ABNORMALITIES OF TEETH

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Dental anomalies

defects in tooth developmentenvironmental alterations of teeth

causes - hereditary, systemic, traumatic or local factors (eg. drug) **Developmental alterations**

Idiopathic or hereditary conditions

Alterations in the number of teeth
Alterations in the size of teeth
Alterations in the shape of teeth
Alterations in the structure of teeth

Environmental alterations

Influenced by environmental forces

Developmental tooth defects
Postdevelopmental structure loss
Discoloration of teeth
Localized disturbances in eruption

Environmental effects on tooth structure

- Turner's hypoplasia
- Hypoplasia due to antineoplastic therapy
- Dental fluorosis
- Syphilitic hypoplasia

Postdevelopmental loss of structure

Tooth wear – attrition

 abrasion
 erosion
 abfraction

 Internal and external resorption

Environmental discoloration

- Extrinsic stains
- Intrinsic stains

Localized disturbances in eruption

- Premature eruption
- Retarded eruption
- Premature loss
- Deciduous teeth persistence
- Primary impaction
- Ankylosis +/- reimpaction

Histology of enamel

Formed by ameloblasts 95% mineralized anorganic material 5% organic 98% calcified Consists of enamel rods or prisms Yellow to gravish white

Strong, but prone to splits and chips

 Hardest structure in body

Non-reparative

Subject to caries

Subject to wear

Environmental effects on tooth structure

Ameloblasts in the developing tooth germ highly sensitive to external forces \rightarrow multiple posibilities of enamel abnormalities. \blacksquare No remodeling \rightarrow permanent defects 3 stages: matrix formation mineralization maturation

Factors associated with enamel defects

Systemic

- Birth-related trauma (hypoxia, premature b., prolonged labor)
- Chemicals (chemotherapy, fluoride, Pb, TTC, thalidomide)
- Chromosomal abnormalities (trisomy 21)
- Infections (CMV, varicella, rubella, syphilis, ..)
- Inherited diseases (phenylketonuria, osseous dysplasia, ..)
- Malnutrition (generalized, vit. A, D def.)
- Metabolic diseases (celiac d., hypoparathyroidism, renal d.)
- Neurologic disorders (mental retardation,..)

Factors associated with enamel defects

Local

- Local acute mechanic trauma (falls, traffic accidents, gunshot, mechanical ventilation, ritual mutilation,..)
- Electric burn
- Irradiation
- Local infection (periapical etc.)







Postnatal

Prenatal

Vertical transmission of infection, i.e. rubella, syphilis
Maternal systemic disease

<u>Neonatal</u>

haemolytic disease of the newborn
hypocalcaemia
premature birth/prolonged labour (ischaemia)

Postnatal

- severe childhood infections, esp. viral exanthematic diseases
- chronic diseases in childhood, e.g. congenital heart disease, gastrointestinal and endocrine diseases
- nutritional deficiency, e.g. vitamin D
- cancer chemotherapy
- excess fluoride ions
- trauma

Enamel defects

different causes may result in similar defects
possible timing of cause in deciduous enamel
rough estimate in permanent teeth
very common (≥ 50%)

Enamel defects patterns

Localized x multifocal (number of teeth affected) Partial x global (amount of surface)

- Hypoplasia (pits, grooves, parts missing)
 Diffuse opacities (variations of translucence, white)
- Demarcated opacities (decreased translucence, sharp demarcation, white → brown)



mild type

smooth-surface enamel white, opaque spots, brown after eruption

symmetrical

- horizontal grooves in the enamel surface
 pits in the enamel surface
- general reduction in the thickness of the whole enamel





Turner's hypoplasia (Turner's tooth)

- a local hypoplastic or hypomineralized defect in crown of a permanent tooth

 extension of a periapical inflammatory disease (infection) or mechanical trauma from overlying deciduous tooth, disturbing the ameloblasts of the permanent tooth bud

most common in lower premolars

Localized enamel hypoplasia (Turner's teeth) local or extensive







Turner teeth

- yellowish or brownish pigmentation of the enamel
- pits and irregularity of the surface

smaller crown than normal

Turner tooth







Hypoplasia due to antineoplastic therapy

- childhood cancer
- chemo and/or radiotherapy
- enamel and dentin defects
- hypodontia, microdontia, enamel hypoplasia, ...

Dental fluorosis

Fluorosis (mottled enamel)

- Iuoride in drinking water, toothpaste, supplements hypomineralization, event. enamel hypoplasia
- mostly discoloration, true hypoplasia uncommon
- paper-white patches

own

- permanent teeth (Placenta barrier normally resists fluoride, fluorosis seldom in deciduous dentition.)
- hydroxyapatite
 lcium fluorapatite
- matrix normal
- fluorosis x caries resistance

Dental fluorosis

- fluoride opacities symmetrically around the arch
- faint white flecking of the enamel, white patches or striations
- in severe cases may be associated with loss of the normal tooth form
- the deciduous teeth may be involved in severe cases and in areas of endemic fluorosis
- highly acid-resistant, rapid loss by abrasion and attrition

Fluorosis





bleachingcomposite resin

Congenital syphilis

Congenital syphilis - Hutchinson
later fetal infection, now very rare
dental follicle infection by *T. pallidum*permanent teeth
upper 1. I (*Hutchinson's incisors*) – barrel-shaped fissure on incisal edge

1. M (*mulberry, Moon's molar*) – pitted + bumpy occlusal surface

Congenital syphilis

 30 % of infected fetuses develop dental hypoplasia

Congenital syphilis

Hutchinson's incisors







copy

Postdevelopmental loss of structure

Non-bacterial (x caries) Non-traumatic (x fracture) ■ Tooth wear (enamel) – attrition abrasion erosion abfraction Internal and external resorption (dentin, cement)

Habitual disorders

- Attrition: wearing away of tooth structure during mastication (chewing) through tooth-to-tooth contact
- Incisal, occlusal and interproximal surfaces (contact points)
- Crown shortened, reduction of pulp chamber, canals
- Physiological (contact points and areas, abrasive foods, exposion of dentine → accelerated attrition)
- Dentin sensitivity rare due to slow loss + secondary dentin formation

Attrition



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Habitual disorders

Pathological attrition

- abnormal occlusion (prolongated contact, developmental, acquired – extraction)
- bruxism; long-term use of intraoral abrasives (tobacco or betel chewing)
- abnormal tooth structure (poor quality or absent enamel – fluorosis, amelogenesis or dentinogenesis imperfecta)



Habitual disorders

- Bruxism: an oral habit consisting of involuntary grinding and clenching of the teeth in movements other than chewing.
- Usually performed during sleep, commonly associated with stress or tension.

Bruxism



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Habitual disorders

- Abrasion is the abnormal wearing away of tooth structure caused by a repetitive mechanical habit.
- external cause (friction of a foreign body, abrasive material, pressure)
- improper toothbrushing common, on exposed roots, maxillary > mandibular, anterior > molars, grooves + polished dentine
 gripping objects with teeth babitual (pipe)
- gripping objects with teeth habitual (pipe, pencils), occupational

Toothbrushing injury

- V-shaped groove in cervical area
- Sensitive
- Maxillary premolars >canines
 > incisors
- R-L (mostly) defect at cervical level, well-defined semilunar shapes



Toothbrushing abrasion



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Habitual disorders

Erosion – loss of tooth structure by a chemical process (acid) not associated with bacterial interaction. Possible combination (↑ attrition, abrasion).

- Dietary carbonated soft drinks, fruit juices; shallow polished concavities
- Medication aspirin, vit. C chewing
- Stomach regurgitation involuntary (gastric reflux, pregnancy), voluntary (repeated vomiting)

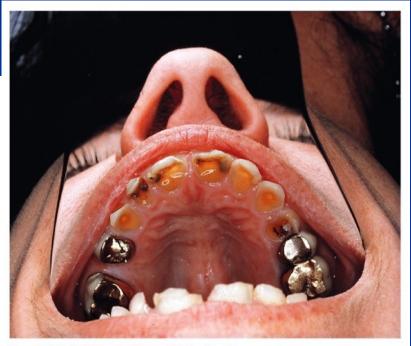
Habitual disorders

- Crown shortened, reduction of pulp chamber, canals
- dentin reactive changes, incl. tertiary reactionary dentine formation
- possible hypersensitive dentin if rapid course

Erosion caused by bulimia.



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Habitual disorders

- Abfraction due to the repeated tooth flexure (occlusion stress) → disruption of enamel crystals → cracked enamel → loss by erosion, abrasion
- wedge-shaped defect on cervical area of the teeth, facial surface
- single tooth often affected

Habitual disorders

commonly multifactorial etiology + resultfunctional, dental sensitivity, aesthetic problems

Histology of dentin

- Formed by odontoblasts
- 70% anorganic matter
- 30% organic matter
- Makes up bulk of tooth
- Dentinal tubules
- Not as hard as enamel
- Somewhat elastic

Pale yellow

- Somewhat transparent
- More radiolucent than enamel
- Can repair itself

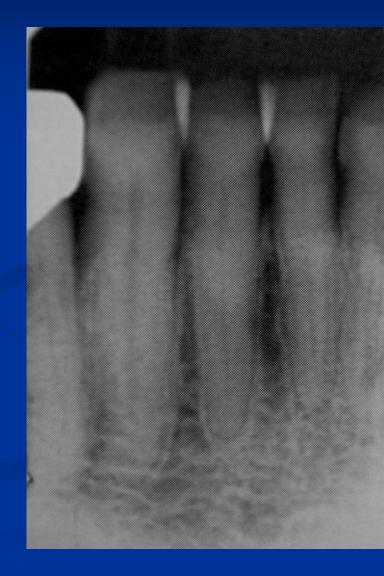
Histology of cementum

- Formed by cementoblasts
- Covering of root
 50% to 55% anorganic material
- 45% organic
- Primary cementum Secondary cementum

 Anchors tooth to socket via periodontal ligaments

Secondary dentin

- Dentin deposited in pulp chamber after primary dentin formed completely
- Normal aging process
- tertiary dentin: pathologic condition after chronic trauma
- Reduction in size of pulp chamber and canals
- Begins in the region adjacent to source of stimuli and alters normal shape of chamber



Internal and external resorption
resorption of dentin or cementum
internal surface – cells in the pulp
external surface – cells in the periodontal ligament

- Internal resorption macrophages (dentinoclasts) on pulpal surface, vital pulp necessary
- loss of odontoblasts
- asociated with pulpitis, physical trauma
- **rare** idiopathic
- less common than external resorption
- pulp tissue visible through enamel pink spot

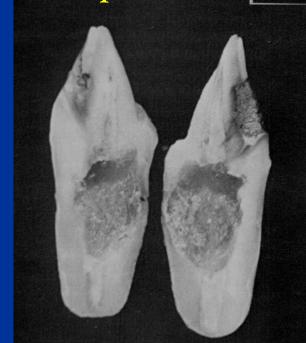
Internal resorption

- 2 main patterns:
- inflammatory resorption: replacement of dentin by granulation tissue, in pulpitis
 metaplastic resorption: replacement by bone or cementum-like bone

Internal resorption

Within the pulp chamber or canal, involves resorption of surrounding dentin, results in enlarged pulp space ■ M>F, commonly begins during 3rd-5th decade Radiographs reveal symptomless early lesions of IR Radiolucent, round, oval, or elongated within root or crown and continuous with pulp chamber or canal Sharply defined and smooth or slightly scalloped \rightarrow irregular widening of the pulp chamber or canal Metaplastic bone may lead to partial obliteration of the canal

Internal resorption







External resorption

- from root surface
- variable individual susceptibility to external resorption – most important factor
- extremely common, in 10% serious
- variable radiolucency (moth-eaten)
- resorption by multinucleated dentinoclasts, inflammatory reaction + woven bone, may lead to ankylosis

External resorption

- inflammatory res. periapical inflammation, root res., layer of granulation tissue (later fibrotic), layer of woven bone
- pressure/mechanical res. ? aseptic necrosis → repair
- idiopathic burrowing from root surface into dentine → granulation tissue → bone (event. ankylosis); invasive cervical resorption

Apical ER:

-blunting with normal bone and lamina dura -root shortening, except due to periapical inflammatory lesions

canal is visible and abnormal wide at apex

Lateral root surface ER:

-presence of an unerupted adjacent tooth

External resorption risk factors

- cysts
 - periodontal, keratocysts, pressure notching
- dental trauma
- excessive external forces (mechanical, occlusal)
- therapy (orthodontic, bleaching, teeth reimplantation, ...)
- local diseases (periradicular inflammation, herpes zoster, Paget's bone disease, tumors...
- generalized disorders (hormonal imbalances)idiopathic



Lateral root surface ER

Hypercementosis

Cementum hyperplasia

- cellular cementum deposition in the region of the root apex or on the cementum surface
- postinflammatorry changes, Paget disease, etc.
- normal during aging
- roundish apex thickening, possible problems during extraction

Teeth discoloration

extrinsic – surface deposits bacterial stain iron, other metals tobacco, betel food + beverages gingival haemorrhage restorative materials medication

Teeth discoloration

Intrinsic:

 changes in the structure or thickness (amelo-, dentinogenesis imperfecta, developmental enamel hypoplasia, caries)

diffusion of pigments after formation of tissues
 1 in preexisting enamel or dentin changes (root filling material, pulp necrosis + haemorrhage)

Teeth discoloration

Intrinsic:

pigment incorporation during formation of enamel/dentin

- congenital hyperbilirubinemia (greenish)
- congenital porphyria (red-brown, UV red fluorescence)

TTC pigmentation (yellow dentin bands, UV yellow, later brown)

TTC pigmentation





Pigment disorders therapy

composite resinbleaching



Composite resin

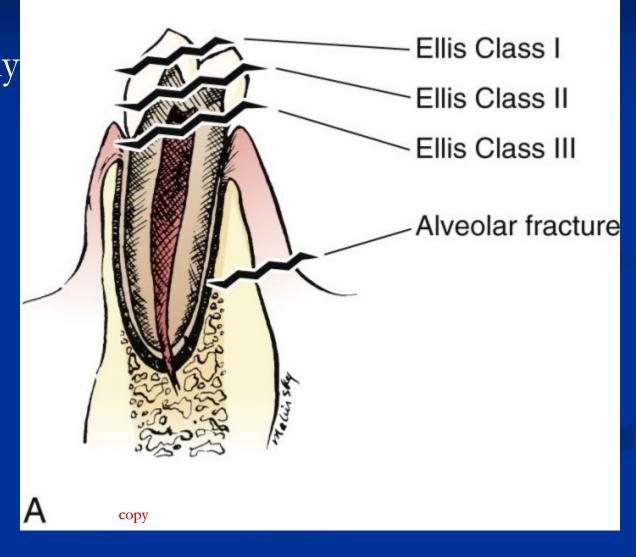


Tooth trauma

- Maxillary central incisors 70-80% of all fractured teeth
- Complications: failure to complete eruption, color change of the tooth, abscess, loss of space in the dental arch, ankylosis, abnormal exfoliation, root resorption.

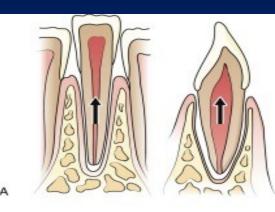
Tooth fracture

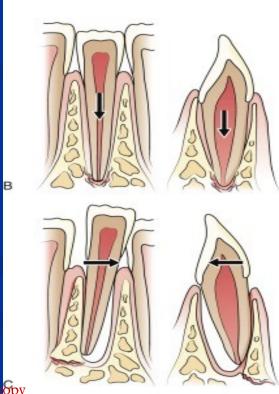
■ I.- enamel, usually no complication ■ II. – enamel+dentin: risk of pulp necrosis ■ III. – into pulp, 10-30% necrosis



Tooth luxation

- A. extrusive luxation partially out of socket
- B. intrusive luxation pulp compression, bone crush
- C. lateral luxation commonly + alveolar bone fracture
- Complete luxation (complete avulsion) – entire tooth out of socket





Tooth trauma - intrusion

The missing tooth could be lost, fully intruded, aspirated or swallowed.

■ upper tooth into maxillary sinus → recurrent sinusitis

into the nasal cavity

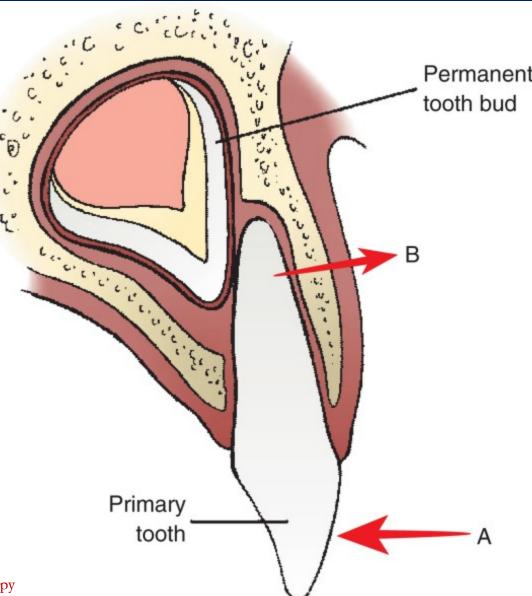
 → infection or
 bleeding

 aspiration into the airway



Tooth trauma

Deciduous tooth trauma: typical direction of force in a forward fall (A), the apex of the deciduous tooth levered away from the developing tooth bud (B).



Root fracture

- Multiple factors affecting healing: location, degree, fragment position and mobility
- Sterile x infected
- Sterile: similar to bone fracture healing organisation of haematoma by granulation tissue
 maturation + calcification.
- Malposition: fragments rounded, covered by cementum, more or less separated, gaps filled by fibrotic tissue
- Infected: abscess, gangrene