

A comparative study on the effect of Emdogain and Calcium Hydroxide on apical closure in young permanent teeth of dogs.

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Traumatic injuries to young permanent teeth are not uncommon and are said to affect 30% of children.⁽¹⁾ The majority of these incidents occurs before root formation is complete⁽²⁾ and may result in pulpal inflammation or necrosis.

Apexification is a method of inducing root end closure of an incompletely formed non vital permanent tooth by removing the coronal non vital radicular tissue just short of the root end and placing in the canal a suitable biocompatible agent such as calcium hydroxide⁽³⁾ or MTA.⁽⁴⁾ The rationale is to establish an apical stop that would enable the root canal to be filled.

Because of the important role of Hertwig's epithelial root sheath in continued root development after injury, every effort should be made to maintain its viability. It is thought to provide a source of undifferentiated cells that could give rise to further hard tissue formation. It may also protect against the in growth of ligament cells into the root canal, which would result in intracanal bone formation and arrest of root development.⁽⁵⁾ Complete destruction of Hertwig's epithelial root sheath results in cessation of normal root development. This does not however mean that there is an end to deposition of hard tissue in the region of the root apex. Once the sheath has been destroyed there can be no further differentiation of odontoblasts. However, hard tissue can be formed by cementoblasts that are normally present in the apical region and by fibroblasts of the dental follicle and periodontal ligament that undergo differentiation after the injury to become hard tissue producing cells.⁽⁶⁾ Cooke and Robotham⁽⁷⁾ hypothesize that the remnants of Hertwig's epithelial root sheath, under favorable conditions, may organize the apical mesodermal tissue into root components.

Although a variety of materials have been proposed for induction of apical barrier formation, calcium hydroxide has gained the widest

acceptance. As the calcium ions from calcium hydroxide dressing do not come from calcium hydroxide but from the blood stream^(8,9), the mechanism of action of calcium hydroxide remains controversial.

Holland et al⁽¹⁰⁾ has demonstrated that the reaction of the periapical tissues to calcium hydroxide is similar to that of pulp tissue. It produces a multilayered necrosis with subjacent mineralization. Schroder and Granath⁽¹¹⁾ have postulated that the layer of firm necrosis generates a low-grade irritation of the underlying tissue sufficient to produce a matrix that mineralizes. Calcium is attracted to the area and mineralization of newly formed collagenous matrix is initiated from the calcified foci. It appears that the high pH of calcium hydroxide is an important factor in its ability to induce hard tissue formation.⁽¹²⁾

Controversy exists as to whether or how often the calcium hydroxide should be changed.⁽¹³⁻¹⁶⁾

Abbot⁽¹⁷⁾ suggests that the ideal time to replace a dressing depends on the stage of treatment and the size of the foramen opening. This must be assessed for each individual tooth at each stage of development.

Enamel matrix derivative (EMD) has been shown to enhance new connective tissue attachment, defined as new cementum with inserting collagen fibres, in addition to new alveolar bone formation in both animals and humans.⁽¹⁸⁻²²⁾ EMD has been successfully employed to incite natural cementogenesis to restore a fully functional periodontal ligament, and alveolar bone in patients with advanced periodontitis.⁽²⁴⁾ Moreover, EMD was reported to eliminate inflammatory root resorption and infection in young replanted permanent teeth.⁽²⁵⁾

The emdogain induced process actually mimic parts of normal odontogenesis, and it is believed the EMD proteins participate in the reciprocal ectodermal – mesenchymal signaling that control and pattern these processes.^(18,19)

Since exposure to enamel proteins takes place during tooth development in early childhood, tolerance should normally be induced and protein recognized by the immune system as "self" proteins. Changes in the protein structure and conformation or occurrence of unrelated proteins would be possible sources for an immune response against the product. However, enamel proteins have stayed virtually unchanged during evolution.⁽²⁶⁾ Zetterson et al⁽²¹⁾ demonstrated a very low, if any immunogenic potential of EMD when applied to the dental root surface in conjunction with periodontal surgery. The same was reported by Petmaki et al⁽²⁶⁾ whose study indicated a restricted immune response at concentrations far higher than those used in clinical practice.

The present study was performed to test if emdogain could induce apical closure (barrier) without eliciting adverse side effects in immature permanent teeth of dogs. Conventional treatment with calcium hydroxide is commonly used as a control in similar experiments.