydroxide induces the breakdown of the ionic bond that maintains the tertiary structure of proteins. These changes frequently result in the loss of biological activity of the enzyme and disruption of the cellular metabolism[25].

### Damage to the DNA

Hydroxyl ions react with bacterial DNA and induce the splitting of the strands. Genes are then lost. Consequently, DNA replication is inhibited and the cellular activity is disarranged. Free radicals may also induce lethal mutations[25].

Bystrom *et al.*[24] in their *in vivo* study found that root canals treated with calcium hydroxide had less bacteria than those treated with camphorated phenol or camphorated monochlorophenol. They attributed this to the fact that calcium hydroxide can be packed into the root canal system allowing hydroxyl ions to be released over a long period of time. Stevens and Grossman[26], also showed calcium hydroxide to be effective in preventing the growth of microorganisms but to a limited extent when compared to CMCP, stressing the necessity of direct contact to achieve antibacterial effect.

By contrast, DiFiore *et al.*[27] found that calcium hydroxide had no antibacterial effect as a paste, or as the commercial preparation Pulpdent when used against *S. sanguis*. Siqueira *et al.*[28] demonstrated similar results. Bases of alkaline metals, such as NaOH and KOH, show high solubility and thereby may diffuse more than calcium hydroxide across the culture medium. Both bases have pronounced antibacterial activity[28]. But, high solubility and diffusibility increases the cytotoxic effects of these substances on host cells. Because of their high cytotoxicity, these substances are not indicated for endodontic practice.

Calcium hydroxide exerts antibacterial effects in the root canal system as long as the high pH is retained[25]. Although hydroxyl ions possess antibacterial effects, rather high pH values are required to destroy microorganisms. For calcium hydroxide to act as an intracanal dressing, the hydroxyl ions must be able to diffuse through dentine and pulpal tissue remnants. Studies have revealed that hydroxyl ions derived from a calcium hydroxide medication do diffuse through root dentine[6].

Several studies have attested to the inefficacy of calcium hydroxide in eliminating bacterial cells inside dentinal tubules. Haapasalo and Ørstavik[29] reported that a calcium hydroxide paste (Calasept) failed to eliminate, even superficially, *Enterococcus faecalis* in the tubules, and Safavi *et al.*[30] demonstrated that *Enterococcus faecium* remained viable in dentinal tubules after relatively extended periods of calcium hydroxide/saline mixture treatment. Ørstavik and Haapasalo[31] observed that calcium hydroxide can take up to 10 days to disinfect dentinal tubules infected by facultative bacteria. Siqueira and Uzeda[32] demonstrated that calcium hydroxide mixed with saline was ineffective in eliminating *E.faecalis* and *F.nucleatum* inside dentinal tubules even after 1 week of contact. A number of factors can help to explain the inefficacy in disinfecting the dentinal tubules. It has been reported that dentine has buffering ability because of the presence of proton donors such as $H_2PO^-_4$, $H_2CO^-_3$, and $HCO^-_3$ in the hydrated layer of hydroxyapatite to keep the pH unchanged[8]. The arrangement of bacterial cells colonising the root canal walls can also reduce the antibacterial effect of calcium hydroxide, since the cells located at the periphery of colonies can protect those located more deeply inside the tubules[32]. Ramifications, isthmuses and irregularities also protect bacteria from the action of calcium hydroxide due to pH neutralisation.

The period needed for calcium hydroxide to optimally disinfect the root canal system is still unknown. Clinical studies using the root canal sampling procedures have revealed conflicting results. Bystrom *et al.*[24] showed that calcium hydroxide effectively eliminated all microorganisms when the medicament was maintained for 4

weeks. Reit and Dahlen[19] found that infection persisted in 26% of the canals after 2 weeks of dressing with calcium hydroxide. Sjogren *et al.*[33] reported that intracanal dressing with calcium hydroxide for 1 week effectively eliminated bacteria in the root canal in 100% of cases. Barbosa *et al.*[34] reported that 26.7% of the cases dressed with calcium hydroxide for 1 week yielded a positive culture. Variation in the presented results may have methodological origins, and for example be due to the small sample sizes used in these types of studies.

A fundamental role in the cause and maintenance of periapical lesions has been attributed to the bacterial endotoxin, a lipopolysaccharide (LPS)[35]. It appears that LPS, a cell wall component of gram negative bacteria, plays a major role in the periapical bone resorption process[36]. LPS *via* its lipid A moiety stimulates secretion of bone-resorbing mediators such as prostaglandin E2 (PGE2) from a variety of host cells particularly those of immune lineage[37]. LPS is a relatively stable macromolecule. At minute concentrations LPS exerts significant biological effects[38] and thus its inactivation is highly desirable in root canal treatment. Safavi and Nichols[36,37], Barthel *et al.*[39] and Olsen *et al.*[40] demonstrated that calcium hydroxide hydrolyses lipid A, which is a toxic part of endotoxin (LPS). Recently Silva *et al.*[41] reported that calcium hydroxide inactivated the toxic effects of bacterial endotoxin *in vivo*.

They also showed in a radiographic evaluation the effect of endotoxin (LPS) plus calcium hydroxide on apical and periapical tissues of dogs that bacterial endotoxin caused radiographically visible periapical lesions, but when associated with calcium hydroxide, this endotoxin was detoxified[41]. Safavi and Nichols[36,37] reported that calcium hydroxide hydrolysed lipid A *in vitro* and after lipid A hydrolysis, this highly toxic agent released free hydroxy fatty acids that were nontoxic. The contradictions between *in vivo* and *in vitro* findings might suggest that other aspects are involved. One mechanism that can explain the *in vivo* antimicrobial activity of $Ca(OH)_2$ is its ability to absorb carbon dioxide in the root canal systems, which is essential for bacteria such as Capnocytophaga, Eikenella and Actinomyces spp.[42] and is provided by bacteria such as Fusobacterium, Bacteroides, Porphyromonas and Streptococcus spp. If calcium hydroxide absorbs carbon dioxide, $CO_2$-dependent bacteria will not survive[43]. Therefore, the use of an intracanal medicament will disturb established nutritional interrelationships, eliminating some bacteria that might be essential to the growth of others, or leaving some bacteria whose presence will prevent the growth of others.

One of the only shortcomings of calcium hydroxide is its inability to effectively kill enterococcus species, which are often associated with failed root canal treatment[25,44]. The common recovery of *Enterococcus faecalis* (*E.faecalis*) from canals where the treatment has failed suggests it is an opportunistic pathogen whose persistence in the root canal presents a significant therapeutic problem[25,44]. It appears that *E.faecalis* is highly resistant to the medicaments used during treatment and is known to resist the antibacterial effect of $Ca(OH)_2$. Evans *et al.*[44] found that pretreatment by $Ca(OH)_2$ or NaOCl was of minor importance in the ability of *E.faecalis* to survive the high alkalinity of calcium hydroxide. They showed that *E.faecalis* was resistant to $Ca(OH)_2$ at a pH of 11.1 but not of pH 11.5. Pretreatment with $Ca(OH)_2$ at pH 10.3 induced no tolerance to further exposure at pH 11.5. They also stressed that no difference in cell survival was observed when protein synthesis was blocked during stress induction, however, addition of a proton pump inhibitor such as carbonyl cyanide m-chlorophenyl hydrazone (CCCP) resulted in a dramatic reduction of cell viability of *E.faecalis* in calcium hydroxide. In the other words, they concluded that survival of *E.faecalis* in calcium hydroxide appears to be unrelated to stress induced protein synthesis, but a functioning proton pump is critical for survival of *E.faecalis* at high pH.

## Apexification

Apexification is defined as the process of creating an environment within the canal and periapical tissues after pulp death that allows a calcified barrier to form across the open apex[45]. This calcified barrier consists of osteocementum or other bone-like tissue[46].

Creation of a proper environment for formation of the calcified barrier involves cleaning and shaping of the canal to remove debris and bacteria, followed by placement of a paste to the apex[45]. Different materials have been used successfully but the most favoured is a paste of calcium hydroxide and water; the addition of other medicaments to calcium hydroxide has no beneficial effect on apexification[47]. Thorough debridement to remove bacteria and necrotic tissue from the canal system is the primary factor responsible for apical closure. Calcium hydroxide is used as a temporary obturating material and it is its bactericidal effect that may stimulate apical calcification[45]. Although apexification had been attempted in the past, the technique was given impetus by the description of three cases by Frank[48]. Frank cleaned and irrigated canals and sealed them with a paste of camphorated chlorophenol and calcium hydroxide. Radiographic examination 3 and 6 months later showed evidence of a root apex cap or barrier, following which the root canals were obturated. Actual root growth does not occur as a result of apexification, but radiographic evidence of a calcified mass at the root apex gives that impression[46].

Although apexification occurs with many materials, it has been reported even without the presence of canal-filling material after the removal of necrotic pulp tissue[49].

According to Kleier and Barr[50], the usual time

required to achieve apexification is 6 to 24 months (average l year ± 7 months). However, in one case it has been reported that 4 years of treatment was required for complete apexification[46].There is disagreement in the literature on how often the canal should be refilled with calcium hydroxide paste to produce apexification, and the decision seems to be empirical. According to Tronstad *et al.*[6] refilling every 3 to 6 months is favoured. Some of the other researchers favour refilling only if there is radiographic evidence of resorption of the paste[51]. Chosack[52] suggested that after initial root filling with calcium hydroxide there was nothing to be gained by repeated root filling either monthly or after 3 months for at least 6 months.

If any signs or symptoms of reinfection or pathology occur at any time during the periodic recall of apexification, the canal is cleaned again and refilled with calcium hydroxide paste[49]. If apexification is incomplete, the canal is repacked with the calcium hydroxide paste, and the periodic recall continues[49].

From the histological viewpoint, the calcified tissue that forms over the apical foramen has been identified as an osteoid or cementoid material[53]. Histological studies consistently report the absence of Hertwig's epithelial root sheath (HERS). However, according to Grossman[46] the residual undamaged pulp tissue, if there is any, and the odontoblastic layer associated with the pulp tissue resume. The matrix formation and subsequent calcification is guided by the reactivated HERS. He also stressed the fact that the HERS and the pulp tissue that were once damaged may explain why some of the apical formations appear atypical.

Pitt Ford[45] stated that the type of barrier that forms depends on the extent of pulp necrosis at the start of treatment. Vital (but probably inflamed) pulp may be present at the root end; following pulpectomy, canal preparation, irrigation, and insertion of calcium hydroxide, some continued root formation may be expected from the surviving HERS. However, if there is severe inflammation (or even abscess formation) in the periapex (with or without sinus tract), HERS has probably been completely destroyed.

## Apexogenesis

Apexogenesis is defined as a treatment of a vital pulp in an immature tooth to permit continued root growth and apical closure[45]. Treatment techniques for apexogenesis include shallow pulpotomy (Cvek pulpotomy) and cervical pulpotomy.

Calcium hydroxide has been the drug of choice for use in pulp capping and pulpotomy. Although suspensions of calcium hydroxide are highly alkaline, other compounds, such as ammonium hydroxide with the same alkalinity cause liquefaction necrosis of the pulp when placed on exposed pulp tissue. The calcium ions delivered to the site of the exposure by the calcium hydroxide suspension are not utilised in the repair of the exposure. Sciaky and Pisanti[54] have shown by means of radioautographs that the calcium ions present in the dentine bridge that is formed during repair come from the systemic circulation. The applied calcium hydroxide which in their experiments contained radioactive calcium did not enter into the formation of the bridge.

According to Schroder and Granath[55] the mechanism for the induction of dentine formation and repair under calcium hydroxide may be that it causes a superficial coagulation of the pulp tissue on which it is placed. The necrosis is apparently initiated by damage to the blood vessels. The initial damage from calcium hydroxide occurs in the capillaries closest to the region of the capping or pulpotomy[56]. Because of its high pH, calcium hydroxide helps to keep the immediate region in a state of alkalinity, which is necessary for bone and dentine formation. Under this region of calcium hydroxide-induced coagulation necrosis, which is saturated with calcium ions, cells from the underlying pulp tissue differentiate into odontoblast–like cells which then begin to elaborate matrix.

Schroder and Granath[57] examined the coronal surface structure of calcium hydroxide-induced bridges with both the light and the scanning electron microscope. They found tubular openings surrounded by collagen bundles similar to those found in normal predentine. It has been reported that saturated calcium and barium hydroxide completely inhibited alkaline phosphatase and lactic dehydrogenase activity, but calcium hydroxide preparations at lower pH levels were much less inhibitory[56]. Seltzer and Bender[56] stated that two undesirable side effects had been attributed to calcium hydroxide when used as a pulp capping or a pulpotomy agent: one is the possibility of eventual complete calcification of the tissue in the root canal. If this occurs, subsequent endodontic therapy, if needed, becomes a difficult and often impossible procedure. The second adverse effect is persistence of induced inflammation, eventually causing internal resorption.

## Application of calcium hydroxide for root resorption

Root resorption is defined as resorption affecting the cementum and/or dentine of the root of a tooth. On the basis of the site of origin of the resorption, it may be referred to as internal, external, or root-end resorption[58].

Calcium hydroxide has an active influence on the local environment of resorption area by reducing osteoclastic activity and stimulating repair[6]. This is directly related to the alkaline pH of the calcium hydroxide, which spreads through the dentine. Hard tissue resorption, with its enzymatic activity, takes place in an acidic pH. $Ca(OH)_2$ creates an alkaline environ-

ment in which this reaction is reversed and hard tissue deposition can take place. The phenomena of pH change to the periphery is increased, especially where resorption has exposed the dentine[6].

Frank and Weine[59] reported on a technique using the $Ca(OH)_2$-CMCP mixture for the nonsurgical treatment of perforating internal resorption. In such situations other similar techniques have been used in which the deposition of a cementum-like or osteoid tissue at the site of the defect is the end result.

Treatment of choice for internal root resortption is to pack the canal and the resorption lacuna with calcium hydroxide paste. By the next visit, the calcium hydroxide will have necrotised any remaining tissue in the lacuna, and the necrotic remnants are readily removed by irrigation with sodium hypochlorite[58]. When lateral resorption is noticed from the outset, pulp extirpation, debridement and $Ca(OH)_2$ therapy are preferred. In addition, when the lateral resorption process reaches the dentine or perforates the root canal, the $Ca(OH)_2$ procedure should be attempted after canal debridement[60]. According to Chivian[58], calcium hydroxide should be placed in to the resorption defect at 3-month intervals until there is evidence of hard tissue repair, confirmed by both radiographs and direct examination through the access cavity. When the physical barrier has been established, the defect can be filled with gutta percha. Cvek[61] stated that the arrest of external root resorption related to necrotic pulp can be attributed exclusively to removal of necrotic pulp and antibacterial treatment of the root canal. When external resorption is noticed from the outset of luxation injuries, pulp extripation, debridement, and $Ca(OH)_2$ therapy are necessary[58]. In some situations when root resorption continues after the completion of active and retentive phases of orthodontic treatment, intentional extripation and $Ca(OH)_2$ therapy seems to be successful in abating resorption[62]. Andreasen[63] was able to arrest inflammatory root resorption in nine out of ten cases by using intracanal $Ca(OH)_2$ dressing[63].

## Application of calcium hydroxide for avulsion

Endodontic treatment of avulsed teeth with closed apex and an extra-oral time of more than 60 minutes is initiated 7–10 days after the emergency visit. According to Trope[64], in cases where endodontic treatment is delayed or signs of resorption are present, 'long-term' calcium hydroxide is the treatment of choice before obturation. Trope[64] also stressed that if therapy is initiated at the optimum time (7–10 days after emergency visit), endodontic therapy with an effective interappointment antibacterial agent such as $Ca(OH)_2$ over a relatively short period of time (7–10 days) is sufficient to ensure effective disinfection of the canal. The advantage of long-term calcium hydroxide is that it allows the clinician to have a temporary obturating material in place until an intact periodontal ligament space is confirmed. In long-term calcium hydroxide therapy, $Ca(OH)_2$ is changed every 3 months within a range of 6–24 months[64]. Cvek[65] stated that long-term $Ca(OH)_2$ for avulsed teeth will result in an extremely high rate of success. However, Hammarstrom *et al.*[66] and Lengheden *et al.*[67] have warned that use of calcium hydroxide in the root canals of teeth with damaged root surfaces might be contraindicated because of its necrotising effect on the cells repopulating the root surface. Trope *et al.*[68] in another study found that there were no differences between infected teeth which were obturated with gutta-percha after 1 week of calcium hydroxide therapy.

According to Andreasen *et al.*[69] fracture strength of calcium hydroxide-filled roots would be reduced to half in about a year due to the root filling. The finding may explain the frequent reported fractures of immature teeth filled with calcium hydroxide for extended periods. They also stressed that decreased fracture strength of teeth with long-term calcium hydroxide is due to the alkaline nature of calcium hydroxide which neutralises, dissolves, or denatures some of the acidic components acting as bonding agents and thereby weakens the dentine.

## Other applications of calcium hydroxide

### *Indirect pulp capping (IPC)*

Indirect pulp therapy is a technique for avoiding pulp exposure in the treatment of teeth with deep carious lesions in which there exists no clinical evidence of pulpal degeneration or periapical disease. The main purpose is to arrest the carious process by promoting dentinal sclerosis, remineralisation of carious dentine as well as preserving pulp vitality. Many materials and drugs have been used as pulp capping agents. One of the most favourable agents is calcium hydroxide.

Calcium hydroxide is an ideal pulp protectant but it should be used only where indicated and in a very thin layer. Regular aqueous or methylcellulose calcium hydroxide fails as a base. It is biocompatible but, unfortunately, has a very low compressive strength, allowing the filling material to be crushed into it. It should be noted that in the case of IPC calcium hydroxide is being used as an antibacterial agent and mild pulp stimulant to produce irritation dentine.

Warfving *et al.*[70] found that to achieve these two objectives calcium hydroxide paste in saline was much more effective than a commercial hard-setting calcium hydroxide cement (LIFE). Another variation of calcium hydroxide is Prisma VLC Dycal, which consists of calcium hydroxide and fillers of barium sulphate dispersed in a specially formulated urethane dimethylacrylate resin containing initiators (camphoroquinone) and activators.

According to Stanley and Pameijer[71], Prisma VLC Dycal has a number of advantages over regular water- or methylcellulose-based calcium hydroxide: "dramati-

cally improved strength, essentially no solubility in acid, minimal solubility in water, control over working time, and reaching the maximum physical properties almost immediately".

### *Direct pulp capping (DPC)*

DPC involves the application of a medicament, dressing, or dental material to the exposed pulp in an attempt to preserve its vitality. Several materials have been used as pulp capping agents, but calcium hydroxide has been the standard by which all others were judged and it is generally accepted as the agent of choice. For this purpose, different forms of calcium hydroxide are available such as pure calcium hydroxide and various hard-setting $Ca(OH)_2$- containing cements. When $Ca(OH)_2$ is applied directly to pulp tissue, there is necrosis of the adjacent pulp tissue and an inflammation of the contiguous tissue.

According to Meadow and associates[72], pure calcium hydroxide necroses about 1.5mm of pulp tissue. Schroder[73] found that the high pH of 12.5 of calcium hydroxide causes a liquefaction necrosis in the most superficial layers of the pulp and the toxicity of $Ca(OH)_2$ appears to be quickly neutralised as deeper layers of pulp are affected (causing coagulative necrosis at this level). The coagulative necrotic tissue causes a mild irritation to the adjacent vital pulp tissue. This mild irritation will initiate an inflammatory response and in the absence of bacteria will heal with a hard tissue barrier. Conversely, Stanley and Lundy[74] reported that hard-setting calcium hydroxide formulations will not necrose the superficial layers of pulp and initiation of healing with a hard-tissue barrier occurs only with pure calcium hydroxide.

According to Stanley *et al.*[74] the reactions to Dycal, Prisma VLC Dycal, Life, and Nu-Cap were similar. However, in another study Stanley *et al.*[71] found that in contrast to regular Dycal, which caused a thickness of pulp mummification of 0.3-0.7mm at the exposure site, Prisma VLC Dycal caused no inflammation.

$Ca(OH)_2$ dressings of hard-setting Life and Dycal dissolve clinically within 1–2 years. Since the majority of dentine bridges under capping appear to contain tunnels, about 50% of the pulps may show infection or become necrotic due to microleakage[75]. Another problem with Dycal and Life is that they are degraded by etching and rinsing prior to restoration[76]. In newer products, such as Prisma VLC Dycal, the $Ca(OH)_2$ is incorporated in urethane dimethacrylate with initiators and accelerators by which they bind to dentine and have a higher resistance to acid dissolution[77].

## References


1. Hermann BW. Calcium hydroxyd als Mittelzurn, Behandeln und Fullen von Wurzelkanalen [thesis] Wurzburg, 1920.
2. Fava LRG, Saunders WP. Calcium hydroxide pastes: classification and clinical indications. *Int Endod J* 1999 **32:** 257–282.
3. Estrela C. Analise quimica de pastas de hidroxido de calcio frente a liberacao de ions calcio, de ions hidroxila efomacao de carbonato de calico na presenca de tecido coni untivo de cao. [thesis] Sao Paulo, 1994.
4. Rehman K, Saunders WP, Foye RH *et al.* Calcium ion diffusion from calcium hydroxide-containing materials in endodontically treated teeth: An *in vitro* study. *Int Endod J* 1996 **29:** 271–279.
5. Wang JD, Hume WR. Diffusion of hydrogen and hydroxyl ions from various sources through dentine *J Endodont* 1988 **21:** 17–26.
6. Tronstad L, Andreasen JO, Hasselgren G *et al.* pH changes in dental tissue after root canal filling with calcium hydroxide. *J Endodont* 1981 **7:** 17–21.
7. Fuss A, Szajkis S, Tagger M. Tubular permeability to calcium hydroxide and to bleaching agent. *J Endodont* 1989 **15:** 362–364.
8. Nerwich A, Figdor D, Messer HH. PH changes in the root dentine over a 4-week period following root canal dressing with calcium hydroxide. *J Endodont* 1993 **19:** 302–306.
9. Pashley DH. Dentine permeability: theory and practice. In Spangberg LSW(ed) *Experimental endodontics.* 1st ed.pp 19–49. Boca Raton, FL: CRC press, 1990.
10. Maisto OA, Capurro MA. Obtureacion de conductos radiculares con hidroxido, de calcio-iodoformo. *Revista de la Asociacion Odontologica Argentina* 1964 **52:** 167–73.
11. Berbert A. Comportamento dos tecidos apicais e periapicais apos biopulpectomia e obturacao canal com AH-26. hidroxido de calcio oumistura de ambos. Estudo histological em dentes de caes [thesis]. Bauru, 1978.
12. Holland R, Souza V, Nery MJ, *et al.* A histological study of the effect of calcium hydroxide in the treatment of pulpless teeth of dogs. *J Endod B Soc* 1979 **12:** 15–23.
13. Maisto OA. Endodoncia. 3rd ed. Buenos Aires: Mundi, 1975.
14. Estrela C, Pesce HF. Chemical analysis of the liberation of calcium and hydroxyl ions from calcium hydroxide pastes in connective tissue in the dog. Part I. *Brazilian Dent J* 1996 **7:** 41–46.
15. Barbosa SV, Spangberg LSW, Almedia D. Low surface tension calcium hydroxide solution is an effective antiseptic. *Int Endod J* 1994 **27:** 6–10.
16. Ozcelik B, Tasman F, Ogan C. A comparison of the surface tension of calcium hydroxide mixed with different vehicles. *J Endod* 2000 **26:** 500–502.
17. Stamos DG, Haasch GC, Gerstein H. The pH of local anaesthetic/calcium hydroxide solutions. *J Endod* 1985 **11:** 264–265.
18. Bystrom A, Sundqvist G. Bacteriological evaluation of the efficacy of mechanical root canal instrumentation in endodontic therapy. *Scand J Dent Res* 1981 **89:** 321–328.
19. Reit C, Dahlen G. Decision making analysis of endodontic treatment strategies in teeth with apical periodontitis. *Int Endodo J* 1988 **21:** 291–299.
20. Ørstavik D, Kerekes K, Molven O. Effect of extensive apical reaming and calcium hydroxide dressing on bacterial infection during treatment of apical periodontitis: a pilot study. *Int Endod J* 1991 **24:** 1–7.
21. Sigurdsson A, Stancill R, Madison S. Intracanal placement of $Ca(OH)_2$. *J Endod* 1992 **18:** 367–370.
22. Staehle HJ, Thoma C, Muller HP. Comparative in vitro investigation of different methods for temporary root canal filling with aqueous suspensions of calcium hydroxide. *Endod Dent Traumatol* 1997 **13:** 106–112.
23. Heithersay GS. Calcium hydroxide in the treatment of pulpless

teeth with associated pathology. *J Brit Endod Soc* 1975 **8:** 74–92.
24. Bystrom A, Claesson R, Sundqvist G. The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals. *Endod Dent Traumatol* 1985 **1:** 170–175.
25. Siqueira Jr JF, Lopes HP. Mechanisms of antimicrobial activity of calcium hydroxide: a critical review. *Int Endod J* 1999 **32:** 361–369.
26. Stevens RH, Grossman LI. Evaluation of the antimicrobial potential of calcium hydroxide as an intracanal medicament. *J Endod* 1983 **9:** 372–374.
27. Difiore PM, Peters DD, Setterstrom JA. The antibacterial effect of calcium hydroxide apexification pastes on streptococcus sanguis. *Oral Surg Oral Med Oral Pathol* 1983 **55:** 91–94.
28. Siqueira JF Jr, Lopes HP, Uzeda M. Recontamination of coronally unsealed root canals medicated with camphorated paramonochlorophenol or calcium hydroxide pastes after saliva challenge. *J Endod* 1998 **24:** 11–14.
29. Haapasalo M, Ørstavik D. *In vitro* infection and disinfection of dentinal tubules. *J Dent Res* 1987 **66:** 1375–1379.
30. Safavi KE, Spangberg LSW, Langelang K. Root canal dentinal tubule disinfection. *J Endod* 1990 **16:** 207–210.
31. Ørstavik D, Haapasalo M. Disinfection by endodontic irrigants and dressings of experimentally infected dentinal tubules. *Endod Dent Traumatol* 1990 **6:** 142–149.
32. Siqueira JFJr, Uzeda M. Disinfection by calcium hydroxide pastes of dentinal tubules infected with two obligate and one facultative anaerobic bacteria. *J Endod* 1996 **22:** 674–676.
33. Sjogren U, Figdor, Spangberg, *et al.* The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing. *Int Endod J* 1991 **24:** 119–125.
34. Barbosa CAM, Goncalves RB, Siqueira JFJr, *et al.* Evaluation of the antibacterial activities of calcium hydroxide, chlorhexidine and camphorated paramonochlorophenol as intracanal medicament. A clinical and laboratory study. *J Endod* 1997 **23:** 297–300.
35. Schein B, Schilder H. Endotoxin content in endodontically involved teeth. *J Endod* 1975 **1:** 19–21.
36. Safavi KE, Nichols FC. Effect of calcium hydroxide on bacterial lipopolysaccharide. *J Endod* 1993 **19:** 76–78.
37. Safavi KE, Nichols FC. Alteration of biological properties of bacterial lipopolysaccharide by calcium hydroxide treatment. *J Endod* 1994 **20:** 127–129.
38. Morrison DC, Ryan JL. Endotoxins and disease mechanisms. *Ann Rev Med* 1987 **38:** 417–432.
39. Barthel CR, Levin LG, Reisner HM, *et al.* TNF-alpha in monocytes after exposure to calcium hydroxide treated Escherichia coli LPS. *Int Endod J* 1997 **30:** 155–159.
40. Olsen M, Difiore PM, Dixit SN, *et al.* The effects of calcium hydroxide inhibition on LPS induced release of IL-lβ from human monocytes in whole blood [Abstract]. *J Endod*on 1999 **25:** 289.
41. Silva LAB, Nelson-Filho P, Leonardo MR, *et al.* Effect of calcium ydroxide on bacterial endotoxin in vivo. *J Endod* 2002 **28:** 94–98.
42. Huang T-JG, Schilder H, Nathanson D. Effects of moisture content and endodontic treatment on some mechanical properties of human dentine. *J Endod* 1992 **18:** 209–215.
43. Kontakiotis E,Nakou M, Georgopoulou M. *In vitro* study of the indirect action of calcium hydroxide on the anaerobic flora of the root canal. *Int Endod J* 1995 **28:** 285–289.
44. Evans M, Davies JK, Sundqvist G, *et al.* Mechanisms involved in the resistance of Enterococcus faecalis to calcium hydroxide. *Int Endod J* 2002 **35:** 221–228.
45. Pitt Ford TR. Apexification and Apexogenesis. In Walton RE. Torabinejad M (eds) *Principles and practices of Endodontics.* 2nd ed, pp 373–384. Philadelphia: W.B. Saunders, 2002.
46. Grossman LI. *Endodontic practice.* 11th ed, pp 102–115. Philadelphia: Lea & Febiger, 1988.
47. Gutmann JL, Heaton JF. Management of the open (immature) apex. 2. Non-vital teeth. *Int Endod J* 1981 **14:** 173–177.
48. Franks AL. Therapy for the divergent pulpless tooth by continued apical formation. *J Am Dent Assoc* 1966 **72:** 87–92.
49. England MC, Best E. Non-induced apical closure in immature roots of dog's teeth. *J Endod* 1977 **3:** 411–415.
50. Kleier DJ, Barr ES. A study of endodontically apexified teeth. *Endod Dent Traumatol* 1991 **7:** 112–118.
51. Cohen S, Burns RC. *Pathways of the pulp.* 8th ed. pp 797–844. Mosby, 2002.
52. Chosack A, Cleaton-Jones P. A histological and quantitative histomorphometric study of apexification of nonvital permanent incisors of vervet monkeys after repeated root filling with a calcium hydroxide paste. *Endod Dent Traumatol* 1997 **13:** 211–216.
53. Ham JW, Patterson SS, Mitchell DF. Induced apical closure of immature pulpless teeth in monkeys. *Oral Surg* 1972 **33:** 438–442.
54. Sciaky I, Pisanti S. Localization of calcium hydroxide placed over amputated pulps in dogs teeth. *J Dent Res* 1960 **39:** 1128–1133.
55. Schroder U, Granath LE. Early reaction of intact human teeth to calcium hydroxide following experimental pulpotomy and its significance to the development of hard tissue barrier. *Odont Rev* 1971 **22:** 379–385.
56. Seltzer S, Bender IB. *The dental pulp.* 2nd ed, p 260. Philadelphia: J.B Lippincott, 1975.
57. Schroder U, Granath LE. Scanning electron microscopy of hard tissue barrier following experimental pulpotomy of intact human teeth and capping with calcium hydroxide. *Odont Revy* 1972 **23:** 211–216.
58. Chivian N. Root resorption. In Cohen S, Burns RC (eds) *Pathways of the pulp.* 5th ed. pp 504–547. St. Louis: Mosby, 1991.
59. Frank AL, Weine FS. Non-surgical therapy for the perforating defect of internal resorption. *J Am Dent Assoc* 1973 **87:** 863–868.
60. Stewart GG. Calcium hydroxide-induced root healing. *J Am Dent Assoc* 1975 **90:** 793–797.
61. Cvek M. Clinical procedures promoting apical closure and arrest of external root resorption in non-vital permanent incisors, In Transactions of 5th international conference on endodontics. 1973; University of Pennsylvania.
62. Gholston LR, Mattison GD. An endodontic-orthodontic technique for aesthetic stabilization of externally resorbed teeth. *Am J Orthod* 1983 **83:** 435–439.
63. Andreasen JO. Treatment of fractured and avulsed teeth. *J Dent Child* 1971 **38:** 29–32.
64. Trope M, Chivian N, Sigurdsson A, *et al.* Traumatic injuries. In Cohen S, Burns RC (eds) *Pathways of the pulp.* 8th ed. pp: 603–650. St.Louis: Mosby, 2002.
65. Cvek M, Granath LE, Hollenbder L. Treatment of non-vital permanent incisors with calcium hydroxide. III. Variations of occurrence of ankylosis of reimplanted teeth with duration of extra-alveolar period and storage environment. *Odontol Rev* 1974 **25:** 43–56.
66. Hammarstrom L, Blomlof L, Feiglin B, *et al.* Replantation of teeth and antibiotic treatment. *Endod Dent Traumatol* 1986 **2:** 51–57.

67. Lengheden A, Blomlof L, Lindskog S. Effect of delayed calcium hydroxide treatment on periodontal healing in contaminated replanted teeth. *Scand J Dent Res* 1991 **99:** 147–153.
68. Trope M, Yesilsoy C, Koren L, *et al.* Effect of different endodontic treatment protocols on periodontal repair and root resorption of replanted dog teeth. *J Endod* 1992 **18:** 492–496.
69. Andreasen JO, Farik B, Munksgaard EC. Long-term calcium hydroxide as a root canal dressing may increase the risk of root fracture. *Dent Traumatol* 2002 **18:** 134–137.
70. Warfving J, *et al.* Effect of calcium hydroxide treated dentine on pulpal responses. *Int Endod J* 1987 **20:** 183–190.
71. Stanley HR, Pameijer CH. Pulp capping with a new visible light-curring calcium hydroxide composition (Prisma VLC Dycal). *Oper Dent* 1985 **10:** 159–163.
72. Meadow D, Needleman H, Lindner G. Oral trauma in children. *Pediatr Dent* 1984 **6:** 248–255.
73. Schroder U. Reaction of human dental pulp to experimental pulpotomy and capping with calcium hydroxide. *Odont Rev* 1973 **24:** 97–105.
74. Stanley HR, Lundy T. Dycal therapy for pulp exposure. *Oral Surg* 1972 **34:** 818–825.
75. Cox CF, Bergenholtz G, Heys DR, *et al.* Pulp capping of dental pulp mechanically exposed to oral microflora: a 1–2 year observation of wound healing in the minkey. *J Oral Pathol* 1985 **14:** 156–168.
76. Olmez A, Oztas N, Basak F, *et al.* A histopathologic study of direct pulp capping with adhesive resins. *Oral Surg Oral Med Oral Pathol Oral Radio Endod* 1998 **86:** 98–103.
77. Pameijer CH, Stanley HR. The disastrous effects of the "total etch" technique in vital pulp capping in primates. *Am J Dent* 1998 **11:** S45–54.

Correspondence to: Dr. Alireza Farhad, Assistant professor of Endodontics, Faculty of Dentistry, University of Esfahan, Esfahan, Iran. P.O.Box. 81465-1596 Esfahan, Iran.
Email: Farhad@dnt.mui.ac.ir