Pathology of teeth
Developmental disorders

Changes of numbers, size and shape

• Hypodontia (dif.dg retention!) – small conically shaped teeth
• Anodoncia
• Supernumerary teeth (cleft palate, cleido cranial dysplasia)
• Variation in dates of eruption (race, socio economic environment, delayed eruption – Down sy, endocrine def.)
• Mikrodontia, macrodontia (inherited, STH, not related to metabolic factors)
• Variation in cusp and root (extra root, extra cusp)
• Fused teeth
Resorption of teeth

- deciduous teeth – physiological process
- permanent teeth – always pathological
- sign of pathological process in the vicinity of the tooth (cyst, tumor, etc.)
- idiopathic resorption (pink spot) – dentine is resorbed from within the pulp – localized process – opening of pulp cavity (infection)
- idiopathic peripheral resorption – rare, usually multiple teeth – resorption of roots near apex
Developmental disorders

Enamel and dentine formation disturbances
Hypoplasia (enamel imperfect, but hard)
Hypocalcification (soft and eroded enamel)

Acquired conditions:
- local infection – pitted enamel of permanent teeth in association with a deciduous teeth
- generalised infections and trophic disturbances – (bands around teeth)
- generalised metabolic diseases
- endocrine disorders (hypoparathyroidism)
- fluorosis
Enamel and dentine formation disturbances

Acquired conditions:

Prenatal

- infections (rubella, syphilis, gross maternal metabolic disturbances)

Neonatal

- hypocalcemia, severe neonatal hypoxia
- Severe neonatal hyperbilirubinemia

Infantile

- malnutrition, GIT diseases (coeliac disease infections, endocrine disorders, fluorosis)
Developmental disorders

Enamel and dentine formation disturbances

• Developmental conditions
  – Amelogenisis imperfecta
  – Hypoplastic type (matrix defect)
  – Hypocalcification
  – Dentinogenisis imperfecta
  – Osteogenisis imperfecta
Developmental disorders

Discoloration of the teeth

• widespread coloration – abnormal blood pigment
  – Infantile jaundice- blue-green color of deciduous teeth – laying down the pigment in the immediate postnatal dentine zone
  – Rarely in haemolytic anemia by Rh incompatibility
  – Porphyria
• Tetracyclin – yellow, brown, irreversible, altered mineralisation
• Fluorids – grey spots, erosions
• Betel chewing – red color
Resorption of tooth tissue

External
• Periapical inflammation
• Tumors – mechanical usuration (pressure)
• Cysts (odontogenis, keratocyt)
• Reimplantace a transplanace
• Unerupted teeth – resorption of neighbour teeth

Internal (idiopathic) – internal granuloma
• Pulp hyperplasia
Regressive changes

**Abrasion** – loss of dental hard tissue by non-physiological mechanisms
- Necks and chewing surfaces
- Shape „V“ defect
- Sharp demarcation

**Erosion** – loss of dental hard tissue by chemical proces which does not involve bacteria
- Not sharp margins
- Local acidosis- citrus juice, fruit, acidid beverages, mecidines of low pH
- Self induced vomiting (bulimia nervosa)
Regressive changes

Cementum hyperplasia – hypercementosis

- Cementum deposits around an apex (associated with inflammation)
- Complication in extraction

Cementicle – lesion in periodontium

Tooth ankylosis – bone bridge between a tooth and alveolar bone
Bacterial plaque

- organic matrix (polysaccharides) + bacteria
- biofilm enables much higher concentrations of acids than those in saliva
- resistant to immunological defences
- plaque visible after 12-24 hours
- translucent film with matt surface
- staining with disclosing agent
- forms rapidly and abundantly in high-sucrose diet
- resists friction during chewing – removal only by tooth brushing (persistence in pits and fissures!)
- plaque minerals – calcium, phosphorus, fluorides
Plaque

- acid production in plaque (lactic acid)
- sucrose diffusion into plaque (sweet drinks x caramel) – repeated small doses more cariogenic!
- fermentation into acids - in minutes (!)
- lasts 20 min. after washing-out sugar,
- returns to normal after 60 min.
Diet and plaque

• sucrrose rich diet – main source of plaque (sweets, snack-bars)
• reverse association between malnutrition and caries
• baby bottle caries
• newly erupted teeth more susceptible – progressively
• increases resistance (maturation – deposition of minerals from saliva)
Baby bottle syndrome
Role of saliva

- complex secretion, multiple factors – buffering power (bicarbonate content)
- salivary flow – clearing of cariogenic foods. Xerostomia!
- immune deficiencies do not have significant role on dental caries
Dental caries

• most common disease
• major cause of loss of teeth

Multifactorial disease:
• Tooth plaque
• Food – sugar content (sacharosis – increase adhesion of plaque – progressive bacterial damage)
• Predisposing factors (sex, age, systemic disease, chewing)
Other factors

• Reduced saliva is associated with increased caries since the buffering capability of saliva is not present to counterbalance the acidic environment created by certain foods.

  salivary glands diseases:
  • Sjögren's syndrome
  • diabetes mellitus,
  • diabetes insipidus
  • and sarcoidosis
  • Medications, such as antihistamines and antidepressants, stimulants, most notoriously methylamphetamine – Tetrahydrocannabinol, the active chemical substance in cannabis.
  • Radiation therapy of the head and neck may also damage the cells in salivary glands, increasing the likelihood of caries formation.
Other factors

- Tobacco may also increase the risk for caries formation. Some brands of smokeless tobacco contain high sugar content, increasing susceptibility to caries. Tobacco use is a significant risk factor for periodontal disease – risk of root-surface caries.
- Intrauterine and neonatal lead exposure promote tooth decay.
- Cadmium exposure mimics the calcium ion and therefore exposure may promote tooth decay.
Dental caries

• Long history – over a million years ago, hominids (Australopithecus) suffered from cavities.
• The largest increases in the prevalence of caries have been associated with dietary changes.
• The increase of caries during the neolithic period may be attributed to the increased consumption of plant foods containing carbohydrates.
• The beginning of rice cultivation in South Asia is also believed to have caused an increase in caries.
• A Sumerian text from 5000 BC describes a "tooth worm" as the cause of caries.
Dental caries

• a bacterial disease
• decalcification caused by acid production by plaque bacteria
• not present in germ-free animals
• Streptococcus mutans, sobrinus, salivarius, mitior, sanguis,
• Lactobacilli
• different ability of different bacteria to:
  – attach to different types of tissue
  – produce different polysacharides
  – ferment sugars (sucrose)
  – produce acids (S. mutans) pH<5
• S. mutans stores sucrose intracellularly (metabolic reserve)
• omitting of sucrose in diet – disappearance of S. mutans
• new supply – recolonization of plaque
• stagnation areas (pits, fissures, interstitially)
• **Streptococcus mutans**
• ability to polymerise sucrose into sticky dextran-like polysaccharides (glucans) – strongly related to cariogenicity
• adheres to tooth surface
• produces glycans – matrix of the plaque
• plaque adheres to teeth and persists there
• produces acid
• survives at the low pH (4.2)

• **Lactobacilli** – less cariogenic (fissure caries -plaque formation less important); contribute to tooth destruction later, after the process has been started
The stages of tooth decay

1. Healthy tooth with plaque
2. Decay in enamel
3. Decay in dentin
4. Decay in pulp
Dental caries

1) Fissural caries
   • Chewing surfaces (occlusal) + bucal and lingual of molars and premolars
   • Lingual surfaces of incisors

2) Smooth surface caries
   • Contact surfaces – approximal caries
   • Dental necks

3) Cememtal caries (root ca – elderly people with periodontitis

4) Dentinal caries
Dental caries

Enamel

• **Initial stage – reversible**
  ✓ 4 zones
  ✓ Grossly – superficial erosion

• **Progressive stage – irreversible**
  ✓ Proteolysis of organic parts of prizmatic connections
  ✓ Fissures and creckles
Pit and fissure caries
• same changes, different shape
• ring around the pit
Dental caries

Clinical course
- Primary caries
- Secondary
- Acute
- Chronic
- Arrested
Enamel caries

Early lesion
- white opaque spots, chalky appearance
- hard to probe
- changes seen best in polarized light - demineralization -
  - conical in shape - apex towards dentine

4 zones - (from inside out):
• translucent z. - loss of 1% of minerals -demineralization
• dark z. - remineralization
• body of the lesion - loss of 5-25%
• surface z. - lost in cavitation - very important one - remineralized (salts from plaque or by ions escaping from deeper structures)
Enamel caries

- enamel – usual site of initial lesions
- calcium apatite, minute organic content barrier to bacterial attack
- once enamel defence is breached – dentin represents a poor barrier
- prevention - to stop caries at the enamel level, increasing the resistance of enamel
- crystalline lattice of Ca apatite crystals – impermeable
- organic matrix – high water contents – permeable for H+ ions
- dynamic process – alternation of de- and remineralization
- progression slower in adults, fast in children
Enamel caries

4 phases

• early (submicroscopic)
• non-bacterial enamel crystal destruction
• cavity formation
• bacterial invasion of enamel
Cavity formation

- prisms disappear - formation of pathways large enough for bacteria to enter
- bacteria reach amelodentinal junction – spread laterally - enamel undermining
- pinhole lesion in enamel - large underlying cavity
- fragmentation of enamel on the surface – clinically obvious cavity
- bacterial attack of dentine enabled
Dentinal caries

• initial (non-bacterial) lesion - demineralization
• invasion of bacteria - role of Lactobacilli (later on mixed population)
• dentinal tubules - ideal pathway - colonization - spread
• demineralization - proteolysis
• distension of tubules - coalescence – liquefaction foci
• lateral spread
Protective reactions of dentine and pulp under caries
• odontoblast activity
• reactions are not specific (any trigger - attrition, erosion, abrasion, restorations)
• more prominent in slowly progressing caries
• formation of sclerotic dentine (tubular sclerosis)
• reactionary dentine - regular and irregular
• vulnerability - no barrier against pulpal infection
Arrested caries

- white spot can be arrested – remineralized from saliva
- dentine caries - destruction of enamel - dentine exposed to saliva
- use of fluorides and less cariogenic diet
- 50% of early interproximal lesions do not progress
Protective mechanisms

Effect of fluorides
- drinking water
- toothpaste
- mouth rinses
- tablets
- fluoride incorporated into the teeth during development
- reduction of enamel solubility
- favorization of mineralization
- possible reduction of acid production within plaque

Dietary changes
Xylitol (a sugar alcohol)

Oral hygiene
Dental caries, complications

- breakdown of enamel and dentine
- opening way of bacteria to infection of pulp (acute pulpitis)
- spread into periapical tissue (periodontitis)
- toothache
- periapical abscessus
- infection spreads into jaw – osteomyelitis – sepsis
- maxillary sinusitis
- odontogenic cyst (radicular)
Pulpitis

- most common cause of dental pain and loss of teeth in younger persons
- usually due to dentine caries
- other causes: open trauma (cavity preparation!), fracture of crown, cracked tooth syndrome, thermal or chemical irritation (protective layer under filling!)
- untreated pulpitis > necrosis (gangrene), spread of infection through root channel - periapical inflammation (periodontitis)
- Types:
  - acute X chronic
  - open X closed
  - focal X diffuse
Acute pulpitis

- clinically transient sensitivity to hot or cold
- later on - toothache becomes persistent
- pain is poorly localized (felt in other teeth)
- pressure on the irritated nerve
- pain producing substances released by inflammatory cells
Acute closed pulpitis

• initially hyperemia
• infiltration by inflammatory cells
• localized abscess or necrosis, formation of granulation tissue
• later - diffuse inflammation of the pulp, obliteration of blood flow - necrosis
Chronic closed pulpitis

- mononuclear cell infiltrate
- chronic abscess, wall made of granulation tissue
- pulp calcifications (dentine bridges)
Open pulpitis

- pulp exposed
- massive infection
Chronic hyperplastic pulpitis (pulp polyp)

- pulp replaced by granulation tissue
- polyp may become epithelialised by squamous cells
Periodontitis

- Acute X chronic
- Associated with:
  - Pulpitis
  - Trauma
Acute apical periodontitis

- Hyperemia and serous exsudation
- Suppuration and osteoclastic bone transformation,
- Previous pulpitis in history
- Escape of exsudate into periodontal tissues - extrudes tooth - bite falls more heavily

- 4 phases
  - Periodontal
  - Endoseal
  - Perisostal
  - Sumbucous
Acute apical periodontitis

• uncomfortable feeling - tender tooth - pain
• reddening of gingiva
• X-ray not too much helpful, penetration of overlying bone - swelling of surrounding soft tissues (upper canine - eyelids, face)
• swelling is due to oedema
• enlargement of regional lymph nodes
• complication - acute osteomyelitis,
• phlegmone of soft tissues (cellulitis)
Chronic apical periodontitis

- Secondary to acute periodontitis
- Primary chronic (more common)

**Forms**
- Granulomatous
- Granulomatous – progressive fistulation (mucoses, skin)
- Diffuse – degradation of alveolar bone
Chronic apical periodontitis

- low grade infection
- inadequately treated or silent acute infection
- non-vital tooth
- X-ray - "apical granuloma" - 5 mm
- chronic inflamm. infiltrate (lymphocytes, plasma, cells, macrophages) + granulation tissue
- osteolysis - formation of "cavity" - radiolucency
- no spontaneous healing (reservoir of infection in root canal)
- proliferation of epithelial rests of Malassez – cystic degeneration - formation of radicular cyst
- rarely formation of fistula (sinus tract to buccal gingiva)