SEQUELAE OF INJURIES TO THE PRIMARY DENTITION

Complications following traumatic injuries to primary teeth may appear shortly after the injury (e.g., infection of the PDL or dark discoloration of the crown) or after several months (e.g., yellow discoloration of the crown and external root resorption).

- **Pulpitis**

The pulp's initial response to trauma is pulpitis. Capillaries in the tooth become congested, Teeth with reversible pulpitis may be tender to percussion if the PDL is inflamed (e.g., following a luxation injury). Pulpitis may be totally reversible if the condition causing it is addressed, or it may progress to an irreversible state with necrosis of the pulp.

- **Infection of the Periodontal Ligament**

Infection of the PDL becomes possible when detachment of the gingival fibers from the tooth in a luxation injury allows invasion of microorganisms from the oral cavity along the root to infect the PDL. Loss of alveolar bone support can be seen on a periapical radiograph . This diminishes the healing potential of the supporting tissues. Subsequently, increased tooth mobility accompanied by exudation of pus from the gingival crevice will require extraction of the injured tooth.

- **Pulp Necrosis and Infection**

Two main mechanisms can explain how the pulp of injured primary teeth becomes necrotic: (1) infection of the pulp in cases of untreated crown fracture with pulp exposure, and (2) interrupted blood supply to the pulp through the apex in cases of luxation injury leading to ischemia. Periapical radiolucencies indicative of a granuloma or cyst are frequently evident radiographically in necrotic anterior teeth.

- **Coronal Discoloration**

As a result of trauma, the capillaries in the pulp occasionally hemorrhage, leaving blood pigments deposited in the dentinal tubules. In mild cases, the blood is resorbed and very little discoloration occurs, or that which is present becomes lighter in several weeks. In more severe cases, the discoloration persists for the life of the tooth .

From a diagnostic standpoint, discoloration of primary teeth does not necessarily mean that the tooth is nonvital, particularly when the discoloration occurs 1 or 2 days after the injury.

Pink discoloration that is observed shortly after the injury may represent intrapulpal hemorrhage.

Yellow discoloration of primary incisors can be seen when the dentin is thick and the pulp chamber narrower than usual. This condition is termed pulp canal obliteration

Dark discoloration of primary teeth is the most controversial posttraumatic complication in terms of the significance of the change in tooth color. The term "dark" refers to a variety of shades, including black, gray, brown.

When the pulp becomes necrotic or when pulpal hemorrhage occurs, red blood cells lyse and release hemoglobin. Hemoglobin and its derivatives, such as hematin molecules that contain iron ions, invade the dentin tubules and stain the tooth dark. If the pulp remains vital and eliminates the pigments, the dark discoloration may fade with subsequent restoration of the original color. If the pulp loses its vitality and cannot eliminate the iron-containing molecules, the tooth may remain discolored.

- **Inflammatory Resorption**

Inflammatory resorption can occur either on the external root surface or internally in the pulp chamber or canal .It occurs subsequent to luxation injuries and is related to a necrotic pulp and an inflamed PDL.

Internal Resorption

The predentin, an unmineralized layer of organic material, covers the inner aspect of the dentin and protects it against access of osteoclasts. When the pulp becomes inflamed, as in cases of traumatic injury, the odontoblastic layer may lose its integrity and expose the dentin to odontoclastic activity, which is then seen on radiographs as radiolucent expansion of the pulp space. Eventually this process reaches the outer surface of the root, causing root perforation.

External Resorption

The cementoblast layer and the precementum serve as a shield protecting the root from involvement in the perpetual remodeling process of the surrounding bone. In nontraumatized primary teeth, external root resorption is part of the physiologic process of replacing the primary dentition with permanent teeth. In primary incisors sustaining traumatic injuries, external root resorption may appear as an accelerated unfavorable pathologic reaction.

- **Pulp Canal Obliteration**

Pulp canal obliteration is the result of intensified activity of the odontoblasts that results in accelerated dentin apposition. Gradually, the pulp space narrows to a state in which it cannot be seen on a radiograph. PCO is a common finding in primary incisors following traumatic injuries and is often associated with yellow coronal discoloration

- **Replacement Resorption**

Replacement resorption, also known as ankylosis, results after irreversible injury to the PDL. Alveolar bone directly contacts and fuses with the root surface. As the alveolar bone undergoes normal physiologic osteoclastic and osteoblastic activity, the root is resorbed and replaced with bone). Ankylosis occurs more often in intruded primary teeth, and they eventually become infraoccluded

Injuries to developing permanent teeth

Injuries to the permanent successor tooth can be expected in 12-69% of primary tooth trauma and 19-68% of jaw fractures. Intrusive luxation causes most disturbances. Most damage to the permanent tooth bud occurs under 3 years of age during its developmental stage. However, the type and severity of disturbance are closely related to the age at the time of injury. Changes in the mineralization and morphology of the crown of the permanent incisor are most common, but later injuries can cause radicular anomalies. Injuries to developing teeth can be classified as follows:

1. White or yellow-brown hypomineralization of enamel.

2. Crown dilaceration.

3. Odontoma-like malformation.

4. Root duplication.

5. Vestibular or lateral root angulation and dilacerations.

6. Partial or complete arrest of root formation.

7. Sequestration of permanent tooth-germs.

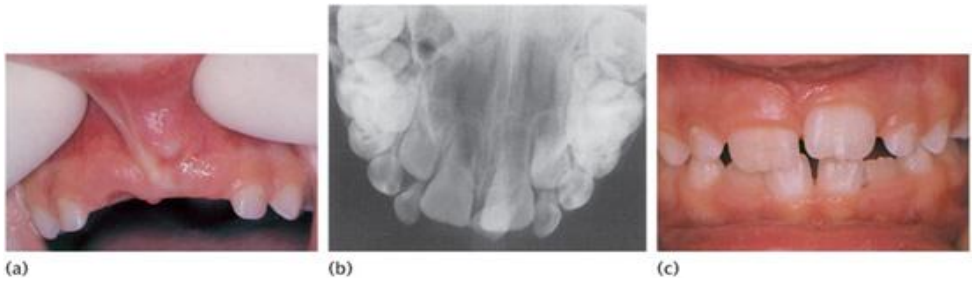
8. Disturbance in eruption: Eruption disturbances may involve delay because of thickening of connective tissue over a permanent tooth-germ, ectopic eruption due to lack of eruptive guidance, and impaction in teeth with malformations of crown or root.

The term dilaceration describes an abrupt deviation of the long axis of the crown or root portion of the tooth. This deviation results from the traumatic displacement of hard tissue, which has already been formed, relative to developing soft tissue.

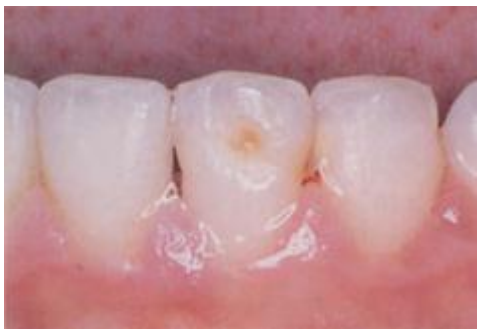
The term angulation describes a curvature of the root resulting from a gradual change in the direction of root development, without evidence of abrupt

displacement of the tooth-germ during odon-togenesis. This may be vestibular (i.e. labiopalatal) or lateral (i.e. mesiodistal).

Evaluation of the full extent of complications following injuries must await complete eruption of all permanent teeth involved. However, most serious sequelae (disturbances in tooth morphology) can usually be diagnosed radiographically within the first year post-trauma.



(a) Investigation of delayed eruption of the permanent upper central incisors revealed (b) an intruded upper left primary central incisor on radiograph. (c) Following removal of the retained primary incisor, the permanent successor erupted spontaneously with a white hypoplastic spot on the labial surface.



Brown hypoplastic area on the lower left permanent central incisor

Treatment of injuries to the permanent dentition

Yellow-brown hypomineralization of enamel with or without hypoplasia

1. Acid–pumice micro–abrasion.

2. Composite resin restoration: localized, veneer, or crown.

3. Porcelain restoration: veneer or crown . Conservative approaches are preferred whenever possible.

Crown dilaceration

1. Surgical exposure + orthodontic realignment.

2. Removal of dilacerated part of crown.

3. Temporary crown until root formation complete.

4. Semi-permanent or permanent restoration.

Vestibular root angulation

Combined surgical and orthodontic realignment.

Injuries To The Permanent Dentition

Trauma cases require follow-up to identify any complications and institute the correct treatment. In the review period the following schedule is a guide: 1 week; 1, 3, 6, and 12 months; then annually for 4–5 years.

1. Enamel infraction

Infraction is defined as an incomplete fracture (crack) of the enamel without loss of tooth structure. Clinical examination reveals normal gross anatomic and radiographic appearance: however, upon closer examination, craze lines are apparent in the enamel, especially with transillumination. A periapical radiograph of the involved area is taken to rule out root fracture and injuries to the supporting tooth structure. Pulp sensibility testing is recommended in adult teeth to monitor pulpal changes, however, results in primary teeth may be unreliable. Test results may be negative initially indicating transient pulpal damage.

These incomplete fractures without loss of tooth substance and without proper illumination are easily overlooked. Review is necessary, as above, as the energy of the blow may have been transmitted to the periodontal tissues or the pulp.

2. Enamel fracture

In some cases, minor enamel fractures can be smoothed with fine disks. Larger fractures should be restored using an acid-etch/composite resin technique.

3. Enamel–dentine (uncomplicated) fracture

1. The primary issue in managing fractures that expose dentin is to prevent bacterial irritants from reaching the pulp. Standard care in the past called for covering exposed dentin with calcium hydroxide (CaOH) or glass ionomer cement to seal out oral flora. Recent research indicates that sealing exposed dentin with a bonding agent enables the unexposed pulp to form reparative dentin. Some clinicians are thus advocating simultaneous acid etching of dentin and enamel followed by dentin and enamel bonding without placement of CaOH or glass ionomer. However, a recent review of pulp capping with dentin adhesive systems reported that these systems are not indicated owing to increased inflammatory reactions, delay in pulp healing, and failure of dentin bridge formation. Its recommends covering the deepest portion of dentin fractures with glass ionomer cement, followed by a dentin-bonding agent . The tooth can then be restored with an acid-etch/composite resin technique .If adequate time is not available to restore the tooth completely, an interim covering of resin material (a resin "patch") can temporize the tooth until a final restoration can be placed. Some dentists routinely place such a partial restoration to ensure an appropriate post-treatment evaluation when the patient returns for the final restoration. This is a reasonable strategy, provided that care is taken to ensure an adequate seal.

2. Reattachment of crown fragment. Few long-term studies have been reported and the longevity of this type of restoration is uncertain. In addition, there is a tendency for the distal fragment to become opaque or require further restorative intervention in the form of a veneer or full-coverage crown . If the

fracture line through dentine is not very close to the pulp, the fragment can be reattached immediately. However, if it runs close to the pulp, it is advisable to place a suitably protected calcium hydroxide dressing over the exposed dentine for at least a month while storing the fragment in saline, which should be renewed weekly.

Technique

1. Check the fit of the fragment and the vitality of the tooth.

2. Clean fragment and tooth with pumice-water slurry.

3. Isolate the tooth with rubber dam.

4. Attach fragment to a piece of sticky wax to facilitate handling.

5. Etch enamel for 30 seconds on both fracture surfaces and extend for 2mm from fracture line on tooth and fragment. Wash for 15 seconds and dry for 15 seconds.

6. Apply bonding agent ± dentine primer according to the manufacturer's instructions and light cure for 10 seconds.

7. Place appropriate shade of composite resin over both surfaces and position fragment. Remove gross excess and cure for 60 seconds labially and palatally.

8. Remove any excess composite resin with sandpaper discs.

9. Remove a 1mm gutter of enamel on each side of the fracture line both labially and palatally to a depth of 0.5mm using a small round or pear-shaped bur. The finishing line should be irregular in outline.

10. Etch the newly prepared enamel, wash, dry, apply composite, cure, and finish.

4. Enamel-dentine–pulp (complicated) crown fracture

Management of crown fractures that expose the pulp is particularly challenging .Pertinent clinical findings that dictate treatment include the following:

1Vitality of the exposed pulp

2Time elapsed since the exposure

3Degree of root maturation of the fractured tooth

4Restorability of the fractured crown

The objective of treatment in managing these injuries is to preserve **a vital pulp** in the entire tooth. It is not always possible to maintain vital tissue throughout the tooth. Three treatment alternatives are available, based on the clinical findings just noted:

1. Direct pulp cap
2. Pulpotomy
3. Pulpectomy

The direct pulp cap is only indicated in small exposures that can be treated within a few hours of the injury. The chances for pulp healing decrease if the tissue is inflamed, has formed a clot, or is contaminated with foreign materials..

A rubber dam is applied, and the tooth is gently cleaned with water. Commercially available CaOH paste or mineral trioxide aggregate (MTA) is applied directly to the pulp tissue and to surrounding dentin. It is essential that a restoration be placed that is capable of thoroughly sealing the exposure to prevent further contamination by oral bacteria. As in the management of dentin fractures, it is acceptable to use an acid-etch/composite resin system for an initial restoration. A calcific bridge stimulated by the capping material should be evident radiographically in 2 to 3 months**.**

In fractures exposing pulps of immature permanent teeth with incomplete root development, a direct cap is no longer the treatment of choice. Failure in these cases leads to total pulpal necrosis and a fragile, immature root with thin dentinal walls. Thus the preferred treatment in pulp exposures of immature permanent teeth is pulpotomy.

Vital pulp therapy: pulpotomy

In pulpotomy a portion of exposed vital pulp is removed to preserve the radicular vitality and allow completion of apical root development (apexogenesis) and further

deposition of dentine on the walls of the root. This procedure is the treatment of choice following trauma where the pulp has been exposed to the mouth for more than 24 hours. The amount of pulp that is removed depends on the time since exposure, which will also determine the depth of contamination of the pulp. Attempts must be made to remove only the pulp that is deemed to be contaminated. If the patient presents within 24–48 hours of the incident, it is safe to assume that the contaminated zone is no more than 2-4mm around the exposure site and only the pulp in the immediate vicinity of the exposure is removed, in a procedure also termed partial pulpotomy (Cvek's technique). For more extensive exposures all coronal pulp can be removed down to the cervical constriction of the tooth .