

CHAPTER 1
INTRODUCTION

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1.1 Introduction/Background

Endodontic treatment of non-vital immature permanent teeth is often complicated. This is due to the fact that the teeth have wide canal which are frequently divergent and open apices, thereby making the classic instrumentation of root canal is difficult.

Immature permanent teeth can become non vital due to few reasons. Dental caries or developmental defects such as *dens invaginatus* may result in pulp necrosis in immature teeth (Sheehy & Roberts, 1997). Injuries to the dental pulp due to trauma can also lead to pulp necrosis. It has been reported that dental trauma is the most common cause of pulp necrosis in immature anterior permanent teeth. The Children's Dental Health Survey in the United Kingdom (2003) stated that the proportion of children sustaining accidental injuries to their incisors increased with age from 5% at age eight to 13% by age 15 (Lader *et al.*, 2003).

Erdogan (1997) reported that the maxillary central incisors are the most commonly traumatized teeth in children. Trauma usually occurs between 8-10 years of age when the roots of the incisors are developing. Trauma at this time can lead to pulp necrosis, hence no further development or/and maturation of the root will take place, resulting in a non vital incisor with open apex and incomplete root development (Mackie *et al.*, 1988).

Prevalence of pulp necrosis varies according to the type of injury, 0-3.5% for enamel infraction, 0.2-1.0% for enamel fracture without dentinal exposure, 1-6% for crown fractures involving enamel and dentine and 15-59% for luxation injuries (Andreasen & Andreasen, 1994). Crown fractures together with luxation injuries have a higher risk of pulp necrosis, being 25%

for subluxation, 30% for lateral luxation, 4% for extrusion and 100% for intrusion (Andreasen & Andreasen, 1994).

For the incompletely developed tooth with an open apex that is diagnosed with pulp necrosis, an apexification procedure has been shown to be consistently successful. Apexification is a method of treatment that induces apical closure of the open apex with a hard-tissue barrier. The use of calcium hydroxide paste to promote the formation of a calcific apical barrier prior to the placement of a gutta-percha root filling is well documented. Fava & Saunders (1999) reported that 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. Frank (1966) was among the first clinician 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 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 other studies in the literatures that support the use of calcium hydroxide for induction of apical healing in non-vital immature permanent incisors (Walia *et al.*, 2000, Kinirons *et al.*, 2001, Dominguez *et al.*, 2005 and Felipe *et al.*, 2005).

It is believed that the mode of action of calcium hydroxide is dependent on its ability to rapidly dissociate into hydroxyl (OH^-) and calcium (Ca^{2+}) ions, which then diffuse into the surrounding tissues. Such diffusion through dentine to the external surface has been demonstrated by many authors (Tronstad *et al.*, 1987 and Nerwich *et al.*, 1993). The action of

Ca^{2+} and OH^- ions on vital tissues and bacteria generates the induction of hard tissue deposition and gives the antibacterial effect.

Calcium hydroxide has the property of inhibiting bacterial enzymes by means of OH^- ions that act on the cytoplasmic membrane of the bacteria and kill the bacteria (Byström *et al.*, 1985 and Siqueira & Uzeda, 1998). The antibacterial property is most effective when there is direct contact between the antibacterial agent and the microorganisms. Apart from its bacterial enzymatic inhibition, calcium hydroxide also has the capability of activating tissue enzymes, such as alkaline phosphatase, which favour tissue restoration through mineralization (Estrela *et al.*, 1999).

A plethora of substances have been used as vehicles for calcium hydroxide. The vehicle plays an important role in the overall process of ionic dissociation because it determines the velocity of ionic dissociation causing the paste to be solubilized and resorbed at various rates by the periapical tissues (Fava & Saunders, 1999). Generally, there are three types of vehicles; aqueous, viscous and oil. The calcium hydroxide powder can either be prepared with the preferred vehicle at chairside or using the premixed paste or proprietary brands.

Healing of teeth with necrotic pulp is largely dependent on the degree of contact between the antibacterial material and the microorganisms (Safavi & Nichols, 1993). Hence, placement of the material in close proximity to the appropriate tissue is of paramount importance. In the case of apexification of non vital immature permanent teeth, calcium hydroxide should be placed inside the canal till 1-2mm short of the apex (Mackie, 1998).

Accurate placement of the material at the apical area of the tooth is crucial so that the dissociation and diffusion of OH^- ions occurred at the ideal location to promote mineralization. Thus, filling the root canal using an effective method may enhance the healing process. The

placement effectiveness of calcium hydroxide with different vehicles and different methods in non-vital immature permanent teeth has not been reported or compared.

CHAPTER 2

LITERATURE REVIEW