Apexification of a primary central incisor: 6-year follow-up

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Removal of abscessed primary teeth has been suggested because they have been shown in some studies to create developmental defects with underlying permanent successors.[1–3] However there might be a rationale for attempting pulp therapy in some carefully selected cases, i.e., abscessed incisor prior to eruption of primary cuspids, as space closure may occur.[4] In necrotic immature teeth, the wide and funnel-shaped root canals cause difficulties for endodontic treatment, especially removal of all necrotic tissues from the canal walls and adequate obturation. A number of treatment solutions have been described. Only when apexification using calcium hydroxide was introduced[5] could favorable results be obtained. Apexification promotes elongation of the root and formation of a calcific closure across the enlarged apex of immature permanent teeth. Whether this procedure can be used successfully in primary teeth is unknown.

The purpose of this article is to describe the apexification of a nonvital, primary maxillary central incisor treated with calcium hydroxide followed for 6 years until its permanent successor erupted.

Case report

A 14-month-old Thai girl was referred by another dentist. Antibiotics had been prescribed for emergency treatment of upper lip swelling extending from the gingiva of the upper incisors. The swelling subsided in 1 week. Clinical examination revealed that the maxillary right and left primary central incisors had soft carious lesions on the labial, incisal, palatal, and mesial surfaces. An occlusal radiograph (film size 2) revealed deep carious lesions, possibly with pulp exposure of both teeth (Fig 1). The periapical area, periodontal space, and lamina dura were normal. Roots of both teeth were about 70–80% formed, with wide open apices.

The patient was wrapped in a restraint board, and caries on the maxillary right and left primary central incisors were removed under local anesthesia using a rubber dam. The maxillary left primary central incisor had no pulp exposure; it was lined with Dycal (Kerr, Orange, CA) and restored with composite. The pulp of the maxillary right primary central incisor was exposed, and fetid odor and purulent exudate were noted when the pulp chamber was opened. Calcium hydroxide apexification was the treatment of choice, as obliteration of the canal of this tooth would be difficult with its open apex.

Root length was determined by taking radiographs with a file placed in the root canal. After pulp remnants had been removed with barbed broaches, the canal was irrigated with chlorinated soda after instrumentation with files. The canal was dried with large paper points and filled with calcium hydroxide powder mixed with

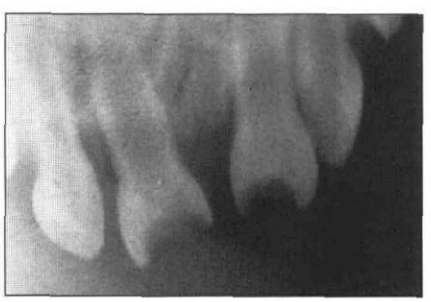

Fig 1. Fourteen months. Pre-op radiograph of teeth E and F taken when the patient presented with swelling of the upper lip, shows wide open apices.

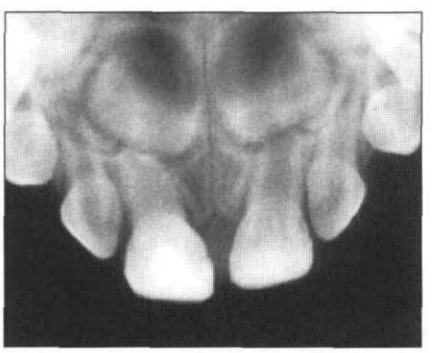

Fig 2. Seventeen months. Apexification of tooth E with calcium hydroxide powder mixed with distilled water, the crown restored with composite at 3 months.

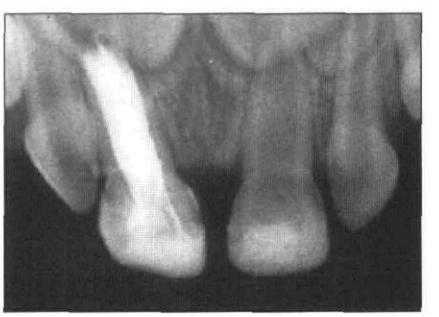

Fig 3. Twenty months. Root canal of tooth E, filled with zinc oxide eugenol paste at 6 months.

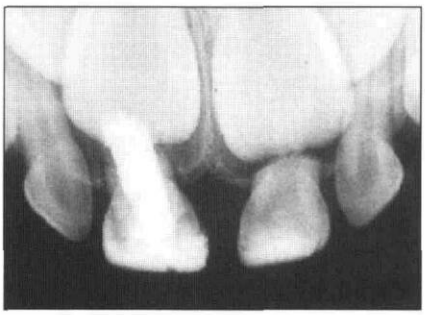

Fig 4. Twenty-six months. Thickening of periodontal space of tooth E after trauma 6 months after root canal filling.

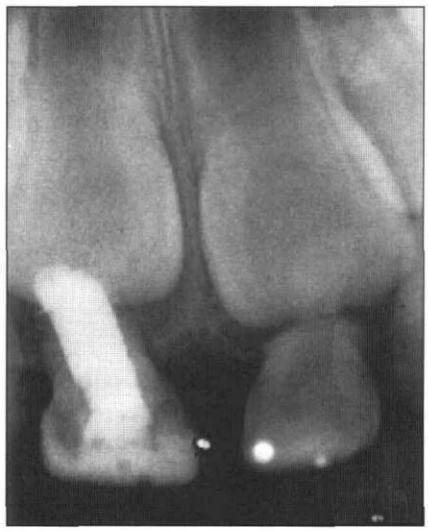

Fig 5. Six years old. Teeth E and F resorbed at approxiamtely the same rate at 5 year follow up.

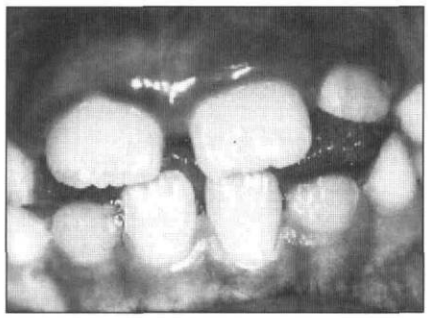

Fig 6. Seven years old. Tooth #8's surface and color appeared normal, similar to tooth #9 at 6 year follow up.

distilled water using a file and damp cotton pellets. A cotton pellet was placed over the calcium hydroxide and the seal was completed with intermediate restorative material (IRM, L.D. Caulk). The procedure was repeated after 3 months and the tooth was restored with the composite crown (Fig 2). After a further 3 months, the root canal was reopened, cleaned, and upon probing with a file, there appeared to be an apical stop. The root canal was then filled with zinc oxide eugenol paste, zinc phosphate cement, and composite filling at the palatal opening (Fig 3). The apexification was considered successful, as there were no untoward clinical signs and symptoms and the radiograph showed no pathologic changes. Oral hygiene and feeding instructions were given at the beginning of treatment. By the third appointment, she was weaned from the bottle completely. A sodium fluoride tablet (0.25 mg) was sucked once a day before bed.

Six months after the root canal of the maxillry right primary central incisor was filled, the patient had a fall and second degree mobility with fracture of the composite crown was observed. Thickening of the periodontal space was seen in a radiograph (Fig 4). The composite crown was replaced. The parents were informed of the possibility of a poor prognosis and to give her soft food for a few weeks. At follow-up there was less mobility of the incisor. The patient did not complain of any pain or discomfort and no abnormal root resorption was seen in the radiograph.

At the 5-year follow-up, a radiograph showed that the roots of the maxillary right and left primary central incisors had resorbed at approximately the same rate, (Fig 5) with 10–20% of the cervical roots left. The only difference was the bulk of zinc oxide eugenol beyond the level of the maxillary right primary central incisor's resorbed root. Six months later, the maxillary left primary central incisor exfoliated followed by eruption of the maxillary left permanent central incisor. The maxillary right primary central incisor exfoliated shortly afterwards, and the maxillary right permanent central incisor had erupted by the 6-year follow-up, with its surface and color normal and similar to the maxillary left permanent central incisor (Fig 6).

Discussion

If the maxillary right primary central incisor was left untreated, the tooth bud of the maxillary right permanent central incisor might have developed a hypoplastic lesion. Because the apex of the maxillary right primary central incisor was open, it was possible that the infection could continue to be chronic and cause a localized type of hypoplasia as described by Turner.[6] Baur[7] concluded, from a study of autopsy material, that the periapical inflammatory processes of primary teeth extends toward the buds of the developing permanent teeth and affects them during their prefunctional stage of eruption. The infection fails to stimulate the development of a fibrous wall that would localize the lesion. Instead, the infection spreads diffusely through the bone around the buds of the successors, thereby affecting the important protective layer of the young enamel—the united enamel epithelium.

The purpose of apexification with calcium hydroxide is to achieve formation of an apical hard-tissue barrier, against which an adequate root filling can be placed. The success rates range from 94–96% in immature permanent incisors,[8–10] as a result of the strong antibacterial property of calcium hydroxide (which is related to its high pH, 12.5).[11] Ninety-nine percent of bacteria from common root-canal flora are killed within a few minutes upon direct contact with calcium hydroxide.[12] Elimination of bacteria from the root canal enables healing of the surrounding vital tissue and the formation of an apical cementoid barrier, indicating that periodontal tissues are involved in the process.[13] Necrotic pulp remnants are also dissolved by calcium hydroxide, rendering the root-canal walls clean;[14] this is important, as the thin dentinal walls of immature teeth do not permit intensive reaming.

Because this procedure has not been studied before in primary teeth, one should not overlook some po-

tential side-effects that occasionally develop after pulp therapy has been completed, such as iatrogenic damage to the developing tooth bud[1] or possible reinfection of the primary incisor.[15]

Even though apexification was successful in this child, periodic observation was necessary to intercept if early or delayed exfoliation interfered with normal eruption of the permanent successor. Sometimes extraction of primary incisors is necessary to correct lingual eruption of the permanent incisors, usually in the maxillary arch. Starkey[16] believes that this phenomenon occurs when normal physiologic exfoliation is delayed by the bulk of cement contained in the pulp chamber. Though the material is resorbable, resorption is impaired significantly when large quantities are present.

In the case of possible space loss, one may attempt to perform apexification of necrotic immature primary incisors rather than extraction. More studies should be conducted on the success rate—and its consequences—on the formation and eruption of the succedaneous teeth before its use generally can be recommended.

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References

1. Matsumiya S: Experimental pathological study on the effect of treatment of infected root canals in the deciduous tooth on growth of the permanent tooth germ. Int Dent J 18: 546–59, 1968.
2. Valderhaug J: Periapical inflammation in primary teeth and its effect on the permanent successors. Int J Oral Surg 3:171–82, 1974.
3. Brook AH, Winter GB: Developmental arrest of permanent tooth germs following pulpal infection of deciduous teeth. Br Dent J 139:9–11, 1975.
4. Kapala J: Interceptive orthodontics and management of space problems. In Textbook of Pediatric Dentistry 1st Ed. RL Braham, ME Morris EDS. Baltimore: Williams & Wilkins, pp 336, 1980.
5. Frank AL: Therapy for the divergent pulpless tooth by continued apical formation. J Am Dent Assoc 72:87–93, 1966.
6. Turner JG: Two cases of hypoplasia of enamel. Br J Dent Sci 55:227–28, 1912.
7. Baur WH: Effect of periapical processes of deciduous teeth on the buds of permanent teeth. Am J Orthod 32:232–41, 1946.
8. Kerekes K, Heide S, Jacobsen I: Follow-up examination of endodontic treatment in traumatized juvenile incisors. J Endod 6:744–48, 1980.
9. Mackie IC, Bentley EM, Worthington HV: The closure of open apices in non-vital immature incisor teeth. Br Dent J 165:169–73, 1988.
10. Cvek M: Prognosis of luxated non-vital maxillary incisors treated with calcium hydroxide and filled with gutta-percha. Endod Dent Traumatol 8:45–55, 1992.
11. Javelet J, Torabinejad M, Bakland L: Comparison of two pH levels for the induction of apical barriers in immature teeth of monkeys. J Endod 11:375–78, 1985.
12. 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 1:170–75, 1985.
13. Binnie WH, Rowe AHR: A histological study of the periapical tissues of incompletely formed pulpless teeth filled with calcium hydroxide. J Dent Res 52:1110–16, 1973.
14. Andersen M, Lund J, Andreasen JO, Andreasen FM: In vitro solubility of human pulp tissue in calcium hydroxide and sodium hypochlorite. Endod Dent Traumatol 8:104–108, 1992.
15. McDonald RE, Avery DR: Treatment of Deep caries, Vital Pulp Exposure and Pulpless Teeth. In Dentistry for the child and Adolescent, 6th Ed. RE McDonald, DR Avery EDS. St. Louis: Mosby-Year Book, Inc. pp 451, 1994.
16. Starkey PE: Treatment of pulpally involved primary molars. In Current Therapy in Dentistry, vol 7, RE McDonald, WCHurt, HW Gilmore and RA Middleton EDS. St Louis: CV Mosby, pp 430, 1980.