Stimulation of root formation in incompletely developed pulpless teeth

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treatment of teeth in which the dental pulp has become totally necrotic before completion of root development is difficult if the operator depends on a coronal approach and attempts to seal the patent canal via the root. Because of this, many endodontists have favored a surgical approach and have endeavored to seal the apex by using a retrograde root-filling material.

However, surgical management is not entirely satisfactory because of inherent difficulties in the operative procedure and because of the physical and psychologic trauma to a young patient. Furthermore, a tooth treated in this manner often has inadequate root length to withstand masticatory forces, and the periapical tissues may fail to adapt to the wide and relatively irregular surface of the retrograde root-filling material. Thus, the long-term prognosis is generally doubtful.

In order to overcome the difficulties just mentioned, some writers have essayed a conservative approach and have used calcium hydroxide, either alone or in combination with other agents, to stimulate further root development. To date there have been few case reports, and as yet the results of a histologic examination have not been published.

The purpose of this article is to present case reports of twenty-one teeth which have been treated by a conservative method and to provide details of histologic examination of a tooth which received conservative treatment but which had to be extracted later because of a vertical root fracture. In presenting this series, I hope to provide further support for conservative treatment of the pulpless “blunderbuss” incisor.

MATERIALS AND METHODS

A. Clinical series

The patients in the present series were referred for endodontic treatment of teeth which had suffered pulp death and consequent periapical involvement

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at a stage of incomplete root development. A total of twenty-one teeth were treated in seventeen patients whose ages ranged from 7 to 11 years.

The causes of pulp death were trauma (eighteen teeth), caries (one tooth), and deep palatal invagination with subsequent bacterial invasion (two teeth).

All teeth had evidence of periapical pathology on radiographic and clinical grounds. In four cases (Teeth A3, A8, A9, and A11) the patients had acute periapical osteitis and associated cellulitis of the lip and face.

**Clinical procedure**

In each case, the root canal was sterilized by routine endodontic procedures. Mild medicaments were used for irrigation and root canal dressing. The solution used for both purposes in this series was an aqueous solution of 0.03 per cent chlorhexidine and 0.3 per cent cetrimide (Savlon). After sterilization of the canal had been achieved, the walls were dried with a cotton pellet attached to the tip of a large file or with the blunt ends of paper points. Calcium hydroxide and methyl cellulose (Pulpdent) paste was then introduced by means of a Pulpdent syringe with a Luer-Lok needle attached. Excess paste was removed from the pulp chamber, and the coronal opening was sealed with Cavit and amalgam. Patients were recalled after 1 month and 3 months, and at 6-month intervals thereafter. The observation times ranged from 14 to 75 months.

**B. Histologic specimen**

A central incisor tooth treated by the above method became available for histologic examination when a vertical root fracture made extraction necessary. The patient had been treated at the Dental Department of the Royal Adelaide Hospital in April, 1965. He had failed to report for follow-up examinations but returned in pain in May, 1968. The tooth was extracted, decalcified in a formic acid–sodium formate solution, and serially sectioned at 7 microns. Sections were stained with either hematoxylin and eosin or Mayer's hematoxylin and eosin.

**RESULTS**

**Clinical series**

The results of treatment are summarized in Table I.

Representative cases (Teeth A9, A13, A17, and A7) are shown in Figs. 1 through 8. In each instance the case histories are included in the legends.

Of the twenty-one teeth treated by this method, only Teeth A6 and A18 (Figs. 5 and 6) did not show evidence of further root development. The observation times for these two cases were 55 months and 18 months, respectively. Periapical repair was complete in Tooth A6 and, on clinical investigation, viable tissue was present within the root canal. In Tooth A18 there was a breakdown of the coronal seal after 10 months, and bacterial contamination resulted. Subsequently, the tooth was re-treated, and the 18-month follow-up examination disclosed positive evidence of periapical repair, although this was not complete. No further root development could be demonstrated at this stage.

Of the nineteen teeth which demonstrated further root development, five
showed partial apical root formation and fourteen showed complete apical root formation. In all cases, root formation had proceeded to such a stage that the apical opening was convergent rather than divergent.

There did not appear to be any difference in the reparative response in those cases in which the patient presented in an acute inflammatory state (Teeth A3, A8, A9, and A11) and the remaining cases in which there were associated chronic periapical lesions.

A definite fine root canal could be observed in all teeth that had progressed to completion of root formation. No complete calcific barrier could be observed at the coronal extremity of the apical canal in these cases. In four teeth permanent root fillings of gutta-percha and AH-26 were placed after apical development was complete. In each case bleeding tissue was encountered within the root canal in the apical region.

**Histologic specimen**

Histologic examination revealed that the newly deposited apical tissue not only encircled but extended into the original root. The tissue forming the apex of the specimen consisted of remnants of pulp tissue, dentine, and thick deposits of cementum with attached periodontal membrane (Fig. 9).

A buckling of the original root apex was evident (framed areas in Fig. 9
calcium hydroxide and methylcellulose paste

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and Figs. 10 and 11). In the region of this buckling there was evidence of resorption on the outside of the root. This had subsequently been repaired by the deposition of cellular and acellular cementum which also extended in thick layers over the entire apical plug of calcific tissue. The major part of this apical plug consisted of interglobular dentine. In some areas there was a tendency toward regular tubule formation, but in the region of the outward buckling of the original root the dentine was of a particularly irregular character.

Within the old canal, two distinct layers had been deposited. There had been some separation of these layers during the histologic preparation and thus slight artifacts were evident (Figs. 9 and 10). Both layers which extended 3 to 4 mm. into the old canal consisted of interglobular dentine, but the first layer was thin and seemed less tubular than the thicker superimposed layer. No calcific barrier bridging the original canal was evident at the coronal extremity of these layers.

A fine root canal was present in the newly formed apical region. This contained tissue resembling the dental pulp, but infiltration with chronic inflammatory cells had occurred. No normal pulp tissue could be observed in the junction between the fine apical canal and the wide original canal. A lateral canal containing chronically inflamed pulp tissue was present in this region. Scattered carbon artifacts were present in many sections as a result of the pencil lead which had been inserted into the canal by the patient.
Fig. 1. Tooth A9. Upper left central incisor of an 8-year-old boy. The patient, who presented with an acute periapical osteitis, gave a history of a blow 2 months earlier. A radiolucent periapical lesion was evident. Root treatment was followed by the insertion of calcium hydroxide-methylocellulose paste.

Fig. 2. Tooth A9. Two years later the tooth showed considerable root development and periapical repair.

Fig. 3. Tooth A13. Upper right lateral incisor of a 10-year-old girl. The cause of the chronic periapical osteitis was a deep palatal invagination of the cingulum with subsequent intrapulpal bacterial invasion. A periapical radiolucency was evident (see Fig. 1).

Fig. 4. Tooth A13. One year later, following the placement of a calcium hydroxide and methylocellulose paste, apical development and periapical repair have occurred.
Fig. 5. Teeth A17 and A18. A 7-year-old girl with a history of traumatic injury to the upper right central and lateral incisors 2 months previously. On initial examination, both teeth were pulpless and there was an acute periapical osteitis associated with the lateral incisor. Both teeth were treated and filled with calcium hydroxide and methylcellulose paste. The coronal seal in the central incisor was deficient, and contamination of the canal occurred after 4 months. Subsequently, the canal was re-treated and filled with the same paste.

Fig. 6. Teeth A17 and A18. Ten months later complete root development was apparent in the upper right lateral incisor, and there were signs of periapical repair in the central incisor.

Fig. 7. Tooth A7. Upper right central incisor in an 8-year-old boy. There was a chronic periapical osteitis, with an associated draining sinus. The tooth was treated and filled with the calcium hydroxide and methylcellulose paste.

Fig. 8. Tooth A7. The patient returned 2½ years later, having failed to keep appointments for follow-up examinations. The coronal seal had been lost; caries and a vertical root fracture with an associated lesion on the mesial aspect of the root were present. In addition, the patient had pushed the lead of a pencil into the canal. Considerable apical root development had occurred since the initial treatment. This tooth was extracted and prepared for histologic examination.
DISCUSSION

The clinical series described here provides support for conservative endodontic treatment in incompletely developed teeth where the aim is stimulation of further root formation.

Clinical evaluation, based on evidence of periapical repair and root development, revealed that in nineteen of the twenty-one cases treatment was considered successful. Two cases (Teeth A6 and A18) were classified as doubtful although periapical repair was complete in one (Tooth A6) and was progressing satisfactorily in the other (Tooth A18). No tooth in the present material was considered to represent a treatment failure.

The clinical features of the root formation in the present series were essentially the same as those in previously published case reports, although Steiner and associates indicated that a definite bridge or calcific barrier was present. They reported that a permanent root filling could be placed without any risk of extending the root-filling material apically. In teeth with apical development a definite fine apical canal could be seen radiographically, but there was no definite apical barrier. The clinical observation of bleeding tissue within the canals of those four teeth in which roots were filled offers additional evidence that no calcific barrier existed between the fine apical canal and the original wider canal.
The histologic specimen similarly did not demonstrate any calcified barrier between the newly formed apical plug and the old canal. Indeed, in this histologic specimen, the absence of such a barrier can be assumed to be the reason for the unimpeded bacterial invasion which followed the breakdown of the coronal seal. This would account for the chronic inflammatory changes observed in the apical pulp, but it would seem reasonable to assume that prior to bacterial invasion this tissue had been in a healthy state.

The histologic findings show that apical development had occurred by the formation of relatively normal dental tissues of pulp, dentine, and cementum. Not only was there development apically, but there was also deposition of new dentine within and also on the surface of the original canal, indicating that the replacement of the calcium hydroxide by ingrowing pulp tissue may have been a continuing process.

The repair reactions that have been observed are interesting, and some speculation can be made as to their nature. Epithelium is thought to be resistant to inflammatory changes. Thus, it is possible that in these cases Hertwig's
Fig. 12. Section through the root canal in the apical region. (Hematoxylin and eosin stain. Magnification, ×40.) Pulp contains many chronic inflammatory cells following contamination of the root canal with bacteria. Black carbon artifacts from the lead of a pencil inserted into the canal by the patient are scattered over the field. Thick apical deposits of cementum can be observed.

Fig. 13. Higher magnification (×100) showing chronically inflamed pulp tissue at the apical foramen.

Sheath can survive and so remain able to continue its role of organization of root development when the inflammatory process is eliminated. Accordingly, if bacteria are removed and a material which favors repair of the periapical tissues is subsequently introduced into the root canal, Hertwig's sheath may be expected to continue more or less in its normal fashion. The cells in the periapical region of an incompletely developed tooth may perhaps be considered to be pluripotential and thus subject to differentiation into cells which can form normal dental tissues after the inflammatory reaction resolves.

The material used in filling the root canal may aid the process of differentiation. Calcium hydroxide has been considered by some workers to have an osteogenic potential. However, this same development has been reported by Cooke and Rowbotham, who used an antiseptic paste containing zinc oxide and eugenol, and also by Ball, who used an antibiotic paste. Calcium hydroxide may exert a favorable effect by virtue of its high alkalinity altering the pH of the region, or perhaps the calcium ions may increase the local capillary permeability, and this may favor repair. Histochemical studies may help to solve the mode of action of calcium hydroxide in its present application.

The absence of an apical calcific barrier and the presence of ingrowing pulp
tissue, as shown by the clinical and histologic material, pose additional clinical problems. If a permanent root filling of gutta-percha and a suitable sealer are inserted directly onto the apical pulp tissue, an adverse pulpal response can be expected. Should a permanent root filling be necessary for restorative purposes, the ingrowing pulp tissue can be removed down to the level of the entrance of the fine apical canal and the pulp wound dressed with a thin layer of calcium hydroxide. The rest of the canal can then be filled with gutta-percha and a sealing agent. The pulp remnant may then undergo the normal repair reactions which follow partial pulpectomy using calcium hydroxide. An alternative and probably safer method would be to extend the root filling to the apex of the tooth after removal of the pulp tissue and widening of the fine apical canal.

It is hoped that most of the teeth in the present series will not require permanent root fillings for some years. Further observation may reveal additional calcific changes and furnish material for subsequent reports.

Conservative treatment of incompletely developed pulpless teeth with calcium hydroxide provides a simple, nontraumatic solution to an otherwise difficult problem. The method has the advantage that greater root length is attained, with its obvious advantages in the long-term stability of the tooth. Once root development has been completed, an orthodox root filling can be placed without technical difficulty.

SUMMARY

A series of twenty-one incompletely developed pulpless human teeth have been treated conservatively with calcium hydroxide and methylcellulose as a root-filling material. Observation times varied from 14 to 75 months.

Elimination of bacteria was achieved by routine endodontic procedures in conjunction with irrigation and dressing with an aqueous solution of 0.03 per cent chlorhexidine and 0.3 per cent cetrimide.

Of the twenty-one cases, fourteen showed complete, five partial, and two no root development during the period of observation. Periapical repair was complete in twenty of the twenty-one cases and is proceeding satisfactorily in the remaining case. A clinical evaluation of success showed nineteen cases to be successful and two doubtful, with no failures over the period of observation.

Histologic material has been presented which revealed that new tissue had been formed, both apically and within the old canal. This consisted of pulp, interglobular dentine, cementum, and attached periodontal membrane fibers. Two calcified layers of interglobular dentine extended into the old canal and lined it. However, no calcific barrier was present at the coronal extremity of the canal. Thick deposits of cellular and acellular cementum not only covered the newly formed tissue but extended beyond the junction with the old root.

The results of the clinical series and the histologic material support the conservative approach to the treatment of pulpless, incompletely developed teeth. The method is indicated because of its simplicity, the lack of surgical trauma, and the improved prognosis afforded by the further root development.

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REFERENCES


ERRATUM

In the article entitled “Pathology of Oral Tonsils” by Milton J. Knapp, which appeared on pages 295-304 of the February, 1970, issue of the JOURNAL, the first reference on page 304 should read as follows: