SCIENTIFIC ARTICLES

A Clinical Report on Partial Pulpotomy and Capping with Calcium Hydroxide in Permanent Incisors with Complicated Crown Fracture

Miomir Cvek, DMD, PhD, Stockholm

The exposed pulps of 60 permanent incisors with a complicated crown fracture were treated with partial pulpotomy and calcium hydroxide dressing. The interval between accident and treatment varied from one to 2,160 hours and the size of the pulpal exposure varied from 0.5 to 4.0 mm. Of the teeth, 28 had immature and 32 had mature roots. The treatment was successful in 58 teeth or 96% according to the following criteria: no clinical symptoms, no radiographically observed intraradicular or periradicular pathologic changes, continued development of an immature root, radiographically observed and clinically verified hard tissue barrier, and sensitivity to electrical stimulation. The follow-up examination varied from 14 to 60 months, with an average of 31 months.

An exposed pulp in young crown-fractured teeth is usually treated with either capping or pulpotomy, depending on the degree of pulp exposure, the interval between accident and examination, and the stage of root development. Calcium hydroxide is regarded as the preferred dressing.

Capping of the pulp is recommended when the exposure is small and when it can be treated shortly after the accident. These indications apply to only a limited number of teeth and, in the majority of cases, pulpotomy is therefore performed.

Pulpotomy involves removing the injured coronal pulp, with the incision placed at the level of or near the coronal opening in the root canal. Because of objections of a biologic as well as technical nature,[1-6] the treatment has been looked on as temporary, to be followed by pulpectomy when the root has matured, which is unsatisfactory per se.

Partial pulpotomy, that is, removal of only part of the coronal pulp adjacent to the exposure, has been recommended for those crown-fractured teeth that have a pinpoint exposure and can be treated within 15 to 18 hours of the accident.[7] These indications, however, have never been studied experimentally and no comprehensive investigation of the therapeutic results is on record.

Little is known about tissue reactions, especially during the first days, in a pulp exposed through a crown fracture to the oral environment. Changes in tissue have been described either as destructive, such as formation of abscesses or necrosis as a result of mechanical injury and contamination of damaged pulp tissue,[8] or as proliferative, seen clinically as hyperplastic tissue in the pulpal lesion or later as a polyp of the pulp.[9] It has been said that a hyperplastic response of the pulp to acute inflammation occurs in very young teeth that have a richly vascularized pulp[10] but never in teeth of older persons.[8] It has also been clearly shown that a necrotizing process in the exposed pulp of rat teeth is caused by oral microorganisms,[11] whereas the factors underlying a proliferative reaction of the dental pulp have never been assessed experimentally.

Clinical observations during a

period of two years before the current investigation disclosed, however, that not one of the permanent incisors with a complicated crown fracture that were examined 12 hours or more after the accident showed any sign of necrotic disintegration, whereas they all had a proliferative response with the clinical appearance of granulation tissue or hyperplasia of the pulp. This observation corroborates the results reported recently by Smukler and Tagger.[12] When the roots of multirooted teeth in adults were amputated and the pulp was exposed (in a way that resembles the exposure caused by a fracture) and left untreated for two weeks, they found clinically a polyp of the pulp in nearly half of the teeth and found histologically in the remaining pulp of all teeth changes consistent with a histopathologic diagnosis of hyperplastic pulpitis restricted to the coronal pulp. The findings thus confirm earlier observations that in the pulps with hyperplastic pulpitis, only superficial layers are involved by inflammatory processes.[13]

These results seem to justify the assumption that the area of exposed pulp tissue and the clinically relevant interval between accident and treatment are not critical for primarily healthy pulp, provided superficially inflamed tissue is removed and the technique used minimizes the injury to the remaining pulp. A gentle technique, inflicting only insignificant injury to the underlying connective tissue, has been described by Granath and Hagman.[14]

The purpose of the current investigation was clinically and radiologically to assess the frequency of healing of accidentally exposed pulp treated by partial pulpotomy and calcium hydroxide dressing, taking into account the size of pulpal exposure, the interval between accident and treatment, and the stage of root development of permanent crown-fractured incisors.

MATERIAL AND METHODS

The original sample consisted of 83 traumatized permanent incisors with a complicated crown fracture of 74 patients that were referred to the Eastmaninstitutet, Stockholm, between 1973 and 1976 and treated there with partial pulpotomy and calcium hydroxide dressing.

Twenty-two teeth were excluded because the pulpal lesion previously had been capped temporarily with a dressing by the referring dentist. A 12-month follow-up was adopted as a minimum time and one tooth did not meet this requirement. After this selection, the sample consisted of 60 teeth, 51 maxillary and nine mandibular incisors. Of the patients, 16 were girls and 38 were boys, aged 7 to 16 years.

At the examination before treatment, the teeth showed no mobility or slightly increased mobility as a result of accidental injury, with or without tenderness to percussion. The interval from the accident and the size and appearance of the pulpal lesion were registered, and the crown-fracture area was photographed. The crown fracture was transverse or oblique in all teeth except four, which had longitudinal crown-root fractures. In no case was necrotic tissue disintegration seen in the lesion but all cases had a bleeding wound or a proliferated pulp tissue. All teeth were sensitive to electrical stimulation.

For treatment, the tooth was isolated with a rubber dam and cleaned with a 0.5% chlorhexidine solution. Proportional to the area of exposed pulp tissue, only part of the coronal pulp and surrounding dentin

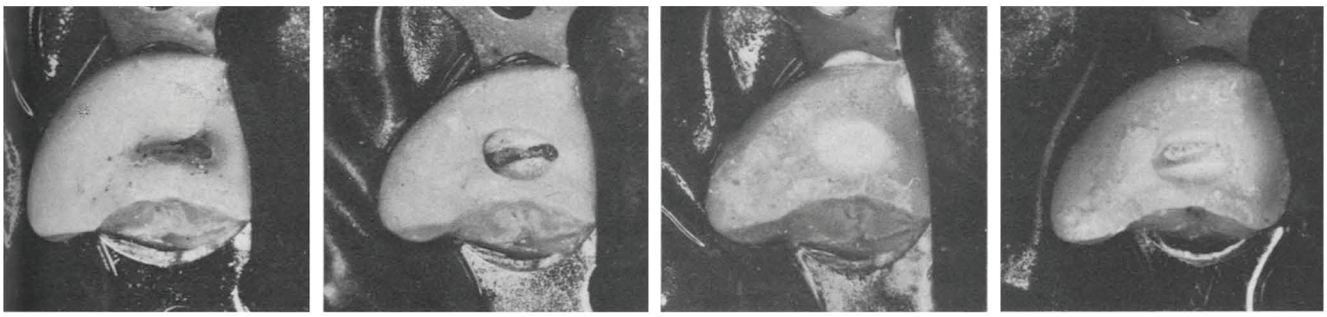

Fig 1—Boy, aged 10, right central incisor. From left to right: exposure of 2.5 mm of pulp, 37 hours after accident, granulation tissue in pulp lesion; physiologic hemostasis after amputation; cavity sealed with zinc oxide-eugenol cement; and hard tissue barrier as seen at clinical examination six months after treatment.

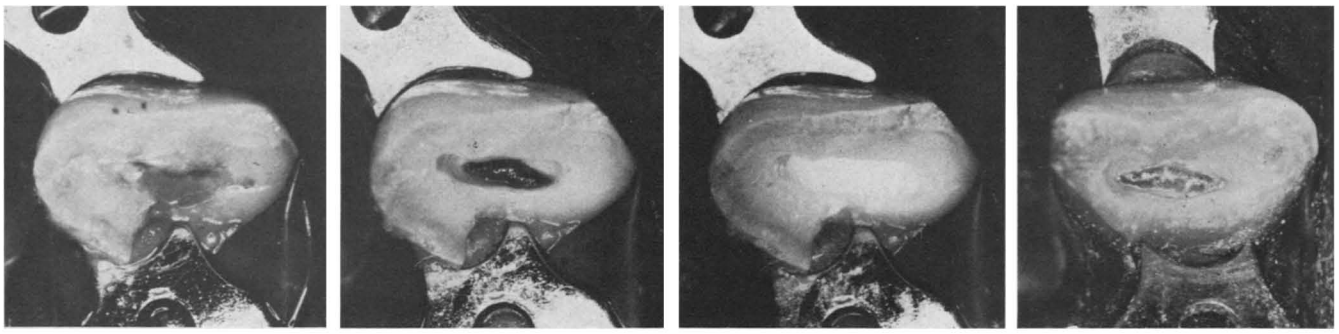

Fig 2—Boy, aged 13, left central incisor. From left to right: exposure of 4 mm of pulp, three months after accident, hyperplasia of pulp; hemostasis after amputation; pulp capped with calcium hydroxide (proposed gingivectomy was not accepted by the patient); and hard tissue barrier four months later.

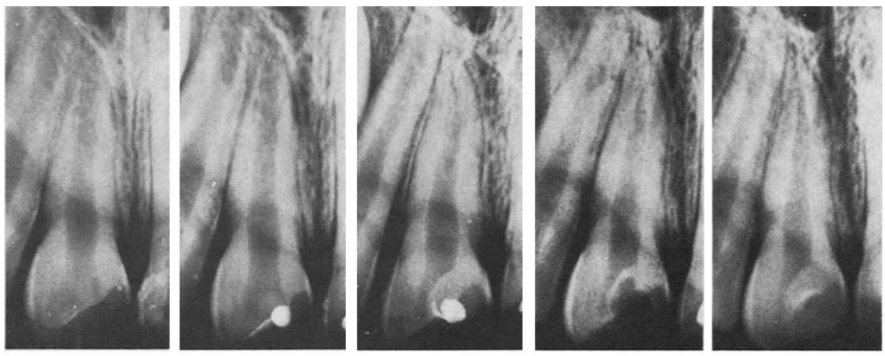

Fig 3—Radiographs of same case as shown in Fig 1. From left to right: 37 hours after accident; immediately after treatment, calcium hydroxide and zinc oxide-eugenol cement used as sealing material; before clinical check-up six months later; at follow-up examination six months after treatment; and at follow-up 36 months after treatment.

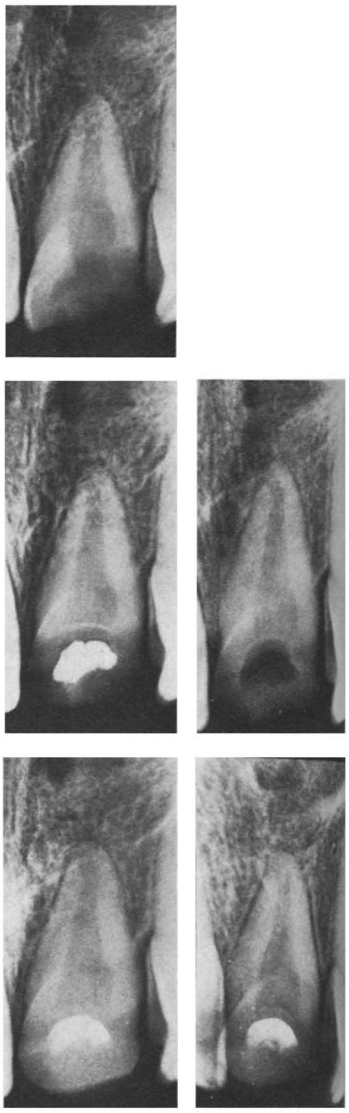

Fig 4—Radiographs of same case as shown in Fig 2. Top: three months after accident; middle: before and at time of clinical control four months after treatment; bottom: nine (left) and 18 (right) months' follow-up.

was removed, in a manner described by Granath and Hagman,[14] with a sterile diamond instrument (cylindrical or cone-shaped, no. 2 or 3) in a high-speed handpiece and cooled by ample flushing with sterile saline solution. A cavity about 2 mm deep remained, sufficient to hold the wound dressing and sealing material. Bleeding was controlled with physiologic saline solution, the pulpal wound was covered with Calasept, a calcium hydroxide product, and the cavity was sealed with sterile zinc oxide-eugenol cement (Fig 1-5). In three teeth with crown-root fracture and a wide lateral pulpal exposure, the entire pulp tissue coronal to the lesion was removed to avoid its constriction by hard tissue development and subsequent necrosis, as described by Stanley and Lundy[15] (Fig 6). The treatment was performed by 11 dentists.

The teeth were checked clinically and radiographically three weeks after treatment and thereafter during the first year at three-, and later, at six-month intervals. Radiographs obtained before treatment and at the follow-up examinations were taken with at least two standardized projections. The observation period ranged from 14 to 60 months, with an average of 31 months.

When a continuous hard tissue barrier was seen in the radiographs, the cavity seal and calcium hydroxide were removed under aseptic conditions and the continuity of the barrier was explored clinically with a sharp probe in a manner explained by Nyborg[16] and Masterton.[17] After this examination, the barrier and the exposed dentin were covered with a hard-setting calcium hydroxide compound (Dycal) and the crown was

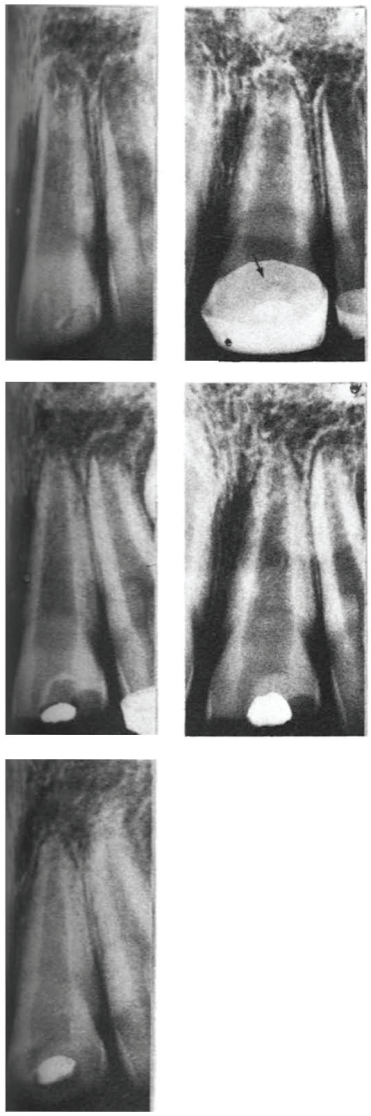

Fig 5—Boy, aged 9, right central incisor. Top left: three hours after accident; top right: three weeks after treatment, initial hard tissue formation (arrow); middle: before and after clinical inspection six months later; bottom: follow-up 60 months after accident.

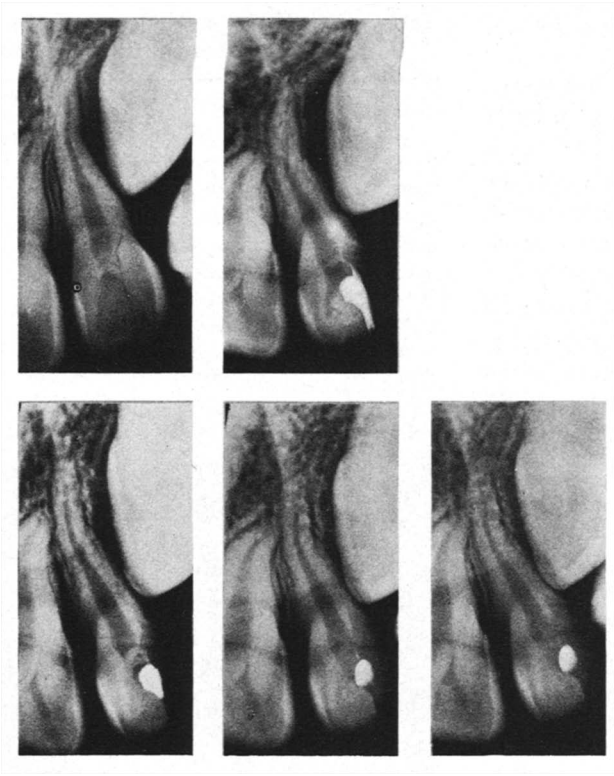

Fig 6—Girl, aged 12, left lateral incisor. Top left: complicated crown-root fracture 16 hours after accident; top right: 12 weeks after initial hard tissue formation; bottom: 6-, 9-, and 30-month follow-up.

restored with a composite material, by use of the acid-etch technique.

For evaluation, the teeth were subgrouped according to the interval between accident and treatment (1 to 8, 9 to 30, 31 to 100, and 101 to 2,160 hours, respectively) and the size of exposed pulp tissue (0.5 to 1.0, 1.1 to 2.0, 2.1 to 3.0, and 3.1 to 4.0 mm, respectively). The teeth were also grouped according to the stage of root development: the apical opening of the root canal was measured as described by Cvek;[18] when it was 0.5 mm or less, the tooth was classified as mature, otherwise as immature. The distribution of the material is presented in the Table.

Healing was considered to have taken place if there were no clinical symptoms; no radiographically demonstrable intraradicular or periradicular pathological changes; a continued root development of immature teeth; a radiographically seen and clinically verified continuous hard tissue barrier at the site of the surgical incision; and sensitivity to electrical stimulation.

RESULTS

The results are given in the Table, from which it is clear that the variables had no influence on the frequency of healing, which amounted to 96% or 58 teeth (Fig 1-6).

The initial formation of hard tissue at the site of incision was visible in the radiographs three to 12 weeks after treatment and a complete barrier was noticed after three to six months. In one case, the clinical exploration of the hard tissue barrier resulted in puncture-like

bleeding, which indicated a discontinuous demarcation; calcium hydroxide was reapplied as a dressing, and clinical inspection four months later showed a clinically continuous barrier. In the remaining teeth, the check-up showed a clinically continuous hard tissue barrier, often sensitive to exploration with a probe.

The treatment was not successful in two teeth. In one, necrosis of the pulp occurred after four days. In the second case, the patient said, 40 months after treatment, that he had recurrent pain in the tooth, which was clinically tender to percussion and sensitive to electrical stimulation; radiographs disclosed a continuous barrier and no signs of pathological changes (Fig 7). These teeth were treated 17 and 30 hours, respectively, after the accident. The size of pulpal exposure was 1 mm and the roots were mature.

Table • Distribution and outcome of 60 permanent incisors with complicated crown fracture subjected to partial pulpotomy and capping with calcium hydroxide.

Size of exposed pulp tissue in mm	Interval from accident to treatment (hours)				
	1 to 8	9 to 30	31 to 100	101 to 2,160	Total
0.5 to 1.0	9	11*	2	1	23
1.1 to 2.0	10	5	4	2	21
2.1 to 3.0	6	3	1	...	10
3.1 to 4.0	3	...	2	1	6
Total	28	19	9	4	60
Immature root	16	6	5	1	28
Mature root	12	13*	4	3	32

*One separate case of failure.

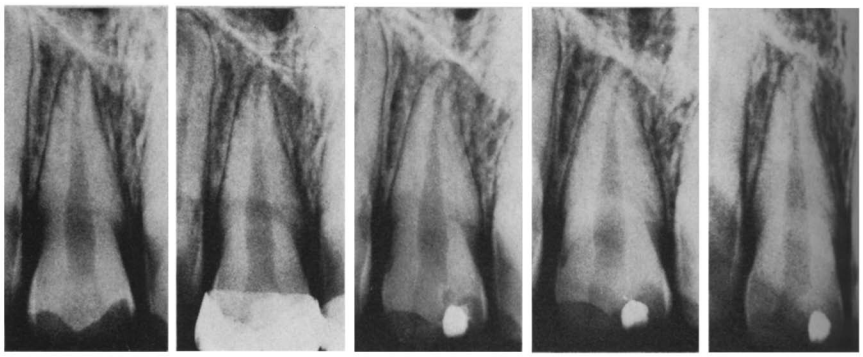

Fig 7—Girl, aged 15, left central incisor. From left to right: 30 hours after accident; three weeks after treatment; before clinical inspection six months later; after clinical follow-up six months later; 40 months after treatment, immediately before pulpectomy performed for recurrent pain in tooth.

DISCUSSION

The assumption that the size of pulp exposure and the interval between accident and treatment are not critical for healing of primarily healthy pulp was confirmed by the favorable results of the current study. The frequency of healing appears to be among the highest reported for pulp treatments performed under similar clinical conditions. The results reported by Schröder,[19] who used the same surgical technique and wound dressing in experimental pulpotomy of intact permanent premolars and found histological healing in 89% of the teeth, indicate that the remaining pulp of teeth in the current study was either healthy or capable of healing after removal of inflamed superficial layers. The frequency of healing should, therefore, be judged against the regular findings of proliferative tissue in the pulp exposure before treatment, which is a superficial, clearly defensive, and reparative reaction.[20] The decisive factors responsible for this reaction of the pulp tissue have not been investigated experimentally. The results reported by Smukler and Tagger,[12] however, indicate that the age of the patient is not determinative. It is possible that the reason for a proliferative reaction lies in the free exposure, which permits continuous salivary rinsing of the pulp exposed by a fracture and does not involve the impaction of contaminated food debris that occurs in carious and artificial cavities. Another explanation for the positive results may be that the ability of inflamed young pulp to heal was increased by the gentle surgical technique for the removal of irritants and superficial tissue. These hypotheses are currently being tested experimentally.

The frequency of healing might have been less favorable if the teeth with a follow-up shorter than average (31 months) had been checked after a longer period. However, most failures, that is, internal dentin resorption and obliteration of the root canal, that are detectable radiographically can be observed within the first year after treatment. Another conceivable objection is that the

material should have included more teeth treated eight days or later after the accident. Such cases, however, are seldom seen in clinical practice.

The reason for the failure in the first case may have been the thermal injury during surgical procedure. In the second case, four months before the last check-up the tooth was involved in a minor injury in which the composite restoration was lost and, later, the crown was again restored. Any explanation for the failure in this case must be purely speculative.

In summation, it may be concluded that in children and young adults, most teeth that have the pulp exposed by a crown fracture can be treated successfully by the method described. The advantage of the method as compared with capping lies in better control of the surgical wound and retention of the sealing material. Compared with pulpotomy, the pulp is not deprived of the possibility of continued physiologic dentin production in the crown and the cervical area of the tooth. This, coupled with the possibility of sensitivity testing, makes it unnecessary to perform pulpectomy routinely.

This investigation was supported by a grant from the Stockholms läns landsting (Bidrag till Odontologisk utvecklingsarbete, Dnr. 603-0300, 1977).

Dr. Cvek is in the department of pedodontics, Eastmanistitutet, Dalagatan 11, S-113 24 Stockholm, Sweden.

References

1. Cabrini, R.I.; Maisto, O.A.; and Manfredi, E.E. Internal resorption of dentine. Histopathologic control of eight cases after pulp amputation and capping with calcium hydroxide. Oral Surg 10:90 Jan 1957.

2. James, V.E.; Englander, H.R.; and Massler, M. Histologic response of amputated pulps to calcium compounds and antibiotics. Oral Surg 10:975 Sept 1957.

3. Kalnins, V., and Frisbie, H.E. The effect of dentine fragments on the healing of exposed pulp. Arch Oral Biol 2:96 July 1960.

4. Masterton, J.B. Internal resorption of dentine: a complication arising from unhealed pulp wounds. Br Dent J 118:241 March 16, 1965.

5. Patterson, S.S. Pulp calcification due to operative procedures—pulpotomy. Int Dent J 17:490 June 1967.

6. Langeland, K.; Dowden, W.E.; Tronstad, L.; and Langeland, L.K. Human pulp changes of iatrogenic origin. Oral Surg 32:943 Dec 1971.

7. Massler, M., and Malone, A.J. Fractured anterior teeth—diagnosis, treatment and prognosis. Dent Dig 58:442 Oct 1952.

8. Seltzer, S., and Bender, I.B. The dental pulp: biologic considerations in dental procedures, ed 2. Philadelphia, J. B. Lippincott Co., 1976, pp 252-266, 320.

9. Andreasen, J.O. Traumatic injuries of the teeth. Copenhagen, Munksgaard, 1972, pp 64-66.

10. Stanley, H.R. Diseases of the pulp. In Tiecke, R.W. Oral pathology. New York, McGraw-Hill Book Co., 1965, p 108.

11. Kakehashi, S.; Stanley, H.R.; and Fitzgerald, R.J. The effects of surgical exposures of dental pulp in germ-free and conventional laboratory rats. Oral Surg 20:340 Sept 1965.

12. Smukler, H. and Tagger, M. Vital root amputation. A clinical and histological study. J Periodontol 47:324 June 1976.

13. Gustafson, G., and Granath, L.E. Pathology of the pulp. In Gustafson, G. Oral pathology for students. Stockholm, Esselte Studium, 1975, p 14.

14. Granath, L.E., and Hagman, G. Experimental pulpotomy in human bicuspids with reference to cutting technique. Acta Odontol Scand 29:155 June 1971.

15. Stanley, H.R., and Lundy, T. Dycal therapy for pulpal exposures. Oral Surg 34:818 Nov 1972.

16. Nyborg, H. Pulpaöverkappning och vitalamputation. In Nordisk Klinisk Odontologi. A/S Forlaget for Faglitteratur, Copenhagen 1965, pp 10-II:16-17.

17. Masterton, J.B. The healing of wounds of the dental pulp. An investigation of the nature of the scar tissue and of the phenomena leading to its formation. Dent Pract 16:325 May 1966.

18. Cvek, M. Treatment of non-vital permanent incisors with calcium hydroxide. Follow-up of periapical repair and apical closure of immature roots. Odontol Revy 23:29 No. 1 1972.

19. Schröder, U. Evaluation of healing following experimental pulpotomy of intact human teeth and capping with calcium hydroxide. Odontol Revy 23:329 No. 3 1972.

20. Shroff, F.R. The healing powers of dental pulp. Oral Surg 12:1249 Oct 1959.