Lec. 11 Trauma/3

Reaction of the Tooth to Trauma

Traumatic dental injuries (TDIs) can produce a wide range of pulpal and periodontal responses. The outcome depends on the intensity of impact, stage of root development, and integrity of the neurovascular supply. Understanding these biological reactions is essential for prognosis, diagnosis, and appropriate management. Little relationship exists between the type of injury to the tooth and the reaction of the pulp and supporting tissues.

1) Pulpal Hyperemia

A transient increase in pulpal blood flow following trauma.

Pathophysiology

- Even mild trauma disrupts pulpal microcirculation → vascular congestion.
- Because the pulp lacks collateral circulation, even a temporary hyperemic state may result in ischemia and eventually necrosis.

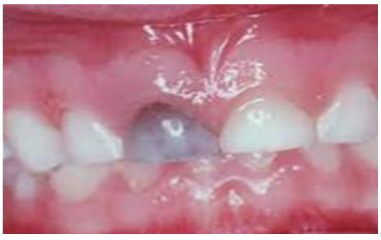
Clinical Signs

- Tooth may appear reddish when transillumination is used.
- Color change may persist for several weeks.
- Sensitivity to cold may be heightened.

Prognosis

- Persistent hyperemia with prolonged discoloration often indicates unfavorable prognosis, especially when associated with additional symptoms (e.g., loss of sensibility response).

2) Internal hemorrhage



- **Mechanism**
  Increased pulpal pressure →rupture of capillaries→RBC extravasation.
  Breakdown of hemoglobin pigments →discoloration.
- **Clinical Features**
  Color change appears 2–3 weeks after injury.
  Mild hemorrhage: color change often temporary or reversible.
  Severe hemorrhage: pigments enter dentinal tubules→long-term discoloration.

- **Vitality Considerations**

  Some pulp tissues recover.

  Dark-gray discoloration in primary teeth is strongly associated with pulp necrosis.
- **Important Note**

  Gray-black discoloration shortly after trauma = likely necrotic pulp.

  New discoloration months or years after trauma = necrosis until proven otherwise.
- ****Discoloration that becomes evident for the first time months or years after an accident, however, is evidence of a necrotic pulp.**

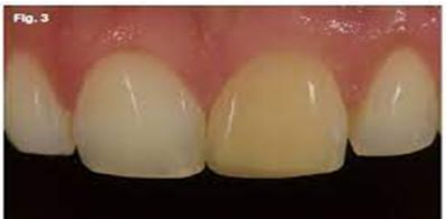
3) Calcific metamorphosis of dental pulp (Progressive Canal Calcification or Dystrophic Calcification)

Pathophysiology

- Trauma stimulates odontoblasts (or odontoblast-like cells) →excessive dentin deposition.
- Gradual obliteration of pulp chamber and canals (partial or complete).

Clinical Signs

- Crown appears yellowish, opaque.
- Pulp space may look fully obliterated on radiographs ("ghost canal"), though microscopic remnants remain.



Management

- Primary teeth: usually undergo normal root resorption.
- Permanent teeth: regarded as a potential focus of infection so it need monitor closely; treat only if:
  - symptoms develop,
  - periapical pathology appears,
  - pulp testing suggests necrosis (rare).

Long-Term Risk

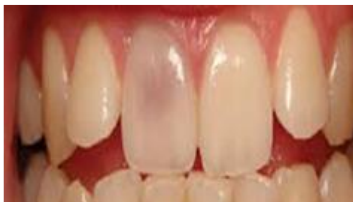
- A small percentage develop late pathology (years after trauma).

4. Internal resorption

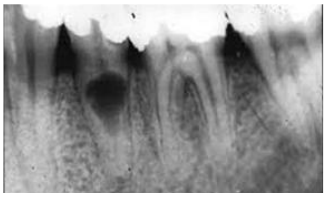
Etiology

- Odontoclastic activity triggered by pulpal inflammation.

Signs



- **Pink spot** visible coronally when resorption thins remaining enamel/dentin.
- This is because when the crown is affected, the vascular tissue of the pulp shines through the remaining thin shell of the tooth.
- Radiographically: well-circumscribed radiolucency within pulp chamber or canal.



Rate

- Can be slow or rapid, potentially perforating the root or crown. This perforation is referred as "perforating hyperplasia of the pulp."

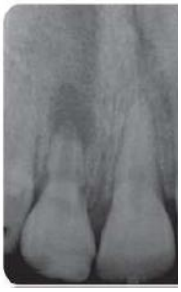
Management

- Immediate **endodontic treatment** before perforation.
- If perforation occurs, prognosis decreases.

5. Peripheral (External) root resorption

Cause

- Damage to periodontal ligament (PDL) during luxation (displacement) injuries, and the pulp may not be involved.
- Often occurs without direct pulpal involvement.



Features

- Progressive resorption of root surface.
- May stabilize in some cases; often continues until significant root structure is lost.

Prognosis

- It depends on severity of trauma and rate of resorption.

6.Pulpal necrosis

Mechanism

- Disruption of apical blood supply →ischemia →necrosis.
- Bacteria may enter via gingival sulcus or bloodstream or both even in intact crowns.

Clinical Features

- Many affected teeth remain asymptomatic initially.
- Radiographs may appear normal early on.
- Later: periapical radiolucency, pain, swelling.

Relationship to Injury Type

Risk of pulpal necrosis from lowest to highest:

Concussion → Subluxation → Extrusion → Lateral luxation → Intrusive luxation (highest).

Intrusive luxation carries near-100% risk of pulp necrosis and external resorption in immature teeth.

Prognostic Insight

- Teeth with crown fractures may have a better pulpal prognosis than teeth sustaining a strong blow without fracture, because energy dissipates through the fracture rather than all the energy's being absorbed by the tooth's supporting tissues. Thus, the periodontium and the pulp of the injured tooth sustain fewer traumas when the crown fractures

Management

- Endodontic treatment or extraction depending on:
  - extent of root development,
  - associated resorption,
  - long-term prognosis.

Note:

*In some cases, necrosis may not occur until several months after the injury. The tooth with a necrotic pulp should therefore be extracted or treated with endodontic procedures, whichever is indicated.

* A necrotic pulp in an anterior primary tooth may be successfully treated if no extensive root resorption or bone loss has occurred. The treatment technique is essentially the same as that for permanent teeth. However, trauma to the periapical tissues during canal instrumentation must be carefully avoided.

7. Ankylosis

Mechanism

- Injury to the PDL →inflammation →osteoclastic activity →loss of PDL →fusion of root with alveolar bone.

Clinical Signs

- "Submerged" appearance due to failure of ankylosed tooth to follow vertical growth of adjacent teeth.
- Percussion: high, metallic sound.
- Radiographically: loss of PDL space (interruption in the periodontal membrane and often the dentin may appear to be continuous with alveolar bone), replacement resorption appearance.

Management

- Primary teeth: extract if causing delayed/ectopic eruption of permanent successor.

Permanent teeth: if ankylosis occurs during growth, it often requires extraction or surgical repositioning (especially if the ankylosis occurs during the preteen or early teen years) due to loss of arch length.

Restoration of Fractured Teeth

To aid in the recovery of the pulp after the trauma, the restoration of a fractured tooth is as important as the emergency treatment designed. Several restorations have been advocated, and although the dentist has a wide choice of techniques and types of restorations, the following factors may affect the type of the restoration:

1. The circumstances surrounding the case often dictate the type of restoration for a given patient.
2. The prognosis of pulp healing,
3. The amount of tooth structure remaining,
4. The stage of eruption of the tooth and adjacent teeth,
5. The size of the dental pulp and degree of root closure,
6. The normalcy of the occlusion,
7. The wishes of the patient.

All of these factors must be considered in the selection of a temporary restoration, an intermediate restoration, or the permanent restoration. In the young patient, although it is often desirable to wait for continued eruption of the tooth or to determine the outcome of a vital pulp procedure, a delay of even a few weeks is often sufficient to allow the tipping of adjacent teeth, overeruption of opposing teeth, or other undesirable changes in the occlusion.

Notes

- Permanent filling in young children should not be done from the beginning (delayed), because:

  1) The dentinal tubules are widely open. So, any cut in the crown or cementation for the crown might affect the pulp because it produces irritation to the pulp.
  2) The full length of the clinical crown is not yet established.
  3) The pulp chamber of newly erupted tooth is wide and any cutting might get pulp exposure.
  4) The root of the tooth continues to develop 3-4 years after eruption.

Treatment of traumatized teeth usually divided into 3 stages:

1. Emergency treatment
2. Intermediate or semi-permanent treatment
3. Permanent treatment

Types of semi-permanent restorations:

1. Colloid crown
2. Chrom steal crown with window labeling
3. Pin with filling material
4. Cold cure acrylic temporary crown (good esthetic, keep it 4-6 weeks in the month)

In case of Class III fracture where fracture line below the level of root (with the present of sufficient amount of root structure), try to extend the root orthodontically and do R.C and make post and core.

Reaction of Permanent Tooth Buds to Injury

The dentist who provides emergency care for a child after an injury to the anterior primary teeth must be aware of the possibility of damage to the underlying developing permanent teeth. The close anatomic relationship between the apices of primary teeth and their developing permanent successors explains why injuries to primary teeth may involve the permanent dentition. The dentist

and the physician should also be aware of the possibility of trauma to permanent tooth buds from other unusual injuries so that parents may be informed of the possibility of defective permanent tooth development. Some injuries to the face and jaws may not appear to have caused any dental injuries initially, but the problem may be noticed several months or years later. The reaction could be hypocalcification, reparative dentine or dilacerations.

1) Hypocalcification and Hypoplasia

Gross malformed crown, small pigmented hypoplastic areas referred as (**Turner tooth**). Small hypoplastic defects may be restored by the resin-bonding technique.

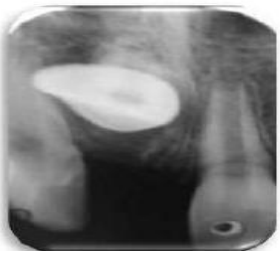
Note:

- **Hypoplasia** = defective matrix formation (thin enamel, pits), which is characterized by irregular thickness of enamel and presence of pits and grove in the enamel surface.
- **Hypocalcification** = normal enamel thickness but poor mineralization. It can be found in both primary and permanent dentition.

2) Reparative Dentine Production

This condition may occur in cases in which the injury to the developing permanent tooth is severe enough to remove the thin covering of developing enamel or cause destruction of the ameloblasts, the subjacent odontoblasts have been observed to produce a reparative type of dentin. The irregular dentin bridges the gap where there is no enamel covering to aid in protecting the pulp from further injury.

3) Dilaceration



Tooth with sharp bend in crown or root is known as dilacerated tooth.

Dilacerations is most commonly seen in maxillary permanent central incisors. Most common cause of dilacerations is trauma to the deciduous teeth (Occasionally occurs after the intrusion or displacement of an anterior primary tooth. The developed portion of the tooth is twisted or bent on itself, and in this new position growth of the tooth progresses. Germination may appear in the part of the tooth formed after the injury).

Dilacerated tooth usually fails to erupt but may sometimes erupt into an abnormal position and can cause displacement of adjacent teeth.

**Crown of a permanent tooth or a portion of it develops at an acute angle to the remainder of the tooth ---------->

Treatment

1. Unerupted dilacerated tooth usually require surgical extraction.
2. Erupted teeth with root dilacerations should be extracted, if they are in abnormal position. Since, it is difficult to move by orthodontic force.
3. Following extraction, extracted space should be maintained by prosthesis or closed by orthodontic treatment.