Torsten Neuber, Frank C Setzer

Long-term treatment of a maxillary central incisor with uncomplicated crown fracture: a case report

Torsten Neuber
Dr med dent
Private Practice
Münster, Germany

Frank C Setzer, MS
Dr med dent
Lecturer
Department of Endodontics
School of Dental Medicine
University of Pennsylvania
Philadelphia, Pennsylvania, USA

Correspondence to:
Dr Torsten Neuber
Kompetenzteam Endo
Bischopinkstrasse 24-26
48151 Münster
Germany
Email:
neuber@kompetenzteam-endo.de
Tel: +49 251 791007
Fax: +49 251 791008

Key words *apexification, apexogenesis, crown fracture, immature root, MTA, pulpotomy*

Pulpal necrosis after dento-alveolar trauma of immature permanent teeth can cause a substantial problem for both patient and practitioner. Traditional therapy includes apexification with calcium hydroxide or the one-step apical plug procedure with mineral trioxide aggregate. The case presented here illustrates how an immature central incisor in a young individual, in which treatment was planned for a one-step apical plug technique after trauma and subsequent pulp necrosis, displayed root maturation due to the long-term course of the treatment.

Introduction

Patients with dental trauma make up a small, but nevertheless, important fraction of patients presenting to the dental office. According to Andreasen[1], up to 30% of all children and juveniles have experienced an oral traumatic injury, with the highest incidence between the ages of 9 and 12 and a higher frequency in males than females. Falls and traffic accidents are the most common injuries, followed by accidents during playing and sports[2]. Table 1 illustrates the classification of dento-alveolar trauma in the permanent dentition according to the most recent nomenclature of the International Association of Dental Traumatology (IADT). Traumas not only occur as isolated hard-tissue injuries or dislocation injuries, but also in combination. Thus, depending on the severity of the injury, hard tissue or the pulpo-dentinal complex or both structures can be involved.

Flores et al[3] gave an excellent review of therapeutic options depending on the type of trauma. For enamel fractures, the simplest procedure can be the removal of sharp edges and restoration of the lost structure according to the patient's aesthetic or functional needs. The priority for other uncomplicated crown fractures with dentin exposure is to maintain pulp vitality by covering the dentinal wound with a definitive restoration of the fractured crown with an appropriate restoration material. Complicated crown fractures have to be treated according to the stage of root maturation and the time that had passed since the pulp exposure. Mature teeth in older patients or teeth with a necrotic pulp have to receive root canal therapy to maintain the tooth. The treatment of choice for patients with immature roots and young patients with fully developed roots is pulp capping or partial pulpotomy to preserve pulp vitality. The level of pulpotomy is dictated by the point where the remaining pulp

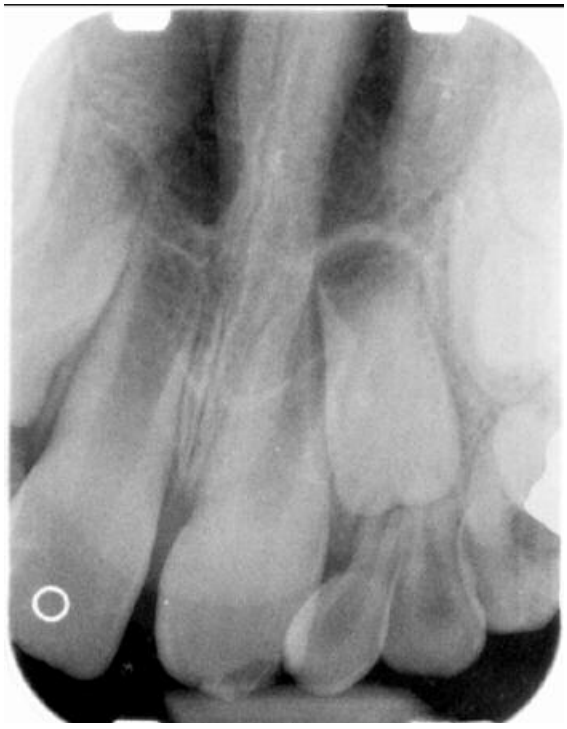

Fig 1 Preoperative radiograph. Traumatic injury to distal incisal edge and open apex.

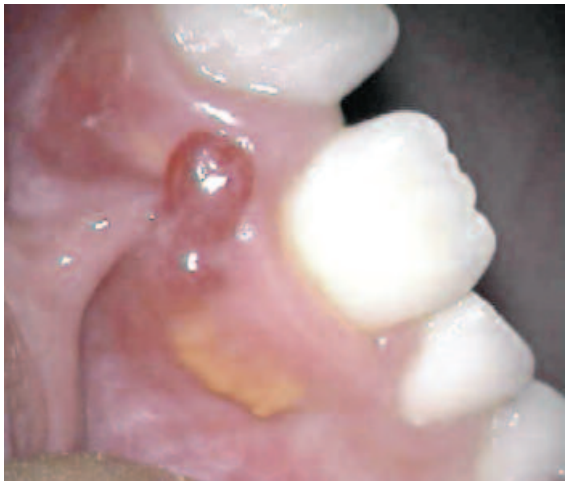

Fig 2 Clinical situation after 3 months. Fistula tract between the maxillary central incisors.

stump ceases to bleed. Overall, treatment success is determined by the practitioner's ability to seal pulp and dentin completely to prevent reinfection[12]. Pulp survival rates range from 93% for uncomplicated crown fractures[12-16] to 90% for complicated crown fractures where pulpotomies were performed[17-22].

Case report

A 7-year-old boy presented to the dental office one day after dental trauma. According to the mother he had received a blow to his teeth while drinking from a glass bottle and a part of the maxillary left central incisor had been chipped off. The patient had already been to an emergency clinic where the missing part of the tooth had been restored. A phone call with the dentist on duty revealed that it had apparently been an uncomplicated crown fracture with a dentinal wound, but no pulp exposure. The patient's medical history was non-contributory. The dental history showed two amalgam fillings on the maxillary left deciduous molars and good oral hygiene. The incisal edge of the maxillary left central incisor had been restored with a composite resin material. The reaction to a cold test with carbon dioxide snow was questionable. The tooth was slightly percussion and palpation sensitive. There was no increased mobility, no signs of alveolar bone fracture and all soft tissues as well as the periodontal probing depths were within normal limits. Radiographically, the fracture of the incisal edge was visible (Fig 1). There was no other vertical or horizontal root fracture apparent. The root end was immature, although radiographic interpretation was limited due to the unerupted left lateral incisor. The maxillary right central incisor was sensitive to the cold test; all other tests as well as the radiographic and clinical appearance were within normal limits. The patient was asymptomatic.

A diagnosis of uncomplicated crown fracture of the maxillary left central incisor was made. The pulpal status was questionable. At this point, no further therapy was initiated. The patient's parents were advised to return for systematic recall (after 1, 3, 6, 12, 18, 24 and 48 months) to have the pulpal status and clinical appearance checked regularly to detect any complications early on. In case of pain or any abnormality, the parents were instructed to contact the office immediately. The patient did not receive any antibiotics or analgesics.

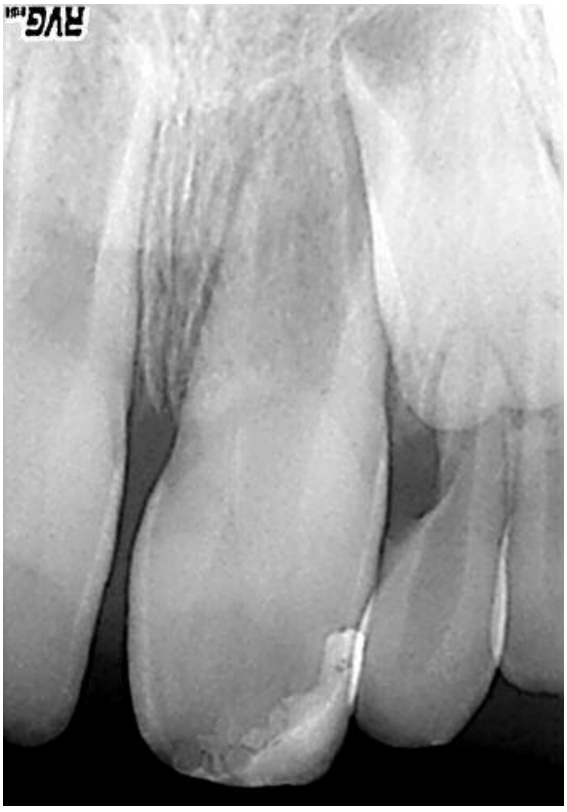

Fig 3 Radiographic control after 3 months. Composite resin *in situ*. The incomplete root development is evident.

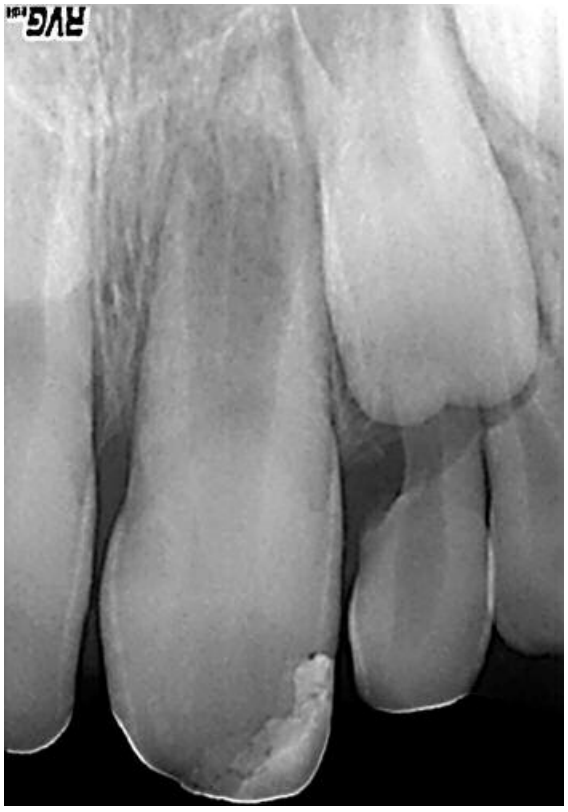

Fig 4 Radiograph immediately prior to root canal therapy. Apex is beginning to close. Continuity interruption of the periodontal ligament 3 mm coronal to the apex.

The patient returned for the first regular recall visit after 4 weeks. He was still asymptomatic, the tooth responded negative to percussion and palpation but still not positive to the cold test. Mobility, periodontal status, soft tissues and tooth colour appeared within normal limits. The parents were informed that the negative cold test might be a sign of pulp necrosis and that, although in teeth with a history of concussion cold sensitivity might still return even after a period of up to six weeks, further treatment might be necessary at the next recall visit, and could include apexification with calcium hydroxide or a one-step procedure with mineral trioxide aggregate (MTA).

Three months after the trauma, the patient remained asymptomatic. The tooth was negative to cold and demonstrated a greyish discoloration. A fistula tract was present at the level of the frenulum, about 2-3 mm below the gingival margin (Fig 2). The cold response of the maxillary right central incisor was positive. A radiograph showed that the root development of the maxillary left central incisor was not complete (Fig 3). At this appointment, the patient was uncooperative to undergoing further treatment, so that an attempt to trace the fistula tract with a gutta-percha cone was unsuccessful.

Unfortunately, the patient did not return for another 8 months. The fistula was still present. Radiographically, the root development had continued since the trauma had occurred. The continuity of the periodontal ligament was interrupted approximately 3 mm coronal to the apex (Fig 4). Conventional root canal therapy was initiated. The patient received a buccal infiltration with 4% articaine/adrenaline 1:100,000 (Ubistesin™, 3M ESPE, Seefeld, Germany). The maxillary left primary lateral incisor was removed for better access to the area. The maxillary left central incisor was accessed after rubber dam isolation. The pulp was necrotic, but a massive light-coloured bleed erupted from the pulp chamber. The root canal was irrigated with 3% sodium hypochlorite by inserting the irrigation needle (NaviTip®, Ultradent, South Jordan, UT, USA) to a depth of 15 mm, with no further instrumentation. The use of an apex locator (Root ZX, J. Morita, Dietzenbach, Germany) was ineffective due to

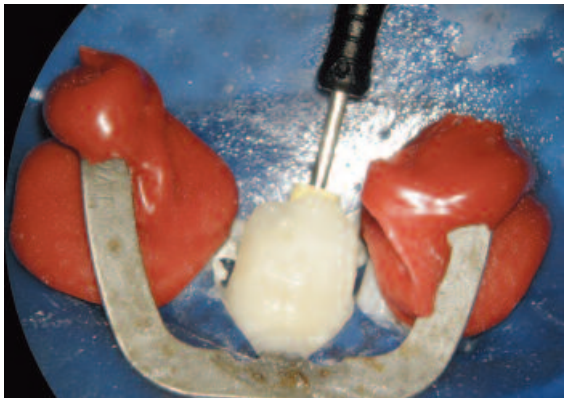

Fig 5 Clinical situation during root canal therapy. To increase retention, the rubber dam clamp was held in position on the adjacent teeth by a thermoplastic material (GC Bite Compound, Girrbach, Pforzheim, Germany) and additionally on the maxillary left central incisor with a composite resin (Tetric Flow). The hand instrument was fixed in position with composite resin to avoid movement in the root canal.

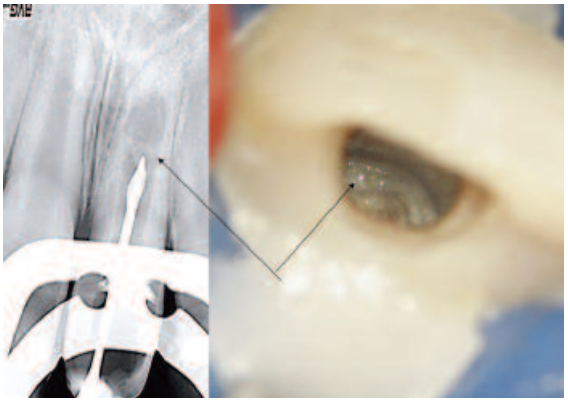

Fig 6 Hard tissue barrier. Clinical situation and radiograph (arrows).

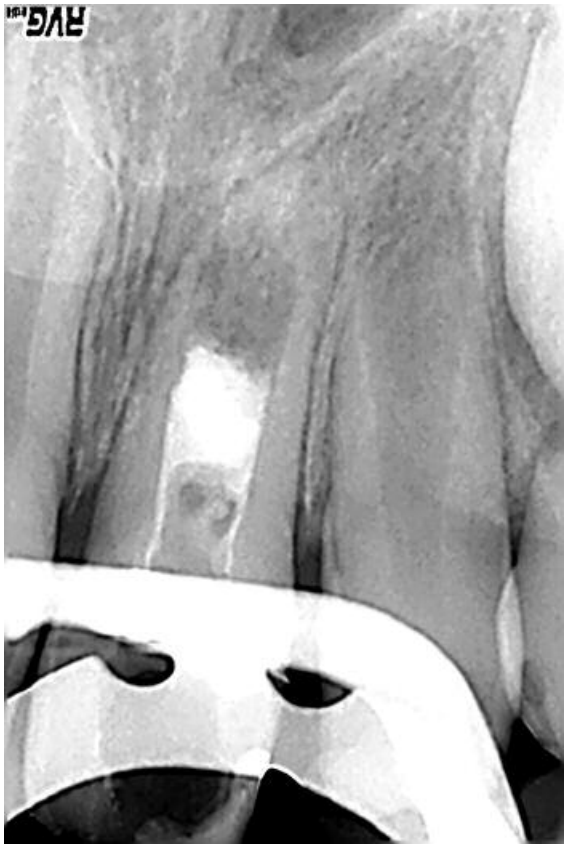

Fig 7 Radiographic control after MTA placement.

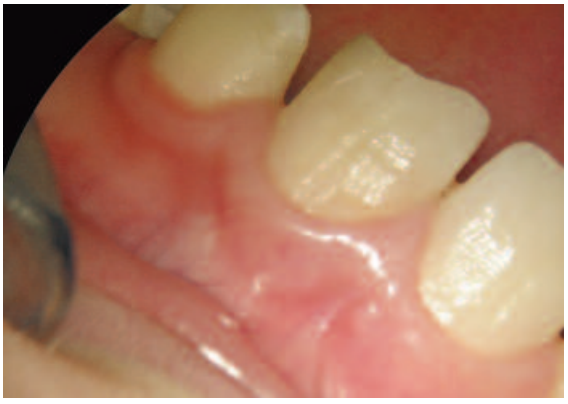

Fig 8 Clinical situation at the recall 32 months after the trauma, 22 months after access to the root canal system and 12 months after final obturation.

the exudate. An aqueous suspension of calcium hydroxide (UltraCal®, Ultradent) was placed in the canal and the cavity was temporarily sealed with Cavit™ (3M ESPE) and a composite resin (Tetric® Flow Chroma, Ivoclar Vivadent, Schaan, Liechtenstein). On two subsequent appointments, after three and nine weeks, the same treatment was repeated. By the second appointment, the fistula tract had disappeared and the intracanal bleeding was reduced to a moderate level. Electronic length determination and complete instrumentation was still impossible due to the bleeding, so that calcium hydroxide was placed and the access cavity was again sealed with Cavit and Tetric Flow Chroma.

The patient repeatedly missed appointments and did not present to the office again until 21 months after the initial trauma. He was asymptomatic and the clinical situation was within normal limits. After local anaesthesia the tooth was reopened and the root canal irrigated with sodium hypochlorite (Fig 5). Careful probing with a #10 hand instrument (FlexoFile®, Dentsply Maillefer, Ballaigues, Switzerland) demonstrated a mechanical resistance at 15 mm from the incisal reference point. Electronic length measurement gave a clearly reproducible reading at this level. A

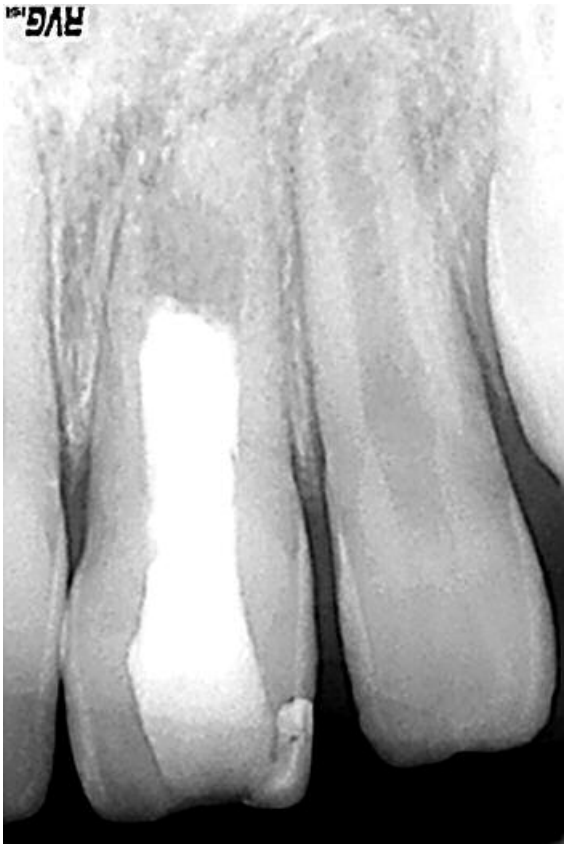

Fig 9 Radiographic control at the recall 32 months after the trauma, 22 months after access to the root canal system and 12 months after final obturation. Slight discontinuity in the periodontal ligament.

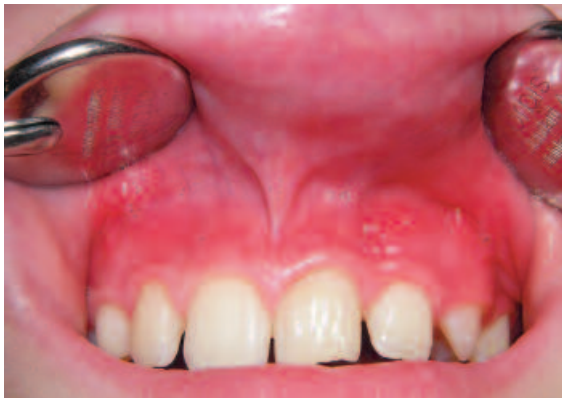

Fig 10 The clinical situation at the recall 3.5 years after the trauma was within normal limits.

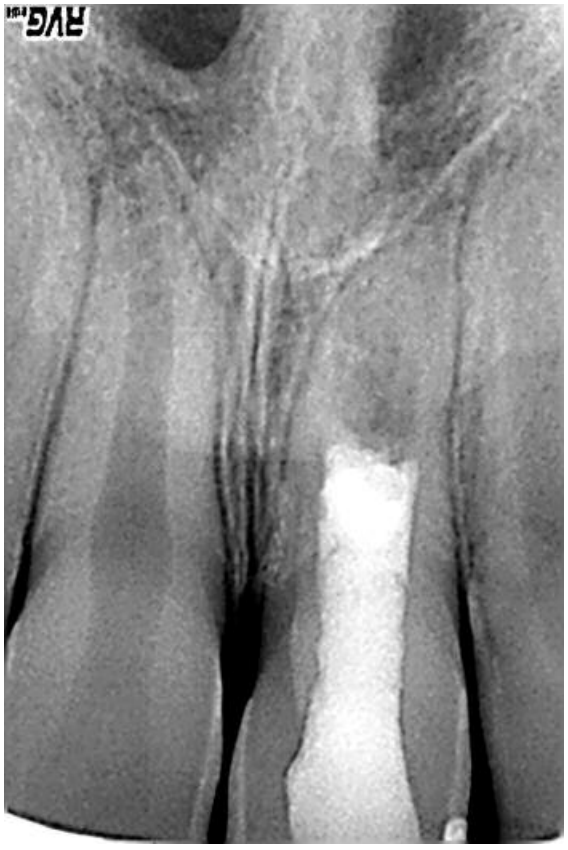

Fig 11 Radiographic control at the recall 3.5 years after the trauma. The periodontal ligament was intact. The root canal lumen apical to the hard tissue barrier was continuing to narrow.

working length radiograph at 15 mm revealed a newly formed hard tissue barrier at that level (Fig 6). This was also confirmed under high magnification using a dental microscope. No further mechanical instrumentation was rendered and the canal received a final irrigation with ultrasonically activated 3% sodium hypochlorite, 17% EDTA and 96% ethanol solutions. Approximately 2 mm of MTA (ProRoot™, Dentsply Maillefer) was placed on top of the hard tissue barrier using an MTA placement gun (Dentsply Maillefer) (Fig 7), covered with a wet cotton pellet and temporarily sealed. At the following appointment the MTA was completely set. The previously unfilled portion of the root canal was bonded (A.R.T. Bond; Coltene-Whaledent, Langenau, Germany) and filled with a dual curing composite resin (LuxaCore®, DMG, Hamburg, Germany). The access cavity was sealed with a composite resin restoration (Tetric® Ceram, Ivoclar Vivadent).

One week later the patient presented with a clinically normal appearance and no symptoms (Fig 8). The radiograph demonstrated almost complete root formation, with only a minor interruption in the continuity of the periodontal ligament (Fig 9).

On a recall visit six months later, the clinical situation was normal (Fig 10). Radiographically, the periodontal

Table 1 Classification of dento-alveolar trauma in the permanent dentition with incidence, modified from Flores et al[3].

Hard tissue injuries	Incidence
Uncomplicated crown fractures (infractions, enamel or dentin and enamel, no pulp exposure)	26–76%[4]
Complicated crown fractures (dentin and enamel, pulp exposure)	
Crown–root fracture	5%[5]
Root fracture	0.5–7%[6]
Alveolar bone fracture	16%[7]
Dislocation Injuries	
Concussion	23%[8]
Subluxation	21%[8]
Lateral dislocation	11%[9]
Extrusive dislocation	7%[9]
Intrusive dislocation	0.3–1.9%[10]
Avulsion	0.5–3%[11]

ligament space appeared physiologically normal and the root canal space apical to the hard tissue barrier demonstrated a narrowing tendency (Fig 11).

Discussion

The case presented seemed to require simple treatment procedures. The primary treatment goal for traumatised immature teeth with hard tissue injuries and no damage to the periodontal ligament is to maintain pulp vitality in order to ensure continued root development[23]. The fate of the tooth depends on events that occur in the pulp, including complications such as pulp necrosis, calcifications due to the deposition of tertiary dentin and resorptive processes. To avoid or treat these complications, the necessary approaches will vary depending on the grade of damage to the pulpo-dentinal complex. For uncomplicated crown fractures with a dentinal wound the immediate seal with a restorative material is the treatment of choice to prevent bacteria or their endotoxins from accessing the pulp through open dentinal tubules. This can be achieved by direct dentin bonding and restoration, or application of hard setting calcium hydroxide and subsequent restoration in order to cover the dentin in close proximity to the pulp. In the present case, all of those treatment modalities had been considered. Pulp necrosis may result from trauma, even in cases with uncomplicated crown fractures. Ravn et al[12] reported the incidence of pulp necrosis being 8% with a calcium hydroxide base of the dentinal wound near the pulp chamber versus 54% without. Similar results were shown demonstrating incidences of pulp necrosis of 7% after uncomplicated crown fractures with dentin exposure. Teeth with immature roots showed a superior prognosis, which is a result of better vascularisation of the pulp compared to teeth with completed root development[16].

In the present case, the pulp necrosis that had occurred may be explained by bacteria or their toxins that had invaded the pulp space through open dentinal tubules[12], either before the composite restoration or after, due to micro-leakage of the restoration. There is also the possibility that the pulp had been exposed by the trauma and had been directly infected, but the exposure had gone unnoticed during the emergency visit. If an exposure was evident, hard-setting calcium hydroxide or MTA are the materials of choice for direct pulp capping and partial or full pulpotomies. Recent research favours MTA due to its biocompatibility, tight seal and its ability to set in a moist environment[24]. Direct pulp capping should be restricted to small pulp exposures with an exposure time less than 24 hours[25]. In these cases, the pulp should be healthy and not inflamed. If control of the bleeding cannot be accomplished, a partial pulpotomy is indicated to remove damaged and inflamed tissue. Thus, the level of the pulpotomy depends on exposure time and size[22]. Success rates after direct pulp capping, partial and total pulpotomy range from 72-96%[25].

In the case presented, disruption of the neuro-

vascular supply due to a concussion might have contributed to a reduced immune response of the pulp tissue, facilitating easier access and increased destructive potential of bacteria and endotoxins. According to Robertson et al[16] a compromised blood circulation in the pulp tissue after crown fractures with a concomitant luxation is a primary factor influencing the healing of the pulp. In the case presented, an intrusive or lateral dislocation could have been a component of the trauma, severing or disrupting the blood supply to the pulp. Due to the minor hard tissue injury, an irreversible pulpitis seemed unlikely when the patient initially presented to the dental office. The negative reaction to the cold test after 4 weeks could have been an indication for pulp necrosis, but as a single entity, it had been insufficient to induce root canal therapy, due to the high regenerative potential of the dental pulp in immature teeth[11]. Immature teeth that initially react negative to sensitivity or vitality tests after trauma, can recover after at least 36 days[26]. A similar reaction is reported for mature teeth after two to three months, however, it is far more unlikely. The discoloration of the tooth after three months, together with the negative cold response and appearance of a fistula tract were regarded as clear indications for pulp necrosis (Fig 2). Discoloration of traumatised teeth is a widely reported phenomenon[27-28]. Gray colours indicate pulp necrosis[29-30], as, after iron is released during haemolysis, it can react with bacterially produced hydrogen sulphide to form black ferric sulphide, which is responsible for the discoloration[31]. The patient did not keep his appointments for a period of 8 months after the diagnosis of pulp necrosis. As a dislocation injury might have been part of the trauma and could have lead to destruction of the root cementum, root resorption, in combination with the infection in the pulp space, could have been a result. Complete root resorption in immature teeth with thin root canal walls has been reported frequently[32].

In the event of pulp necrosis, conventional root canal therapy is indicated to eliminate infection from the root canal system and to prevent apical periodontitis. Since apexogenesis will not occur in immature teeth where the pulp is necrotic, apexification with calcium hydroxide[33], one-step apical plug procedure with MTA or pulp revascularisation[34-36] are alternative options. If calcium hydroxide is used over a long time period, it stimulates the formation of an apical hard tissue barrier[37]. Success rates for this therapy are reported to be between 90 and 95 %[38-39]. As this treatment protocol requires numerous appointments over a period of several months, patient compliance is a prerequisite. Besides the long treatment period, possible root fractures due to the thin root canal walls can be a disadvantage[40-41].

Due to the patient's lack of cooperation and the tendency to miss appointments, the decision was made for a one-step apical plug procedure with MTA, which can be undertaken in one or two visits[42-43]. A two-visit procedure is recommended after the previous intracanal application of calcium hydroxide[44]. If MTA is the only material being used, then a 5 mm plug is recommended for optimal sealing capability[45]. In the present case, a 2 mm plug seemed appropriate as the MTA only functions as a biocompatible barrier between the hard tissue formation and the adhesive seal of the intracanal dual-curing composite. MTA was preferred over gutta-percha in combination with a sealer or resin-based root filling materials to minimise the risk of irritation of the soft tissue structures apical to the hard tissue barrier, given the fact that porosities in the newly formed mineralised tissue barrier could have facilitated the penetration of less biocompatible materials. Besides excellent biocompatibility, MTA has osteoinductive properties stimulating the formation of cementum-like hard tissue structures[42]. *In-vitro* studies by Mitchell et al[46] showed that MTA induces the release of cytokines from osteoblasts, indicating its ability to stimulate hard-tissue formation. Although long-term results are still unavailable, several studies favour MTA for the closure of immature roots[47-50]. In the case presented, grey MTA was used because of the lack of aesthetic concerns in the apical third of the root. Due to the excellent sealing ability of MTA, the short remaining root canal and for better stability, the root canal was sealed completely with an adhesive composite resin technique. The benefit of adhesive stabilisation of immature teeth after apexification has been demonstrated[51].

Besides coronal instrumentation with Gates Glidden burs no further mechanical instrumentation of the root canal had been carried out to minimise the loss of tooth structure of the immature root. For disruption of possible biofilm formation, all irrigation solutions had been ultrasonically activated.

Although the pulp had been necrotic for over 8 months, there was clinical evidence of continuation of

root development (Fig 4). This lead to the presumption that vital pulp tissue had been present apically and the pulp could have been in a stage of partial pulpitis and partial necrosis. Because of this and the general risk of a sodium hypochlorite accident in wide open root canals, irrigation was only carried out up to a depth of 15 mm. Due to the severe bleeding, calcium hydroxide had to be placed in the root canal space as a temporary medication. During the 8-month period when the patient did not keep his appointments, the calcium hydroxide remained in place and induced the production of a hard tissue barrier, which was evident clinically and radiographically (Fig 6). The exact composition of this hard tissue remains speculative and can only be proven histologically, however, a compound mineralised tissue consisting of osteocementum, osteodentin, cementum, atubular dentin or bone is possible[52]. Its formation might have been a result of the action of pluripotent mesenchymal stem cells or progenitor cells originating from Hertwig's epithelial root sheath or from remaining apical vital pulp tissue after the disinfection with sodium hypochlorite where the long-term medication with calcium hydroxide lowered the number of bacteria within the root canal system below a critical concentration. These cells might also have induced the continuation of root development, despite the necrosis of the coronal aspects of the pulp (Fig 4). Besides the existence of remaining pulp tissue, another possibility could have been a revascularisation of necrotic pulp space below the hard tissue barrier at the terminal level of the calcium hydroxide placement. There are case reports demonstrating the feasibility of inducing vital tissue growth within immature teeth via the formation of a blood clot under an MTA layer, after the application and subsequent removal of an antibiotic paste containing a mixture of minocycline, metronidazole and ciprofloxacin[35].

The prognosis of the tooth is very good. If it is assumed that vital pulp tissue had remained after careful irrigation short of the physiologic apex, the treatment rendered closely resembled an apexogenesis procedure in the form of a pulpotomy, or vital pulp therapy performed to result in regular root development and physiological root end formation. Thirty months after treatment the patient was asymptomatic, all tissues appeared healthy and there were no signs of altered function. According to Cvek[22] most treatment failures after pulpotomies occurred after 26 months. Several studies have shown excellent results with pulpotomies in immature teeth[20,53].

It was interesting to note, that the present case, due to the time factors involved, illustrated many different facets of dental traumatology and the complexity in treatment planning and execution of treatment of trauma cases. It can serve as a reminder that, especially with traumatic injuries, time is an important factor and that a constant vigilance by the care provider has to be maintained in order to be able to react to changes in pulp vitality or the periapical status of a tooth with the appropriate therapy at a specific point in time. Thus, patients have to be encouraged to follow a strict recall regimen. If ideal treatment protocols are followed as recommended, the high regenerative potential of the pulp and the periapical tissues in growing patients, combined with the use of modern materials and equipment, such as MTA and the dental operating microscope, render the ability to maintain previously hopeless teeth for the patient. This provides natural occlusion, aesthetics and phonetics at an age when dental implantology is not an option due to the unfinished skeletal development of the craniofacial tissues.

References

1. Andreasen JO. Etiology and pathogenesis of traumatic dental injuries. A clinical study of 1298 cases. Scand J Dent Res 1970;78:329-342.
2. Adekoya-Sofowora CA. Traumatized anterior teeth in children: A review of the literature. Niger J Med 2001;10:151-157.
3. Flores MT, Andersson L, Andreasen JO, Bakland LK, Malmgren B, Barnett F et al. Guidelines for the management of traumatic dental injuries. I. Fractures and luxations of permanent teeth. Dent Traumatol 2007;23:66-71.
4. Andreasen FM, Andreasen JO. Crown Fractures. In: Andreasen JO, Andreasen FM, Andersson L (eds). Textbook and color atlas of traumatic injuries to the teeth, ed 4. Copenhagen: Blackwell Publishing, 2007:280-313.
5. Andreasen JO, Andreasen FM, Tsukiboshi M. Crown-Root Fractures. In: Andreasen JO, Andreasen FM, Andersson L (eds). Textbook and color atlas of traumatic injuries to the teeth, ed 4. Copenhagen: Blackwell Publishing, 2007:314-336.
6. Andreasen FM, Andreasen JO, Cvek M. Root Fractures. In: Andreasen JO, Andreasen FM, Andersson L (eds). Textbook and color atlas of traumatic injuries to the teeth, ed 4. Copenhagen: Blackwell Publishing, 2007:337-371.
7. Andreasen JO. Injuries to the Supporting Bone. In: Andreasen JO, Andreasen FM, Andersson L (eds). Textbook and color atlas of traumatic injuries to the teeth, ed 4. Copenhagen: Blackwell Publishing, 2007:489-515.
8. Andreasen FM, Andreasen JO. Concussion and Subluxation. In: Andreasen JO, Andreasen FM, Andersson L (eds). Textbook and color atlas of traumatic injuries to the teeth, ed 4. Copenhagen: Blackwell Publishing, 2007:404-410.

9. Andreasen FM, Andreasen JO. Extrusive Luxation and Lateral Luxation. In: Andreasen JO, Andreasen FM, Andersson L (eds). Textbook and color atlas of traumatic injuries to the teeth, ed 4. Copenhagen: Blackwell Publishing, 2007:411-427.
10. Andreasen JO, Andreasen FM. Intrusive Luxation. In: Andreasen JO, Andreasen FM, Andersson L (eds). Textbook and color atlas of traumatic injuries to the teeth, ed 4. Copenhagen: Blackwell Publishing, 2007:428-443.
11. Andreasen JO, Andreasen FM. Avulsions. In: Andreasen JO, Andreasen FM, Andersson L (eds). Textbook and color atlas of traumatic injuries to the teeth, ed 4. Copenhagen: Blackwell Publishing, 2007:444-488.
12. Ravn JJ. Follow-up study of permanent incisors with enamel-dentin fractures after acute trauma. Scand J Dent Res 1981; 89:355-365.
13. Stalhane I, Hedegard B. Traumatized permanent teeth in children aged 7-15 years. Sven Tandlak Tidskr 1975;68:157-169.
14. Robertson A. A retrospective evaluation of patients with uncomplicated crown fractures and luxation injuries. Endod Dent Traumatol 1998;14:245-256.
15. Zadik D, Chosack A, Eidelman E. The prognosis of traumatized permanent anterior teeth with fracture of the enamel and dentin. Oral Surg Oral Med Oral Pathol 1979;47:173-175.
16. Robertson A, Andreasen FM, Andreasen JO, Noren JG. Long-term prognosis of crown-fractured permanent incisors. The effect of stage of root development and associated luxation injury. Int J Paediatr Dent 2000;10:191-199.
17. Andreasen JO. Traumatic injuries of teeth. Copenhagen: Munksgaard, 1981:1-462
18. Ravn JJ. Follow-up study of permanent incisors with complicated crown fractures after acute trauma. Scand J Dent Res 1982;90:363-372.
19. Fuks AB, Bielak S, Chosak A. Clinical and radiographic assessment of direct pulp capping and pulpotomy in young permanent teeth. Pediatr Dent 1982;4:240-244.
20. Cvek M. A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated crown fracture. J Endod 1978;4:232-237.
21. Fuks AB, Cosack A, Klein H, Eidelman E. Partial pulpotomy as a treatment alternative for exposed pulps in crown-fractured permanent incisors. Endod Dent Traumatol 1987;3:100-102.
22. Cvek M. Results after partial pulpotomy in crown fractured teeth 3-15 years after treatment. Acta Stomatol Croat 1993;27:167-173.
23. Hülsmann M, Schäfer E. Probleme in der Endodontie. Berlin: Quintessenz, 2007;500.
24. Bakland LK. New endodontic procedures using mineral trioxide aggregate (MTA) for teeth with traumatic injuries. In: Andreasen JO, Andreasen FM, Andersson L (eds). Textbook and color atlas of traumatic injuries to the teeth, ed 4. Copenhagen: Blackwell Publishing, 2007;658-668.
25. Cvek M. Endodontic Management and the Use of Calcium Hydroxide in Traumatized Permanent Teeth. In: Andreasen JO, Andreasen FM, Andersson L (eds). Textbook and color atlas of traumatic injuries to the teeth, ed 4. Copenhagen: Blackwell Publishing, 2007:598-657.
26. Andreasen JO, Borum M, Jacobsen HL, Andreasen FM. Replantation of 400 avulsed permanent incisors II. Factors related to pulp healing. Endod Dent Traumatol 1995;11:59-68.
27. Auslander WP. Discoloration, a traumatic sequela. N Y State Dent J 1967;33:534-538.
28. Andreasen JO, Andreasen FM. Textbook and color atlas of traumatic injuries to the teeth, ed 3. Copenhagen: Munksgaard, 1994.
29. Schröder U, Wennberg E, Granath L-E, Möller H. Traumatized primary incisors follow-up program based on frequency of periapical osteitis related to tooth color. Swed Dent J 1977;1:95-98.
30. Andreasen FM. Pulpal healing after luxation injuries in the permanent dentition. Endod Dent Traumatol 1989;5:11-31.
31. Attin T, Paque F, Ajam F, Lennon AM. Review of the current status of tooth whitening with the walking bleach technique. Int Endod J 2003;36:313-329.
32. Trope M. Root resorption due to dental trauma. Endod Top 2002;1:79-100.
33. De Cleen M. Apexifikation – eine Literaturübersicht und klinische Empfehlungen. Endodontie 1994;3:39-49.
34. Banchs F, Trope M. Revascularization of immature permanent teeth with apical periodontitis. J Endod 2004;30:196-200.
35. Thibodeau B, Trope M. Pulp revascularization of a necrotic infected immature permanent tooth: case report and review of the literature. Pediatr Dent 2007;29:47-50.
36. Jung IY, Lee SJ, Hargreaves KM. Biologically based treatment of immature permanent teeth with pulpal necrosis. J Endod 2008;34:876-887.
37. Ham JW, Patterson SS, Mitchell DF. Induced apical closure of immature pulpless teeth in monkeys. Oral Surg Oral Med Oral Pathol 1972;33:438-449.
38. Tronstad L. Clinical Endodontics, ed 2. Stuttgart: Thieme, 2003:120-122.
39. Finunce D, Kinirons MJ. Non-vital immature incisors: factors that may influence treatment outcome. Endod Dent Traumatol 1999;15:273-277.
40. Andreasen JO, Farik B, Munksgaard EC. Long-term calcium hydroxide as a root canal dressing may increase risk of root fracture. Dent Traumatol 2002;18:134-137.
41. Cvek M. Prognosis of luxated non-vital maxillary incisors treated with calcium-hydroxide and filled with gutta-percha. A retrospective clinical study. Endod Dent Traumatol 1992;8: 45-55.
42. Torabinejad M, Chivian N. Clinical applications of mineral trioxide aggregate. J Endod 1999;25:197-205.
43. Fayad M, Montero MJ. Multiple vs. one-step apexification. Endodontie Journal 2005;3:19-21.
44. Matt GD, Thorpe JR, Strother JM, McClanahan SB. Comparative study of white and gray mineral trioxide aggregate (MTA) simulating a one- or two-step apical barrier technique. J Endod 2004;30:876-879.
45. Al-Khatani A, Shostad S, Schifferle R, Bhambhani S. *In-vitro* evaluation of microleakage of an orthograde apical plug of mineral trioxide aggregate in permanent teeth with simulated immature apices. J Endod 2005;31:117-119.
46. Mitchell PJ, Pitt Ford TR, Torabinejad M, McDonald F. Osteoblast biocompatibility of mineral trioxide aggregate. Biomaterials 1999;20:167-173.
47. Giuliani V, Baccetti T, Pace R, Pagavino G. The use of MTA in teeth with necrotic pulps and open apices. Dent Traumatol 2002;18:217-221.
48. Rafter M. Apexifikation – a review. Dent Traumatol 2005;21:1-8.
49. Maroto M, Barberia E, Planells P, Vera V. Treatment of non-vital immature incisor with mineral trioxide aggregate (MTA). Dent Traumatol 2003;19:165-169.
50. Simon S, Rilliard F, Berdal A, Machtou P. The use of mineral trioxide aggregate in one-visit apexification treatment: a prospective study. Int Endod J 2007;40:186-197.
51. Rabie G, Trope M, Garcia C, Tronstad L. Strengthening and restoration of immature teeth with acid-etch resin technique. Endod Dent Traumatol 1985;1:246-256.
52. Hülsmann M. Endodontie – Checklisten der Zahnmedizin. Stuttgart: Thieme, 2008:232.
53. El-Meligy OA, Avery DR. Comparison of mineral trioxide aggregate and calcium hydroxide as pulpotomy agents in young permanent teeth (apexogenesis). Pediatr Dent 2006;28:399-404.