(1) Permanent and deciduous dentitions

Deciduous teeth

The first set of teeth in the growth development of humans and other mammals. They form during the embryonic stage of development (6th week of intrauterine life) and mineraulte from the 5th month of pregnancy until after birth

The process of development starts at the midline and then spreads back into the posterior region.

There are (20) teeth which erupt from the age of (6) months → 25-33 months
2 years ~ 2.5 years

The process of shedding deciduous teeth and replacement by permanent teeth is called exfoliation

(eupcion (months)	central incisor 6 - 8	Lateral incisor 9 - 10	canine 16 - 20	1st molar 12 - 16	2nd molar 20 - 24
Shedding (years)	6 - 7	7 - 8	10 - 12	9 - 11	10 - 12

By the end of 1st year of life, there should be 8 teeth and by the end of 2nd year of life, there should be 16 teeth. And the whole deciduous dentition by the 30th month

- Dentitio precox: premature eruption by the end of 3rd month
- Dentitio tarda: delayed eruption after the 10th month

Permanent teeth

Some of the permanent teeth are formed up as early as during intrauterine life, others are formed after birth. Development of crowns lasts for up to 6 years. The first of the adult teeth to erupt are the permanent first molars that come through the gums at the back right behind the last milk molars. These first permanent molars are the most important teeth for correct development of an adult dentition.

Up to the age of 13 years, (28) of the 32 permanent teeth will appear. The full permanent dentition is completed much later: the 4 last adult teeth, one at the back of every arch, will appear around 21 years (wisdom teeth)
21 - 25 yo

Exfoliation (years)	central incisor	lateral incisor	canine	1st premolar	2nd premolar	1st molar	2nd molar	3rd molar
	7 - 8	8 - 9	10 - 12	10 - 11	11 - 12	6 - 7	12	13 - 25

② Changes in the oral cavity accompanying systemic diseases

Pathological changes affecting the periodontium may occur at milky temporary, mixed and permanent dentition. Various forms of gingivitis or gingivostomatitis are the most common diseases. Diffuse alteration of periodontal tissues that result in a premature loss of deciduous / permanent teeth. Progress of destructive changes is rapid.

A systemic / metabolic disease is usually the cause

Inflammation of periosteum

- Syphilis - syphilitic periostitis (non-gummous) - tenderness and swelling of bone + aching pain
- TB - oral granulomas
- HIV - leukoplakia, oral candidiasis, necrotising gingivitis, periodontitis, Kaposi's sarcoma, NH lymphoma
- ⊙ Sjögren's syndrome - lymphocytic inflammation of mucous glands
- Leukoplakia (keratosis) - precancerous epithelial dysplasia. white plaque (hyperkeratosis) that can't be scraped away
- Aphtous ulcers - crohn's and celiac disease, patients with fever
- Erythroplakia - red velvety patches of epithelial atrophy are pronounced dysplasia. Mainly in ♂ elderly on buccal mucosa / palate. Precancerous dysplasia associated with heavy tobacco use.
- Angular cheilitis / glossitis / stomatitis - in Fe-deficiency anemia, DM, Sjogren's
- vitamin B deficiency - burning sensation, specially on the tongue, angular cheilitis, recurrent aphtous stomatitis, chronic atrophic mucosal candidosis, atrophic glossit
- Iron deficiency - fungal infections, tongue redness, swelling, bores and pale tissue in the mouth
- vitamin C deficiency - bleeding gums, gingivitis

① Leukemia - ↓ blood platelets (petechia, bleeding from gums)

- suppressed WBC (infected tonsils, sores, opportunistic infections)

② Agranulocytosis - sore throat, gingival bleeding, ↑ saliva, halitosis, osteoporosis, destruction of periodontal lig.

③ Polycythaemia vera - symptoms are due to ↑ blood thickness + clotting

④ systemic: + diabetes + congenital ♡ (cyanosis → blue + violet mucosa)

③ Classification of orthodontic anomalies

Congenital anomalies can be either hereditary or appear during intrauterine development as a result of harmful influences. These influences can be toxic chemicals, physical, viral infections (rubeola). Hereditary disorders include anomalies in #, shape and position of the teeth. A primary position of the tooth germ, the shape + size of jawbones are genetically determined as well.

Acquired anomalies appear after a child's birth by effects of internal and external influences. Internal causes include metabolic disorders, rachitis, endocrine disorders, etc.. External causes are for example: sucking fingers, putting foreign objects inside the mouth, biting of lips, tongue, cheeks, breathing by mouth. Dental decays and premature losses of teeth may lead to formation of orthodontic anomalies, too.

Odontic anomalies

→ Anomalies of teeth positions

Teeth inclination: tilting of a tooth along its longitudinal axis. Its forms are mesial, vestibular, oral and distal. Vestibular inclination at the front area is often called protrusion whereas an oral tilt is called retrusion.

Teeth rotation: turning of a tooth along its longitudinal axis. It's frequent for single root teeth (incisors, canine teeth) if there's not enough room at the dental arch.

Supraocclusion: a tooth overhangs the occlusal plane

Infraocclusion: a tooth doesn't reach the occlusal plane

Shift: a tooth changed its position while maintaining its longitudinal axis (electric

Transposition ~ 2 teeth exchange their locations

Dystopia ~ a tooth out of the dental arch at vestibular or oral side

Retention ~ a tooth did not cut during a period of physiological teething. The most often cause of retention is horizontal position of the germ or construction of the dental arch.

→ Anomalies in tooth (size): microdontia, macrodontia

→ Anomalies in teeth (number): hyperdontia, hypodontia, anodontia

→ Anomalies in teeth (shape): dilaceration (angulation of the root), peg-shaped (conical),

→ Anomalies of dental arches relationships

These anomalies can be evaluated at sagittal, transversal or vertical directions

At a sagittal plane, the lower dental arch can be positioned by 3 ways relative to the upper arch
↳ normocclusion - Angle class I
↳ distocclusion - Angle class II (1)
↳ mesiocclusion - Angle class III (2)

(1.) Distal shift of the lower jaw. First lower molar is shifted relatively to the upper molar distally. overbite

(2). Mesial shift of the lower jaw (toward the middle). The first lower molar is shifted mesially relative to the upper molar. Commonly accompanied by reverse bite

At a vertical plane, mutual position of jaws can be hindered by a deep or an open bite. A deep bite is such a bearing when upper incisor teeth overlap by more than 2/3 of the labial surface of lower incisor teeth. (sorriso arburaquintos / dentes de chuche)
The open bite occurs when a vertical gap appears between groups of 2 or + adjacent teeth

→ Disorders of the facial skeleton structure and growth

Some of the dental arches mutual position anomalies are determined by deviations in size, position and bearing of jawbones. They are normally hereditary although they may be caused by external factors as well.

- **Prognathia maxillaris**: characterized by an excessive growth of the upper jaw in forward direction while the lower jaw has a normal shape and size. The upper frontal teeth don't touch their lower antagonists and protrude out the mouth, quite often
- **Progenia mandibularis**: large lower jaw while the upper jaw's size is normal. ↑ occlusion + enlarged lower jaw's body, resulting in the horizontal overlap. Normally corrected after growth of the jaws is completed, by surgery
- **Pseudoprogenia**: small upper jaw while lower jaw's size is at normal range. Accompanied by reverse bite
- **Microgenia**: small lower jaw, accompanied by distocclusion. Chin projection is absent and the chin changes over to the neck's upper pt. almost imperceptibly. Nose + upper lip protrude from face profile
- **Laterogenia**: caused by an ↑ or ↓ in size of one half of the lower jaw. It occurs w/ face asymmetry

④ Prevention of the dental caries

Caries prevention is the most important task of stomatology. It should begin in childhood as at this time it's possible to influence the development of dental tiss and to learn how to care for the teeth. 3 main approaches are possible:

→ Removal of bacterial plaque by physical and chemical means. Regular tooth br shing in the morning and evening after the last meal with toothpast conta ning fluoride, dental flossing and use of interdental brushes

→ Dietary advice depends on reduction of the microbial substrate and the fre- quency of consumption by confining sugar to mealtimes. It's also importa to maintain vitamin, mineral and albumin nourishment. food supporting sali excretion and selfcleaning of the teeth, like coarse grain food, vegetables

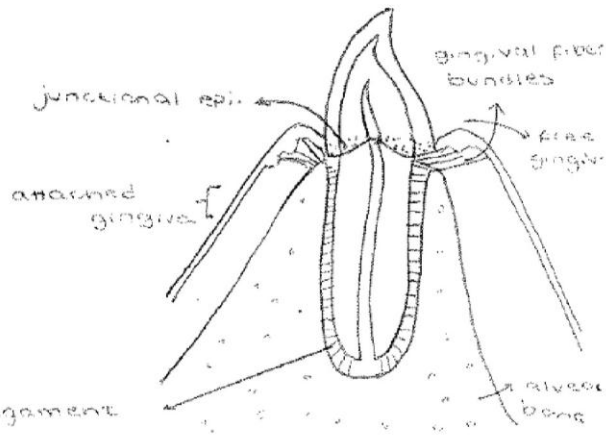
→ Tooth strenghtening by means of fluoride or fissure sealing. Fluoride may b administered to the organism → systemically by fluoridation of drinking water by fluoridation of salt or milk by means of NaF tablets

→ locally by application to the surface teeth i the form of toothpast, gels, solution, chewin gum

⑤ Periodontium

It is a complex of tissues connected with the tooth either functionally or ana - tomically. It's a functional system of # tissues holding and supporting the tooth including the cementum, periodontal ligament, alveolar bone and gingiva

Anatomically, the term is restricted to the tissues interposed between the tooth and its bony socket



⑥ Focal infection

Region of localized infection causes pathological spread in remote organs.

⊙ can lead to: endocarditis, myocarditis, phlebitis, subfebrile status, rheumatic disease, nephritis, migraine, eczeme

⊙ caused by: gangrenous teeth, chronic pulpitis, sirmoitis, dental cyst, retained teeth, periodontal abcesses - ATB prophylaxis - amoxicilin, erythromicin

⑦ Periodontal diseases

Periodontal diseases are diseases affecting either a part of the periodontium or the periodontium as a whole.

Etiological factors of periodontal diseases, are local and general. The most comm diseases of the periodontium are represented by inflammatory alterations in the gingiva (or the whole periodontium) induced by local microbial infection.

Dental microbial plaque represents the source of this infection. It's a soft, structured yellow-grayish substance adhering to the teeth which may be removed mechanically and other means. The plaque is composed of microorganisms and their products which subsist in the macromolecular matrix of bacterial and salivary origin.

→ **Classification of the periodontal diseases**

- Gingival diseases
- Periodontitis
- Gingival recession (ușor)

Gingival diseases

Plaque induced gingivitis is caused by an increased amount of microorganisms, specially anaerobic GR rods and oral spirochets. The inflammed gingva is red, sensitive and oedematous. The gingiva bleeds easily on various impulses and even spontaneously. worst in puberty, pregnancy, oral contraception

Acute necrotising ulcerative gingivitis is caused by a predominance of anaerobic spirochets, bacteroides and oral fusiform bacteria in the plaque. Manifests as progressive necrotising destruction of the interdental papilla, sometimes even of the whole gingival margin. Painful, expressive inflammation, accompanied by odor. It affects mostly young people between 17-24 yo with poor oral hygiene.

Therapy: debridement, thourough rinses with oxidants (H_2O_2) and oral hygene instruction. In more severe cases, systemic penicillin / nitroimidazole antimicrobial drug may be used as an adjunctive therapy.

without treatment, it may progress and involve deeper periodontal tissues (ulcerative gingivoperiodontitis).

Gingival hyperplasia in acute leukemia, developed from storing of leukemic infiltrate in the gingiva. It may also be a result of drug effect like phenytoin, cyclosporin A and Ca^{2+} channel blockers.

Periodontitis → necrosis → gangrene → reach periosteum → fracture → severe pain

Inflammatory disease of the tooth-supporting apparatus. It usually develops from a pre-existing plaque elicited gingivitis.

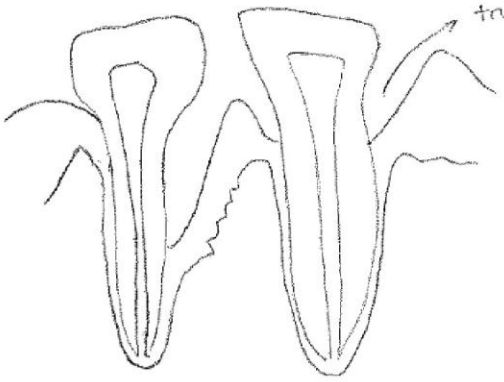
Symptoms: inflammatory resorption of alveolar bone, loss of the periodontal fiber bundles and necrosis of the cementum on the root surface contributing to the formation of the true periodontal pocket which is the most important clinical symptom of periodontitis.

⇓

crevicular space between the gingva and a part of the dental root not covered with the bone of the socket

the plaque penetrates into this space where it multiplies and causes further progression of the disease.

Periodontitis may be accompanied by further symptoms: tooth mobility, gingival shrinkage, spontaneous elimination of teeth, acute painful states like abscess and retrograde pulpitis. The earlier the manifestation, the more aggressive



- Adult periodontitis: slowly progressing from untreated plaque induced gingivitis in adults
- Rapidly progressive periodontitis: in persons between 19-30 y with a rapid clinical course
- Localized juvenile periodontitis: rapidly progressing occuring during the period of puberty, affecting the periodontium of the earliest erupting teeth
- Pre-pubertal periodontitis: very rapidly progressing periodontitis both in deciduous and permanent teeth

Gingival recession

Characterized by an apical regression of the periodontium without the formation of the pockets. The gums are pale, inflammation-free. The margin of the alveolar process slowly decreases but the bone maintains a normal structure

It doesn't proceed to the loss of teeth if the oral hygiene is sufficient.

Primary causes: facial plate of the bone overlying the root is supposed to be insufficiently developed

Secondary causes: traumatization of gingiva by horizontal brushing can cause gingival recession

(8) Hygiene of the oral cavity

Tooth brush: short, fitted with bundles of plastic fibers rounded at their ends (≈ 3 months)

Tooth paste: slightly acidic but not too abrasive

Dental floss: clean interdental spaces and sulcus.

Gum massages: ↑ blood supply and metabolism, support keratosis, remove plaque, ↓ edema and prevent formation of C.T.

Roll and sweap method 45° ∠ with gingiva, 2x daily for 2 minutes.

(9) Dental treatment of patients requiring a special care

- Allergic reactions
- cardiovascular complications
- Bleeding
- ↑ susceptibility to infections
- impaired wound healing
- attack states
- oncologic patients
- Hepatitis B, C, HIV infections

Detailed anamnesis: hospitalization
pregnancy
drug usage

Allergic reactions - local anaesthetics
- Atb's
- disinfections
- filling prosthetic materials
- latex of surgical gloves

Check for necessity of anti-allergic prophylaxis - antihistaminics / corticosteroids

CV complications

Expected in patients with : IHD, post myocardial infarction, hypertension, hyperthyroidism

Main risk : ↑ supply of endogenous or exogenous adrenalin, HR disorders, attack of angina pectoris or MI. ↑ Risk of acceleration of hypertension, risk of ♡ attack, vascular cerebral incident or cardiac decompensation

Prevention of stress in dental office (contigent benzodiazepine prophylaxis...)

Reduction of adrenaline in local anaesthetics

Remember potential antithrombotic medication (bleeding

Av 1 surgical treatment at least 6 month after ♡ attack

Bleeding

Disorders of haemocoagulation : haemophilia, von willebrand's disease, hemoblastosis, anti

- anticoagulant therapy
- antiaggregation therapy

coagulant therapy, heparinization at hemodyalysis or use of acetylsalicylic acid (antiaggregation therapy)

- heparinization

Risk of post-operative haematomas endangered by infectious inflammations.

↑ susceptibility to infection

In immunosupressed patients : oncotherapy, after organ transplantation, RA, multiple sclerosis, inflammatory bowel disease, AIDS, risk of infective endocarditis - due to bacteremia

ATB prophylaxis necessary (amoxicilin or clindamycin)

Impaired wound healing

Typical in DM, long term corticosteroid therapy, serious nutritional deficiency/cachexia

Enhanced post operative care and atb prophylaxis

Attack states

Diseases that can cause attacks at dental treatment

- Epilepsy: prophylaxis w/ BZP, elimination of adrenalin in anaesthetics
- Glaucoma: use of anaesthetics w/out catecholamines
- Bronchial asthma: ↓ stress and eliminate administration of causing drugs

Attack depends particularly on stress, pain or local anaesthetics

Transmission of viral infection between patient and staff

Highest risk in viral hepatitis and HIV

Vascular space is always opened during dental procedures → ↑ Risk of transmissio

Use of gloves, masks and eye-shields during dental procedures, avoid needle stic injuries and other traumas.

Oncologic patients

- oral mucous membrane manifestations
- ↑ Risk of infection - immunosuppression
- ↑ Risk of post extraction bleeding
- ↓ ability of wound healing and danger of radiosteomyelitis development

other Risks

Hepatotoxicity and nephrotoxicity of some drugs, neurological, endocrine, psychiatric and oth handicapping disorders which can modify the way of dental treatment / be influenced by it.

(10) Dental caries - clinical appearance and treatment what influences them? appearance

Dental caries are the most widespread disease in the population, it affects 80-90% of the population. They're directly related to the food composition and means of preparation.

Factors influencing appearance of the dental caries

- Saliva: mostly produced by glandula submandibularis (40%) and parotis (26%). More than 99% of it is made of water. 0,7% of solid substances (0,5% are organic and 0,2% is inorganic). Potassium, Mg, Cl, sulfate. CO_2 is an important part of the saliva as well, acting as a buffer to maintain the saliva's pH. Organic compounds of the saliva are represented by mucoids, albumins, globulin and peptides. Urea, uric acid, creatin + ammonia are present in ↓ [%]. Among the most important enzymes are amylase, esterase, lipase and peroxidase. It also contains ery, leukocytes from the gingival grooves + lymphocytes from the tonsils.
- Food intake, nutrition, hereditary factors: ↓ molecular saccharides, time interval of food ingestion
- Microorganisms of the oral cavity: S. mutans

Clinical manifestations

Dental caries are linked to the hard dental tissues. It starts with the enamel surface's decalcification during which the hydroxyapatite crystals loose its orientation, change their shape and the interprismatic spaces are enlarged. When more rods become affected, a macroscopically detectable defect of the enamel can be observed. After the decay crosses the enamel-dentine boundary, it spreads more rapidly through dentine towards the pulp.

The carious focus consists of large cavities on the surface which are filled w/ numerous microorganisms producing acids and proteolytic enzymes. As the caries process widens, it affects the enamel, dentine and cement. The primary caries occurs on those teeth surfaces that have not yet been treated yet, the secondary caries depends on the physical + chemical properties of the filling materials, faults during its preparation and during caries preparation and filling.

Enamel caries are manifested as a white spot where the enamel looses its gloss. It may be brown coloured by deposition of a pigment. Not painful. A large carious defect, extending to a various depth into dentine, is usually painful when a tooth is irritated directly.

The affected tooth has clinical signs of rough surfaces or sharp-edged cavities which may accumulate food residues.

Caries close to pulp are a result of the carious process. Dental pulp defends itself by producing a tertiary/transparent dentine. The tertiary dentine is synthesized as a response of odontoblasts to an irritation and it contains more of the basal substance and less dentine tubules. It contains mineralized processes of odontoblasts. The pulp doesn't usually display any signs of inflammation and a patient may not have any problems. Pain caused by cold, salt, sweet + sour irritation is often reported however, this pain is relieved after the causing effect stops.

Caries close to the pulp are treated by the method of indirect pulp capping. This treatment is based on supporting the pulp's resistance by calcium hydroxide that's applied to the pulp wall of a prepared cavity after soft dentine (tertiary) has been removed. Calcium hydroxide has antimicrobial + antiinflammatory effects and stimulates the pulp's resistance mechanisms. Ca^{2+} and OH^- ions penetrate the pulp. OH^- ions cause coagulation of protein components of the pulp and neutralize acidic inflammatic products. Ca^{2+} ions stimulate phagocytosis, decrease permeability of capillaries and upon reaction with CO_2 forms calcium carbonate in the tissue.

This treatment provides for successful results in up to 90% cases. If it fails, it's accounted as a false diagnosis caused by leaving a rather thick layer of softened dentine or low resistance abilities of the pulp. In cases when the pulp chamber is exposed either by injury or by careless preparation of a cavity, the direct pulp capping method is indicated. Here, the most appropriate is also Ca^{2+} hydroxyde.

This material is applied directly on the exposed pulp providing a perforation is not too large.

By these means, a layer of a coagulation necrosis is formed, and the layer of CT. barrier forms underneath it. Non-differentiated mesenchymal cells produce new odontoblasts that differentiate further. A dentine bridge is gradually formed above the perforation which takes 4-6 weeks.

(1) Diseases of the dental pulp, diagnostics and therapy

caused usually by a mixed bacterial flora that enters the pulp from a carious focus through the dentine tubules. Less frequently, an infection enters the pulp retrogradely via foramen apicale (in case of deep periodontal pockets) or by the blood stream.

Silica-phosphate fillings that lack bases and to the lesser extent also composite fillings may cause chemical irritation. The pulp inflammation may occur after an acute trauma or by a chronic traumatic irritation.

⊙ Regressive changes of the pulp

Google pic.

As a reaction of the pulp to either external/internal irritation, the true pulp stones arise from odontoblasts. Their structure is similar to the tertiary dentine. Mock pulpstones are formed by mineralizing deposits surrounding an organic core. Pulp stones may be located freely inside the pulp's interstitium or adhere to the walls of the pulp's chamber or a root canal. The proof of pulp stone presence is done by an x-ray examination. Clinically, pulp stones may be a cause of neuralgic pains.

⊙ Pulp hyperemia

Short and well localised pain in response to physical/chemical stimuli which diminishes after a causing effect stops. Treatment is an indirect pulp capping.

⊙ Acute pulp inflammation

May affect part of the pulp or the whole pulp. May be purulent or serous. The pain at serous pulpitis is caused by an external irritation at the beginning, later it may be spontaneous - blunt, radiating, pulsating. At a total pulpitis, the pain is often hard to localize - has a neuralgic character.

Treatment: pulpotomy / extirpation of the pulp by the vital/mortal methods
partial total

The purulent form of pulpitis is accompanied by formation of small abcesses inside the pulp chamber. Pain is relieved by cooling

Treatment: pulpotomy

⊙ Chronic pulp inflammation

Takes forms of a closed inflammation of the pulp chamber (clausa, granulomat... internal) or an open one (aperta hypertrophica, ulcerosa)

Closed pulpitis often have a course w/out significance. Diagnosis is based on... objective finding completed by an X-ray examination (resorption of the pulp chamber / root canal).

Chronic pulpitis is characterized by growing of the pulp through a cavity, its ulceration on the surface and sometimes by an outgrowth of the granulomatous tissue through a cavity to the oral cavity + formation of the pulpous polyp

Clinical signs: bleeding occurs as a result of the pulp injury.

Treatment: partial removal of the inflammed pulp or total removal. It may be... under a local / block anesthesia (vital method) or after a devitalization (mortal method). Substances used for devitalization: arsenic trioxide, cobalt paste or para formaldehyde are often utilized. Devitalization is recommended for all kinds of pulp inflammation except the purulent, ulcerous and polypous forms.

⇒ How arsenic trioxide works:

Affects blood vessels, cells of the pulp and nerve endings. Its application results ... necrosis of the pulp as possible or directly on an exposed pulp and a cavity is then sealed hermetically by a temporary filling.

Complications of pulpitis ⟹ pulp necrosis, pulp gangrene

↳ a patient with gangrene feels pain during a heat test

(12) Apical periodontitis

characterized by spreading of an infection into the periodontium. They are caused mostly by the root canal infection that proceeds into pendontium through f. apicale and by trauma, chemical effects (devitalization / disinfecting agents)

→ Acute apical periodontitis

characterized by dilation of vessels near the root apex and an edematous infiltration of periapical tissues. It has the following stages:

- Inflammation is located at the apex area - periodontal phase
- serous exudation and formation of a cellular infiltrate with a pus colliquation - endosseal phase
- inflammatory process spreads under the periosteum of an alveolar ridge - subperiosteal phase
- after periosteal necrosis, the pus containing exsudate leaks under a mucosa, the submucosal phase. Accompanied by formation of an abcess that sometimes empties by an intra-oral fistula

It often originates by exacerbation of a Chronic periodontitis due to decrease of an organism's immunity or after a treatment of an infected root canal

(C.F) Aching tooth, sensitivity of apical area by percussion, submucous tissue infiltrate Redness, swelling and fluctuation at a vestibule surrounding the affected tooth Enlargement of regional lymph nodes, fever in more severe cases

Pain is severe during the early phases of the inflammation, after the submucosal inf. trate or a fistula are formed, an obvious pain relief comes.

Treatment - ensure exsudate drainage. This may be achieved by a tooth trepanation (at periodontal / endosseal phases) and by making a passage through the root canal At the subperiosteal and submucosal phases, the tooth trepanation has to be complemented by an intra-oral incision

Atb should be prescribed if general status of patient is altered and if he has a fever.

→ Chronic periodontitis

The underlying cause of the disease is formation of a granulomatous tissue at the tooth apex area. The course of disease is either in the form of non-limited, diffuse process or localized. A focus is then encapsulated by a fibrous membrane, with small abcesses cavities or a single continuous abcess cavity inside

Malassé's epithelial cells, remnants of the Hertwig's sheath occuring during tooth development can often be found, these cells grow as a result of an inflammatory irritation.

chronic periodontitis usually runs with no clinical difficulties and it's diagnosed by a X-ray examination. An x-ray image shows large or small, diffuse or sharp-edged radiolucency (lighter white) around the tooth apex.

Treatment →conservative: mechanical widening of the root canal, treatment of a microbial infection and filling the root canal with a filler that prevents re-infection and allows for healing of periapical tissues

→ surgically: root canal is filled with zinc oxide-phosphate cement after a previous root canal treatment. The surgery - tooth apex resection (apicectomy, amputation) - basis of which is a Removal of a treated tooth apex, excochleation of the granulomatous tissue:

(13) Disturbances of the tooth development

Anomaues exist in number, shape, size, structure, position of teeth as well as temporal disorders of teeth development and teething

⇒ Reduction of teeth # / Hypodontia (agenesis) : upper second incisor teeth and wisdom teeth are the most commonly absent.

A condition when groups of teeth aren't formed is called oligodontia. If the teeth aren't formed, we call it anodontia

⇒ Increase in teeth # / Hyperodontia : usually found between the central incisor teeth.

⇒ Shape change : affecting crown / root / pulp chamber. The most common shape alterations are peg-shaped (conical) teeth or teeth with accessorey cusps.

Preoportional reduction in teeth size is called microdontia, enlargement of teeth is macrodontia.

⇒ Defects of hard dental tissues formation

① disorders that are caused by non-specific effects on a developing tooth

- genetically determined anomalies

① Hypomineralization / Enamel hypoplasia } maybe caused by common diseases of upper respiratory tract otitis, diarrhoea, deficiency (specially vitamin deficiency) stat or endocrinopathies. They are also a result of tetracycline atb's administration during the enamel formation and mineralization. The first few years of life are risky since crowns of most permanent teeth are formed and mineralized at this age. Affected teeth are yellow / brownish and have some depects that make them prone to decays and fractures

Enamel composition defects occur as a consequence of erythroblastosis, or as a result of fluorine overdose.

② Hereditary imperfect dentinogenesis / Hereditary imperfect amelogenesis } affect all teeth of both dentitions

(14) Significance of deciduous dentition treatment

- In temporary teeth destruction - digestive system suffers - affected development of organism.
- Periosteal inflammation caused by milk teeth may be a life threatening situation
- Temporary dentition's periapical changes may damage permanent teeth resulting in some orthodontic anomalies
- Treatment can be prophylactic / restorative / surgical

(15) Pain in oropacial region

Pain felt in face, mouth, nose, ears, eyes, neck and head

origin of pain

↳ non-masticatory: • atypical: non-organic cause
- trigeminal neuralgia
- glossopharyngeal neuralgia
- temporal arteritis
- periodic migrainous neuralgia - cluster

↳ odontogenic: gingivitis, pulpitis, bruxism, cancer, infection

↳ Neurogenic: post-herpetic neuralgia, CN V, IX neuralgia, bell's palsy

↳ Musculoskeletal. TMJ disorders, sinusitis, osteomyelitis

↳ Psychogenic

(16) Viral diseases in the oral cavity

- HSV : gingivostomatitis herpetica (primary herpetic stomatitis)
- EBV : infective mononucleosis - ulcerations and hemorrhages in mouth
- HPV : verruca vulgaris, condyloma, viral acanthomas
- coxsackievirus : hand - foot - oral
- Mumps, variola, varicella : blisters
- Measles - Koplik spots - moles
- rubella - catarrhal inflammation of mucosa

(17) Microbial inflammations of the oral mucous membrane. Diseases of oral mucous membrane of obscure ethiology

- candida albicans : most frequently oral mycotic infection

syphilis : acquired (all 3 stages have oral manifestations) → Treponema pallidum

1st - hard chancre

2nd - generalized macula and papula

3rd - gumma - in tongue - atrophy, sclerosis

- TB : oral symptoms : ulcers - after infection of mucosa from patient's sputum
- Actinomycosis
- Sarcoidosis

→ From immune system disorders

- Recurrent aphtous stomatitis - minor aphtous ulcers
  - major aphtous ulcers

- Erythema exsudativum multiforme hebral – recurrent disease of skin and mucous membranes with fever and general alteration of organism. Blisters

- Pemphigus vulgaris – affects both skin and mucous membranes
  in oral cavity: widespread bullae – breaks down → irregular erosions
  ↳ Ab against desmosome protein

- Benign pemphigus and bullous pemphigoid – subepitheloid blister

- Sjögren's syndrome – chronic inflammatory disease; affects endocrine gl. (most) salivary and lacrimal. Glandular parenchyma is gradually replaced by lymphocytic infiltration.
  ↓ secretion of both glands – impairment of food processing and difficulty swallowing

  - primary – sicca syndrome; only exocrine gl. involved
  - secondary – sicca syndrome + systemic disease of c.t.

(18) Professional damage to the oral mucous membrane

There are 3 causes of oral lesions elicited by work environment / process

1. Chemical burns of oral mucosa:
   - by strong acids → coagulative necrosis → rinsing with sodium bicarbonate
   - by strong alkaline → colliquative necrosis → rinsing with weak citric acid

2. Long-term exposure to harmful effects of chemicals, solvents, gases
   Results usually in increased epithelial keratinization, damage of enamel
   ↑ caries incidence and marginal inflammation of gingiva in bakers, confectioners and chocolate manufacturers.

3. General chronic intoxication - with heavy metal salts, characterized by specific oral symptoms. Rare.
   oxides of the metals circulate in blood and meet in gingiva with hydrogen sulphide which is produced by plaque microorganisms - they fuse into sulphides which are deposited under epithelium and form coloured border in area of free gingiva

- lead intoxication: gray gingiva + hypersalivation
- zinc intoxication: blue gray gingiva and ulcerative gingivitis

(19) Candidiasis of the oral mucous membrane

Factors enhancing expression of C. albicans / decreasing immune system activity

- steroids
- HIV
- chemotherapy
- transplant
- elderly
- dry mouth
- dentures
- smoking
- nutritional deficiency

Complications → Spread distally - esophagitis
- endocarditis
- meningitis
- arteritis

Features:

⊙Acute — Pseudomembranous candidiasis (thrush): white patches

— Atrophic candidiasis: smooth red shiny patches on tongue

⊙Chronic — Atrophic candidiasis: common with dentures, mucosa is red and swolle or hard palate

— Hyperplastic candidiasis: resembles leukoplakia / premalignant white patches in cheek / tongue..

Candidal angular cheilitis - bright red skin at angle of mouth w/ dry white coating sometimes bleeding - DM ♀. Treatment: keto/itraconazole, chlorhexidine

(20) odontogenic cysts (diagnosis, treatment)

Cyst : pathological cavity with solid, fluid or gaseous contents

Cyst formation : • proliferation of ep. lining and C.T. capsule

• accumulation of fluid w/in cyst

• Resorption of surrounding bone and incomplete compensatory repair

→ odontogenic cysts of jaw

Inflammatory dental cysts (apical / lateral / residual)

• Necrotic pulp

• stimulates formation of cyst and ep. lining originates from cells of Malasser

• surgical therapy

Dentigerous cysts

• From around crown of unerrupted permanent tooth and arise from reduced enam

ep. Bony expansion often Therapy : surgical
occurs

Keratocysts

• Are lined with parakeratinized ep. derived from remnants of dental lamina

therapy curettage + enucleation

→ Non-odontogenic cysts of jaw

Fissural cyst

• Arise from embryogenic junctional epithelium. Rare, include incisive canal cyst, incisive papilla cyst and vasolabial cyst

Therapy : Surgical - enucleation

→ Cyst of soft tissues in cervicofacial region

- Mucoceles

usually mucous extravasating cysts - saliva leaks from traumatised duct and pools

Ranula

Mucocele in floor of mouth arising from sublingual gland. Therapy: surgical excision of cyst and associated sublingual gl.

Dermoid and epidermoid cysts

Also developmental. May cause swelling of floor of mouth, submental region and

Therapy: conservative excision

(21) Salivary gland diseases

→ Sialadenitis: inflammation of major salivary glands

- Acute bacterial sialadenitis (s.aureus, oral streptococcus)

Painful swelling with purulent discharge from duct

May also develop as an exacerbation of chronic bacterial sialadenitis which often exists

-> complication of duct obstruction. usually unilateral

Therapy: Atb's, stimulation of salivary flow - chewing, massage of gland. Rarely: drainage

removal of gl.

- Viral sialadenitis (mumps → parotid (bilateral) - fever, pain, swelling)

Tends to be more severe in ♂ adults leading to orchitis

- Recurrent sialadenitis

Severe recurrent infection of parotid → dilation and ballooning of duct + alveoli → Sialectasis. Parotidectomy is indicated

→ Sialolithiasis

symptoms: recurrent pain and swelling in obstructed fluid, mainly before and during meals

X-ray: radiopaque (super bright!) Stones, but doesn't exclude radiolucent Stones

Most common in Wharton's duct (submandibular)

Removal under local anaesthesia is indicated, if too large extirpation of gl. is necessary

→ Sialosis

Painless swelling of salivary gl., mostly bilateral.

↓ saliva production → xerostomia: predisposes mouth, pharynx and salivary gl. to infection and caries

(22) Temporomandibular joint disorders

causes of joint difficulties are : - occasional / chronic traumas

- recurring luxations
- lowering of the vertical mutual position of jaws
- psychogenic factors causing bruxism + muscle spasm
- infectious diseases (acute rheumatic disease)
- degenerative processes (osteoarthritis)
- congenital disorders (joint head hyperplasia)

Symptoms: jaw pain, headache, jaw noise, difficulty opening/closing jaw/chewing + ting difficulty

Classification

→ Masticatory muscle disorders (myofascial pain, myospasm, myositis)

→ Temperomandibular joint disorders (hypermobility, inflammatory and metabolic disorders

→ chronic mandibular hypomobility (ankylosis, muscle contracture)

→ Growth disorders (congenital, developmental, incuding neoplasia)

Management of temporomandibular joint disorder

All initial treatment should be conservative, reversible and non-invasive.

Pharmacotherapy: analgetics, anti-depressants, tranquilizers and sedatives, muscle relaxants, local anaesthetic agents. Pharmacotherapy is rarely the only treatment. It's usual adjunctive to other measures such as rest, reassurance, orthetics (splint therapy, dietary changes and physical medicine (thermotherapy, coolant therapy, ultrasound)

(23) Sinusitis maxillaris odontogenes

odontogenic sources of maxillary sinusitis should be considered in people:

- with history of odontogenic infection and/or dentoalveolar surgery
- Resistent to standard therapy

Diagnosis: dental and clinical evaluation, X-rays

Cause: dental abcess and periodontal disease perforation schneiderian membrane/ sinus perforation during tooth extraction; irritation and secondary infection caused intra-antral foreign body; odontogenic infection (strep., proteus, coliform bacilli)

Treatment: 3-4 weeks Atb ⊕ surgical removal of foreign body ⊕ treatment of odontogenic condition ⊕ if oroantral communication - surgery to avoid chronic sinus disease

(24) Tumours of the oro-facial region (principles of treatment)

1/2 of all tumours

4/5 inside mouth → mucous membranes cheeks, ant. 2/3 of tongue, alveolar ridge, palate ⇒ squamous cell carcinoma

⊕ from lymphoid tissue waldeyer's ring

Etiology: smoking, food, alcohol, combination (smoking + alcohol), viral infections, immunodeficiency, poor nutrition, UV light, occupational exposures

Signs and symptoms: usually painless and difficult to detect in early stages

- mouth sore that fails to heal or bleeds easily
- white or red patch in mouth that don't rinse out
- a lump, thickening or soreness in mouth, throat, tongue
- difficulty chewing / swallowing

① Tumour staging (TNM)

② Tumour biology and differentiation

③ General condition and age of patient

④ other illness

⑤ social situation and profession

⑥ Perspectives of psychosomatic rehab.

Treatment

Surgery

- extirpation
- excision
- partial resection
- subtotal resection

radiation

- pre- or postoperative for suppression of residual malignancies (54 - 60 Gy)

Chemotherapy

- intra-arterial infusion of metotrexate + bleomycin and vincristin (usually int. carotid a.)

(26) Implantation in dentistry

Dental implants : alloplastic materials which can be incorporated into jaw bone and are able to replace natural teeth and wear prosthodontic appliance (crown, bridge)

≥ 6 months after extraction

Materials : titanium, titanium + hydroxyapatite, plasma - Sprayed titanium

osteointegration : implants are locked solidly into bone by virtue of a direct interface direct connection between living bone and load bearing endosseous implant at light microscope level - essential for implant "survival"

Bone needs to have sufficient depth and quality to accept an implant

Types of implants

- ✓ subperiosteal
- ✓ Transosteal
- ✓ endosteal blade (plateform)
- ✓ endosteal root-form : 2 stage implants. Most common cylindrical and may have external thread

Process of 2 stage implantation

Treatment planning (clinical examination, X-rays, casts, photos)

⊙ 1st surgery - implant placement into bone. Soft tissue and bone healing for
lower jaw - 3-4 months required
upper jaw - 6 months

⊙ 2nd surgery - implants recovered and 2nd component is placed. 4 weeks healing

Prosthetic restorative procedures - crowns, bridge or prosthesis fabrication

contraindications to implant

- acute illness
- terminal illness
- pregnancy
- uncontrolled metabolic disease
- drug abuse
- Local pathological process
- inadequate hygiene

(27) Precancerous lesions in oral cavity

- Leukoplakia: 20% of lesions show evidence of dysplasia or carcinoma at first clinical recognition - Hyperkeratosis
  - Erythroleukoplakia: leukoplakia with a red component
- Erythroplakia: red lesions that can't be classified as another entity. 97% probability of dysplasia or malignancy. Flat, macular, velvety appearance and may be speck. with white spots representing foci of keratosis
- Lichen planus: difficult to differentiate from ep. dysplasia
- Other lesions: premalignant changes arising in other oral lesions - Linea alba, leukoedema and frictional keratosis

(28) Surgical treatment of orthodontic anomalies

orthodontic therapy is based on the assumption that bone tissues adjust themselves to gradual changes of their functional load. The field can be divided into 2 branche conservative and surgical orthodontics.

The best time at which the orthodontic therapy should start is the period of mixt. teeth, when cutting teeth can be lead to a correct position at the dental arch and when the growth of jaws may affect the teeth development in a ⊕ manner

Surgical therapy

The basic element of surgical orthodontics is orthodontic extractions. They ena ble for faster and more stable incorporation of teeth into the dental arch in ca se of tightened and lack of space. It's used mainly for correcting of congenital ano- maues of jaws where the orthodontic therapy alone is not sufficient

Surgeries of jawbones are performed after their growth is completed (around 18y until this age, attempts to adjust the teeth positions, height of bite and mutual positions of jaws should be carried out so that the best possible conditions for a Surgery are achieved.

Surgical therapy is most frequently applied in cases of mandibular progenia, open bite, maxillar prognathia, microgenia and laterogenia

Myotherapy - serves for exercising of particular groups of muscles. It's not very demanding method that should create a proper muscular tonus. Favorable condition for normal development of the teeth are created by ↑ the tonus of flacid muscles or by releasing a hypertonic

(29) Dentitio difficilis molari inf. tertii

3rd molars (wisdom teeth) erupt usually at 18 - 24 years

Position of the 3rd molars in posterior part of oral cavity (near mandibular ramus is unfavourable as the high density of surrounding bone.

Eruption is often impossible due to malposition of tooth (mesial, distal, buccal or lingual incunation, rotation or horizontal position)

Pericoronitis begins under gum flap overlying crown of tooth. Sinus is formed - re tention of plaque and food is possible - tissue around wisdom tooth has beco me inflammed: Definition: inflammation of the soft tissues surrounding the crow of a partially erupted tooth including gingiva and dental follicle.

↳ Acute pericoronitis

Either a sequela of primary infection or exacerbation of chronic form. Occurring swel ling of soft tissues may spread into lower face or into floor of mouth. Masticatory muscles become inflammed resulting in inflammatory trismus (reduced opening of jaws caused by spasm of mastication muscles)

Symptoms: fever, headache, dysphagia, lymphadenitis → more severe: purulent exsudatic losing of teeth and pain in percussion

Abcess may form in pterygomandibular, submasseteric or submandibular and para- phryngeal spaces

Differential diagnosis: tonsilitis, pentonsillar abcess, acute lymphadenitis

Therapy: wide spectrum Atb
abcess incision
extraction of 3rd molar

↳ Chronic pericoronitis

- Variable and infrequent pain
- Gum flap is swollen + red
- Regional lymphadenitis

Therapy – conservative: irrigation + draining of pe ricoronal space
– surgical: excision of gum flap or extra tion

(30) Epulis

Is the name given to any lesion that develops on gingiva / alveolar mucosa. These lesions are reactive in tooth bearing area leading to localized tumor function with exception of epulis congenita - benign myofibroblastic soft tissue tumour of newborn

Histologically - 4 different types → Epulis granulomatosa (1)
→ Giant cell epulis (2)
→ Epulis fibromatosa (3)
→ Epulis fissurata (4)
④ Epulis congenita

(1) Also found in other parts of mouth

Related to trauma or minor injuries - present as tumour like tissue reaction

Mainly in young ♀

Can develop during pregnancy (epulis gravidarum)

Painless, red / blue lesions covered by normal mucosa, sometimes ulcerated

(2) Exclusively on gingiva or toothless alveolar ridges

Mostly ♀ - 40 - 60y

Macroscopically can't be differentiated from epulis granulomatosa

(3) Mainly occurs in interdental papillae

very often surface is ulcerated

lesion is connected to periosteum union should also be excised

(4) Reactive tissue alteration related with persistent traumatisation by ill-fitting dentures

Is most commonly found in elderly patients

Single or multiple firm folds in anter. alveolar mucosa

(5) True soft tissue neoplasm that occurs exclusively on alveolar ridges of new born children. It presents as a fleshy pedunculated mass lateral to midline and more often in maxilla than in mandible. Benign. More common in ♀

(31) Mandibular fractures

A fracture with no dislocation of fragments has its fracture line extended clearly along the whole bone's width, no shifting or deformation is apparent on its edges. Breakage is narrow sharp, and has interspersed fragments.

Fractures with fragment dislocation have their edges visibly shifted, the breakage is widened and often contains a tooth root or a retained tooth.
According to the # of fracture lines, fractures can be divided into single, double, triple and multiple. The breakages character may be either multiple or comminuted. If a bone tissue is lost, a defective fracture occurs.

Clinical findings: facial distortion, oral occlusion of teeth, abnormal mobility of portion of mandible or teeth

can occur in: body, symphisis, angle, coronoid process, condyle, subcondylar, ramus

when double fractures occur, they are usually on contralateral sides of symphisis
Ring bone rule - look for another fracture or dislocation also.

Classification of the mandibular fractures

1. Fractures of the alveolar ridge

Most often at the front section. There are usually more fracture lines that descend through the teeth sockets vertically and a horizontal one dividing the ridge from the body at the apex area.

2. Fractures of the mandible toothed section

At the middle part of the jawbone, fractures usually don't run perfectly vertically along the symphysis, but rather sideways. Lateral fractures result either from a direct impact at a place of its effect or on the jaw bone by transferring its force there. Muscle tension is ultimate for dislocated of fragments. In general, a short fragment is pulled upwards, specially when the dental arch is shortened on the affected side and an inferolligation of antagonists is not present.

3. Fracture behind rows of teeth

At the teethless distal sections (a retained 3rd molar is often placed here), the bone's thickness is reduced and its fracture can occur after hitting w/ a fist. If there's a retained tooth present at the fracture line, surgical treatment including tooth extraction followed by osteosynthesis is the only correct way of treatment.

4. Fractures of articular process

Quite frequent. Can be either extracapsular or intracapsular. A fracture with no significant dislocation should be treated by resting for 3 weeks followed by rehabilitation of the mouth opening

5. Fractures of the muscular process

very rare

6. Fractures of a edentulous or sparsely toothed jaw bone

After reposition of fragments, it's not possible to make any immobilization with dental splints or intermaxillar fixation. They are often treated surgically, currently using mini-spunts or functionally stabilizing bone splints and screws

7. Defective fractures

characterized by a bone loss during a gunshot wound / explosion. Reconstruction is made by a combined treatment including the transper of a bone transplant and fixation of the fracture.

(32) odontogenic tumours

- Ameloblastoma

Aggressive epithelial tumour from precursor cells of enamel

Mandible around angle and unerupted 3rd molar deforms mandible and makes it thinner by expansive growth – radical surgery. Risk of malignization.

- Cementoma

from cementoblasts ↗ biological cell that forms from the follicular cell around the root of a tooth and whose function is cementogenesis (periodontal membrane). Occurs around 1st mandibular molar

X-ray – homogenous shadow around root. Complete excision of tumour and tooth is required

- odontoblastoma

Hamartoma of odontogenic origin composed of enamel, cement, dentin, pulp

Hamartoma – benign, focal malformation. Composed of tissue elements normally found at that site but growing in a disorganized mass.

- Adenomatoid odontogenic tumor (ep. of enamel)
- Ameloblastic fibroma / fibroodontoma (benign mixed ep + mesenchymal)

(33) some complications during and after the tooth extraction

* During

- Breaking of a tooth's crown, breaking an alveolus wall
- luxation or sub-luxation of an adjacent tooth (dislocation)
- opening of the maxillar sinus during extractions of premolars or molars in the upper j.
- injury of the mandibular n. during difficult extractions of deeply positioned lower molars, eventually breaking the lower jaw

Breakage of an extracted tooth is quite common complication that requires finishing the extraction by surgical means. A surgical extraction starts by cutting the mucosa and the periosteum and continues by forming so called mucoperiosteal flap. A cortical part covering the broken tooth's root is removed by bone surgery instruments (elevator?). The root is pulled up by extraction levers and after trimming the bone wound, edges of the mucosa are joined by a stitch.

* After

• Impaired heaung of an extraction wound - healing process may be hamped by absence o a blood clot at hemocoagulation disorders or by washing off a coagulum during extensive mouth rinsing after an extraction, or by infection of a coagulum followed by it decay. This leads to <u>post-extraction syndrome</u> causing prolonged heaung of an extraction wound. The syndrome has 2 forms:

→ Alveolitis sicca (dry) caused by traumatization of tissues after a difficult extraction or by a tissue ischemia after the use of excessive amounts of anthetic. A sharp pain shoots to temples, ears and cheeks, occurs the 2nd/3rd days after an extraction

→ Alveolitis purulenta is caused by infection and subsequent decay of a blood coagulum. It's characterized by a large general alteration, fever and reaction of local lymph nodes.

• Bleeding

Should stop in 5 – 10 mins under physiological conditions. Prolonged bleeding may be caused by local or general factors. <u>Local causes</u>: granulous tissue inside and extraction wound, a root or its fragment after an unfinished extraction, foreign objects (pieces of metal fillings) in a wound, bruised mucous edges

<u>General causes</u>: heavy bleeding may appear at patient with hypertension, RT diseases w/ a fever when a permeability of blood vessel walls is ↑ due to infection. Diabetic angiopathies cause heavy bleeding at patients w/ diabetes. Menstruation tends to ↑ bleeding, so surgeries aren't usually planned for this period

(34) Limited mouth opening (cause, treatment) = Trismus

Maximal physiological distance between incisors of upper and lower jaw [40 - 60mm]

Causes

- Inflammatory: inflammation close to masticatory muscles may cause infiltration that limits mandibular movement. Therapy: treating primary cause - parotitis, penmandibular abcess, permaxillary abcess or suppurative cervical lymphadenitis

- Myogenic: may follow inflammatory trismus, if there's fibrotisation of contractile elements of muscles. Can also be because of mechanic irritation of muscle after application of inf. dental block.

- Aethrogenic: after inflammation or trauma of TMJ
  jaw immobilitation is indicated

- Neurogenic: tetanic origin - toxic tonic spasm of masticatory muscles
  Risus sardonicus appearance - smile
  Therapy: atb and serum

- Cicatrical: follows severe burn and injury
  Treatment: warming of scar place by active and passive rehabilitation
  If not successful → surgical therapy → excision of scar

(35) Polytraumas

Polytrauma → multiple injuries with an immediate damage to a vital organ, followed by failure of vital functions. By their nature, polytraumas don't allow for an immediate treatment of all injured parts.
Main origin of polytraumas is road accidents

Most common injuries occur to brain (58%), lower limb (25%) and upper limb (24%) chest injury (10%)

Treatment strategy is based on a 5-step therapeutic plan:

1. Reanimation
2. 1st surgical phase (life-saving activities)
3. stabilization
4. 2nd surgical phase (final treatment)
5. Recovery and rehabilitation phase

Treatment of combined injuries of the pacial area should assure the following:

- Firstly → making airways free (releasing of stuck tongue, foreign object inside the mouth, mucous scraps and clotts). If the airways can't be freed, breathing has to be enabled by tracheotomy or long-term intubation

  Secondly → bleeding control + suturing situationally soft tissues. No long-term actions may be undertaken (such as osteosynthesis), bone fragments are fixed temporarily mainly by fixtures and dental splints

- stabilization → sufficient fixation, control damaged soft tissues + direct rigid intermaxillary fixation shouldn't be used (because it doesn't allow intubation by mouth, draining from airway is difficult, lung ventilation is worsened
- Final treatment: osteosynthesis with miniplints + complex stomatological treatment + active rehabilitation

Trauma to mouth can produce lacerations of soft tissues, fractures of alveolar bone + fractures of tooth crown and root

Injuries opening pulpal space acquire endodontic treatment of tooth usually and conserva tive or prosthetic reconstruction of tooth crown

(36) Anaesthesia in dental surgery

↳ Local anaesthesia

Infiltration method : blocks directly sensory n. endings in operative field. If area is sm and w/out inflammatory involvement, this method is preferred

Nerve blocking : n. trunk is blocked in its course somewhere between brain and operativ field. Inferior dental block, long buccal block, mental n. block, post. sup. alveolar bloc infraorbital block, palatinal, nasopalatinal block

Individual teeth can be rendered pain free by injecting local anaesthetic along perio-dontal membrane using specially designed system - high pressure syringe for intraligamentary anaesthesia - rapid onset and specific analgesic for isolated teeth

Drugs used

(6) patients INJ

Tarmecaine, Gdocaine, bupivocaine, mepivocaine, articaine with adrenaline, noradrenalin or vasopressin

sterile disposable needles

↳ General anaesthesia

Absence of sensation and paralysis of all nerve centers except those of circulatory and respiratory systems (6) unconsciousness

Fundamental problem for any dental, oral or maxillofacial general anaesthesia is that both surgeon and anaesthetist need to have access to same anatomical site

Drugs used

Sombrevin and propofol

↳ Sedation

Beneficial to anxious or mildly uncooperative patients

Drugs used

Benzodiazepines, nitrous oxide

(37) Treatment of jaw fractures

↳ Conservative

only absolutely non-dislocated fractures with sufficiently firm entanglement of fragments at their normal anatomical positions don't require any repositioning

- Manual Repositioning
- Dental splint
- Monofixation (splint reinforced by resin)
- IMF (intermaxillary fixation) · possesses a danger of aspiration of blood, vomits or food

↳ Surgical

- osteosynthesis : connecting fragments by means of artificial mostly metallic materials. Connecting elements : wires, bone, splints, screws, clinches and other parts.
- osteosutures : bone fragments joined together by bone wire stitches. Stitches can be single, double, cross-like, etc. They are inserted into apertures made at bone fragments edges. They hold the fragments together. Connection of fragments can't be made firm enough to ensure keeping fragments together w/out dental splints or intermaxillary fixation

(38) osteomyelitis of the jaws

osteomyelitis : diffuse extension of an inflammatory process to a large extent. Pyogenic destructive process affecting periosteum, cortex, marrow of spongy bone

In dentistry, the localized demarcated forms of bone inflammations are usually called ostitis

It begins in spongy bone and is followed by vascular enlargement, edema, leukocytic infiltration with necrosis of bone and bone marrow. Mandible is the most frequently affected due to its anatomical formation and position.

More common in lower jaw, except in infancy (more in maxilla)

Causes - infections (carious teeth with gangrenous pulps, fractures, dry socket, inflammatory changes in cysts, sinusitis, dermatologic infections, infections via bloodstream

- physical factors : osteoradionecrosis after oncological therapy

- chemical factors : as a result of devitalization of pulp w/ arsenic

Symptoms and signs : septic fever, swelling of soft tissues (abscess pointing intraorally or extraorally), pain, headache, dysphagia, ↑ WBC and ↑ESR, cervico-facial lymphadenitis

If it lasts more than 1-2 weeks → chronic osteomyelitis

X-ray results present rarefaction, sequestration

Necrotic tissues drift out through fistulas. Inflammatory process subsides by reduction of suppuration and edema. A portion of mandible dies out and nature tends to exfoliate sequestrum. Regeneration of bone: 12 - 18 months

Therapy

Intensive and early therapy is indicated

Wide spectrum Atb - lincosamides i.v.

If abscess is present - intraoral or extraoral excision and draining

Gangrenous teeth - extracted

In acute form - great amount of fluids, vitamins, minerals

osteomyelitis can be a sign of immunodeficiency

If it occurs during suckling age may lead to damage of growth zones, which results in asymmetric development of facial skeleton

(39) Inflammations of fascial spaces

- Periapical abcesses (localized collection of pus w/in tissue)
- Cellulitis (edematous inflammation process that spreads diffusely through C.T fascial plaques) - may extend to pharyngeal and cervical spaces - airway obstruction!

Bacteria invade periapical tissues (swelling and mid - severe pain) → inflammation spreads further → to fascial planes (chills, fever, nausea, headache, lymphadenopathy)

Potential spaces: - mental (between mental and mm. platysma)

- submental (mylohyoid / platysma)
- sublingual (floor of mouth / mylohyoid) ⇒ mandibular teeth problems
- buccal (lat. surface of buccinator and skin of cheek → maxilar / mandibular post. teeth
- submasseteric (lat. surface of ramus mandibular / masseter → mandibular 3rd molar

Inferior mid. face - can spread to cavernous sinus → thrombosis → infected thrombi may emanate → life-threatening condition

(40) Management of teeth injuries - dentoalveolar traumatology

Concussion, subluxation, loose, luxation

→ Concussion - tooth is sensitive to pressure and thermal changes. Lead to pulpitis and/or periodontitis. Sometimes pulp undergoes necrosis and must be removed

→ Dislocation - partial (subluxation) - tooth remains in place but has considerable dislocation and pathological movement. Teeth may be slightly displaced when crown is pushed inside or outside of dental arch while apex remains in position. If blood supply is impaired, pulp will be necrotic

Treatment: reduction of dislocation, fixation with splint and/or casts to surrounding teeth

→ Loose tooth - periodontal lig. are completely torn, tooth is extruded, feels movable and will likely fall out from socket

→ Dislocation - total (luxation of tooth, tooth out of socket)

Tooth is removed completely from its normal position

It may be driven into the spongy portion of jaw or maxillary sinus or if the outer or inner alveolar plate has fractured off it may slide under periosteum.

Tooth should be reinserted under some conditions, when no periodontitis is present and when it can be appropriately fixed to the surrounding teeth

!! Exclusion of pulmonary aspiration of luxated teeth particularly if there is history of loss of consciousness or the destiny of tooth is unknown

(41) Benign epithelial tumours of the oral cavity → from surf. or glandular epithelium

well demarcated and encapsulated

- Papilloma: from papillary layer of cutaneous or mucous surfaces. Typically in gums, lip, tongue, soft palate. Removal by surgery, cryosurgery or laser vaporization
- Verruca (wart) and acanthoma cutaneum: keratinized. on skin surfaces only! Never on mucosa
- Adenoma: from salivary gl. epithelium

(42) Fractures of middle 3rd of face

1. Maxillary alveolar process
2. Zigomaticomaxillary complex fracture (tripod)
3. Blow out fracture of orbit
4. Nasal bone fractures
5. Lefort fractures

(1) Associated with dislocation of several teeth
fracture of alveolar process of maxilla
Treatment: Secure viability of teeth

(2) Most common

- involve separation of all 3 major attachments of zygomatic bone & diastasis of zygomaticofrontal suture → frontal, temporal, maxilla
- Usually due to direct strike to body of zygoma
- Extraocular mm may become entrapped in zygomaticomaxillary component of fracture complex. Displaced fragment may restrict motion of mandible
- force may propagate along the long axis of lat. orbital wall and involve orbital apex or optic canal → ↓ vision
- Flatness of lat. cheek area and inability to open mouth
- CT scan

③ • orbital floor fracture - downward into maxillary sinus

• usually due to a blow to the eye

• enophthalmus, diplopia

• accompanying injuries of ethmoids and eyes

Treatment : eye must be dressed first.

Atb and blow not nose → can cause emphysema in case there's accumulation betwe ethmoid and medial wall of orbit

Presence of retrobulbar hemorrhage requires urgent treatment in order to preserve vision.

• Surgical intervention

④ • most frequently injured structure in face because of prominent position

• best seen on film shot with special low kvp nasal bone technique

⑤ - complex bilateral fractures associated with a large unstable fragment an invariably involve pterygoid plates

- 3 main planes of "weakness" in face : - transmaxillary
- subzygomatic
or pyramidal
- craniofacial

→ Le Fort I : transmaxillary fracture / between maxillary floor and orbital floor

→ Le Fort II : pyramidal or triangular fracture. CSF leakage is generally presen due to ethmoidal cribriform involvement

→ Le Fort III : most severe, craniofacial dissociation. Fracture of zygomaticofrontal junction and fracture of frontonasal junction. Soft tissue injury and CSF leakage

⚠ Look carefully at orbits because 60-70% of facial fractures involve orbit. Bilateral symmetry can be very helpful trace along lines of orbits, sinuses and zygoma.

(43) Malignant epithelial tumours of the oral cavity

- Basal cell carcinoma (basaliorna, ulcus rodens, carcinorna terebrans)

From stratum basale of epithelium

usually on face skin in >70y

Good prognosis - grows locally - no metastases

- sq. cell carcinoma

From stratum spinosum of ep.

it infiltrates into surrounding tissue or attacks into other structures via perivascular, endovascular and perineural propagation

can metastite via L.N. and vessels into bone, brain, lungs

- Adenocarcinoma

originates from glandular ep.

Prognosis depends on primary staging, Localization and grading

- undifferentiated carcinoma (anaplastic, lymphoepithelioma

Rare tumour of salivary gl. and nasopharyngeal mucosa.

(44) Salivary stones (sialolithiasis)

Sialolithiasis is a disease where concrements (sialoliths) are formed inside the ducts or parenchyma of salivary glands thus slowing down or disabling flow of saliva. In 90% of cases, submandibular gland is affected.

Salivary stones originate from changes (mucin) of the saliva, by precipitation of salts around a core formed in the duct by a foreign object, cluster of epithelial cells and leukocytes at the time of salivary gland inflammation. The stones may be round or spindle shaped

Clinical manifestations: repeated swelling of gland area, specially before a meal when salivation increases and a stone prevents the saliva to pass freely. Saliva retention may result in an ascendant transfer of infection and occurence of a secondary sialoadenitis (inflammation of salivary gland)

Upon long term illnesses and repeated infections, the gland may become fibrously modified

Treatment: Surgical removal from the duct after discision of its wall. If a stone is positioned intra-parenchymatously at the submandibular gland, an extirpation of the whole gland from the extra-oral side is indicated

(45) Benign mesenchymal tumours of the orofacial region

⊙ Hemangioma

- Arises from normal vessel tissues
- very frequent in children
- can be flat or prominent red-purple or violet blue lesions which bleed if traumatized
- Excision is contraindicated
- cryosurgery is the main therapy

⊙ Lymphangioma - from lymphatic vessel tissue

⊙ Fibroma - small, localized, slightly raised

- typically in vestibule
- smooth surface, composed of hypocellular fibrotic tissue
- excision is curative

⊙ Osteoma

- composed of mature compact or spongious bone
- most common in frontal sinuses[1], ethmoid[2], antrum[3], jaws[4] (in order)
- removal is necessary only when symptomatic obstruction of sinus ostium w/ sinusitis and headache

(46) Cysts of the soft tissues of the oro-facial region

① Mucoceles

② Ranula

Question 20 for more details! ☺

③ Neck cysts

④ Dermoid and epidermoid cyst.

(47) Malignant mesenchymal tumours of the oro-facial region

① Fibrosarcoma - malignancy of fibroblasts

- most common in extremities, only 10% in head and neck
- can arise anywhere in head and neck but most frequently in nose and para nasal sinuses
- slowly growing w/ only 10% of metastatic rate
- often have obstructive symptoms
- most frequent in young adults + children

② osteosarcoma

- 5% of osteosarcomas occur in head + neck and half of these arise in maxilla
- Most often in 30-40 y, mainly ♂
- Most often in 30-40 y, [illegible] ♂
- Arise in several clinical settings including pre-existing bone abnormalities: Paget's disease, fibrous dysplasia, giant cell tumour, chronic osteomyelitis, osteogenesis imperfecta
- Depending on amounts of osteoid, cartilage or collagen fibers, produced by the tumour it can be osteoblastic, chondroblastic and fibroblastic
- Radical surgical excision and chemotherapy / radiotherapy
- lung metastasis

(48) Salivary gland tumours

occur mainly during the 4th – 6th decade.

Clinical manifestations: slow, non-painful growth, gradual deformation of the face

Most commonly found in parotid gland (90%), submandibular (9%) and rarely in the sublingual / small salivary glands.

Classification

1. Epithelial tumours
   - Adenomas – pleomorphic
     - " monomorphic
   - Mucoepidermoid tumor
   - Acinocellular
   - Carcinomas – adenoid cystic carcinoma
     - adenocarcinoma
     - epidermoid carcinoma
     - non-differentiated carcinoma
     - carcinoma in the pleomorphic adenoma
2. Non-epithelial tumors
3. Non-classified tumors
4. Tumor-like states – benign lymphoepithelial lesion
   - sialoses
   - oncocytosis
5. Malignant lymphoma
6. Secondary tumours.

Pleomorphic adenoma – mixed tumour

- Most common salivary gland neoplasm, 85% in parotid
- slowly enlarging, painless tumour, round w/ smooth surface
- 50 y, ♀ > ♂
- surgical excision via lat. parotidectomy (facial n. stays intact)

Warthin tumour - adenolymphoma

- 60y ; ++ ♂
- almost exclusively in parotid

Carcinomas - rapid growth, facial and tongue paralysis and pain

- acinic cell carcinoma commonly in parotid gl.
- mucoepidermoid carcinoma
- adenoid cystic carcinoma - infiltrative, common in intraoral minor salivary gl.
  - perineural or perivascular spread
  - 3 growth patterns: glandular, tubular, solid

Malignant lymphomas

- lymphoid tissue of salivary glands is part of MALT and may be involved as part of systemic spread of malignant lymphomas
- usually non-Hodgkin and associated with chronic immunosuppression

(49) Injuries of orofacial region (emergency measures)

1st - free airways

2nd - stop bleeding

A, B, C - airways, breathing, circulation.

save patient's life: technical (remove chemical/physical influences)

medical (free airway/stop bleeding)

(1st) free airway from foreign object, blood, vomit and place person at recovery position (prevent choking, aspiration)

(2nd) stop bleeding with bandage by pressure on maxillary a., supp. temporal a., ext. caroti artery

Airway obstruction from: foreign bodies

trismus

gross edema of tongue

obstructed oropharynx

Massive bleeding in head and neck may cause: obstruction of upper airways, aspiration of blood into upper airways and lungs, haemorrhagic shock

(50) Bleeding in the oral cavity (etiology/treatment)

Etiology - trauma
- gingivitis
- pulpitis
- tumours
- teeth extraction
- vit. C deficiency
- coagulation disorders

Treatment: compression

antifibrinolytic drugs (iv./mou

(51) luxation of the mandible

luxation = displacement of mandibular condyle from mandibular fossa

Mechanism of luxation

If the head leaves the pit by shifting in front of tuberculum articulare, the lower jaw luxation occurs. luxation w/out bone damage happens at excessive mouth opening (yawning, screaming, cramps, vomiting) or during a forceful mouth opening (medical treatments: intubation, insertion of probe, etc.)

Conditions such as shallow tuberculum and loose articular capsule predispose for recurring luxations

Trauma: hit, sometimes after falling.

Classification

- Acute luxation at ventral direction (w/ no bone damage)
- Recurring luxation at ventral direction
- Traumatic central luxations, happen by penetration of mandibular condyle through damaged glenoid/mandibular fossa in to the middle cranial spaces (intracranial hemorrhage)

M ~~ir~~estations and examination

Patient can't close mouth, pain and tension are felt in chewing muscles and the mandibular joint. Saliva may leak from the mouth since swallowing is difficult. An empty pit of the joint can be felt.

Therapy

Timely repositioning of luxated lower jaw is crucial for the feasibility to use a simple maneuver. Soon, a spasm of chewing muscles causes a very strong resistance that can be surpassed under general anesthesia and after muscular relaxation only!

Reposition ⟶ fixation by an external sling bandage/ wire intermaxillar bond for several day

(52) General symptoms of jaw fractures

- Pain : appears in the forms of spontaneous / functional or pressure pain. At the moment of an injury / an affected person feels a blunt or sharp pounding pain. Pain is later sensed during movement of jaws, opening, closing and biting. Mostly located in the wounded area. In cases of non-dislocated fractures, pain can be initiated by pulling the jaw forward by a finger hooked in it

- Deformation of the face : only in highly dislocated fractures.

- occlusal defects : impairment of chewing is always present at fractures of the alveolar ridge / teeth - containing parts of the jaw. Teeth don't touch each other and food mastication is hampered

- Inability to open / close mouth

- Swallowing function is restrained mostly by pain which may make difficult swallowing saliva that'll ooze from the mouth

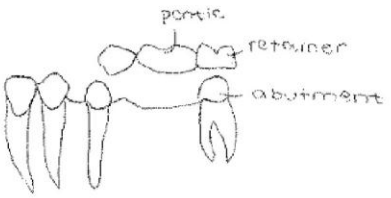
- Pathological motion and crepitation of fragments

(53) Prosthetic dentistry (types of dentures)

↳ Fixed

↳ Removable (partial removable and complete dentures)

Prosthodontics: branch of dentistry belonging to restoration and maintenance of oral function, comfort, appearance and health by restoration of natural teeth or replacement of missing teeth with artificial substitutes



Abutment: teeth to which prosthesis is fixed

Retainer: the restoration, usually a crown, rebuilding prepared tooth by which bridge is attached to abutment and to which pontic is connected

Pontic: substitute for the lost tooth, aesthetically and functionally

→ Fixed prosthesis - pillar constructions

- Inlays - replace lost crown (part/whole) + cast filling - gold
- Outlays - Replace whole occusal part
- Crowns - Replace part or whole damaged crown, reach to marginal gingiva

1. Jacket crowns - metal (gold, silver), ceramic (aluminium oxide), resin
2. veneer crowns - metal in combination with resin / ceramic
3. Pin crowns - replace whole crown of a pilar tooth

Advantages: no support from mucosa, occupy some space as natural tooth. Greater masticatory beds than dentures

Disadvantages: time consuming, expensive, not easy to clean (prone to caries, periodontal disease

→ **Partial removable dentures** – replace one/⊕ natural teeth, occupy more space, intimate contact with mucosa

classifications:

1. Tooth born – "occlusal rests" placed on occlusal surface of natural teeth so that vertical masticatory and clenched loads are transmitted via teeth bearing the rests
2. Mucosa born – load transmitted through interposed layer of mucosa
3. Tooth and mucosa born

Advantages – constructed in any case, cheaper, easily cleaned / repaired

Disadvantages – caries, damages supporting tissue, may lose natural teeth. Hyperemia/ hyperplasia / ulceration of mucosa

→ **Complete dentures** – must replace lost teeth and restore impaired functions (eating, speaking, facial expression) and appearance

→ retention depends on environment between denture and prosthetic bed, state of mucous membrane and quality of saliva

→ stability depends on artificial teeth position at body

(54) Prevention of damage of oral tissues caused by dental prosthesis

- Damage to abutment periodontium due to functional overloading
- wrongly adhering / unstable total removable dentures – decubitus ulcers
  - chronic inflammation (hyperplasia, granuloma, fissuration of prosthesis bed mucosa
  - ↓
  - retention of prosthesis
- stomatitis prosthetica: palate under resin denture (contact allergy to acrylic compound)
- candidiasis – in total dentures
- Angular infection – chronic inflammation of mouth commissures

Prevention: technically correct prosthesis ⊕ regular check ups ⊕ proper oral hygiene