CHAPTER 2

LITERATURE REVIEW

2.1 Immature permanent teeth

The British Society of Paediatric Dentistry defined an immature permanent tooth as one where the apex can be considered to be opened (Mackie, 1998). An open apex is found in the developing root of immature tooth until apical closure occurs approximately 3 years after eruption. Hertwig's epithelial root sheath is responsible for determining the shape of the root/roots (Rafter, 2005). The epithelial diaphragm surrounds the apical opening to the pulp and eventually becomes the apical foramen.

Trauma to immature permanent teeth is not uncommon. It has been estimated that 6% of traumatized immature incisor teeth will become non-vital and require endodontic treatment (Andreasen & Andreasen, 1994). The majority of trauma incidents to developing permanent teeth occur before root completion and this may result in pulpal inflammation and necrosis.

Other causes of pulpal infection and necrosis are caries, tooth anomalies such as *dens invaginatus* and *dens evaginatus*. Dental caries is an infectious, communicable, multifactorial disease in which bacteria dissolve the enamel surface of a tooth (Featherstone, 1999). Untreated caries can lead to incapacitating pain, bacterial infection that leads to pulpal necrosis, tooth extraction and loss of dental function, and may progress to an acute systemic infection. The

condition is even worse in an immature permanent tooth due to the complicated treatment in preserving it.

Dens invaginatus is a malformation of teeth probably resulting from infolding of the dental papilla during tooth development in which it shows a deep infolding of enamel and dentine starting from the foramen coecum or even the tip of the cusps and may extend deep into the root (Hülsmann, 1997). Synonyms for this malformation are *dens in dente*, invaginated odontome, dilated gestant odontome, dilated composite odontome, tooth inclusion and *dentoid in dente* (Hülsmann, 1997). This anomaly is rare in primary teeth but accounts to about 1-5% in permanent teeth (Laskaris, 2000). The teeth most affected are maxillary lateral incisors and bilateral occurrence is not uncommon and occurs in 43% of all cases (Hülsmann, 1997). The invagination allows entry of irritants into an area which is separated from pulpal tissue by only a thin layer of enamel and dentine and presents a predisposition for the development of dental caries. Hence, pulp necrosis often occurs rather early, within a few years of eruption, sometimes even before root end closure (Ferguson *et al.*, 1980 and Nik-Hussein, 1994). Nik-Hussein (1994) reported on a case of a non-vital immature infected *dens invaginatus* of maxillary lateral incisor which has been treated by apexification using calcium hydroxide paste. However, the author reported that even after apical closure was achieved, infection persisted and resolution was only achieved after apical curettage and apicectomy.

Dens evaginatus is a developmental dental anomaly that can be defined as a tubercle or protuberance from the involved surface of the affected tooth (Uyeno & Lugo, 1996). It consists of an outer layer of enamel, a core of dentine and may contain a slender extension of pulp tissue. Fracture or wear of the tubercle frequently leads to the major complication of *dens evaginatus* ie. pulp necrosis and early periapical infection, often before completion of root formation (Hill &

Bellis, 1984 and Gallagher *et al.*, 1988). In 1984, Hill and Bellis reported a case that was successfully treated, using calcium hydroxide apexification technique and followed by conventional nonsurgical endodontic therapy. Nik-Hussein (1986) reported a case of apexification of a non-vital *dens evaginatus* premolar on a 10 year old Chinese girl who was presented with abscess on the buccal mucosa associated with the non-vital immature dens evaginated tooth with fractured tubercle. Shay (1984) and Su (1992) also reported cases successfully treated using apexification, followed by endodontic therapy.

Rafter (2005) indicated that Hertwig's epithelial root sheath plays an important role in continued root development after pulpal injury, hence, every effort should be made to maintain its viability. It is believed that these tissues provide a source of undifferentiated cells that could give rise to further hard tissue formation (Rafter, 2005). However, this does not mean that there will be no hard tissue deposition in the apical region if the Hertwig's epithelial root sheath is destructed. Torneck (1982) stated that cementoblasts that are normally present in the apical region, fibroblasts of the dental follicle and periodontal ligament that undergo differentiation can become hard tissue producing cells.

2. 2 Treatment

There are few methods of treating a tooth that has a necrotic pulp and an open apex. It ranges from controlling the infection, filling the canal short of the apex using gutta-percha, custom fitted gutta-percha, surgical technique, apexification and one-visit apexification.

2.2.1 Control of infection

Reports on cases of root-end closure of non-vital immature permanent teeth without pulp therapy or endodontic therapy are limited. Das (1980) reported a case of apical closure in a non-vital tooth by control of infection. The author concluded that minimal mechanical intervention and removal of infection alone would be sufficient to induce apical closure. Similarly, Lieberman & Trowbridge (1983) suggested that debridement of the pulp chamber may not be a prerequisite for apical closure of non-vital immature permanent teeth. However, this line of treatment is not shared by other authors who felt that thorough cleaning of the root canal may be the primary factor responsible for apical closure (England & Best, 1977 and McCormick *et al.*, 1983).

2.2.2 Short-fill

Short-filling technique in managing non-vital immature permanent tooth has been proposed by Moodnick in 1963. He advocated removal of the bulk of the necrotic tissue from the canal and filling the root canal short of the apex using gutta-percha. However, using this technique, microorganism may remain within the canal system, thus healing may not take place or breakdown of periapical could occur later (Morse *et al.*, 1983).

2.2.3 Customized cone

Stewart (1963) described the use of custom fitted gutta-percha cones in managing the non-vital immature permanent teeth. However, this is not advisable as the apical part of the root is usually wider than the coronal part, thus making proper condensation of the gutta-percha impossible. In addition assessing the point of root development radiographically would also be difficult because root formation in the buccolingual plane is less advanced than it is in the mesiodistal plane (Rafter, 2005).

2.2.4 Periapical surgery

Periapical surgery in non-vital immature permanent teeth can be done via apicectomy and/or periradicular curettage. However, endodontic treatment and root end closure should be attempted before surgery is done. These combination of treatment approaches have many drawbacks and not surprisingly, several authors (Frank, 1966, Heithersay, 1970, Michanowicz & Michanowicz, 1967 and Steiner *et al.*, 1968) did not recommend this method of treatment due to the following reasons :-

- Further reduction of the already shortened roots could result in an inadequate crown-to-root ratio
- Thin walls of an immature permanent tooth could complicate the retrograde condensation of material
- Surgery could remove the root sheath and prevent the possibility of further root development
- Cooperation of young patient to undergo surgery since surgery could be physically and psychologically traumatic to them

Other disadvantages of surgical intervention include the difficulty of obtaining the necessary apical seal in the young pulpless tooth with its thin, fragile, irregular walls at the root apex (Rafter, 2005). These walls may shatter during retrograde preparation or condensation of the filling material.

2.2.5 Apexification

Apexification is a method of inducing apical closure through the formation of mineralized tissue in the apical pulp region of a non-vital immature tooth (Morse *et al.*, 1990). This induction is promoted by stimulating the formation of mineralized tissue (osteocement) at the end of the root, either with or without radicular growth (Reyes *et al.*, 2005).

Apexification has been used with great success by many researchers (Heithersay, 1975, Ghose *et al.*, 1987, Kleier & Barr, 1991, Morfis & Siskos, 1991, Erdorgan, 1997, Walia *et al.*, 2000, Kinirons *et al.*, 2001, Reyes *et al.*, 2005 and Felippe *et al.*, 2005). The technique involves removal of the necrotic tissue followed by debridement of the canal and placement of a medicament. If the Hertwig's epithelial root sheath has not been irreversibly damaged,

apexification allows apical closure and promotes radicular lengthening (Heithersay, 1975). However, some believe that instrumentation may in fact hamper root development and that preparation of these canals should be done carefully, if at all (Das *et al.,* 1997). Some authors hypothesized that debridement of the root canal and removal of the necrotic tissue and microorganisms along with decrease in pulp space are the critical factors in apexification (McCormick *et al.,* 1983).

The mechanism of action in apexification is still unclear. Some authors believed that the root sheath remains intact and resumes its function once the source of infection is eliminated (Frank, 1966 and Heithersay, 1970). It was thought that the cells of the dental sac at the apical region maintain their genetic code that predisposes them to differentiate into cementoblast (Klein & Levy, 1974).

Hertwig's epithelial root sheath is responsible for determining the shape of the root/roots (Rafter, 2005). This root sheath of Hertwig is usually sensitive to trauma but because of the degree of vascularity and cellularity in the apical part, root formation can continue even in the presence of pulpal inflammation and necrosis (Andreasen & Hjorting-Hansen, 1967). Therefore every effort must be taken in order to maintain its viability even after pulpal injury.

Thorough debridement to remove bacteria and necrotic tissue from the canal system is the primary factor responsible for apical closure (Farhad & Mohammadi, 2005). 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 into the canal (Pitt Ford, 2002). Calcific barrier that forms at the end of the apexification could be consisted of osteocementum, osteodentine, or bone or some combination of the three. The calcific barrier can be a complete or an incomplete hard tissue bridge at the apex or for a few milimeter short of the

apex, or the bridging can be an irregular mass of calcification traversing the apical one third of the root (Morse *et al.,* 1990).

2.2.6 One-visit apexification

Morse *et al.* (1990) defined one-visit apexification as the non-surgical condensation of a biocompatible material into the apical end of the root canal. The rationale of doing this is to create an apical stop that would allow the root canal to be filled immediately, without attempting a natural root end closure. However, it only fulfills one aspect of apexification which is the creation of an apical stop, hence, this technique cannot be used for teeth with excessively short roots (Morse *et al.,* 1990). A number of materials have been proposed for this technique, with reported favourable results. These include tricalcium phosphate (Coveillo & Brilliant, 1979 and Harbert, 1996), calcium hydroxide (Coviello & Brilliant, 1979 and Schumacher & Rutledge, 1993), freeze-dried bone (Rossmeisl *et al.*, 1982a) and freeze-dried dentine (Rossmeisl *et al.,* 1982b).

Recently, mineral trioxide aggregate (MTA) has been reported to be used in one-visit apexification. MTA can be packed down to the apex and the canal can be filled in on the same visit. The MTA will act as an apical barrier and induce bony formation around it. A number of authors have reported clinical success using MTA for one visit apexification (Shabahang & Torabinejad, 2000, Giuliani *et al.,* 2002 and Maroto *et al.*, 2003). A more detailed review on MTA will be discussed in chapter 2.3.4.

One-visit apexification may shorten the treatment time between the patient's first appointment and the final restoration. The importance of this approach lies in the expedient

cleaning and shaping of the root canal system, followed by its apical seal with a material that favours regeneration (Steinig *et al.,* 2003).

2.3 Materials for induction of apexification

Several materials have been used to induce apexification in teeth with immature apices. These include antiseptic pastes, antibiotic pastes, tricalcium phosphate, osteogenic protein-1, mineral trioxide aggregate and calcium hydroxide. However, although a variety of materials have been proposed, calcium hydroxide has gained the widest acceptance. The most favoured material is a paste of calcium hydroxide and water; the addition of other medicaments to calcium hydroxide has no beneficial effect on apexification (Gutman & Heaton, 1981). To date, there are many studies in the literatures that support the use of calcium hydroxide for induction of apical healing in non-vital immature permanent teeth (Walia *et al.,* 2000, Kinirons *et al.,* 2001, Dominguez *et al.,* 2005 and Felippe *et al.,* 2005).

2.3.1 Antiseptic and antibiotic pastes

Many of the early work in the area of inducing apical closure focused on the use of antiseptic and antibiotic pastes (Rafter, 2005). Cooke & Rowbotham (1960) described a method of using an antiseptic paste for the treatment of non-vital teeth with open apices, avoiding interference in its apical 2-3mm. The antiseptic paste consisted of zinc oxide, cresol, iodoform and thymol. Presumably, by this method, avoiding further irritation of the apical tissues, the sheath of Hertwig was enabled to effect repair and further growth of the root took place.

Ball (1964) successfully reproduced the apical closure using an antibiotic paste. This method avoided the possibility of chemical irritation of the apical tissues. Winter (1966) used polyantibiotic paste in treating a non-vital permanent incisor in an 11-year-old child before filling the apical portion of the root canal with a sectioned silver point and the remainder with Grossman's sealer and gutta-percha. However, Ball (1964) and Winter (1966) considered this apical paste to be a protoplasmic poison that would further irritate the periapical tissue.

The polyantibiotic paste was reportedly made up from:

Neomycin sulphate	2.0 gm.
Polymyxin B sulphate	100 mg.
Bacitracin	500 mg.
Nystatin	500,000 units
Polyethylene glycol base	

2.3.2 Tricalcium phosphate

Another material that has been used to induce apical healing is tricalcium phosphate (Coviello & Brilliant, 1979 and Koenigs *et al*., 1975). Tricalcium phosphate ($Ca_3 (PO_4)_2$) is a

bioceramic material which is resorbable. It degrades gradually over time, and is replaced by the host tissue (Roberts & Brilliant, 1975). Tricalcium phosphate is also biocompatible, so there is no interference between the biological system and the ceramic.

Koenigs and coworkers (1975) simulated conditions of an open apex in 24 monkey teeth and packed the apical 3 to 4 mm with tricalcium phosphate. At the end of a 24-week period, they reported that most of the material had been resorbed and replaced by a mineralized tissue. However, histologically, it was noted that the mineralized tissue or the bridge was not completed and a small channel containing connective tissue and blood vessels was observed on the lateral wall.

2.3.3 Osteogenic protein-1 (OP-1)

Osteogenic protein-1 (also known as bone morphogenetic protein-7) is a member of the bone morphogenetic protein family. Bone morphogenetic proteins and related members of the TGF-ß (transforming growth factor-ß) superfamily are involved in the development and repair of bone. Bone morphogenetic proteins are secretory signal molecules which have a variety of regulatory functions during morphogenesis and cell differentiation.

Shabahang *et al.* (1999) and Sampath *et al.* (1992) believed that OP-1 could induce apical hard tissue formation by attracting and recruiting mononuclear phagocytes to ectopic sites of bone formation. OP-1 stimulates the proliferation of mesenchymal cells that subsequently differentiate into osteogenic lineages. OP-1 has also been used as a pulp capping agent to induce dentine formation (Rutherford *et al.,* 1993).

Shabahang *et al.* (1999) conducted a study on root-end induction using OP-1, calcium hydroxide and mineral trioxide aggregate (MTA) in dogs and they reported that although OP-1

induced an apical hard tissue formation with the same frequency but in larger quantities as seen in calcium hydroxide, however, statistical analysis did not reveal a significant difference.

2.3.4 Mineral Trioxide Aggregate (MTA)

Mineral trioxide aggregate (MTA) is a new endodontic material that has been recently used in dentistry. It is a biomaterial that has been investigated for endodontic applications since the early 1990s. MTA was first described in the dental scientific literature in 1993 and was given approval for endodontic use by the U.S. Food and Drug Administration in 1998 (Schmitt & Bogen, 2001). MTA materials are a mixture of a refined Portland cement and bismuth oxide, and are reported to contain trace amounts of silicone dioxide, calcium oxide, magnesium oxide, potassium sulphate and sodium sulphate (Camilleri *et al.,* 2005). There are two types of MTA ie. grey MTA and white MTA. The main difference between the two materials is a reduction in iron content in white MTA, as compared to grey MTA. The MTA has been reported to have favourable sealing ability, superior biocompatibility and is less cytotoxic than other materials currently used in pulpal therapy (Torabinejad *et al.,* 1995, Torabinejad & Pitt Ford, 1996, Torabinejad & Chivian, 1999 and Keiser *et al.,* 2000). It allows osteoblasts to attach and spread on it with little or no tissue inflammation. In root-end filling, MTA shows better adaptation compared with amalgam, Super-EBA and IRM (Scheerer *et al.,* 2001). This improved adaptation allows MTA to provide a better seal when used as retrograde filling. Furthermore, its sealing ability has been shown to be unaffected in the presence of blood (Torabinejad *et al.,* 1994).

MTA can be used in sealing perforation, acts as pulp capping material, produces apical hard tissue formation in immature teeth, and acts as an apical barrier in open apex cases.

Shabahang *et al.* (1999) showed that MTA produced apical hard tissue formation with significantly greater consistency than calcium hydroxide or osteogenic protein-1. For non-vital immature teeth cases, MTA can be packed down to the apex and the canal can be filled in on the same visit. The MTA will act as an apical barrier and allow for bone to grow around it. Maroto *et al.* (2003) reported a case that was retreated with MTA. The case was initially treated with calcium hydroxide and no apical stop was obtained even after 3 years of treatment. At a review 12 months later, the tooth was found to be asymptomatic and radiographically showed initial repair of the radiolucent area. Pradhan *et al.* (2006) carried out a study that compared MTA and calcium hydroxide in non-vital immature permanent teeth and they concluded that both materials were found to be equally efficacious in managing those teeth and healing time for periapical radiolucencies was almost identical. However, the time taken to complete the treatment and the biological barrier formation in MTA group was significantly less than that for calcium hydroxide group.

2.3.5 Calcium hydroxide

Calcium hydroxide has extensive use in endodontic therapies. According to Fava & Saunders (1999), calcium hydroxide was first introduced to dentistry by Hermann in the 1930's to promote healing in many clinical situations and it was introduced as a pulpotomy agent in the United State by Zander in 1939. Matsumiya and Kitamura (1960) clearly demonstrated in the dog that calcium hydroxide packed into the infected root canals would eliminate or reduce microorganisms. However, it took many years before it gained universal acceptance in

endodontics. Frank (1966) was among the first to use calcium hydroxide to induce apical barrier formation.

In 1972, Cvek reported periapical healing and closure of the immature apical foramen by hard tissue in 90% of non-vital immature permanent incisors treated with calcium hydroxide. Ghose *et al.* (1987) reported that apical closure occurred in 96% of non-vital immature incisor teeth treated with calcium hydroxide. Yates (1988) and Kleier & Barr (1991) reported 100% success rate in achieving apical barrier formation using various calcium hydroxide pastes. To date, there are many studies in the literatures that support the use of calcium hydroxide for induction of apical healing in non vital immature permanent incisor (Walia *et al.*, 2000, Kinirons *et al.,* 2001, Reyes *et al.,* 2005 and Felippe *et al.,* 2005).

Nowadays calcium hydroxide is one of the most versatile medications in dentistry especially for its use as an intracanal dressing. It is considered to fulfill many of the properties of an ideal root canal dressing due to its antibacterial action, its ability to induce hard tissue formation and to cause intracanal occlusion, and its tissue dissolving capability (Nerwich *et al*., 1993). Although a variety of materials have been reported for induction of apical barrier formation, calcium hydroxide has gained the widest acceptance.

2.3.5.1 Chemical properties

Calcium hydroxide powder is white in colour and odourless. It has a molecular weight of 74.08, low solubility in water ie. about $1.2gL^{-1}$ at 25°C and has a high pH of about 12.5-12.8 (Fava & Saunders, 1999). The material is chemically classified as a strong base (Estrela *et al.,* 1999 and Estrela & Holland, 2003). Calcium hydroxide is obtained from the calcination/heating of calcium carbonate to calcium oxide. The calcium oxide then is hydrated to form calcium

hydroxide. The main actions of calcium hydroxide come from its dissociation into calcium and hydroxyl ions and the action of these ions on tissues and bacteria generates the biological and antimicrobial properties of this substance (Estrela & Holland, 2003).

2.3.5.2 Biological properties

2.3.5.2.1 Antimicrobial activity

Calcium hydroxide has a marked antimicrobial activity against most of the bacterial species found in root canal infections. *Eubacterium, Peptococcus, Peptostreptococcus, Prevotella, Porphyromonas* and *Fusobacterium* are predominant microorganisms in infected root canals (Sundqvist, 1994). It is believed that the antimicrobial effect of calcium hydroxide is related directly to its high pH. Most of the endodontopathogens are unable to survive in the highly alkaline environment provided by calcium hydroxide (Heithersay, 1975). The endodontopathogens are eliminated shortly after a short period when in direct contact with this material.

2.3.5.2.2 Mechanism of antimicrobial action

Most of endodontic pathogens do not survive in calcium hydroxide's highly alkaline environment (Heithersay, 1975). In 1985, Byström *et al.* showed the elimination of several bacterial species commonly found in infected root canals after a short period of time in direct contact with calcium hydroxide. However, the mechanism of action of calcium hydroxide is not thoroughly understood and several theories have been forwarded to explain its mode of action. Estrela *et al.* (1999) felt that the mechanism of action of calcium hydroxide as an antimicrobial medication may be better understood if knowledge about microbiological and pharmacological

properties of chemotherapeutics and their effects on microorganisms and their sites of action is adopted as reference. They also stated that the antimicrobial substances from any chemotherapeutics affect the microorganisms in two ways ie. inhibit the growth and replication of the microorganisms, and induce cellular inactivation.

Siqueira & Lopes (1999) stated that antimicrobial activity of calcium hydroxide is related to the release of hydroxyl ions in an aqueous environment. The authors advocated that the lethal effects on bacterial cells are probably due to the following mechanisms:-

i. Damage to the bacterial cytoplasmic membrane

Part of the structural components of cellular membrane is phospholipids. Hydroxyl ions induce lipid peroxidation, resulting in the destruction of the phospholipids. Hydroxyl ions remove hydrogen atoms from unsaturated fatty acids, generating a free lipidic radical (Siqueira & Lopes, 1999). The free lipidic radical reacts with hydrogen, resulting in the formation of lipidic peroxide radical. The lipidic peroxide radical in turn will remove another hydrogen atom from a second fatty acid, generating another lipidic peroxide. The peroxides themselves act as free radicals, initiating an autocatalytic chain reaction, and resulting in further loss of unsaturated fatty acids and extensive membrane damage (Siqueira & Lopes, 1999).

ii. Protein denaturation

The cellular metabolism depends on enzymatic activities. The alkaline environment provided by calcium hydroxide induces the breakdown of ionic bonds that maintain the tertiary structure of proteins. This results in the loss of biological activity of the enzyme and disruption

of the cellular metabolism. Structural proteins may also be damaged by hydroxyl ions (Siqueira & Lopes, 1999).

iii. Damage to the DNA

Hydroxyl ions dissociate from the calcium hydroxide react with the bacterial DNA and induce splitting of the strands. Thus, DNA replication and cellular activity of the bacteria are disturbed. Free radicals may also induce lethal mutations (Siqueira & Lopes, 1999).

The influence of pH on growth, metabolism and bacterial cell division is important to explain the mechanism of antimicrobial action of calcium hydroxide. It can change the integrity of the cytoplasmic membrane by means of chemical injuries to organic components and transport of nutrients, or by means of destruction of phospholipids or unsaturated fatty acids of the cytoplasmic membrane, observed in the lipidic peroxidation process (Estrela *et al.*, 1995).

Estrela *et al.* (1995) suggested two hypotheses on the action of the pH of calcium hydroxide on bacterial damage:-

i. Irreversible bacterial enzymatic inactivation

Under extreme pH of calcium hydroxide, bacterial enzymatic activity is interrupted which leads to denaturation of many proteins and destruction of the cytoplasmic membrane.

ii. Reversible bacterial enzymatic inactivation

Renaturation of proteins in the cytoplasmic membrane of the bacteria occurs when an ideal pH is obtained. Thus, enzymatic activity begins again.

A fundamental role in the cause and maintenance of periapical lesions has been attributed to the bacterial endotoxin which is a lipopolysaccharide (LPS) (Schein & Schilder, 1975). LPS, a cell wall component of gram negative bacteria, plays a major role in the periapical bone resorption process via its lipid A which simulates secretion of bone-resorbing mediators such as

prostaglandin E2 from a variety host cells (Safavi & Nichols, 1994). Several authors have demonstrated that calcium hydroxide hydrolysed lipid A, which is a toxic part of LPS, thus inactivating the toxic effects of the bacterial endotoxin (Safavi & Nichols, 1994, Barthel *et al.*, 1997 and Silva *et al.*, 2002).

Kontakiotis *et al.* (1995) suggested that the ability of calcium hydroxide to absorb carbon dioxide may also contribute to its antimicrobial activity. Carbon dioxide is essential for some bacteria such as *Capnocytophaga, Eikenella* and *Actinomyces spp*. and the carbon dioxide is provided by bacteria such as *Fusobacterium, Bacteroides, Porphyromonas* and *Streptococcus spp*. (Huang *et al.*, 1992). If calcium hydroxide absorbs carbon dioxide, carbon dioxide-dependent bacteria will not survive (Kontakiotis *et al.*, 1995). Hence, calcium hydroxide will disturb the 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.

2.3.5.2.3 Induction of repair by hard tissue formation

Calcium hydroxide has the capability of activating tissue enzymes which favour tissue restoration through mineralization. However, the exact mechanism is still unclear. Strauss *et al.* (1990) explained that alkaline phosphatase is a marker for hard tissue forming cells, osteonectin and osteopontin. Alkaline phosphatase is a hydrolytic enzyme that acts by means of liberation of inorganic phosphate from the esters of phosphate (Estrela *et al.*, 1995). The elevated pH of calcium hydroxide activates alkaline phosphatase (Binnie & Mitchell, 1973 and Tronstad *et al.*, 1981). According to Estrela & Holland (2003), the optimum pH value for the activation of this enzyme ranges from 8.6 to 10.3. This condition favours the release of organic phosphate

(phosphate ions) which then reacts with calcium ions from the circulating blood, thus, creating sediment of calcium phosphate on the organic matrix.

It is believed that the hydroxyl ion and the calcium ion of calcium hydroxide act in a synergic way to mineralization. The hydroxyl ions cause activation of alkaline phosphatase favouring mineralization. Foreman & Barnes (1990) described that the calcium ions permit reduction in the permeability of the new capillaries, resulting in increasing of the calcium ions concentration at the mineralization site

2.3.5.2.4 Tissue-dissolving activity

Hasselgren *et al.* (1988) reported that calcium hydroxide has the ability to dissolve necrotic material by a similar action to that of sodium hypochlorite. However, calcium hydroxide performs less effective dissolving ability compared to sodium hypochlorite. Hasselgren *et al.* (1988) cited that Heide & Kerekes (1977) demonstrated in monkey that the use of calcium hydroxide resulted in considerably cleaner root canals compared to root canals where calcium hydroxide had not been used. This could be due to its high pH which is able to denature and hydrolyze proteins, thus breaking down the intracanal soft tissue remnants and rendering a cleaner root canal.

2.3.5.2.5 Effect on physical properties of dentine

Despite its antimicrobial effects and superior activity in endodontic therapy, it has recently been reported that calcium hydroxide treated teeth showed a high failure rate because of an unusual preponderance of root fracture and it has been suggested that this problem may be caused by changes in the physical properties of dentine by the calcium hydroxide medicament

(Doyon *et al.,* 2005, Rosenberg *et al.*, 2007). White *et al.* (2002) reported that a 5-week exposure to calcium hydroxide resulted in a 32% decrease in the strength of bovine dentine. Similarly, it was reported that in sheep dentine treated with calcium hydroxide showed a marked decrease in fracture strength with increasing storage time (Andreasen *et al.,* 2002, Andreasen *et al.,* 2006). This was further supported by Rosenberg *et al.* (2007) who measured the effect of calcium hydroxide root filling on the microtensile fracture strength of teeth and found that there was about 23-43.9% weakening of human dentine following root canal filling with calcium hydroxide. Most importantly, when immersed in a saturated solution of calcium hydroxide for 1 week, a reduction in the flexural strength of human dentine was also demonstrated (Grigoratos *et al.,* 2001).

2.3.5.3 Types of calcium hydroxide vehicles

The dissociation of calcium hydroxide into hydroxyl and calcium ions depends on the vehicle used to make the paste (Simon *et al.,* 1995). According to Fava & Saunders (1999), Fava (1991) reported that the ideal vehicle for calcium hydroxide should:-

i) allow slow and gradual release of calcium and hydroxyl ions;

ii) allow slow diffusion in the tissues with low solubility in tissue fluids;

iii) have no adverse effect on the induction of hard tissue deposition.

Numerous substances have been used as vehicles for calcium hydroxide. Estrela & Pesce (1996) concluded from their *in vitro* study that the type of vehicle has a direct relationship with the concentration and the velocity of ionic liberation as well as with the antibacterial action when the paste is carried into a contaminated area. Fava & Saunders (1999) are also of the opinion

that the vehicle plays an important role in the biological action of calcium hydroxide which is determined by the velocity of ionic dissociation in calcium and hydroxyl ions.

In general, there are three types of calcium hydroxide vehicles; aqueous, viscous or oil. Examples of aqueous vehicles are water, saline, dental anaesthetics solution, Ringer's solution, aqueous suspension of methylcellulose or carboxy-methylcellulose and anionic detergent solution. The second group is represented by glycerine, polyethyleneglycol and propyleneglycol. Common oily vehicles used are olive oil, fatty acids, camphorated monochlorophenol, eugenol and metacresylacetate (Fava & Saunders, 1999).

2.3.5.3.1 Aqueous vehicles

Calcium and hydroxyl ions are rapidly released when calcium hydroxide is mixed with any of aqueous vehicles. This type of vehicle promotes a high degree of solubility when the paste remains in direct contact with the tissue and tissue fluids, causing it to be rapidly solubilised and resorbed by macrophages (Fava & Saunders, 1999). This will cause an emptying of the root canal after a short period and thus delaying the healing process (Fava & Saunders, 1999). Generally, aqueous solutions promote a rapid ionic liberation and should be used in clinical situations involving intense exudation and dental replantation.

Calcium hydroxide powder can be mixed with any of the aqueous vehicles at the chairside. These vehicles include water, sterile water, distilled water, sterile distilled water, bidistilled water, sterile bidistilled water, saline or sterile saline, anaesthetic solutions, Ringer's solution, methylcellulose and carboxymethylcellulose, and anionic detergent solution (Fava & Saunders, 1999). Each vehicle gives different properties and can be used in few dental

procedures such as pulp capping, apexification, apexogenesis, intra-canal dressing after pulp extirpation, and also in treating internal resorption and perforations.

Pre-mixed calcium hydroxide paste in aqueous vehicles are available in the market in different proprietary brands. Examples are *Calxyl (Otto & Co., Frankfurt, Germany), Pulpdent & Tempcanal (Pulpdent Corp., Brookline USA), Calvital (Neo Dental Chemical Products Co., Tokyo Japan), Reogan (Vivadent, Schaan, Leichtenstein), Calasept (Scania Dental AB, Knvista, Sweden), Hypocal (Ellinan Co., Hewlatt, USA), Calcicur (VOCO, Auxhaven, Germany), Hidropulpe (Lab, Zizine, France), Serocalcium (Casa Wild, Basel, Switzerland), Acrical (Bames-Hind Laboratories, USA)* and *Calnex (Associated Dental Products Ltd., London UK).* The usage of pre-mixed calcium hydroxide pastes are similar to those pastes mixed at the chairside.

2.3.5.3.2 Viscous vehicles

Viscous vehicles are also water-soluble substances that release calcium and hydroxyl ions more slowly for extended period of time (Fava & Saunders, 1999). The high molecular weight of these vehicles could minimize the dispersion of calcium hydroxide into tissue and remain the paste in the canal for longer period. Therefore, the calcium and hydroxyl ions will be given off at lower velocity. Examples of viscous vehicles are glycerine, polyethyleneglycol and propyleneglycol (Fava & Saunders, 1999). The use of calcium hydroxide paste with glycerine in its formula was initially reported by Steiner *et al.* (1968) in a paste composed of calcium hydroxide, camphorated parachlorophenol, barium sulphate and glycerine. This paste was used in root-end closure of non-vital immature permanent teeth. Calcium hydroxide-glycerine paste has also been used in cases of chronic abscess with extraoral fistula (Caliskan *et al.,* 1994), internal resorption with or without root perforation (Caliskan & Turkun, 1997), acute or chronic periapical lesion (Caliskan & Sen, 1996 and Gutmann & Fava, 1992), apical periodontitis (Fava,

1998), as an intracanal dressing after pulpectomy (Fava, 1994) and in reparing a root fracture (Caliskan & Pehlivan, 1996).

2.3.5.3.3 Oily vehicles

Calcium hydroxide paste containing oily vehicle may remain within the root canal for longer period than the pastes containing aqueous or viscous vehicles (Fava & Saunders, 1999). These vehicles are non-water-soluble substances that promote the lowest solubility and diffusion within the tissues. In comparison with water-soluble substances, oily vehicles prolong the action of the calcium hydroxide since it gives a very slow ionic dissociation and hence has a low diffusion within the tissues. Thus, pastes containing oily vehicles are employed in those clinical situations that require a slow ionic dissociation such as in apexification. Some examples of oily vehicles are olive oil, silicone oil, fatty acids, camphorated parachlorophenol, metacresylacetate and eugenol. Calcium hydroxide mixed with camphorated parachlorophenol was the most frequently used material in apexification procedures (Steiner *et al.,* 1968, Piekoff & Trott, 1976, Ludlow, 1979 and Kleier & Barr, 1991). Examples of proprietary brands of calcium hydroxide paste in oily vehicles are *Endoapex (Lab. Inodon Ltd., Porto Alegre, Brazil), L & C (Herpo Produtos DentArios Ltda., Rio de Janeiro, Brazil)* and *Vitapex (Neo Dental Chemical Products Co. Ltd., Tokyo Japan).*

2.3.5.3.4 Effect of vehicle

The presence of calcium hydroxide in the root canal elevates the pH, thus producing an alkaline environment by the diffusion of hydroxyl ions through the dentinal tubules (Tronstad *et al*., 1981). In addition, an increased pH is bactericidal and it also inhibits osteoclastic activity (Esberard *et al.,* 1996). The vehicle plays an important role in the process of ionic dissociation

because it determines the velocity of ionic dissociation causing the paste to be solubilised and resorbed at various rates by the periapical tissues and from within the root canal.

2.3.5.4 Dissociation and pH of calcium hydroxide

The diffusion of hydroxyl ions and pH values of calcium hydroxide is a subject of controversy. Few authors claimed that the vehicles mixed with calcium hydroxide can influence the pH of the paste and the velocity of hydroxyl ion diffusion through the dentinal tubules (Simon *et al.,* 1995, Calt *et al.,* 1999). In 1982, Anthony and his co-workers reported that chemical reactions occur when calcium hydroxide is mixed with camphorated parachlorophenol or metacresylacetate. The reaction products could cause a reduction in pH over an extended period of time. However, Solak & Oztan (2003) concluded in their study that four different water-based vehicles demonstrated similar pH changes range within pH 11-12 when mixed with calcium hydroxide. Their finding was consistent with those of Stamos *et al.* (1985) who investigated the pH values of calcium hydroxide preparations combined with lidocaine or mepivacaine. They indicated that there was no significant difference in pH values when calcium hydroxide mixed with normal saline, lidocaine or mepivacaine. Estrela *et al.* (1999) stated that hydrosoluble vehicles (distilled water and saline solution) presented the best chemical characteristics in terms of speed of ionic dissociation and diffusion, which helps in the already known antimicrobial and tissue healing induction powers of calcium hydroxide.

2.3.5.5 Antimicrobial properties of calcium hydroxide

The calcium hydroxide antimicrobial action is related to the alkaline pH. Calcium hydroxide kills bacteria because of the effects of hydroxyl ions, which are extremely reactive,

combining rapidly with proteins and nucleic acids. This leads to lipid peroxidation and increasing the bacterial membrane permeability, which can cause bacterial death. To be effective against bacteria located inside the dentinal tubules, the hydroxyl ions from calcium hydroxide should diffuse into dentine at sufficient concentrations. Siqueira & Uzeda (1998) demonstrated that calcium hydroxide/camphorated monochlorophenol/glycerine paste rapidly kills bacteria and indicated that the camphorated monochlorophenol (CMCP) increased the antimicrobial effect of the calcium hydroxide. However, Spangberg (1994) indicated that the association of calcium hydroxide to CMCP aiming to improve its antimicrobial property should be avoided, as this vehicle can be irritating to periapical tissues.

Recently, Gomes *et al.* (2002) reported that the antimicrobial action of calcium hydroxide pastes prepared with aqueous vehicles had the following descending order: water, saline and anaesthetic solution. Glycerine, which is a viscous vehicle, mixed with calcium hydroxide had larger zone of microbial inhibition compared to the aqueous vehicles. However, they reported that polyethyleneglycol (also a viscous vehicle) had the weakest antimicrobial action.

2.3.5.6 Uses of calcium hydroxide (other than apexification)

Calcium hydroxide is a material that has been used for a variety of purpose since its introduction into dentistry in the early part of the twentieth century (Foreman & Barnes, 1990). It ranges from pulp capping whether indirect or direct, intracanal dressing, pulpotomy or even in prevention of root resorption.

2.3.5.6.1 Indirect pulp capping

Indirect pulp capping is a procedure performed in a tooth with a deep carious lesion adjacent to the pulp. Caries near the pulp is left in place to avoid pulp tissue exposure and is covered with a biocompatible material. The main purpose is to arrest the carious process by promoting dentinal sclerosis, remineralisation of affected dentine as well as preserving pulp vitality (Farhad & Mohammadi 2005). Calcium hydroxide is the most commonly used medicament/material for indirect pulp capping (Llewelyn, 2000). The tooth then is restored with a material that seals the tooth from micro leakage. Bjørndall *et al.* (1997) studied the effect of calcium hydroxide on residual carious dentine after an interval of six to twelve months. The association of microbiological status and clinical dentine alteration was evaluated and they found that there was no evidence of pulpal exposure and only few microorganisms left after removal of residual carious dentine. However, in 1976, Stark and co-workers believed that the type of capping material is not important, but it is the sealing of the tooth that is crucial in order to deprive the bacteria in the remaining caries of nutrient and oxygen, hence prevent future growth of the bacteria.

2.3.5.6.2 Direct pulp capping

Direct pulp capping is a procedure for covering and protecting a healthy pulp tissue that has been inadvertently exposed due to caries excavation or traumatic injury with a biocompatible agent. The treatment objectives are to seal the pulp against bacterial leakage, encourage the pulp to wall off the exposure site by initiating a dentine bridge and maintaining the vitality of the underlying pulp tissue regions (Foreman & Barnes, 1990). It is done in a ‘pinpoint’ mechanical or traumatic exposure of the pulp that is surrounded by sound dentine in an asymptomatic tooth. When pulp capping procedure is successful, a calcific barrier, which is commonly termed as

dentine bridge forms across the exposure. Several materials have been used as pulp capping agents, but calcium hydroxide remains the material of choice for a direct pulp capping procedure (Ricketts, 2001). Different forms of calcium hydroxide are available for this purpose. These include pure calcium hydroxide and hard-setting calcium hydroxide containing cements.

2.3.5.6.3 Pulpotomy

Pulpotomy is a procedure in which the coronal pulp tissues are completely extirpated while the radicular pulp tissues are left intact. A pulp dressing is then placed over the amputation sites and a temporary or permanent restoration is inserted in an effort to preserve its vitality and to allow the tooth to strengthen through continued growth as long as possible. The procedure is indicated when caries removal results in pulp exposure in a tooth with a normal pulp or reversible pulpitis or after a traumatic pulp exposure. The coronal tissue is amputated, and the remaining radicular tissue is judged to be vital by clinical and/or radiographic criteria. Calcium hydroxide, for covering the amputated pulp, may be in the form of a hard setting material, a non-setting material, or slurry of freshly mixed powder and saline (Foreman & Barnes, 1990). The healing process of dental pulp after pulpotomy and treatment with calcium hydroxide is fundamentally characterized by the formation of a hard tissue bridge and maintenance of vital subjacent pulp tissue free of inflammatory cells (Souza & Holland, 1974)). Other materials have been proposed for direct pulp protection, but the results are questionable (Stanley & Pameijer, 1997).

2.3.5.6.3.1 Partial Pulpotomy (Cvek's Pulpotomy)

The American Academy of Pediatric Dentistry (2006) described partial pulpotomy as a procedure in which the inflamed pulp tissue beneath an exposure is removed to a depth of 1 to 3 mm or, in some cases, deeper to reach healthy pulp tissue. The amputation site should then be covered either with a hard setting calcium hydroxide cement, if later direct monitoring of the hard tissue barrier is not anticipated; or with pure calcium hydroxide, when later monitoring is desired (Andreasen & Andreasen, 1994). This procedure is indicated in a vital young permanent tooth with normal or reversible pulpitis and a small (<2 mm) carious pulp exposure in which the pulpal bleeding is controlled in 1 to 2 minutes. Partial pulpotomy is also the treatment of choice for a vital, traumatically exposed, young permanent tooth, especially one with an incompletely formed apex. Pulpotomy procedure using calcium hydroxide as the covering material is a successful treatment in both immature and mature teeth (Blanco, 1996 and Blanco & Cohen, 2002). Fuks *et al.* (1993) reported that partial pulpotomy has a high frequency of long-term success in treating traumatic pulp exposures in crown-fractured permanent incisors. The success rate of calcium hydroxide pulpotomies after one year and two year was reported to be 87.7 and 80.4 percent accordingly, and the result was only influenced by the type of restoration (Gruythuysen & Weerheijm, 1997).

2.3.5.6.3.2 Cervical (full) pulpotomy

Cervical or full pulpotomy involves removal of the entire coronal pulp to the level of the root orifices. Camp (2002) felt that the level of pulp amputation was chosen arbitrarily because of its anatomic convenience. This technique is indicated when the prediction is that the pulp is inflamed to the deeper levels of the coronal pulp such as in large pulpal exposure due to caries, traumatic exposure (after 72 hours), or if haemostasis cannot be obtained during a Cvek

pulpotomy procedure (Camp, 2002). Following coronal pulp amputation, bleeding should appear normal in colour with no excessive bleeding and good haemostasis is achieved. Calcium hydroxide dressing, bacteria-tight seal, and coronal restorations are then carried out as in partial pulpotomy. Because full pulpotomy is performed on pulps that are expected to have deep inflammation and the site of pulp amputation is arbitrary, the prognosis is in the range of 75%, which is poorer than the partial pulpotomy (Gazelius *et al.,* 1988 and Granath & Hagman, 1971).

2.3.5.6.4 Root canal dressing

Elimination of bacteria in the root canal system is of great importance for apical and periapical healing after endodontic treatment (Tronstad *et al.*, 1987) and the use of a root canal dressing has been recommended in teeth with chronic periapical lesions to reach area not accessible by instrumentation (Byström *et al.,* 1985 and Assed *et al.,* 1996). One of the most commonly used materials is calcium hydroxide and it has been recommended as an intracanal dressing because of its antibacterial and biological properties. Calcium hydroxide can be placed in the full length of the canal and if the material is inadvertently forced through the apical foramen, it is soon absorbed (Foreman & Barnes, 1990). Recently, Zerella *et al.* (2005) concluded in their *in vivo* study that root canal dressing with a mixture of two percent chlorhexidine and calcium hydroxide slurry is as effective as aqueous calcium hydroxide on the disinfection of failed root-filled teeth. However, although calcium hydroxide is considered the most effective dressing currently in use, it fails to consistently produce sterile root canals (Reit & Dahlen, 1988 and Ørstavik *et al.,* 1991). More recently, Peters *et al.* (2002) reported that calcium hydroxide and sterile saline slurry only managed to limit but did not totally prevent the

regrowth of endodontic bacteria. The survival of these few microorganisms could be due to the complex anatomy of the root canal that made satisfactory packing of the calcium hydroxide difficult (Sigurdsson *et al.*, 1992 and Staehle *et al.*, 1997).

2.3.5.6.5 Perforation repair

It has been reported that perforations of the root canal wall, either by instruments or by posts, may be treated in a similar way to apical closure, in an attempt to obtain hard tissue formation (Heithersay, 1975). The use of calcium hydroxide to treat root canal perforation has been reported extensively (Martin *et al.*, 1982 and Behnia *et al.*, 2000). In 1986, Beavers and his co-workers used calcium hydroxide sealer, Sealapex, to treat root canal perforations. They noted bone healing and ingrowth of trabeculae into the perforation after 42 days. Calcium hydroxide has also been used together with SuperEBA in repairing iatrogenic root perforations (Bogaerts, 1997). Recently, Kim *et al.* (2001) studied the effect of adding growth factors to calcium hydroxide in the healing of periapical perforations in dogs. The authors claimed that combining platelet-derived growth factor-BB and insulin-like growth factor-I with calcium hydroxide improved the healing of apical perforation in dogs.

2.3.5.6.6 Prevention of root resorption

Root resorption is a condition associated with either a physiologic or a pathologic process resulting in a loss of dentine, cementum and/or bone. On the basis of the site of origin of the resorption, it may be referred to as internal, external or root-end resorption (Chivian, 1991). Calcium hydroxide is frequently used as a dressing for the treatment of both internal and external inflammatory root resorption (Foreman & Barnes, 1990). Calcium hydroxide has osseous reparative properties that make it a good choice to create a barrier. This is directly related to the

alkaline pH of the calcium hydroxide, which spreads through the dentine (Farhad & Mohammadi, 2005). According to Chivian (1991), calcium hydroxide should be placed into 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. Once physical barrier is detected, the defect can be filled with gutta percha. Andreasen (1971) was able to arrest external inflammatory root resorption following replantation, in nine cases out of ten, by the use of calcium hydroxide. However, calcium hydroxide treatment has no effect on replacement resorption (ankylosis) once it has become established (Trope, 2002).

2.3.5.6.7 Treatment of root fracture

Root fractures are relatively uncommon injuries, but represent complex healing patterns due to concomitant injury to the pulp, periodontal ligament, dentine and cementum, which usually result from a horizontal impact (Andreasen & Andreasen, 1994). Few authors reported on treatment of horizontal root fractures using calcium hydroxide (Clark & Eleazer, 2000, Cvek *et al.,* 2004 and Turgut *et al.*, 2004). It has been suggested that calcium hydroxide dressing for 3-6 months may encourage soft tissue healing and possibly mineralization at the fracture site (Foreman & Barnes, 1990).

2.4 Methods of calcium hydroxide placement

Success of endodontic treatment depends on the elimination of the microorganism present in the root canal. Chemomechanical preparation is therefore one of the most important stages of endodontic treatment (Öztan *et al.*, 2002). It has been reported that microorganism may survive within the root canal system even after chemomechanical preparation was carefully done (Byström & Sundqvist, 1985). Therefore, the intracanal dressing using calcium hydroxide as an antimicrobial agent is highly recommended. In order that calcium hydroxide can express its full efficacy, it is necessary to promote the dissociation and diffusion of hydroxyl ions, which requires correct filling of the root canal (Estrela *et al.,* 2002).

Placement of calcium hydroxide into the root canal system can be fairly difficult. It has been reported in the literatures that there are few techniques of intracanal placement of calcium hydroxide (Webber *et al.*, 1981, Kleier *et al.*, 1985, Krell & Madison, 1985, Teplitsky, 1986, Sigurdsson *et al.*, 1992, Deveaux *et al.*, 2000, Öztan *et al.*, 2002 and Torres *et al.*, 2004).

2.4.1 Amalgam carrier

Frank (1966) originally advocated mixing calcium hydroxide powder with camphorated monochlorophenol into a paste and placed it into the root canal with an amalgam carrier before vertical condensation. Webber *et al.* (1981) then reported the use of an amalgam carrier with a plastic or Teflon sleeve that drives calcium hydroxide paste into the root canal of a non-vital immature permanent tooth followed by vertical condensation using endodontic plugger. They made the calcium hydroxide paste by mixing calcium hydroxide powder with sterile water, isotonic saline or dental anaesthetic without constrictor. A case of a 20-year-old woman with a non-vital immature maxillary left central incisor related to trauma 12 years earlier was reported and apexification was carried out by placing calcium hydroxide paste into the root canal system using amalgam carrier and followed by vertical condensation (Webber *et al.*, 1981). Apexification was completed after a year of treatment.

2.4.2 McSpadden compactor

Kleier *et al.* (1985) suggested the use of McSpadden compactor only, whereas Teplitsky (1986) used it together with a vertical condensation. McSpadden compactor is actually an instrument for introducing, thermoplasticizing and compacting gutta-percha into root canals. The compactor resembles a headstrom file in reverse, and it is used on a latch-type, slow-speed contra-angle handpiece. Kleier *et al.* (1985) described a technique of placing calcium hydroxide intracanally. The compactor would place the calcium hydroxide apically into the canal to whatever level the compactor is introduced and as long as the flutes of the compactor do not bind on the canals walls, the paste material will not be pumped ahead of the instrument. Teplitsky

(1986) reported three cases of using McSpadden compactor-vertical condensation combination technique for the delivery of calcium hydroxide in a variety of clinical situations. In this combination technique, calcium hydroxide paste is placed in the canal using McSpadden compactor first. This will be followed by placing a thicker, dryer mix of calcium hydroxide to the canal using a plastic instrument before condensing it with endodontic plugger. The author also claimed that this technique is fast, convenient, effective and reproducible.

2.4.3 Messing gun

Messing gun was originally designed as an amalgam carrier for retrograde fillings (Krell & Madison, 1985). The Messing gun kit comes with three different sizes of nozzles and plungers. Krell & Madison (1985) advocated that the Messing gun has proven to be effective in the placement of calcium hydroxide in all maxillary and mandibular anterior teeth, as well as selected posterior teeth where calcium hydroxide therapy was indicated. They described the use of this instrument in placing calcium hydroxide in powder form, stressing on its simplicity and efficacy of using it. The following advantages of using Messing gun were reported (Krell & Madison 1985) :-

- greater control in the placement of the powder, thereby minimizing undesirable overfills
- greater powder density in the canal(s), thereby minimizing voids
- greater ease in preparation
- greater ease in placement in a short amount of time

2.4.4 Pastinject

Pastinject is a specially designed paste carrier which has similar shape with lentulo spiral and is used in a slow-speed handpiece as well. It has different cross-sectional view compared to lentulo spiral. Deveaux *et al.* (2000) reported that Pastinject provided good root canal placement of calcium hydroxide paste. The special design of Pastinject seems to favour a better intracanal placement of calcium hydroxide in single-rooted teeth (Deveaux *et al.*, 2000). Öztan *et al.* (2002) concluded that Pastinject showed better fillings with calcium hydroxide mixed with water than lentulo spiral. However, placement of calcium hydroxide paste either with a Pastinject or lentulo spiral provided similar satisfactory results when glycerine is used as the vehicle.

2.4.5 Lentulo spiral

Lentulo spiral is an ISO colour-coded instrument which has consistenly spaced spirals that provide outstanding flexibility in distributing root canal sealer or intracanal dressing paste evenly throughout the root canal system. Lentulo spiral usage in intracanal placement of calcium hydroxide has been reported in few studies (Sigurdsson *et al.*, 1992, Torres *et al.*, 2004 and Peters *et al.*, 2005). Sigurdsson *et al.* reported in 1992 that the lentulo spiral performed best for calcium hydroxide paste placed to working length in minimally instrumented (size #25 K-file) curved canals. However, no statistical analysis was included. It was reported that in a curved canal, lentulo spiral-only technique gave significantly greater radiodensity of calcium hydroxide compared with an Ultradent 0.014-inch-diameter polypropylene capillary tip only or followed by lentulo spiral (Torres *et al.*, 2004). Peters *et al.* (2005) studied on intracanal placement of commercially available calcium hydroxide using either lentulo spiral or syringe in different

apical shapes of root. They found that calcium hydroxide was placed with significantly fewer voids using a lentulo spiral compared with the injection technique.

2.4.6 Syringe

The use of syringe system in placing calcium hydroxide paste in straight or slightly curved canals shaped to at least ISO size 50 was found to be better than lentulo spiral or endodontic reamer (Staehle *et al.,* 1997). Staehle and his co-workers (1997) used a syringe with 0.6mm outer diameter of the canula. A study done by Torres *et al.* in 2004 evaluated three placement techniques of calcium hydroxide; (a) injection with a syringe (Ultradent 0.014-inch-diameter polypropylene capillary tip) only, (b) injection with a syringe as in (a) and followed by a lentulo spiral, and (c) lentulo spiral only. However, they found that lentulo spiral technique was superior than using syringe system.

CHAPTER 3