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Dental traumatology: essential diagnosis and treatment planning

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Traumatic dental injuries are for the most part unanticipated events that, if not managed appropriately, can have serious consequences for the patient. The purpose of this review is to describe the current concepts in establishing diagnosis descriptive of specific traumatic entities, and to delineate recommended treatment approaches for these injuries based on available evidence.

Traumatic events involving dental structures

When teeth and their supporting structures are subjected to impact trauma, the resultant injury manifests either as a separation or a crushing injury or a combination of both (1) (Fig. 1).

Separation injuries are exemplified by displacement of teeth during which there is cleavage of tissues such as the periodontial ligament (PDL). This occurs during avulsions and extrusive luxations. Damage to cells in the involved tissues is minimal, so rapid healing is favored following appropriate treatment.

Crushing injuries cause more damage due to the destruction of both tissue cells and intercellular components. This type of injury is exemplified by displacement of teeth against adjacent alveolar bone, the worst of which is intrusive luxation.

During a crushing traumatic event, the cells and associated tissues are damaged and must be removed (by macrophages and osteoclasts) in order to initiate repair. Thus the healing process cannot be expected to proceed as quickly as that observed for separation injuries.

Understanding the difference between separation and crushing injuries is helpful in assessing the extent and the severity of a traumatic injury, planning for the management of the patient, and predicting the likely outcome (1).

Classification of dental injuries

Many attempts have been made over the years to classify dental injuries (2). The currently accepted system is based on the World Health Organization's *Application of International Classification of Diseases to Dentistry and Stomatology* (3), and modified by Andreasen (2).

The classification is applicable to injuries to the teeth and supporting structures and can be applied to both primary and permanent dentitions (Tables 1 and 2).

Examination of the patient with dental trauma

Traumatic dental injuries are unscheduled (and often urgent) events, both for the unlucky patient and for the dentist asked to manage the problem. Because of the often limited time available to examine and treat patients with traumatic dental injuries, having a planned, organized approach to performing such care will help expedite procedures in a timely fashion. Further, providing proper care, based on accurate diagnosis, can be expected to favor successful healing (4, 5).

In an effort to assist dentists in gathering pertinent information, 2 clinical records have been developed to allow the recording of the clinical and historical data associated with the injury (6) (Appendices 1 and 2).

A fact often overlooked when examining a patient with an acute traumatic injury is that the injury site, both intra-oral and extra-oral may be contaminated from the impact and covered with blood and debris. These injury sites must be cleansed with a mild detergent prior to examination.

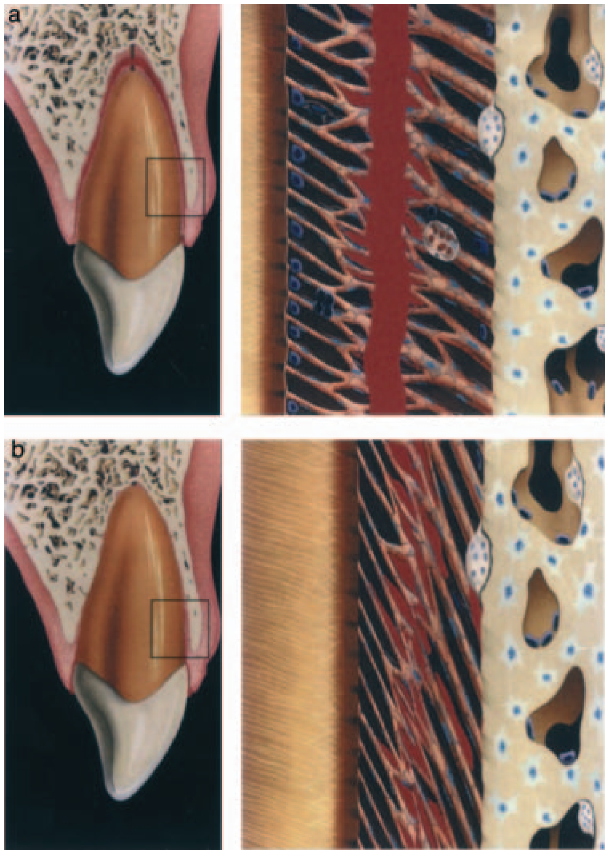

Fig. 1. Basic types of dental traumatic injuries: Separation injury (a) and crushing injury (b). (a) Displacement of the tooth away from the socket wall, such as in extrusive luxations and avulsions, results in separation of the periodontal ligament fibers. (b) When a tooth is forced against the socket wall, such as in intrusive and lateral luxation injuries, the periodontal ligament fibers and associated cells are crushed, causing extensive damage. (From Andreasen JO, Andreasen FM, Bakland LK, Flores MT. Traumatic Dental Injuries. A Manual, 2nd edn, Oxford, UK: Blackwell Munksgaard, 2000: 10)

The following steps in examination will provide necessary information to establish the correct diagnosis and guide the dentist in developing a rational treatment plan (7):

1. Information about the injury: The following questions are intended to elicit essential information about the traumatic event.

When did the injury occur? Time factors are important especially in avulsion and displacement injuries. Also, treatment delay may signal possible child abuse, if the patient is a minor.

Where did the injury occur? For legal and insurance purposes, this information is important in the record.

How did the injury occur? Answers to this question may guide evaluation of the extent of trauma, for example, a blow to the chin may transmit to the condyles.

Has the patient been unconscious? If so, medical attention must be sought, but that does not preclude urgent immediate dental care such as replantation of an avulsed tooth.

Are there previous injuries to the teeth? Some children are accident prone, and participants in various sports will frequently show radiographic indication of previous trauma. Such information can affect treatment options.

Is there a change in the bite? Changes in occlusion following an injury would indicate possible tooth luxation, alveolar/jaw fracture, or condylar fracture.

Is there increased sensitivity to temperature changes? This is typically observed in teeth with crown fractures exposing the dentin.

Pertinent medical history. It is essential to establish any possibility of drug or material allergies, blood disorders or other conditions that may influence treatment.

2. Clinical examination: It is recommended that the findings obtained from the clinical examination be recorded; useful records for that purpose are Appendices 1 and 2. Begin with evaluation of any soft tissue wounds, including examination for the presence of impacted foreign bodies (including tooth fragments) in the wounds. Almost all penetrating lip wounds have impacted foreign bodies (Fig. 2). Next, the teeth are examined for fractures or infractions. The former is easily observed visually, while the latter requires directing the light parallel to the tooth's labial surface (thus shining the light from the mesial or distal aspect of the tooth).

If crown fractures have occurred, note if the pulps are exposed and the extent of exposure and the status of the pulpal circulation (e.g. hyperemic or cyanotic). Any displacement of teeth must be noted and recorded as lateral or axial (extrusive or intrusive).

3. Mobility test: Injuries resulting in mobility of individual teeth (luxation injuries) or mobility of groups of teeth (possible alveolar fracture) must be determined. The degree of mobility should be

Table 1. Traumatic injuries to teeth

Enamel infraction	An incomplete fracture (crack) of the enamel without loss of tooth substance
Enamel fracture	(Uncomplicated crown fracture). A fracture with loss of enamel only
Enamel–dentin fracture	(Uncomplicated crown fracture). A fracture with loss of enamel and dentin, but not involving the pulp
Complicated crown fracture	A fracture involving enamel and dentin, and exposing the pulp
Crown–root fracture	A fracture involving enamel, coronal and radicular dentin, and cementum
Root fracture	A fracture involving radicular dentin, cementum, and the pulp. Root fractures can be further classified according to displacement of the coronal fragment (see luxation injuries)
Luxation injuries	***Concussion.*** An injury to the tooth-supporting structures without abnormal loosening or displacement of the tooth, but with increased reaction to percussion
	Subluxation (loosening). An injury to the tooth supporting structures with abnormal loosening, but without displacement of the tooth
	Extrusive luxation (peripheral dislocation, partial avulsion). Partial displacement of the tooth out of its socket
	Lateral luxation. Displacement of the tooth in a direction other than axially. This is accompanied by comminution or fracture of the alveolar socket
	Intrusive luxation (central dislocation). Displacement of the tooth into the alveolar bone. This injury is accompanied by comminution or fracture of the alveolar socket
	Avulsion (exarticulation). Complete displacement of the tooth out of its socket

recorded thus: 0 = no loosening or change; 1 = horizontal loosening $\leq$1 mm; 2 = horizontal loosening > 1 mm; 3 = axial (vertical) loosening. The type of luxation can be related to the degree of mobility (8).

4. Percussion test: Tenderness to touching or tapping a tooth indicates damage to the PDL. The percussion test is performed to obtain such information and must be done carefully since some teeth are *exquisitively* tender because of the PDL condition: Tap first with a fingertip before proceeding to using a mirror handle.

In addition to establishing PDL tenderness, percussion can provide information about the relationship between the tooth and adjacent bone: a high, metallic tone indicates lateral or intrusive displacement. On later, follow-up examinations, such a percussion tone would indicate ankylosis of the tooth.

5. Pulpal sensibility test: Currently the most useful test to assess the neurovascular supply to the pulp of a traumatized tooth is by the use of an electric pulp tester (EPT) (9). The placement of the electrode should be as close to the incisal edge as possible. It is recognized, however, that the use of the EPT in developing teeth is not always reliable. The lack of response to EPT should not automatically be accepted as proof of pulp necrosis in either developing or mature teeth. The response at the time of first injury examination does provide a good baseline for comparison at subsequent control examinations. With respect to primary teeth, the EPT may be difficult to use due to lack of patient cooperation.
6. Radiographic examination: Multiple radiographic exposures from several angulations provide the most reliable information about changes in the dento-alveolar complex following traumatic injuries (8, 9). It is recommended that one steep occlusal exposure along with three periapical bisecting angle exposures be employed to determine the extent of trauma. For instance, root fractures are often missed

Table 2. Soft tissue and bony injuries

Laceration of gingiva or oral mucosa	A shallow or deep wound in the mucosa resulting from a tear; usually produced by a sharp object
Contusion of gingiva or oral mucosa	A bruise usually produced by impact with a blunt object and not accompanied by a break in the mucosa, usually causing submucosal hemorrhage
Abrasion of gingiva or oral mucosa	A superficial wound produced by rubbing or scraping of the mucosa, leaving a raw, bleeding surface
Fracture of the mandibular or maxillary alveolar socket wall	A fracture of the alveolar process which involves the alveolar socket (see lateral luxation)
Fracture of the mandibular or maxillary alveolar process	A fracture of the alveolar process that may or may not involve the alveolar socket

Fig. 2. Penetrating lip wound containing foreign object (tooth fragment). (a) When a tooth fractures, pieces of it may penetrate soft tissue such as lips. (b) If a soft tissue wound is present, a radiograph of the area should be taken (reduce the expose to about 25% of normal). (c) If a foreign object is noted (such as a piece of tooth), it should be removed as soon as possible to prevent it from becoming encapsulated in fibrotic tissue.

during examination because the typical single exposure fails to demonstrate diagonal root fractures (Fig. 3).

Another application of radiography is to examine for the presence of impacted foreign bodies in penetrating soft tissue wounds. It is not possible to detect by

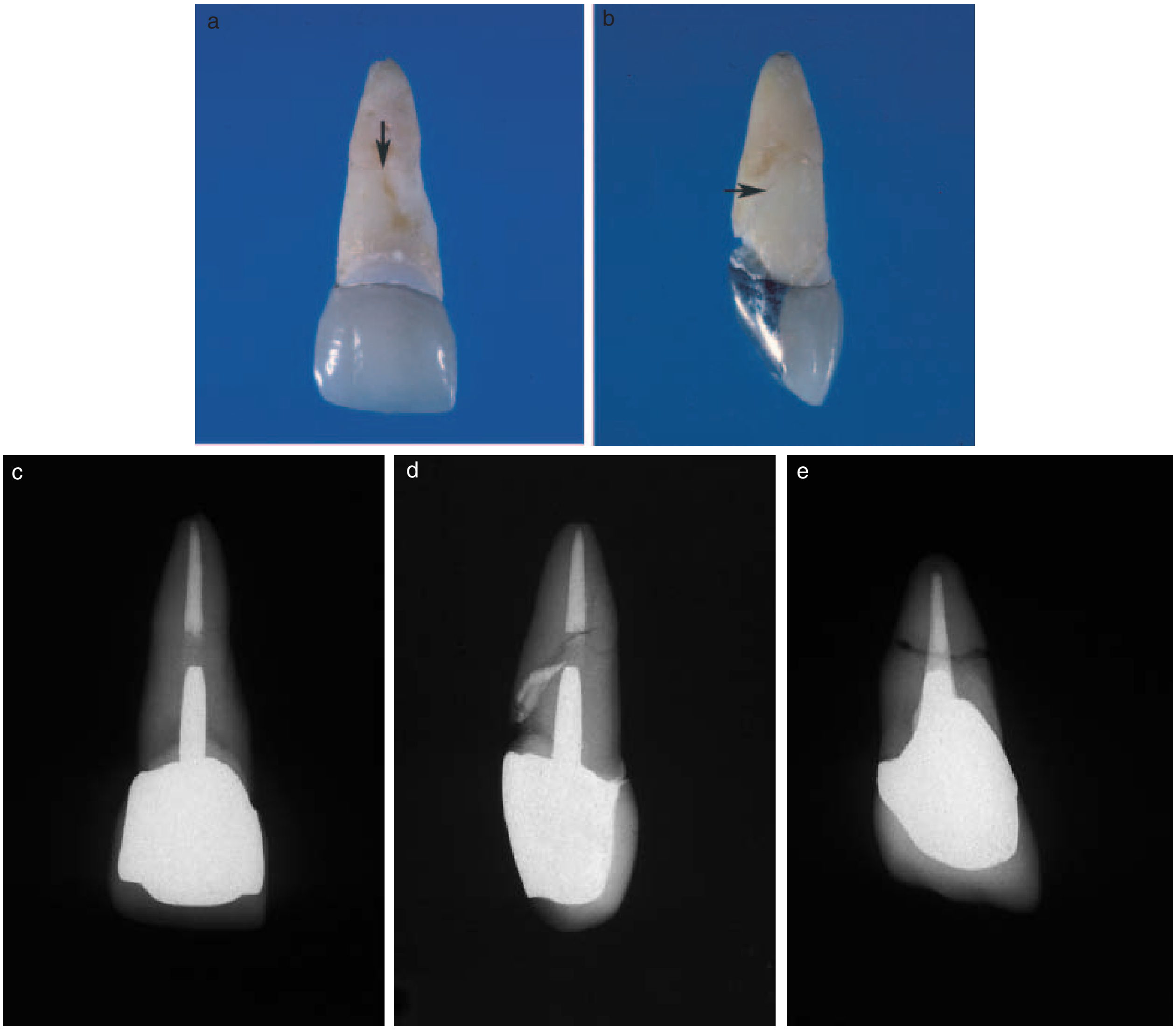

Fig. 3. Radiographic examination for possible root fractures should include at least one steep occlusal exposure in addition to the conventional 90° angulation exposure. The following illustrations demonstrate the importance of this concept. (a) A tooth with a diagonal midroot fracture was extracted. The fracture line on the labial root surface can be seen (arrow). (b) The diagonal nature of the fracture can be seen on the lateral surface of the root (arrow). (c) Radiographic film exposed at 90° angulation fails to show any fracture line. (d) Lateral view of the root shows the fracture (because the X-rays are parallel to the fracture). Such an angulation is not possible, however, in a clinical setting. (e). By changing the angulation, to simulate a steep occlusal one, the fracture line is readily noted.

palpation all foreign bodies in the lips, for example, making it essential to include soft tissue radiography if their presence is suspected (see Fig. 2). The exposure should be reduced to about 25–50% of normal hard tissue exposure allowing detection of non-organic materials (tooth fragments, glass, gravel, and similar materials), while organic material such as wood and cloth will not show on the film.

7. Follow-up evaluation: Management of dental injuries includes follow-up control to complete or confirm the diagnosis, assess response to treatment, determine need for additional treatment or treatment change, and evaluate the treatment outcome or complications. The following recommended schedule can be applied to the management of dental trauma patients (5).

1 week. After 7–10 days, the splint placed on a replanted, avulsed tooth should be removed and endodontic treatment, if indicated, should be initiated.

3–4 weeks. The splint applied to luxated teeth can usually be removed after 3–4 weeks. Radiographic examination should be performed to examine for the possible beginning of root resorption or periradicular lesions.

6 weeks. At this time, clinical and radiographic examinations may reveal evidence of pulp necrosis and infection-related (inflammatory) root resorption.

3–6 months. Examination at this time may be necessary to establish definitive diagnosis of pulpal periodontal healing complications.

1 year and up to 5 years. One year is a minimal control period for traumatic dental injuries; some may require additional observation periods to assess the final outcome.

Fig. 4. Failure to treat. The maxillary right central incisor had been avulsed in a sports accident and replanted within 15 min about 18 months prior to radiographic examination shown in this image. Note the extensive resorption (infection-related) of the root and the apical lesion. Timely endodontic treatment could have prevented the resorption and subsequent loss of this tooth.

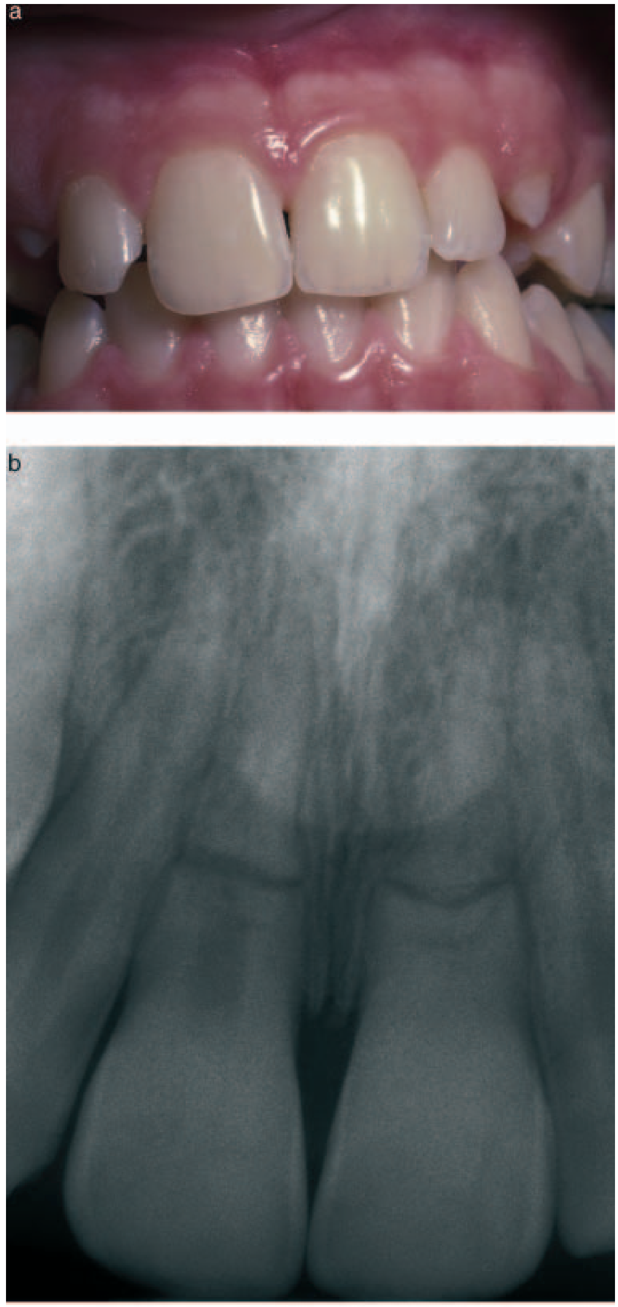

Fig. 5. Healed root fractured teeth in risk of extraction. A 12-year-old boy visited a dentist for the first time. All the teeth were examined radiographically, including the maxillary central incisors, which, according to his mother, had been injured 3 years earlier in an accident. The teeth were asymptomatic, functional and acceptable in appearance, yet both the examining dentist and a subsequent consulting oral and maxillofacial surgeon recommended extraction of both teeth based on the radiographic appearance. Fortunately for the boy, his mother sought additional consultation and was advised by a dentist more familiar with dental trauma that neither tooth should be extracted. (a) The photographs show central incisors. (b) The radiograph indicates that the root fractures have healed satisfactorily.

The effect of trauma on teeth and supporting tissues

The management of dental trauma is a part of practice for most dentists (10). Knowledge about dental traumatology is, however, not universal among dental professionals neither in regards to acute care nor long term treatment (Figs 4 and 5). Since treatment of many traumatic injuries require both immediate as well as subsequent attention, it is useful to look at the effects of trauma on the two tissues that determine the success or failure following injury: The tooth pulp and the PDL (including cementum and lamina dura).

Pulpal responses to traumatic injuries are affected by the degree of injury to the neurovascular supply, which for the most part enter through the apical foramen. The presence of bacteria is also a significant factor in the outcome. Three possible outcomes exist: Pulpal healing, pulpal necrosis, or pulp canal obliteration (4,11–17). It should be noted that all three responses can result at different times, for example, initial healing may be followed by canal obliteration and subsequent pulpal necrosis.

The most desirable outcome after dental trauma is pulpal healing. If the disruption of the neurovascular supply to the pulp is less than total, for example, in subluxation injuries, the pulp function may continue with reduced circulations until complete reconstitution is accomplished, usually within a few weeks. The EPT may provide information, such as demonstrating a change from a relatively high to a low reading, as healing proceeds.

In cases of extensive or total severance of the apical blood supply, pulpal healing is rare if the apical diameter is ≤ 0.5 mm, such as is found in fully formed teeth. Pulp necrosis is the norm with a few exceptions (such as demonstrated by transient apical breakdown situations (18)).

In young patients with immature, developing teeth (apical foramen diameter > 0.5 mm) the possibility of pulpal healing through revascularization exists (12). If a displaced or luxated tooth is repositioned in its normal location, revascularization of the pulp may proceed from the apical opening in a coronal direction at the rate of about 0.5 mm/day (4). Thus, teeth with short roots and large diameter apical openings are more likely to have successful outcomes. It must be recognized that the inhibiting factor of bacterial presence plays a potential role: If a luxated developing tooth also suffers a crown fracture (with or without

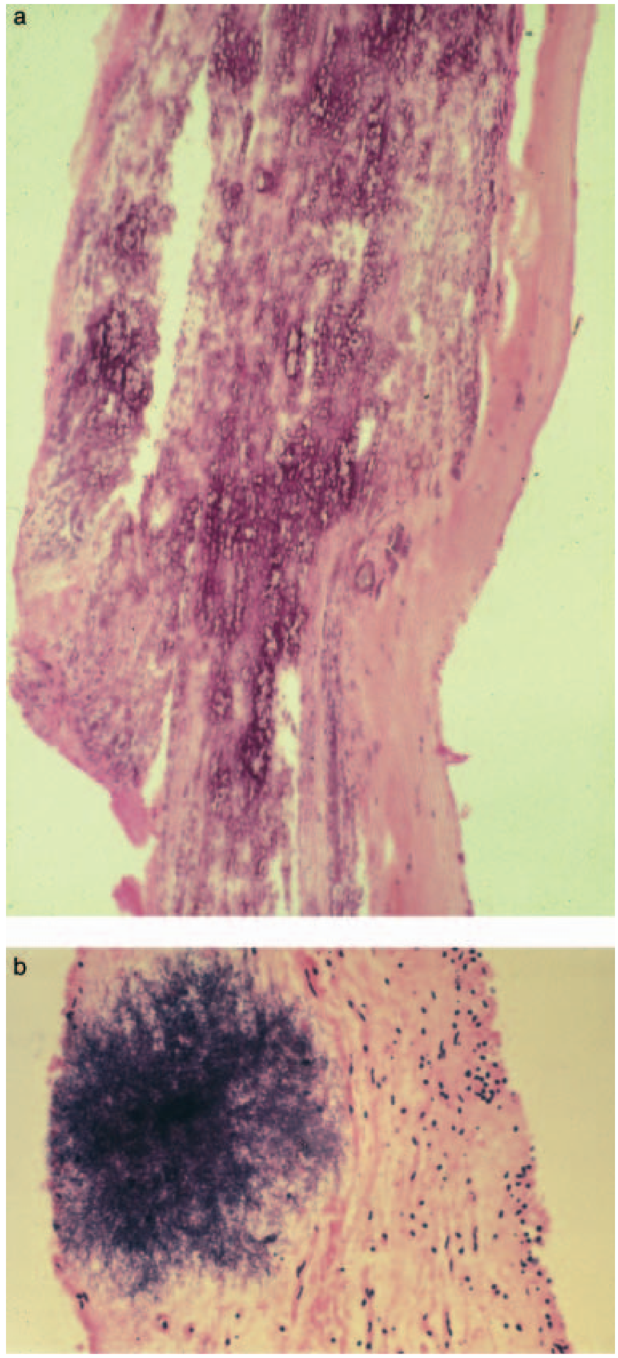

Fig. 6. Examples of pulp necrosis as a result of trauma severing the blood supply to the pulp. (a) Coagulation necrosis occurs when the pulp is deprived of blood. The tissue gradually disintegrates. This photomicrograph shows pulp tissue undergoing coagulation necrosis. (H&E $\times$ 100) (b) When pulp tissue which has undergone coagulation necrosis is exposed to bacteria, gangrenous necrosis results. Such necrosis can result in rapid proliferation of bacteria in the tissue lacking any defense capability (as a result of the severed blood supply). A colony of bacteria can be seen growing on a piece of pulp tissue removed for experimental purposes (H&E $\times$ 100).

pulpal exposure) bacteria can gain entrance to the pulp and the absent or diminished blood supply will allow the bacteria to colonize unhindered. In these situations, pulp necrosis will result rather than pulp healing.

Pulp necrosis following severance of the blood supply may proceed through coagulation necrosis (sterile necrosis) to gangrenous necrosis (infection of infarcted tissue) (Fig. 6). Pulp necrosis as a result of coronal pulp exposure is of the liquefaction type, similar to that seen following carious exposure.

Pulpal necrosis in mature, fully formed teeth is less significant (if treated with proper endodontic care) than necrosis of pulps in developing teeth. Loss of pulp vitality in the latter teeth results in weak, fracture prone roots that may be rendered weaker when subjected to long-term exposure to calcium hydroxide (CH) (such as commonly performed to achieve apexification in roots with large diameter apical openings) (19) (Fig. 7). Thus, in young, developing teeth, every effort should be employed to achieve pulpal survival and healing. Current techniques for vital pulp therapy are quite successful in achieving this goal (20–22).

Of interest, with respect to pulpal necrosis in traumatized teeth, is the risk of infection-related (inflammatory) root resorption (23). This risk emphasizes the need for careful monitoring of pulpal responses to traumatic injuries (see Fig. 4).

The third type of pulpal response to trauma is obliteration of the root canal. This is frequently observed in luxation-type injuries associated with displacement (13, 24). Rarely do such teeth require root canal treatment (13, 24). If pulp necrosis occurs after extensive canal obliteration, the diagnosis can be made based on symptoms and development of periradicular osteitis. While the canals in such teeth may be of small diameter, with modern equipment, endodontists can usually manage these situations with conventional root canal therapy.

The effect on the PDL can be observed in cases where root resorptions take place. These have been identified as repair-related (surface), infection-related (inflammatory), and ankylosis-related (replacement) resorption (23, 25).

Repair-related resorption (also described as surface resorption) is a transient process involving small areas on the root surface following luxation and avulsion injuries and can also be observed associated with root fracture injuries (23). Typically, diagnosis can be made within 4 weeks after injury. As long as conditions are right for healing, that is, absence of bacteria in the root

Fig. 7. Cervical root fracture. (a) The preoperative radiograph of the right lateral incisor in a 12-year-old boy at the beginning of orthodontic treatment. The tooth had been luxated about 4 years earlier and had received no previous treatment. The pulp is now necrotic and a large periradicular lesion is present. Apexification procedure using calcium hydroxide was started. (b) Fifteen months later the lesion has filled in with new bone and there is evidence of apical closure. The patient was scheduled for completion of root canal treatment. (c) Before the appointment to fill the canal, the boy was in a 'pillow fight' with a sibling and the tooth received a mild injury, resulting in cervical fracture, even though the tooth was in part protected by the orthodontic appliance. This case illustrates how susceptible teeth, such as this, are to cervical fracture.

canal, this type of resorption is reversible, as the name implies.

A very aggressive type of injury-related resorption is that which is associated with root canal infection following trauma to a tooth. It has been described as inflammatory resorption (23) and is currently identified as infection-related resorption. Luxation and avulsion injuries resulting in reduction or severance of pulpal blood supply, followed by bacterial invasion of the pulp, is the setting for initiation of external (and sometimes internal) root resorption. Luxation and avulsion injuries are prone to cause damage to the cemental protection of root surfaces, allowing dentinal tubules to become pathways for bacterial toxins within the canal to trigger osteoclastic activity externally.

Failure to remove bacteria from the root canal can result in this type of inflammatory resorption proceeding at a rapid pace, resulting in total root resorption within a very short period of time (23, 25, 26) (see Fig. 4). The less mature the involved tooth is, the faster the resorption proceeds because of the large diameter dentinal tubules. A positive aspect to infection-related resorption is that timely applied root canal treatment can predictably prevent resorption, and if the resorption has already commenced, endodontic intervention can arrest the process (Fig. 8).

A relatively slower, but not necessarily more benign, resorptive process is ankylosis-related resorption, also described as replacement resorption (23). This type of resorption is associated with extensive trauma to the PDL resulting in loss of vitality of the cells and extensive damage to the cementum. Lacking protective covering, the root cementum is exposed to osteoclasts that mistake cementum for bone and proceed to replace the cementum and dentin with new bone, resulting in a fusing of the bone and the tooth (26). An ankylosed tooth can be diagnosed clinically within 1 month by its percussion sound (high, metallic) and radiographically within 2 months (disappearance of PDL space and invasion of bone into the root) (Fig. 9).

Currently, there is no predictable method for arresting ankylosis-related resorption after its initiation. Efforts to make cementum more resistant by various methods (e.g. application of fluoride to the root of an avulsed tooth before delayed replantation) are not, in the long term, successful and may at best slow the process that inevitably will consume the entire root (26). More recently, Emdogain® (Biora, Inc., Chicago, IL, USA) (27) has been recommended as a possible agent for

Fig. 8. Treatment of inflammatory infection-related resorption. (a) The mandibular right first premolar had by error been luxated in preparation for extraction for orthodontic reasons when the dentist realized the error. The dentist stabilized the tooth for 6 weeks when this radiograph was taken. Note the extensive resorption of both bone and root (arrows). (b) Radiograph taken upon completion of root canal treatment. (c) Four-year follow-up examination demonstrates arrest of inflammatory resorption and bony repair. Note that tooth structure lost by resorption cannot be replaced by new tooth structure, emphasizing the need to initiate endodontic treatment at the first sign of infection-related resorption.

Fig. 9. Replacement ankylosis-related resorption. (a) Replacement resorption and ankylosis occurring shortly after replantation of a left maxillary central incisor which had been dry for more than 1 h prior to replantation. Note the disappearance of PDL, indicating bony fusion with tooth structure. (b) The same tooth 20 months later shows almost complete loss of root structure.

promoting development of new cementum into which new PDL fibers can attach. The procedure is still being investigated and cannot yet be deemed predictable.

Treatment concepts

The goal of treatment for traumatically injured teeth is to return the teeth to acceptable function and appearance. Normal function (if present before the traumatic event) requires repositioning of the teeth if they were displaced, and acceptable appearance requires repair of possible dental fractures and proper positioning of peridental soft tissues. A practical question to ask would be – does successful treatment require that every dental injury be treated within the first few hours after trauma? The answer, based on the best available evidence is that various treatment priorities can be selected depending on the type of injury (28). The following guidelines can be applied to treatment priorities.

Acute treatment: There are situations where treatment within a few hours can significantly affect the outcome. In this category belong tooth avulsions, alveolar fractures, extrusive and lateral luxations, and possibly root fractures. Early repositioning and stabilization will promote the best PDL repair (28).

Subacute treatment: Treatment within 24 h after injury allow the following injuries proper care: concussion, subluxations, and intrusive luxation, and crown fractures with pulpal exposure. Pulpal and PDL responses do not seem to be adversely affected by a delay of 24 h (28).

Delayed treatment: Crown fractures without pulpal exposure appear to have the same prognosis whether treatment is performed within a few or several hours (28).

Treatment planning

Management of traumatic injuries include, after examination and diagnosis, urgent care if indicated (involving treatment priorities just discussed) and definitive treatment. The latter requires planning both for the immediate and the long-term care (5).

Immediate care may be initiated with the emergency treatment provided, such as pulp protection for continued root formation in developing teeth with complicated crown fractures. In cases of luxation and avulsion injuries, the immediate concern is to stabilize the tooth in its normal position to allow re-attachment and re-organization of the periodontal ligament support (Fig. 10). This latter group also includes the coronal segment of a root-fractured tooth. With respect to the management of pulpal trauma, every effort must be made to protect pulp vitality or allow for

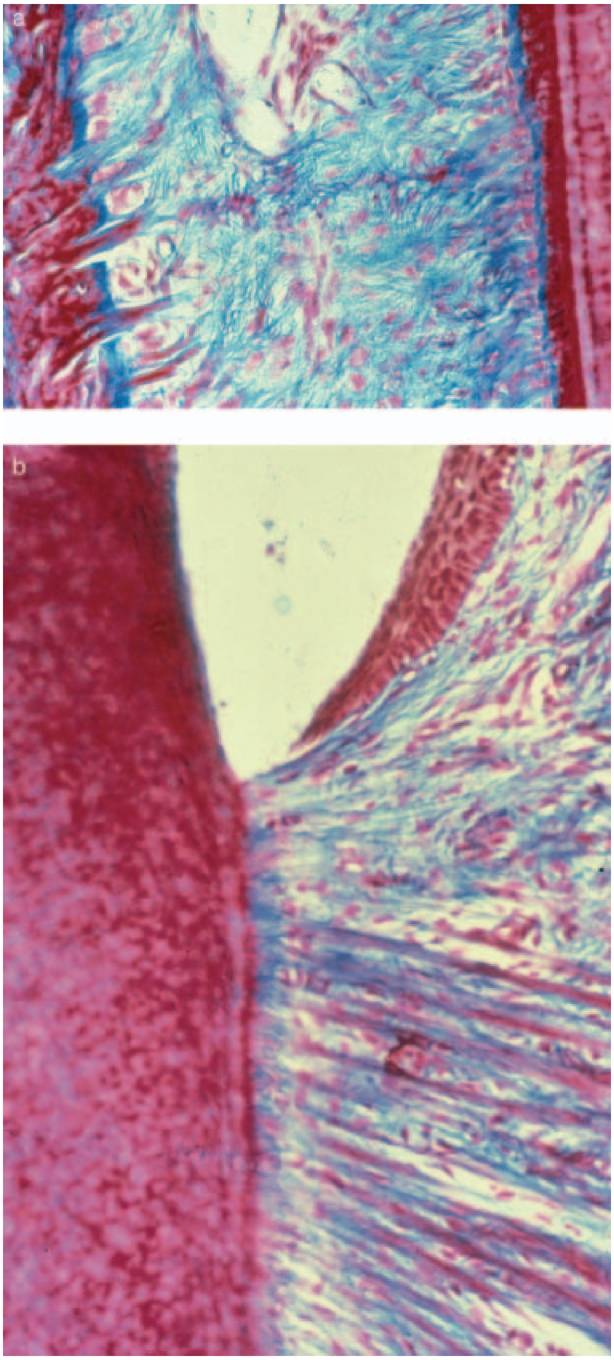

Fig. 10. Reorganization of disrupted PDL fibers. (a) Photomicrograph of PDL after orthodontic extrusion of a tooth in a dog. Note the disarray of fibers (H&E × 100). (b) Photomicrograph of another tooth that also had been extruded, taken after 6 weeks of stabilization. Note the regular arrangement of fibers (H&E × 100).

the possibility of revascularization of pulps in immature, developing teeth (12). This is done to promote continued root formation, the failure of which favors early loss of such teeth due to cervical root fracture (see Fig. 7).

In fully formed mature teeth in which pulpal trauma has occurred, a major concern is that if coagulation-type pulp necrosis develops due to severance of the apical blood supply, subsequent invasion of bacteria into the necrotic pulp tissue stimulates infection-related resorption. Competent root canal therapy can prevent, or if already begun, arrest this type of resorption (11, 23, 25) (see Fig. 8).

Current treatment planning recommendations can be summarized as follows:

- *Uncomplicated crown fractures:* In mature teeth, esthetic and functional restoration will provide good prognosis. In developing teeth, concern must be given to the risk of bacterial contamination involving the exposed dentin with large diameter tubules. Timely care includes disinfection of the dentin and protection with either CH covered with a restoration, or a restoration bonded directly to the broken tooth surface (29, 30) (Fig. 11).
- *Complicated crown fractures:* Fully developed teeth will most likely require a prosthetic crown, thus the patient may wisely choose to have root canal treatment done prior to the restoration. It is, however, acceptable, if a bonded restoration is to be used, to protect the exposed pulp with CH or mineral trioxide aggregate (ProRoot® MTA; Dentsply Int., Tulsa, OK, USA), the procedure recommended for developing teeth to allow continued root formation (29, 31, 32) (Fig. 12).
- *Crown-root fractures:* These complicated fractures often involve pulpal exposure, and in developing teeth, pulpal protection is essential if the tooth is going to continue to develop (Fig. 13). Because the fractures extend to the roots to varying depths, treatment options depend on the level of fracture. After the removal of the loose tooth fragment, one may allow the gingiva to adapt to the exposed dentin by formation of long junctional epithelium, or surgically expose the fracture site, or extrude the tooth orthodontically or surgically (33). In fully developed teeth, all of these procedures are likely to be associated with root canal therapy (34).
- *Root fractures:* This type of injury involves the pulp, dentin, cementum, and the PDL. It is recognized that the coronal segment often has been luxated, thus pointing to a treatment approach different from that recommended in the past, which was rigid

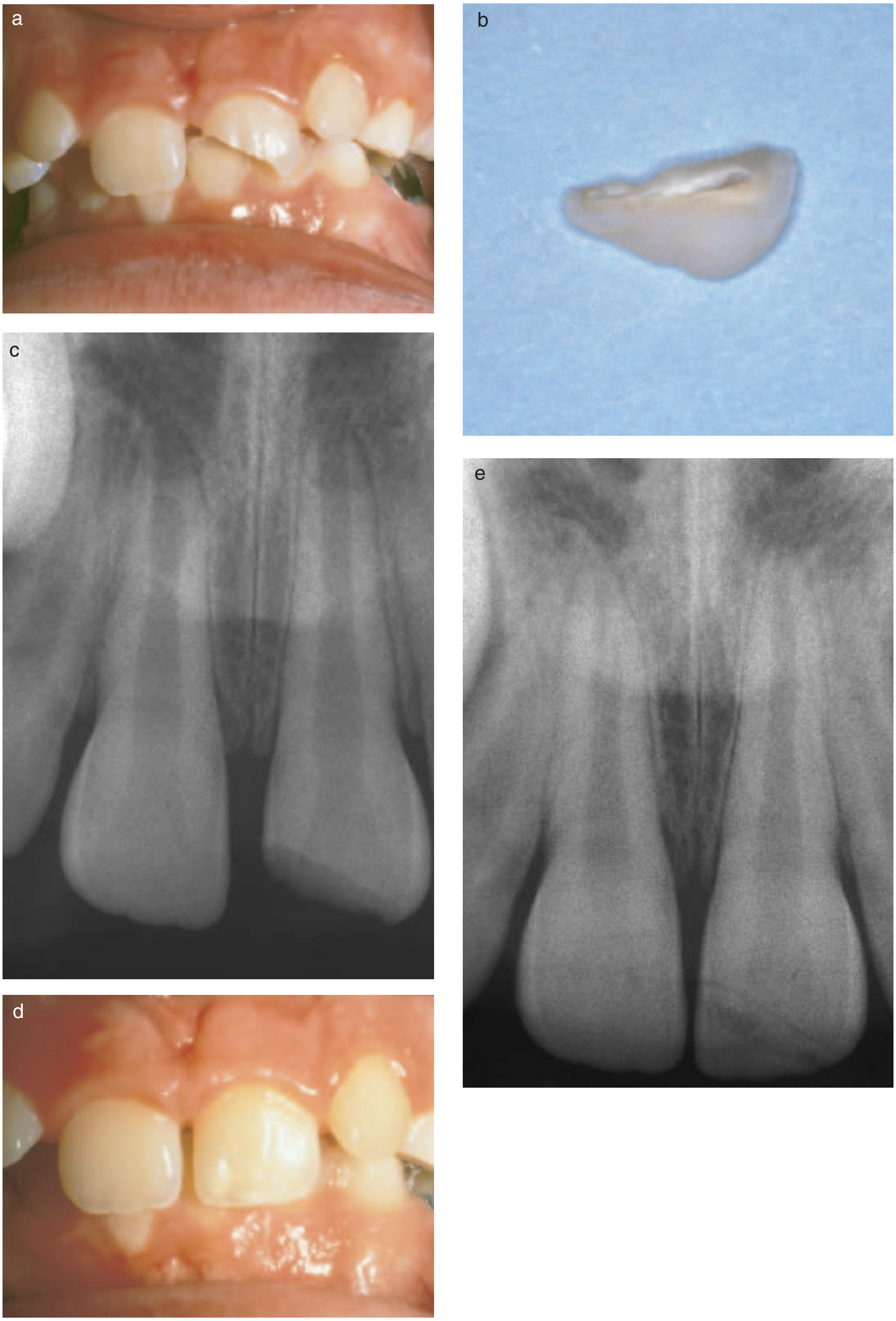

Fig. 11. **Bonding fractured crown. (a) Photo showing maxillary left central incisor of an 8-year-old boy who fractured the tooth in an accident. (b) His mother saved the broken fragment allowing it to be rebonded. (c) Radiograph shows development stage of the tooth; protecting the pulp by bonding the crown fragment is desirable. (d) Photo shows result of bonding. The fracture line can be additionally covered with composite resin. (e) Radiograph 8 months later shows continued root development with apical closure, indicating normal pulpal function. (Courtesy of Dr Todd Milledge, pediatric dentistry, Loma Linda University, Loma Linda, CA, USA.)**

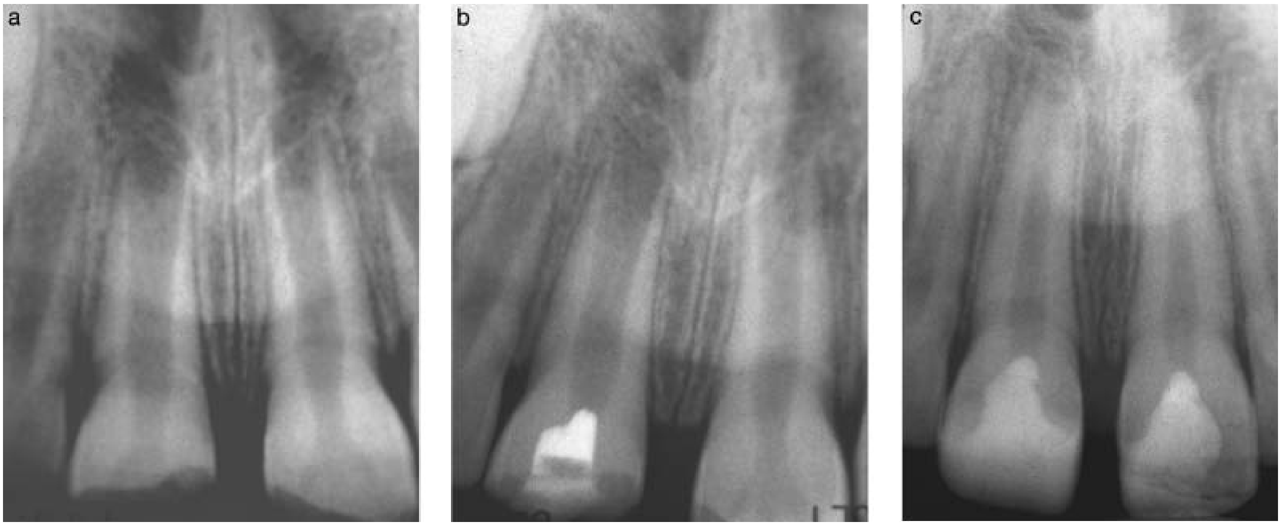

Fig. 12. The use of mineral trioxide aggregate (MTA) for vital pulp therapy. (a) Two fractured central incisors in an 8-year-old boy who suffered a traumatic accident. The pulps in both teeth were exposed. (b) Radiograph shows initial treatment with MTA in the right incisor; the same procedure was performed on the left incisor. (c) Radiograph of the two incisors 3 years post-treatment. Note the continued root development indicating normal pulpal function in both teeth.

splinting for long periods of time, that is, 3 months or more (35). Based on current evidence, the treatment should instead be similar to that given luxation injuries: semi-rigid stabilization for a few weeks (3–4 weeks) to allow re-establishment of the damaged PDL (36). The pulp has a favorable prognosis as well; less than 20% develop necrosis requiring root canal therapy (36–39). And when such treatment is indicated, based on the appearance of osteitis surrounding the fracture site, treatment needs only to be applied to the coronal segment as the apical segment invariably remains vital even when the coronal pulp tissue is necrotic (36).

- *Luxation injuries:* Minor involvement such as concussion and subluxation, requires mostly symptomatic treatment: soft diet and possibly occlusal adjustment to minimize discomfort on biting contact (5, 40). All traumatized teeth must be monitored for delayed pulpal necrosis; endodontic intervention is indicated in such cases (41).

The more severe types of injuries, extrusive and lateral luxations, need both immediate care (repositioning and stabilization for 2–4 weeks) and long-term consideration. In mature, fully developed teeth, pulp vitality is not expected to return (except in a very small percentage of teeth in which revascularization occurs through a process of transient apical breakdown (18)). In luxated teeth with pulp necrosis, root canal therapy is indicated. If neglected, infection-related root resorption is a distinct and dangerous possibility (23, 25, 40) (see Fig. 8).

Developing teeth with immature roots and open apices (>0.5 mm diameter) have the potential for revascularization, which will facilitate continued root development (12). Pulp necrosis in young developing teeth has traditionally been managed by the endodontic technique of apexification using CH. It has recently been demonstrated that long-term (>1 month) use of CH renders root dentin increasingly prone to fracture (19). Current evidence indicates that a short-term (<1 month) use of CH for its disinfecting property, followed by MTA placement in the apical segment of the root canal, leaves the root more resistant to fracture than when long-term CH was used (42).

The most serious type of luxation injury is intrusion (15, 40). Damage occurs to the cementum and the PDL, and the neurovascular pulp supply is crushed. Current treatment approaches include surgical repositioning, orthodontic extrusion, and a combination of both (5, 40, 43). It is not yet evident which approach is most reliable. In any case, root canal treatment is a must (except in very immature teeth) and ankylosis-related resorption is a frequent occurrence (Fig. 14). The only exception to root canal treatment is also the only exception to active repositioning: In very young, developing teeth (probably only below the age of

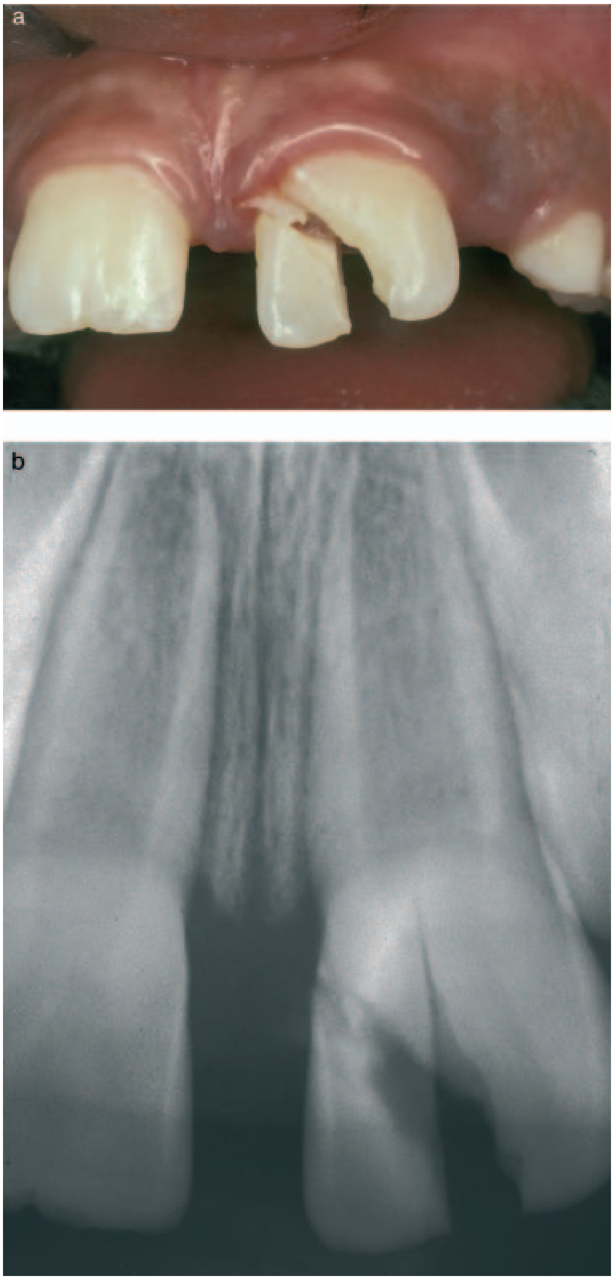

Fig. 13. Crown-root fracture of maxillary left central incisor in a 6-year-old boy who fell off a swing and hit the tooth on a rock. (a) Photo shows the appearance typical of crown-root fractures: Broken, loose crown pieces still attached to PDL. (b) The radiograph shows early development stage of the tooth. Crown root fractures are a severe challenge to manage in young patients with developing roots. The goal is to make every effort to protect the pulp to allow continued root development.

8 or 9) re-eruption can occur and for that reason in such young patients it is prudent to observe for spontaneous re-eruption before initiating active treatment (Fig. 15).

Fig. 14. Ankylosis-related resorption of intruded maxillary left central incisor. (a) Note the extensive infraocclusion as a result of ankylosis at the time of significant growth including the alveolar processes and eruption of adjacent teeth. (b) Radiograph of the tooth pictured in A; interestingly, the pulp survived the traumatic injury, but the PDL and cementum were damaged, allowing ankylosis-related resorption.

- *Avulsions:* All current evidence indicates that immediate replantation favors a successful outcome (44–47). If an avulsed tooth must be transported or

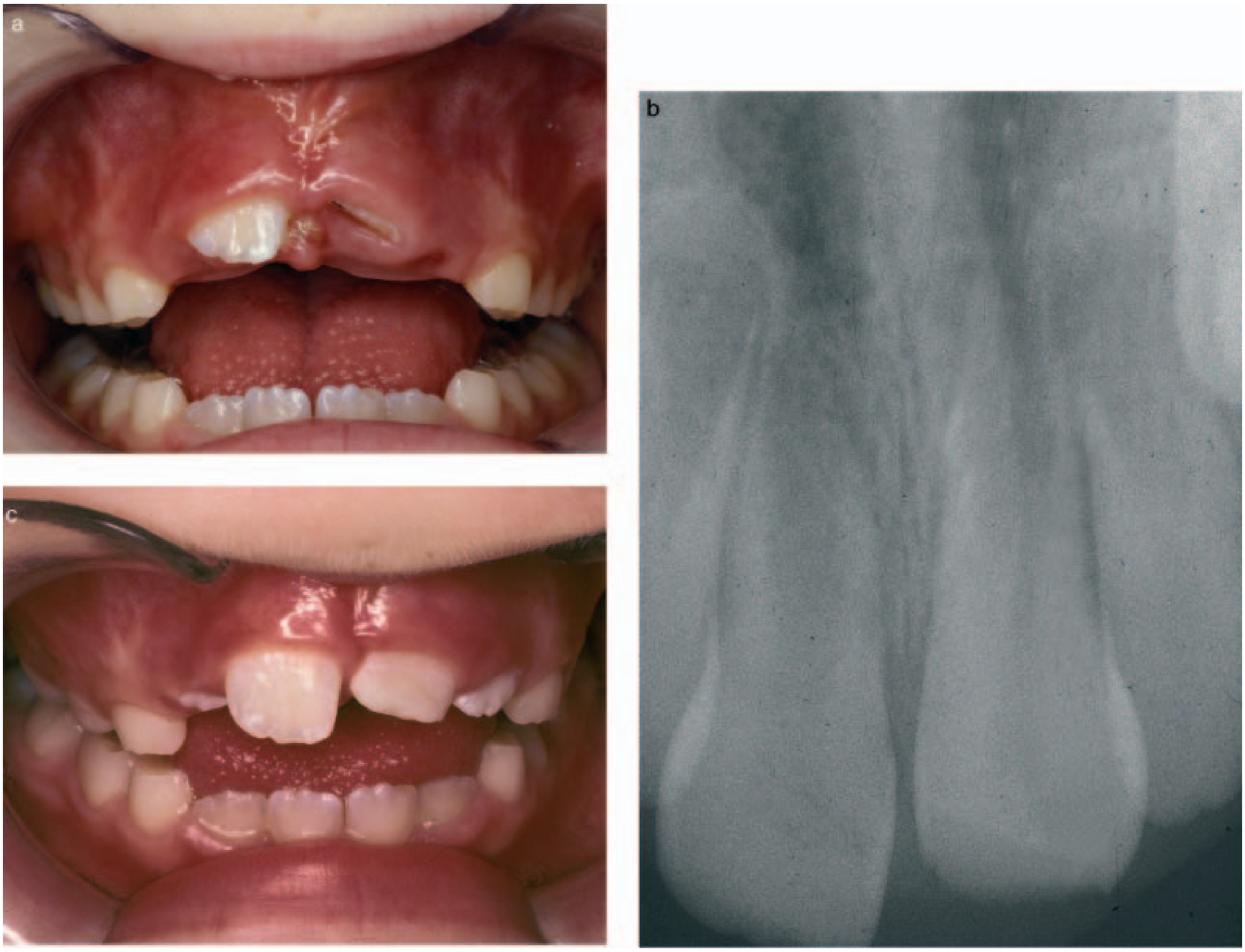

Fig. 15. Intrusive luxation of developing tooth in a 6-year-old boy. (a) Photo taken immediately after intrusion of maxillary left central incisor. (b) Radiograph taken at the time of examination showing very early stages of development of the incisors. Re-eruption is the ideal outcome. (c) Photo taken 6 weeks after intrusion showing satisfactory re-eruption of the intruded tooth, which in time reached the same level of eruption as the adjacent incisor.

stored prior to replantation, specially developed storage media can sustain the vitality of the PDL for several hours (48). Availability of such storage media is a problem, but is has been demonstrated that milk can be a very suitable storage solution for up to several hours (49). Keeping the tooth in saliva is also an option. Plain water or dry storage will result in a quick death of the PDL and its cells.

With the exception of immature, incompletely developed teeth, root canal treatment is an essential component of the treatment strategy (45). Failure to remove the necrotic pulp will result in infection-related resorption (see Fig. 4). Failure to replant the avulsed tooth before PDL death will likely lead to ankylosis-related resorption.

Much attention has been given to the possibility that Emdogain® (27) may promote new periodontal attachment to replanted, avulsed teeth with long dry time (>1 h). The evidence is not yet available to determine if this agent can reliably produce successful outcomes.

- *Fracture of the alveolar process:* Teeth located in the immediate vicinity of a fracture of the alveolar process, may develop pulp necrosis as a result of the effect the fracture has on the blood supply to the teeth. Pulp vitality must be monitored and endodontic intervention is important, when indicated, both for the retention of the teeth, and for the successful healing of the bony fracture (50).

Conclusion

Dental traumatology has progressed in recent years to improve the understanding of the biological considerations involved in both diagnosis and treatment principles. Through public awareness efforts (51–54), lay people are more knowledgeable about dental trauma (e.g. knowing to put an avulsed tooth in milk). Research explores new approaches (e.g. laser doppler flowmeter for pulpal evaluation (55, 56)) and materials (e.g. Emdogain® (27)). The future has promise for even more successful management of traumatic dental injuries.

References

1. Gottrup F, Andreasen JO. Wound healing subsequent to injury. In: Andreasen JO, Andreasen FM, eds. *Textbook and Color Atlas of Traumatic Injuries to the Teeth*, 3rd edn. Copenhagen: Munksgaard, 1993: 13–76.
2. Andreasen JO, Andreasen FM. Classification, etiology and epidemiology of traumatic dental injuries. In: Andreasen JO, Andreasen FM, eds. *Textbook and Color Atlas of Traumatic Injuries to the Teeth*, 3rd edn. Copenhagen: Munksgaard, 1993: 151–177.
3. Application of the International Classification of Diseases to Dentistry and Stomatology *IDC-DA*, 3rd edn. Geneva: WHO, 1995.
4. Andreasen JO. Response of oral tissue to trauma. In: Andreasen JO, Andreasen FM, eds. *Textbook and Color Atlas of Traumatic Injuries to the Teeth*, 3rd edn. Copenhagen: Munksgaard, 1993: 77–112.
5. The International Association of Dental Traumatology. Guidelines for the evaluation and management of traumatic dental injuries. *Dental Traumatol* 2001: **17**: 1–4, 49–52, 97–102, 145–148.
6. Andreasen JO, Andreasen FM, Bakland LK, Flores MT. Emergency record for acute dental trauma, and clinical examination form for the time of injury and follow-up examination. In: *Traumatic Dental Injuries: A Manual*, 2nd edn. Oxford: Blackwell Munksgaard, 2003: 72–75.
7. Andreasen JO, Andreasen FM, Bakland LK, Flores MT. Examination and diagnosis. In: *Traumatic Dental Injuries: A Manual*, 2nd edn. Oxford: Blackwell Munksgaard, 2003: 18–21.
8. Andreasen FM, Andreasen JO. Diagnosis of luxation injuries. The importance of standardized clinical, radiographic and photographic techniques in clinical investigation. *Endod Dent Traumatol* 1985: **1**: 160–169.
9. Andreasen FM, Andreasen JO. Examination and diagnosis of dental injuries. In: Andreasen JO, Andreasen FM, eds. *Textbook and Color Atlas of Traumatic Injuries to the Teeth*, 3rd edn. Copenhagen: Munksgaard, 1993: 196–215.
10. Gift HC, Bhat M. Dental visits for orofacial injury: defining the dentist's role. *J Am Dent Assoc* 1993: **124**: 92–98.
11. Andreasen FM, Wistergaard Pedersen B. Prognosis of luxated permanent teeth – the development of pulp necrosis. *Endod Dent Traumatol* 1985: **1**: 207–220.
12. Andreasen FM, Yu Z, Thomsen BL. Relationship between pulp dimensions and development of pulp necrosis after luxation injuries in the permanent dentition. *Endod Dent Traumatol* 1986: **2**: 90–98.
13. Jacobsen I, Kerekes K. Long-term prognosis of traumatized permanent anterior teeth showing calcifying processes in the pulp cavity. *Scand J Dent Res* 1977: **85**: 588–598.
14. Robertson A, Andreasen FM, Bergenholtz G, Andreasen JO, Norén JG. Incidence of pulp necrosis subsequent to canal obliteration from trauma to permanent teeth. *J Endod* 1996: **22**: 557–560.
15. Humphrey JM, Kenny DJ, Barrett EJ. Clinical outcomes for permanent incisor luxations in a pediatric population. I. Intrusions. *Dent Traumatol* 2003: **19**: 226–273.
16. Lee R, Barrett EJ, Kenny DJ. Clinical outcomes for permanent incisor luxations in a pediatric population. II. Extrusions. *Dent Traumatol* 2003: **19**: 274–279.
17. Nikoui M, Kenny DH, Barrett EJ. Clinical outcomes for permanent incisor luxations in a pediatric population. III. Lateral luxations. *Dent Traumatol* 2003: **19**: 280–285.
18. Andreasen FM. Transient apical breakdown and its relation to color and sensibility changes after luxation injuries to teeth. *Endod Dent Traumatol* 1986: **2**: 9–19.
19. 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.
20. Cvek MA. 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, Gavra S, Chosack A. Long-term follow-up of traumatized incisors treated by partial pulpotomy. *Pediatr Dent* 1993: **15**: 334–336.
22. Lim KC, Kirk EE. Direct pulp capping: a review. *Endod Dent Traumatol* 1987: **3**: 213–219.
23. Andreasen FM, Andreasen JO. Root resorption following traumatic dental injuries. *Proc Finn Dent Soc* 1992: **88**: 95–114.
24. Andreasen FM, Yu Z, Thomsen ML, Andersen PK. Occurrence of pulp canal obliteration after luxation injuries in the permanent dentition. *Endod Dent Traumatol* 1987: **3**: 103–115.
25. Trope M. Root resorption due to dental trauma. *Endod Topics* 2002: **1**: 79–100.
26. Andreasen JO, Borum M, Jacobsen HL, Andreasen FM. Replantation of 400 avulsed permanent incisors. IV. Factors related to periodontal ligament healing. *Endod Dent Traumatol* 1995: **11**: 76–89.
27. Filippi A, Pohl Y, von Arx T. Treatment of replacement resorption with Emdogain® – preliminary results after 10 months. *Dent Traumatol* 2001: **17**: 3–10.

28. Andreasen JO, Andreasen FM, Skeie A, Hjørting-Hansen E, Schwartz O. Effect of treatment delay upon pulp and periodontal healing of traumatic dental injuries – a review article. *Dent Traumatol* 2002: **18**: 1–13.
29. Andreasen FM, Norén JG, Andreasen JO, Engelhardtsen S, Lindh-Strömberg U. Long-term survival of crown fragment bonding in the treatment of crown fractures. A multicenter clinical study of fragment retention. *Quintessence Int* 1995: **26**: 669–681.
30. Robertson A, Andreasen FM, Andreasen JO, Norén JG. Long-term prognosis of crown-fractured permanent incisors. The effect of stage of root development and associated luxation injury. *Int J Paediatric Dent* 2000: **10**: 191–199.
31. Torabinejad M, Chivian N. Clinical applications of mineral trioxide aggregate. *J Endod* 1999: **25**: 197–205.
32. Cvek M. Endodontic management of traumatized teeth. In: Andreasen JO, Andreasen FM, eds. *Textbook and Color Atlas of Traumatic Injuries to the Teeth*, 3rd edn. Copenhagen: Munksgaard, 1993: 517–585.
33. Kahnberg K-E. Surgical extrusion of root fractured teeth – a follow-up study of two surgical methods. *Endod Dent Traumatol* 1988: 45–89.
34. Warfvinge J, Kahnberg K-E. Intraalveolar transplantation of teeth. IV. Endodontic considerations. *Swed Dent J* 1989: **13**: 229–233.
35. Andreasen FM, Andreasen JO. Root fractures. In: Andreasen JO, Andreasen FM, eds. *Textbook and Color Atlas of Traumatic Injuries to the Teeth*, 3rd edn. Copenhagen: Munksgaard, 1993: 297.
36. Cvek M, Mejàre I, Andreasen JO. Healing and prognosis of teeth with intraalveolar fractures involving the cervical part of the root. *Dent Traumatol* 2002: **18**: 57–65.
37. Zachrisson BU, Jacobsen I. Long-term prognosis of 66 permanent anterior teeth with root fracture. *Scand J Dent Res* 1975: **83**: 345–354.
38. Welbury RR, Kinirons MJ, Day P, Humphreys K, Gregg TA. Outcomes for root-fractured permanent incisors: a retrospective study. *Pediatr Dent* 2002: **24**: 98–102.
39. Feely L, Mackie IC, Macfarlane T. An investigation of root-fractured permanent incisor teeth in children. *Dent Traumatol* 2003: **19**: 52–54.
40. Andreasen FM, Andreasen JO. Luxation injuries. In: Andreasen JO, Andreasen FM, eds. *Textbook and Color Atlas of Traumatic Injuries to the Teeth*, 3rd edn. Copenhagen: Munksgaard, 1993: 315–382.
41. de Cleen M. Obliteration of pulp canal space after concussion and subluxation: endodontic considerations. *Quintessence Int* 2002: **33**: 661–669.
42. Andreasen JO, Bakland LK. Comparison of application of calcium hydroxide or MTA in root canals of immature sheep teeth. (in press).
43. Ebeleseder KA, Santler G, Glockner K, Hulla H, Perti C, Quenhenberger F. An analysis of 58 traumatically intruded and surgically extruded permanent teeth. *Endod Dent Traumatol* 2000: **16**: 34–39.
44. Andreasen JO, Borum M, Jacobsen HL, Andreasen FM. Replantation of 400 traumatically avulsed permanent incisors. I. Diagnosis of healing complications. *Endod Dent Traumatol* 1995: **11**: 51–58.
45. Andreasen JO, Borum M, Jacobsen HL, Andreasen FM. Replantation of 400 traumatically avulsed permanent incisors. II. Factors related to pulp healing. *Endod Dent Traumatol* 1995: **11**: 59–68.
46. Andreasen JO, Borum M, Jacobsen HL, Andreasen FM. Replantation of 400 traumatically avulsed permanent incisors. III. Factors related to root growth after replantation. *Endod Dent Traumatol* 1995: **11**: 69–75.
47. Andreasen JO, Borum M, Jacobsen HL, Andreasen FM. Replantation of 400 traumatically avulsed permanent incisors. IV. Factors related to periodontal ligament healing. *Endod Dent Traumatol* 1995: **11**: 76–89.
48. Trope M, Friedman S. Periodontal healing of replanted dog teeth stored in Viaspan, milk and Hank's balanced salt solution. *Endod Dent Traumatol* 1992: **8**: 183–188.
49. Huang S, Remeikis N, Daniel J. Effects of long-term exposure of human periodontal ligament cells to milk and other solutions. *J Endod* 1996: **22**: 30–33.
50. Kahnberg K-E, Ridell A. Prognosis of teeth involved in the line of mandibular fractures. *Int J Oral Surg* 1979: **8**: 163–172.
51. US Department of Health and Human Services. Community and other approaches to promote oral health and prevent oral disease. In: *Oral Health in America: A Report of the Surgeon General*. Rockville, MD: US Department of Health and Human Services, National Institute of Dental and Craniofacial Research, National Institutes of Health, 2000 (Chapter 7).
52. American Academy of Pediatric Dentistry. *Emergency Care*, 2002, http://www.aapd.org/publications/brochures/ecare.asp.
53. American Association of Endodontists. *Your Guide to Traumatic Dental Injuries*, 2002. http://www.aae.org/traumsum.html.
54. International Academy for Sports Dentistry. *Trauma Card*, 2002, http://www.sportsdentistry-iads.org/trauma.htm.
55. Olgart L, Gazelius B, Lindh-Strömberg U. Laser Doppler flowmetry in assessing vitality in luxated permanent teeth. *Int Endodon J* 1988: **21**: 300–306.
56. Ebihara A, Tokita Y, Izawa T, Suda H. Pulpal blood flow assessed by laser Doppler flowmetry in a tooth with a horizontal root fracture. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996: **81**: 229–233.

Appendices

Appendix 1. Emergency record for acute dental trauma. (From Andreasen JO, Andreasen FM, Bakland LK, Flores MT. Traumatic Dental Injuries, A Manual, 2nd edn. Oxford, UK: Blackwell Munksgaard, 2000: 72–74.)

Emergency record for acute dental trauma

page 1

Patient's name
Birth date

Date of examination:	Referred by:
Time of examination:	Referring diagnosis:

General medical history: any serious illness?	yes	no
If yes, explain.		
Any allergy?	yes	no
If yes, explain.		
Have you been vaccinated against tetanus?	yes	no
If yes, when.		
Previous dental injuries:	yes	no
If yes,		
When?		
Which teeth were injured?		
Treatment given and by whom?		
Present dental injury:		
Date: Time:		
Where?		
How?		
Have you had or have now *headache?*	yes	no
Have you had or have now *nausea?*	yes	no
Have you had or have now *vomiting?*	yes	no
Were you *unconscious* at the time of injury?	yes	no
If yes, for how long (minutes)?		
Can you *remember* what happened before, during or after the accident?	yes	no
Is there pain from *cold air?*	yes	no
If yes, *which teeth?*		
Is there pain or tenderness from *occlusion?*	yes	no
If yes, *which teeth?*		
Constant pain?	yes	no
If yes, *which teeth?*		
Treatment elsewhere?	yes	no
If yes, *what treatment?*		

Emergency record for acute dental trauma

page 2

After *avulsion,* the following information is needed:
Where were the teeth found (dirt, asphalt, floor, etc.)?
When were the teeth found?
Were the teeth *dirty?*
How were the teeth *stored?*
Were the teeth *rinsed* and *with what* prior to replantation?
When were the teeth replanted?
Was *tetanus antitoxoid* given?
Were *antibiotics* given?
Antibiotic?
Dosage?

Objective examination

Is the patient's general condition affected?	yes	no
If yes, *pulse*		
blood pressure		
pupillary reflex		
cerebral condition		
Objective findings beyond the head and neck?	yes	no
If yes, *type* and *location*		
Objective findings within the head and neck?	yes	no
If yes, *type* and *location*		

Objective examination – Extraoral findings (contd.)

Bleeding from nose, or rhinitis	yes	no
Bleeding from ext. auditory canal	yes	no
Double vision or limited eye movement	yes	no
Palpable signs of fracture of facial skeleton	yes	no
If yes, *location of fracture*		

Objective examination – Intraoral findings

Lesions of the *oral mucosa*	yes	no
If yes, *type* and *location*		
Gingival lesion	yes	no
If yes, *type* and *location*		
Tooth fracture	yes	no
If yes, *type* and *location*		
Alveolar fracture	yes	no
If yes, *type* and *location*		

Supplemental information:

General condition of the dentition

Caries	poor	fair	good
Periodontal status	poor	fair	good
Horizontal occlusal relationship	undr bite	over jet	norm
Vertical occlusal relationship	deep	open	norm

Radiographic findings
Tooth dislocation
Root fracture
Bone fracture
Pulp canal obliteration
Root resorption

Photographic registration ☐ yes ☐ no

Diagnoses (check appropriate boxes and designate tooth no. or indicate correct anatomical region)

- ☐ Infraction
- ☐ Complicated crown fracture
- ☐ Uncomplicated crown fracture

- ☐ Complicated crown-root fracture
- ☐ Uncomplicated crown-root fracture

- ☐ Root Fracture
- ☐ Alveolar fracture
- ☐ Mandibular fracture
- ☐ Maxillary fracture

- ☐ Concussion
- ☐ Subluxation
- ☐ Extrusion
- ☐ Lateral luxation
- ☐ Intrusion
- ☐ Exarticulation

- ☐ Skin abrasion
- ☐ Skin laceration
- ☐ Skin contusion

- ☐ Mucosal abrasion
- ☐ Mucosal laceration
- ☐ Mucosal contusion

- ☐ Gingival abrasion
- ☐ Gingival laceration
- ☐ Gingival contusion

Supplementary remarks:

Treatment plan

At time of injury:

Repositioning (time finished)
Fixation (time finished)
Pulpal therapy (time finished)
Dentinal coverage (time finished)

Final therapy:

Chart re-read by examining dentist ☐ yes ☐ no

Appendix 2. Clinical examination form. (From Andreasen JO, Andreasen FM, Bakland LK, Flores MT. Traumatic Dental Injuries, A Manual, 2nd edn. Oxford, UK: Blackwell Munksgaard, 2000: 75.)

Clinical examination form for the time of injury and follow-up examinations

	Tooth no.	12		11		21		22	
TIME OF INJURY	Date								
	Tooth color normal yellow red grey crown restoration								
	Displacement (mm) intruded extruded protruded retruded								
	Loosening (0-3)								
	Tenderness to percussion (+/−)								
	Pulp test (value)								
	Ankylosis tone (+/−)								
	Occlusal contact (+/−)								
CONTROL	Fistula (+/−)								
	Gingivitis (+/−)								
	Gingival retraction (mm)								
	Abnormal pocketing (+/−)								

Each column represents an examination of a given tooth. The first column for each tooth gives the values from the time of injury. *Only* the parameters listed in the top half of the form ('Time of injury') are to be recorded at the time of injury. The information from this examination as well as the information collected on the emergency record are used to determine the final diagnoses for the injured teeth. Those parameters *and* the last four (fistula, gingivitis, gingival retraction, abnormal pocketing) are to be registered at all follow-up controls.