Digital method and content development of the hungarian higher education in dentistry in Hungarian, German and English

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Cells involved in acute inflammation and their functions

Classification of inflammation

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Lung volumes. The inspiratory rate depends on chest wall distensibility and the elasticity of the lung tissue. Total lung capacity (TLC) is the largest inspiratory capacity, which depends on the elasticity of the lungs. TLC is increased in emphysema and in diseases associated with scarring of the lung tissue. TLC is increased in emphysema, asthma and chronic bronchitis. In the latter two cases, excessive lung expansion promotes airway dilatation. Functional residual capacity is the volume of air present in the lungs after a passive exhalation when no more passive chest contraction is possible. It is the end of normal expiration. Further exhalation requires the active work of respiratory muscles. Residual volume (RV) is the air volume remaining in the lungs after maximal expiratory effort. RV is increased in emphysema because small airways lose their flexibility and they collapse during exhalation, and in chronic bronchitis which is characterized by the contraction of small airways or inflammation. This is because the increased pressure in the lung tissue helps keep the airway open so they will close later during expiration, thus reducing the residual volume. Vital capacity (VC) depends on the relative changes in RV and TLC, but is generally decreased in lung diseases.

Causes of cough

Allergic and non-allergic factors in asthma

Symptoms of Cushing syndrome, in order of frequency

Side effects of corticosteroid therapy on skeletal system (doses above 10 mg/day)

Symptoms of hypothalamic damage

Theories on atherosclerosis development. According to non-lipid theories, the morpho-functional state of the vessel wall is altered which allows the infiltration and deposition of lipoproteins in the intima.

According to non-lipid theories, the morpho-functional state of the vessel wall is altered which allows the infiltration and deposition of lipoproteins in the intima.

- The fibrof core has a necrotic lipid core, separated from the lumen by a thin, fragile fibrotic (connective tissue) cap (Figure 7). This damage can lead to thrombus formation. With advancing age, high amounts of calcium may deposit in the lipid core, further reducing the elasticity of blood vessels. IEL-internal elastic lamina, E-endothelial layer, LDL- low density lipoprotein, ox-LDL-oxidized LDL.

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Figure 1. – **Dawes model of salivary clearance** (a schematic drawing). Taste buds sense the changing concentration of sugar and acid in the oral cavity. This stimulates salivary glands through the central nervous system to secrete a large amount of watery saliva, which will mix with the saliva already present in the oral cavity. A swallow is initiated when the amount of saliva reaches a maximum volume (swallowing reflex). A residual volume of saliva is left in the mouth after a swallow. This amount of saliva is constantly present in the oral cavity and is responsible for lubricating the mucosal surfaces. The original model is suitable for measuring the speed of oral clearance and was created by Colin Dawes in 1983. CNS: central nervous system

Figure 2. – **Phases of oral clearance.** After a meal (especially when consuming fermentable carbohydrates, acidic liquids and sugar) pH rapidly and significantly drops in the oral cavity in a few minutes. When oral pH falls below the critical range (pH 5.2-5.7), enamel demineralization is initiated. The fall of oral pH significantly stimulates saliva secretion. The saliva thus secreted contains large amounts of sodium bicarbonate, making it slightly alkaline (pH 8) and affording a high buffering capacity. Therefore, stimulated saliva has two major effects: first, its substantial volume significantly dilutes the nutrients in the oral cavity, facilitating their clearance through increased frequency of swallowing. On the other hand, it neutralizes acids due to its buffering capacity. This is the first or rapid phase of oral clearance, its duration is approximately 20 minutes. During this period, the main goal is to increase the oral pH to above the critical level. Subsequently, the rising pH will stimulate saliva excretion less and less, so the amount of secreted saliva gradually returns to the resting level, while oral pH will also return to the initial level. This is the second or slow phase of oral clearance, which takes approximately 40 minutes. The figure is based on the Stephan's curve which shows pH changes measured in the dental plaque after consuming a sugar solution. The curve was published by Robert Stephon in 1943. The dashed line indicates the critical pH value (5.5)
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Cloud-like intensities on the incisal edge 

Incisal halo with mamelon-style 

The application of enamel color gives transparency to the tooth 

Restoration of erosion with enamel color (before) 

Restoration of erosion with enamel color (after) 

Chamfer preparation at the gingival and contact area 

Centripetal build-up technique 

Direct filling constructed with successive cusp build-up technique 

Combination of the axial bevel and the successive cusp build-up techniques (Building-up of the proximal wall) 

Combination of the axial bevel and the successive cusp build-up techniques (Building-up of the oblique cusp) 

Combination of the axial bevel and the successive cusp build-up techniques (The filled-up cavity) 

Combination of the axial bevel and the successive cusp build-up techniques (The polished restoration) 

Prepared teeth (The hue, the enamel value, the shape and place of intensities, the incisal opalescence and other features are determined before the rubber dam placement) 

Conditioning 

Application of bonding agent 

Light-curing of the adhesive 

Dentin core (1. layer: UD4 color) 

Dentin core (2. layer: UD3 color) 

Dentin core (3. layer: UD2 color) 

Shaping of the mamelons 

The dentin core without enamel layer (Transparency and translucence are missing) 

Removal of excess material with diamond bur 

The application of transparent color to create the incisal halo 

UE3 enamel layer 

The layering of enamel color (UE3 enamel color can mimic the young enamel) 

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Intraoperative photograph, mucoperiosteal flap is done, before to the reduction of the bone much harder. One solution should be that we first do the fixation (tooth is still in occlusion). After a few days the blood effusion starts to sink due to the gravitation and makes blue discoloration on the neck. Therefore a sublingual haematoma develops which lifts the tongue and causes slurred speech. After a midface trauma, typical symptoms. At times, an eye-movement uneasiness, which is sometimes transitional but sometimes permanent, can be added to the unfavorable aesthetics. Not only because of the scala formation between the teeth but also because of the commonly torn gingival mucosa from the sulcus. Bleeding into the external world can be a bit scary in the case of fractures. There does not have to be necessarily facial fracture behind the bleeding of the external auditory canal. If the trauma has resulted condylar fractures, it is important to think of the injury and the auditory canal or serious damage of the cranial fossa. The occlusional discrepancy is the most straightforward by the displaced fractures. Sooner or later the bleeding tamponate itself. Therefore a sublingual haematoma develops which lifts the tongue and causes slurred speech. After a few days the blood effusion starts to sink due to the gravitation and makes blue-yellow-green discoloration on the neck.
4.12. Figure 12. – In untreated cases masticatory deficiency and occlusal dysfunction can occur and result in dysphagia or indigestion. The treatment is either conservative or operative, assumes reposition and fixation ................................................................. 951

4.13. Figure 13. – There is a fracture on the picture, which causes significant articular luxation. We put our little fingers into the right and left auditory canal of the patient and ask them to open their mouth. We do not feel the movement of the articular condylar ................................................................. 951

4.14. Figure 14. – In case of that fracture which can be seen on the 14th Figure, a significant sized soft part hernia got into the sinus from the orbit. In association with the bone breakout the bone fragments did not lose their connection with the periosteum. There is no real fracture only a door-like tilt, which is foreshadowed by the coronal record. If the tolerance of the patient lets it, these cases are perfectly suitable for lower restrain of the maxillary aurial balloon, so the bulb gets its original position back. It is favorable to expose the orbit base independently and put the herniated tissues back to the orbit, under eye control. Otherwise the loss of motion can persist despite the aesthetic restoration. Bridging the defect with foil, titanium mesh or own bone, are also good solutions ........................................ 953

4.15. Figure 15. – After the trauma, the explicit swelling and haemorrhage of the injured region and eye periphery covers the symptoms which become obvious later. After trauma the patient usually takes the numbness connected to the infraorbitalis injuries as a matter of course and transient. The dislocation of the bone block can not be seen, even if it is significant, because of the local swelling on the face. After three or five days, when the numbing persists, the patent starts to notice the deformation of the injured side, so the zygomatic fracture gets examined and diagnosed only then. The issue is that, the chance of a successful closed percutan reposition gets smaller due to the soft party, which has been formed in and around of the broken ends. Later intervention needs open exposure and fixation ...................... 954

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4.23. Figure 23. – On the second picture a left-side fracture without explicit dislocation has been diagnosed. In such a case, the ir almost no occlusional compliment, pain is only expressed under load and there can be numbness. It is not sure that such a fracture becomes visualized on a classical AD scull picture or on an off color OP radiograph ................................................................. 959

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4.25. Figure 25. – The radiograph shows well the dislocation of the left side zygoma block. The orbit frames are asymmetric, the eye socket narrows on the pathological side. there are 2-3 millimeter sized stepformations on the lower and lateral eye socket frame. Even the step, which seems to be big, can be diagnosed better according to the radiograph, in case of a freshly injured patient because the periorbital edem and haemorrhage makes non-evaluable the physical examination. We notice sooner the zygoma dislocation, which can be seen on the radiograph, but not always palpable at the steps, since the zygomatic tuber rather takes place inboard, than rising out from there ........................................... 960

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Alginate impression
Alginate mixing machine
In the front region held, lateral flat ridge
Decreased muscle tone of the face and the lips
Examination of sought area becomes easier.
Different intersection plains the connection of the roots and the maxillary sinus could be illustrated in 3D
different from each other and from different perspectives the variant teeth of the jaws’ are visible
The 3D skull image could be examined from the most appropriate aspector we can turn it to every direction of the space
The cephalograph was made by CBCT. The soft tissues could be detected next to the precise bone illustration as well. This is also an important method in the orthodontic diagnostic
Decreased muscle tone of the face and the lips
Positive changes after the reconstruction
Papilla incisive on the vestibular side
High ridge
In the front region held, lateral flat ridge
Position of the buccinator pocket’s lateral wall
Alginate mixing machine
Alginate impression
Material shortage in the alginate impression
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Digital method and content development of the hungarian higher education in dentistry in Hungarian, German and English

Szerkesztő: Dr. Nagy Ákos

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Pécsi Tudományegyetem
Preface

The Lisbon Agenda devised the action and *development plan* in year 2000, for the *economy of the European Union* between 2000 and 2010. It become the most dynamic developing economy, establishing more and better working places and form better society coherence.

The most important of the ten defined fields, was the public health and health business. All the efforts increasing the medical education’s quality has a straight benefit on the economy.

Strengthening the health business and their quality management was based on the medical higher education. Altough the medical students should read abouth 12-16 fresh medical publications every day to follow the development of the medical fields. The available 300 credit points in medical education are definitely not enough to teach everything.

Unfortunately students’ access to the online scientific literature is often restricted at the hungarian universities nowadays. Nevertheless it is impossible to take interest in all different medical disciplines.

It was an urgent need to find methods and establish materials to help the students self-supporting (individual) studies.

A multidisciplinar vertical curriculum was the result of that need. The brief chapters contain the most important information and give the opportunity to find connections between basic, preclinical and clinical studies.

The most important aim was to develop students’ troubleshooting skills.

Beyond that, self-training opens the door for highlighting the most important information at lectures and practices.

It is a matter of course that students can use it online, on their phones or even tablets when they are waiting for the bus!
1.1. Development of tooth germ – Gabor Varga

Teeth develop together with their supporting structures: the bones, the cementum and the periodontal ligament. The tooth is not a homogeneous tissue and it is not derived from a single germ layer. Enamel is secreted by ameloblasts differentiated from oral epithelium, while dentine is the extracellular matrix produced by ectomesenchymal cells of the dental papilla.

**Figure 1.1. Figure 1. – Jawbone in cross section**

Tooth development can be divided into three phases: the initial, the morphodifferentiation and the tissue differentiation phases. The developing tooth goes through the bud, cap and bell morphological stages; then, after mineralization, eruption completes the process.

**Figure 1.2. Figure 2. – Tooth development**

Tooth development starts from ectoderm covering the embryonic oral cavity with the division of the basal cell layer of the epithelium. These cells proliferate faster than the surrounding ones and grow deep into the mesenchyme creating the dental lamina and the vestibular lamina. This later gives rise to the lips and cheeks.

**Figure 1.3. Figure 3. – Tooth development – details 1**
In parallel, neural progenitors migrate to this area from *crista neuralis* and transdifferentiate into mesenchyme. So actually the dental pulp and dentine, despite their connective tissue properties, have ectodermal origin.

Tooth shape is formed during the morphodifferentiation phase as a result of proliferation and migration of different cell types. The morphodifferentiation of the teeth starts in the **bud stage** with the appearance of spherical clusters of cells on the dental lamina. The epithelial cells proliferate fast in the periphery of the bud and invaginate into the underlying ectomesenchyme forming a cap. This cap-shaped epithelial structure constitutes the enamel organ. Under the cap there are dividing but not yet morphodifferentiated cells that subsequently form the dental papilla.

The inner cell layer of the enamel organ facing the ectomesenchyme is called the inner enamel epithelium, while those cells on the outer side are called the outer enamel epithelium. Between the two cell layers is an increasing quantity of *stellate reticulum*. These star-shaped cells are not only responsible for the formation of the tooth shape but they also act as a feeding layer for the secretory ameloblasts formed from the inner enamel epithelium. The indentation of the enamel organ deepens and the tooth germ become **bell-shaped**. During the bell stage the enamel organ loses its connection with the dental lamina and a new cell layer appears within the enamel organ, the *stratum intermedium* that is directly fed by the cells of inner enamel epithelium. From this time point the morphodifferentiation of the undifferentiated, blastoid ectomesenchymal cells starts to create the dental papilla. In addition, in the apical edges of the bell, the generation of mesenchymal periodontal ligament and cementum begins from the dental follicle.

**Figure 1.4. Figure 4. – Tooth development – details 2**
At this stage histodifferentiation begins: two monolayers facing each other are formed. The outer layer differentiates from the inner enamel epithelium and consists of ameloblasts, and the inner ectomesenchymal layer of the dental papilla gives rise to the odontoblasts. The two layers are first separated by basal lamina. Mineralization begins with the secretion of mantle dentine by odontoblasts that is also a trigger for the beginning of amelogenesis. The Hertwig's root sheath derives from the cervical loop of the inner and outer enamel epithelium.

Figure 1.5. Figure 5. – Section of tooth – enamel and dentine formation
Epithelial-mesenchymal interactions during tooth development

It is important to note that both the epithelial enamel and the ectomesenchymal dentino-pulpal complex develop as a result of epithelial-mesenchymal interactions. Transcription and growth factors from the inner enamel epithelium impact the undifferentiated cells of dental papilla make them proliferate and differentiate into an odontoblast monolayer. The developing odontoblasts produce dentine to fulfill the main function of the pulp. On the other hand the inner enamel cells can not turn into ameloblasts without transcription and differentiating factors released from the dental papilla. Animal studies show that the enamel organ and dental papilla can be separated, however this results in the immediate arrest of development.

Figure 1.6. Figure 6. – Enamel organ and dental papilla
The size and shape of the tooth are also the result of epithelial-mesenchymal interactions. The Barx1 gene is expressed in the mesenchyme and its expression pattern follows a gradient along the dental arch. In the area corresponding to the incisors its expression is extremely low. The expression level grows towards the premolars and reaches a maximum in the mesenchymal cells of the molars. The leading role of mesenchyme in controlling tooth shape was proved as following: enamel organ was isolated in vitro from the mesenchyme and than recombined. When mesenchyme from a molar was combined with the enamel organ from an incisor, the tooth became a molar. And vice versa. Dental papilla isolated from an incisor combined with the enamel organ from a molar gave rise to an incisor.

**Figure 1.7. Figure 7. – Basal membrane divide ameloblasts and odontoblasts**

**Figure 1.8. Figure 8. – Control of tooth shape – ectomesenchymal dominance**
Differentiation according to epithelial-mesenchymal interactions is based on classical intercellular communication. Its basis is the secretion of a certain factor and the binding of this ligand to the specific receptor of another cell resulting in modification of the function of the target cell.

**Figure 1.9. Figure 9. – Effects of differentiation and growth factors**

In teeth this intercellular communication is initiated with the release of BMP-4 (bone morphogenic protein 4) and FGF-8 (fibroblast growth factor-8) from the early enamel organ, which cause the activation of the Msx1 gene in the dental papilla and thus activate a multistage cascade reaction. During this reaction, programmed information exchange is going on between the epithelium and the mesenchyme that leads to the development of enamel and dentine, to the building up of the tooth and to its eventual eruption.

**Figure 1.10. Figure 10. – Components of the regulation**
Large number of transcription and differentiation factors participate in this programmed series of information exchanges. There are numerous factors among them of which their function is redundant, such as various BMPs and BMP receptors. Thus the loss of their function does not cause a serious disturbance of tooth development.

Figure 1.11. A model of the molecular regulation of tooth development from initiation to crown morphogenesis.
On the other hand for example the mutation and loss of function of the Runx2 gene arrests the tooth development in a very early embryonic phase, in the bud stage. Loss of both copies of another gene, the ectodysplasin (EDA), does not completely block tooth development but it leads to a reduction in the number of teeth and the teeth that are formed are vestigial and unable to functioning normally.

Figure 1.12. Figure 12. – Oligodontia in a human patient with hypohydrotic ectodermal dysplasia (HED) – The ectodysplasin gene is crucial for tooth development

If the program runs regularly then all the structural elements of the tooth will develop and the tooth will erupt at the right time.

Figure 1.13. Figure 13. – Molar longitudinal section
1. Test – Development of tooth germ (answers)

1. Cell phenotype that is absolutely necessary for tooth formation:
   A. epithelial
   B. mesenchymal
   C. both epithelial and mesenchymal
   D. none of them

2. The first mineralized structure during tooth development:
   A. predentin
   B. mantle-dentin
   C. intertubular dentin
   D. peritubular dentin

3. The borderline of enamel organ facing preodontoblasts:
   A. inner enamel epithelium
   B. outer enamel epithelium
   C. papilla
   D. folliculus
   E. secretory ameloblasts

2. 1.2. Fibers and extracellular matrix of hard tissues – Gabor Varga
The development of hard tissues starts with the synthesis of their organic extracellular matrix by specialized cells (osteoblasts, cementoblasts, odontoblasts and ameloblasts). This protein rich basic structure has an important role both in the initiation and the completion of mineralization. The main component of hard tissues is collagen. Although important in the building of the basic structure collagen does not itself influence the formation of the first crystallites. There is a significantly smaller amount of non-collagenous proteins (glycoproteins, phosphoproteins, proteoglycans, serum proteins) and these are the components that are responsible for the initiation, facilitation, modification and even for the inhibition of mineralization.

**Figure 1.14. Figure 1. – Extracellular matrix of hard tissues**

<table>
<thead>
<tr>
<th>Extracellular matrix of hard tissues</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Inorganic components of bone</strong></td>
</tr>
<tr>
<td>- Water (approx. 5-30%)</td>
</tr>
<tr>
<td>- Minerals (approx. 45%, variable depending on the tissue)</td>
</tr>
<tr>
<td>- Hydroxyapatite and other Ca-phosphate and Ca-carbonate salts (stores easily mobilized from bone)</td>
</tr>
<tr>
<td>- Trace elements (Zn, Cu, Sn, Mg, F)</td>
</tr>
<tr>
<td><strong>Organic components</strong></td>
</tr>
<tr>
<td>- Structural proteins (traces-30%, variable depending on the tissue)</td>
</tr>
<tr>
<td>- Collagenous and non-collagenous proteins</td>
</tr>
<tr>
<td>- Bioactive regulatory peptides - (very small quantity)</td>
</tr>
</tbody>
</table>

The protein content of the enamel secreted by epithelial cells differs considerably from that of the connective tissues that constitute most hard tissues. To read about enamel protein content in detail see the chapter on amelogenesis.

**Figure 1.15. Figure 2. – Most important protein components of bone and dentin**

<table>
<thead>
<tr>
<th>Most important protein components of bone and dentin</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Collagens</strong></td>
</tr>
<tr>
<td><strong>Non-collagen proteins in hard tissues:</strong></td>
</tr>
<tr>
<td>- Glucoproteins</td>
</tr>
<tr>
<td>- Proteoglycans</td>
</tr>
<tr>
<td>- Gla proteins</td>
</tr>
<tr>
<td>- Blood plasma proteins</td>
</tr>
<tr>
<td>- Phosphoproteins</td>
</tr>
<tr>
<td><strong>Most important components of enamel</strong></td>
</tr>
<tr>
<td>- Amelogenin</td>
</tr>
<tr>
<td>- Enamelin</td>
</tr>
</tbody>
</table>

The matrix of connective tissue consists mainly of collagen. This is not a single molecule but rather the name of a family of molecules. **Type 1** collagen is the dominant type in the mineralized tissues. Its structure is built up from three left-coiled helices that are organized in a right-coiled triple helix.

**Figure 1.16. Figure 3. – Collagen – three polypeptide chains forming a rope**
All molecules classified as collagens have a triple helix region. The tissue distribution of different collagens is widespread.

**Figure 1.17. Figure 4. – Types and distribution of collagen**

<table>
<thead>
<tr>
<th>Type</th>
<th>Polypeptide composition</th>
<th>Distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>[α1(I)]₂ [α2(I)]</td>
<td>Skin, bone, tendon, cornea, blood vessels</td>
</tr>
<tr>
<td>II</td>
<td>[α1(II)]₃</td>
<td>Cartilage, intervertebral disk</td>
</tr>
<tr>
<td>III</td>
<td>[α1(III)]₂</td>
<td>Fetal skin, blood vessels</td>
</tr>
<tr>
<td>IV</td>
<td>[α1(IV)]₂ [α2(IV)]</td>
<td>Basement membrane</td>
</tr>
<tr>
<td>V</td>
<td>[α1(V)]₂ [α2(V)]</td>
<td>Placenta, skin</td>
</tr>
</tbody>
</table>

Collagen is produced as a water-soluble precursor. During the conversion of procollagen to collagen, both the C- and N- termini of the molecule are cleaved in the extracellular space. Collagen consists of three independent polypeptide chains, called the α-chains, each comprising 1056 amino acids. Each α-chain has a left-handed helical structure and when they coil around each other they form a right-handed super helix that is similar to a rope. The amino acid composition of the chains has a characteristic periodicity; it contains repeated glycine-X-Y tripeptides, where X and Y are amino acids different from glycine. The residues in the X and Y positions are usually proline and lysine. There are typically 338 Gly-X-Y repeated triplets in the α-chains. The length of the triple helix (consisting of the three α-chains) is 297 nm. The glycine is the smallest amino acid and its predominance in the chains allows tight coiling. Proline and hydroxy-proline are limited in their ability to rotate in contrast to other amino acids therefore these amino acids enhance the stability of the triple helix structure. Collagen molecules combine to form fibers with periodic cross-bonds.

**Figure 1.18. Figure 5. – Structure of procollagen**
The collagen chains are synthetized in the rough endoplasmic reticulum and then undergo posttranslational modifications, hydroxylation and glycosylation. The coiling into a triple helix is catalyzed by specific enzymes. The prosthetic groups of proline hydroxylase and lysine-hydroxylase both contain ferrous ions (Fe$^{2+}$) and use oxygen. Ascorbic acid (vitamin C) is also essential for the function of both enzymes as a cofactor. This is the reason why vitamin C deficiency causes scurvy. An abnormal polypeptide sequence of collagen causes the hereditary disease osteogenesis imperfecta (also known as brittle bone disease).

The conversion of procollagen to collagen happens in the extracellular space and is facilitated by specific proteolytic enzymes. When the cross binding is completed the collagen becomes insoluble.

**Figure 1.19. Figure 6. – Overview of collagen biosynthesis**

**Figure 1.20. Figure 7. – Hydroxylation during collagen biosynthesis**
The stability of collagen is really important. Most of the proteolytic enzymes are unable to cleave it because of its cross-bonds and insolubility. Therefore, special metalloproteases (collagenases) are needed to degrade this molecule. These enzymes can cleave all the three chains at the same time.

**Figure 1.21. Figure 8. – Stages in collagen synthesis – rope formation**

**Figure 1.22. Figure 9. – Enzymatic cleavage of collagen by mammalian collagenases**
Non-collagenous proteins are known to interact with the surface charges of the hydroxyapatite crystals and thus influence the mineralization. There are many similarities but also significant differences between these proteins. Because of their multiple negative charges, these biomineralization proteins are intrinsically disordered proteins, even more so than transcription factors. The disordered structure and its flexibility can play a role in the rapid formation and strength of the interactions due to the fast conformational changes of these proteins.

Figure 1.23. Figure 10. – Interactions between hydroxyapatite crystals and ionic substances

Diagram illustrating the reactive major constituent ions of the hydroxyapatite mineral (OH-Apatite) of calcified bone matrix. Ionic substances in the crystal environment have a high potential for interaction with the mineral

Figure 1.24. Figure 11. – Disorder frequency of amino acid chains of proteins participating in various biological functions
Most of the proteins present in connective tissues are **glycoproteins**. These contain one or more carbohydrate group typically **sialic acid**. Sialoproteins are very acidic and they represent 10% of the non-collagenous proteins of the bone. They contain 20% carbohydrate by weight. The main members of this group, **sialoprotein I** (osteopontin) and **sialoprotein II** also contain large amounts of phosphate.

**Figure 1.25. Figure 12. – Sialic acid, a major constituent of sialoproteins**

Most important proteins of the group: osteopontin, bone sialoprotein

The modification by carboxylase in the γ position of glutamate in the peptide chains leads to the formation of **γ-carboxyl-glutamate** (Gla). This enzymatic reaction requires vitamin K and bicarbonate. Gla groups act as calcium-binding domains. Gla proteins include **osteocalcin** (OC), **bone Gla protein** (BGP) and **matrix Gla protein** (MGP). In vitamin K deficiency Gla proteins are not carboxylated and their incorporation may decrease significantly. However in this case bone structure does not change dramatically, there are only some alterations in the epiphysis. This finding led to recognition that these proteins actually inhibit or at least modulate the calcification. They also slow down the growth of hydroxyapatite crystals in vitro.

**Figure 1.26. Figure 13. – Structure of proteoglycans**
Proteoglycans. Proteoglycans are conjugates consisting of proteins and glycosaminoglycan prosthetic groups. Glycosaminoglycans are built up from repeated units of two different sugars. One subunit is a hexosamine (D-galactosamine or glucosamine) that can be sulphated, and the other unit is glucuronic acid or galactose. They contribute to the jelly-like consistency of the matrix and can slow down mineralisation by their calcium binding property. Proteoglycans accumulate in the non-mineralized connective tissue and their amount decreases significantly during the mineralization process.

Figure 1.27. Figure 14. – Formation of γ-carboxyglutamyl residues

In phosphoproteins phosphate groups bind covalently to the peptide chain as phosphothreonine or phosphoserine. The affinity of phosphate ions for Ca\(^{2+}\) is very strong and the matrix-bound phosphate has a key role in the initiation of mineralization. In addition they serve as phosphate donors in the process.

Two well-known phosphoproteins in bone are osteopontin and osteonectin. The phosphoprotein of dentine, dentine sialophosphoprotein (DSPP), also known as phosforin, only contains phosphoserine.

Figure 1.28. Figure 15. – The most important amino acids in hard tissue phosphoproteins
Plasma proteins can be extracted from both bone and dentine, where significant amounts of albumin and α₂HS glycoprotein can be detected. However, the relevance of this phenomenon is not known. The inert collagens do not have a direct role in the mineralization. The non-collageneous proteins mentioned above are primarily structural components that act as the initiators of mineralization or as enhancers or inhibitors of the process. At the same time, numerous peptides and proteins present in small amounts take part in the mineralization process as transcription and differentiation factors, and as regulators of cell motility or enzymes.

2.1. Test – Fibers and extracellular matrix of hard tissues (answers)

1. Which protein gene mutations may lead to Osteogenesis imperfect?
   A. collagen I
   B. collagen IV
   C. enamelin
2. Which one of the following structures do not contain collagen?

A. Unmatured enamel  
B. Mature dentin  
C. Unmature dentin  
D. Mature enamel

3. The absence of which vitamin leads to incomplete hydroxylation of key amino acids of collagen chain?

A. vitamin A  
B. vitamin B  
C. vitamin C  
D. vitamin K

3. 1.3. Osteogenesis – Gabor Varga

Bone is the organ that has the largest mineralized tissue content. Mesodermal cells play the major role in bone development, even though bones of skull and jaws develop in part by mesenchymal transdifferentiation of ectodermal cells. In addition to the structural function, bones contain bone marrow, where hematopoiesis occurs. Bone also plays a key role in calcium and phosphate metabolism, and in the regulation of their levels in serum.

Figure 1.30. Figure 1. – Bone

<table>
<thead>
<tr>
<th>Bone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specialized connective tissue with mineralized extracellular matrix</td>
</tr>
<tr>
<td>Skeleton and support</td>
</tr>
<tr>
<td>Place of hemopoiesis</td>
</tr>
<tr>
<td>Ca(^{2+}) and phosphate reserve</td>
</tr>
</tbody>
</table>

Bones can be classified as long bones, flat bones (such as certain bones of the skull), short bones and irregular bones. According to their macrostructure, long bones are divided into diaphysis, metaphysis and epiphysis. Bone is not a uniformly solid material. The hard outer layer of bones consists of compact bone (substantia compacta) which surrounds an inner spongy part called trabecular bone (substantia spongiosa), which is composed of a network of rod- and plate-like elements.

Figure 1.31. Figure 2. – Bone anatomy
Compact bones are built up from cylindrical osteons which run parallel to the long axis of the bone. The concentric layers, or lamellae, of osteons surround the Haversian canals that contain the blood vessels and nerves. Bone turnover occurs both during childhood and during adult life. Bone remodeling (bone turnover) is the process of resorption followed by osteon replacement. Within concentric osteon lamellae osteocytes are trapped in the spaces called lacunae. Interstitial lamellae are located between osteons.

**Figure 1.32. Figure 3. – Macromorphological structure of bone**

**Figure 1.33. Figure 4. – Osteocytes in compact bone**
Bone extracellular matrix has a specific composition. Bone contains 20% water, 45% hydroxyapatite, other salts of Ca (phosphate and carbonate), and trace elements (Zn, Cu, Sn, Mg, F). In addition, it contains 35% organic component, mostly (85 to 90%) collagen and also non-collagenous proteins, which play a role in enhancing or inhibiting mineralization.

**Figure 1.34. Figure 5. – Major constituents of bone**

The most important cell types in bone are the *osteoblasts*, *osteocytes*, and *osteoclasts*. Osteoblasts build up, while osteoclasts resorb the bone. After the completion of bone formation some osteoblasts remain within the newly formed bone and become osteocytes trapped in the bone structure that they built.

**Figure 1.35. Figure 6. – Major cellular and matrix constituents of bone**
The two main mechanisms of bone formation are endochondral ossification and intramembranous ossification. Endochondral ossification occurs in three steps performed by specialized cells which can separate and resorb different materials. A soft cartilaginous base is formed first, onto which the bone structure is then deposited. The deposition of calcium and phosphate containing minerals in the cartilaginous structure creates the substructure of bone. Finally this preliminary structure forms and hardens into bone during remodeling. Abnormalities during bone formation lead to malformations and bone disorders.

**Figure 1.36. Figure 7. – Bone formation 1 – intramembranous ossification**

During intramembranous ossification bone is built on sheet-like connective tissue membranes, resulting in the formation of so-called intramembranous bones, examples of which include some of the flat bones of the skull and some irregular bones. At the beginning of intramembranous ossification, connective tissue sheets appear first. Osteoblasts then migrate to the sheets and lay down a bone matrix. Osteoblasts get trapped within the bone matrix and are then called osteocytes.

**Figure 1.37. Figure 8. – Bone formation 2 – Endochondral ossification**
Bone formation 2 – Endochondral ossification

- The process of bone formation occurs in three stages, orchestrated by specialized bone cells that secrete and absorb materials as needed.
- First, a soft cartilage-based foundation is laid, upon which mature bone will solidify.
- Then, minerals containing calcium and phosphate are deposited throughout the foundation, creating a framework for the bone.
- Finally, this raw material is sculpted and hardened into bone.
- Missteps in this process can result in developmental defects and bone diseases.

Figure 1.38. Figure 9. – Endochondral ossification: steps for bone replacement of cartilage

Figure 1.39. Figure 10. – Ossification: long bones continue to grow and elongate (lengthen) though adolescence
Osteoblasts, the cells responsible for the production of bone matrix, differentiate from bone marrow stromal stem cells of mesenchymal origin. The osteogenic lineage includes stem cells, osteoprogenitor cells, preosteoblasts, osteoblasts and osteocytes. The same stromal stem cell is responsible for the initiation of several other connective tissue differentiation pathways. However, these pathways diverge early on and the interconversion of committed cell forms is very limited under normal conditions.

Figure 1.40. Figure 11. – Mesengenic process

A large number of growth factors and differentiation factors are responsible for the differentiation and proliferation of osteoblasts. Extracellular ligands regulate these processes by binding their specific receptors and activating downstream G protein, tyrosine kinase and serine-threonine kinase dependent signalling pathways. Intracellular steroid receptors are also involved in the regulation of these pathways.
Figure 1.41. Figure 12. – Main regulatory factors of osteogenesis

Certain growth factors are released from the bone matrix during tissue remodeling.

Figure 1.42. Figure 13. – Bone formation is regulated by bioactive peptides such as BMPs and also by other growth factors

In addition, the extracellular matrix has an important effect on differentiation via the activation of integrin receptors which influences intracellular, autocrine and paracrine mechanisms.

Figure 1.43. Figure 14. – Role of the extracellular matrix in bone formation and metabolism
The repair of bone after an injury or fracture occurs via several steps. To briefly summarize the process of regeneration, the first proliferative phase is followed by an early remodelling. The original structure, or one very close to it, is finally restored by late remodelling.

**Figure 1.44. Figure 15. – Bone response to injury**

### 3.1. Test – Osteogenesis (answers)

1. **Key peptide group in bone morphogenesis:**
   
   A. AMP  
   B. BMP  
   C. CMP  
   D. DMP
2. Cells of alveolar bone:
   A. osteoblasts
   B. odontoblasts
   C. cementoblasts
   D. ameloblasts

3. Cells that are not characteristic components of tooth formation
   A. odontoblasts
   B. cementoblasts
   C. ameloblasts
   D. osteoclasts

4. 1.4. Dentinogenesis and disturbances; formation of primary-, secondary- and tertiary dentin; dentin permeability – Gabor Varga

Dentin, a tissue of ectomesenchymal origin, forms most of the mass of the teeth. Its properties are similar to bone in many respects, but it has no trapped cells inside and is not involved in calcium phosphate metabolism. Accordingly, its remodelling and regeneration is limited.

**Dentin structure and formation.** Cells produce dentin, cementum and bone first by creating an organic matrix that contains high levels of collagen. Inorganic calcium phosphate is then deposited in this matrix. Dentin contains 70% minerals, 20% organic material and 10% water. The main organic components are collagen and a number of non-collagenous proteins that have a role in the mineralisation process.

**Figure 1.45. Figure 1. – Constituents of dentin**

Types of dentin are classified based on their formation. **Mantle dentin** has an amorphous structure with no tubules. It is produced by newly differentiated odontoblasts with processes not yet developed, located at the enamel-dentin boundary. **Primary dentin** is formed subsequently. It has a characteristic tubular structure, and forms the main mass of the tooth. **Secondary dentin** is produced after root formation is complete, throughout the entire life. **Tertiary or reparative dentin** is produced in response to harmful external stimuli and is a barrier formed at the damaged surface.

**Figure 1.46. Figure 2. – (A) Primary, (B) secondary and (C) tertiary or reparative dentin**
Tooth development starts as a result of ectodermal-mesenchymal interactions. The dentin is produced by dental papilla odontoblasts which form a single layer between the dentin they produced and the pulp. Unlike bone tissue, dentin does not contain cells, only odontoblast processes. Odontoblasts are of ectodermal origin; the differentiation of their blastoid progenitors starts in response to triggers that come from the inner enamel epithelium.

During initial mantle dentin formation, odontoblasts secrete (in addition to collagen) calcium phosphate via matrix vesicles. Simultaneously, odontoblasts become elongated, polarized, and are arranged in a tight columnar pattern. With the thickening of the dentin layer, the odontoblast cell body retreats, leaving behind thin cytoplasmic processes in the newly formed microscopic channels called dentinal tubules. In contrast to ameloblasts, which are of epithelial origin, an odontoblast never forms tight junctions around its entire circumference. The cell body is very rich in endoplasmatic reticulum and in Golgi apparatus. However, beyond the terminal bar, odontoblast processes are devoid of these organelles.

**Figure 1.47. Figure 3. – Differentiation of odontoblasts**

**Figure 1.48. Figure 4. – Formation of mantle dentin during the early phase of mineralization**
Simultaneously with mantle dentin mineralisation, stable odontoblast processes and surrounding dentin tubules are formed by the cells. **Predentin** is located between odontoblast cell bodies and the mineralisation front, and is a partially mineralised ground substance.

**Figure 1.49. Figure 5.** – Dentin is produced by odontoblasts

**Figure 1.50. Figure 6.** – Mature secretory odontoblast
The secretory activity of odontoblasts shows a distinct spatial pattern. Cells produce collagen, proteoglycans and calcium ions at their neck, at the root of the processes. After demineralization, the organic structural framework of dentin, composed of collagen fibers, can be clearly observed. However, the exocytosis of phosphoproteins and proteases occurs from the processes at the mineralisation front. Proteases constantly degrade proteoglycans and phosphoproteins. Decreasing the amount of proteoglycans promotes mineralization, while phosphoproteins are instrumental by serving as phosphate donors.

**Figure 1.51. Figure 7. – Involvement of hard tissue proteins in mineral formation**

Dentin sialophosphoprotein (DSPP) or phosphoforin is a strongly acidic protein produced by odontoblasts. It is the most important non-collagenous component of dentin, and is also essential as a heterogenous mineralisation nodule and a phosphate donor. Its importance is shown by the fact that its genetic mutations cause dentinogenesis imperfecta which is associated with significant dentin damage.

**Figure 1.52. Figure 8. – Dentinogenesis Imperfecta**

Characteristics

- autosomal dominant (1:8000)
- Opalescent teeth
- Irregular structure and hypomineralized dentin, grey or dark yellow/brown
**Dentin permeability.** The tubular structure of dentin makes it permeable if for any reason, such as tooth decay or abrasion, it opens to the surface. The formation of dentin tubules is defined by the odontoblast processes.

**Figure 1.53.** Figure 9. – Collagen fibers around tubules

The diameter of dentin tubules is 1 to 3 µm so they act as a bacterial filter by preventing the entry of microorganisms. Their density is greatest in the upper third of the pulp chamber, and is lower towards the root. The processes of the odontoblasts contain dentin fluid and mineral deposits.

**Figure 1.54.** Figure 10. – Components of dentin

**Figure 1.55.** Figure 11. – The empty dentin tubules provide the basis for permeability longitudinal section
The opening of dentin tubules results in increased activity of sensory nerves in the pulp, thus causing pain, so it is a major problem in dental practice. Through the dentin tubules, fluid and mass transport can occur basically in two ways: \textit{convective transport}, which facilitates mass transfer, and \textit{diffusive transport}, whereby solutes are transported.

**Figure 1.56.** Figure 12. – Permeability: the number and the diameter change depending on the dentin tubules

**Figure 1.57.** Figure 13. – Neuronal network of pulp/dentin
Convective transport has the main role and can be driven by the pressure difference between the outer and inner pulp space, for example in inflammation, or tooth decay caused by high-sugar solutions, or when the dentin is being dried by air or cotton wool, or when biting on a dislocated filling.

The extent of fluid flow is described by the Hagen-Poiseuille equation. The extent of convective transport is determined above all by the diameter of the dentin tubules. The dentin tubule is not an ideal pipe, because the odontoblast processes, collagen fibers, nerves and also mineral and protein precipitates narrow it. Thus blockage or at least narrowing of these tubules can be important in dental practice.

Figure 1.58. Figure 14. – Hagen–Poiseuille equation – fluid movement – basis of the hydrodynamic theory

\[ V = \frac{n\Delta P r^4}{8\eta L} \]

V - fluid movement, \( \Delta P \) – pressure difference, \( r \) – radius, \( \eta \) – viscosity, L - length

Figure 1.59. Figure 15. – Increase of outward fluid movements from the pulp during inflammation

The role of diffusive transport is secondary because the penetration of substances by diffusion is much slower than the development of pressure differences. Diffusive transport results in harmful chemicals appearing in the pulp. Of these, toxins released from the cell wall of bacteria are especially important. Toxins may diffuse through the channels to the pulp. The rate of diffusive transport is described by Fick’s second law. The rate of diffusion is determined especially by the diffusion area, the length of the tubule and the concentration difference.

Figure 1.60. Figure 16. – Diffusion – Fick’s 2nd law

\[ J_s = \frac{D_s \Delta C}{\Delta x} \]

\( J_s \) – diffusion rate, \( D_s \) – diffusion coefficient, \( \Delta C \) – concentration gradient, \( \Delta x \) - length

Figure 1.61. Figure 17. – In a caries lesion, cariogenic bacteria invade the dentinal tubules, demineralizing sclerotic and peritubular dentin in the process
Dentine permeability is extremely important in the so-called hypersensitivity, which is often caused by the very thin or partially missing overlapping area between enamel and cementum at the tooth neck. Treatment options include the closing of tubules with poorly soluble calcium salts or protein precipitates. In addition, a commonly method in dental practice is to hyperpolarize or depolarize nerve fibers by altering the ionic milieu, for example by using a high potassium toothpaste.

**Figure 1.63. Figure 19. – Dentine hypersensitivity – Treatment**

- To obliterate tubules by deposition of calcium salts
- To block tubules by protein precipitation
- To hyperpolarize or depolarize nerve fibers by altering ionic environment

**4.1. Test – Dentinogenesis and disturbances; formation of primary-, secondary- and tertiary dentin; dentin permeability (answers)**
1. Caries induced tooth pain is primarily induced by:
A. bacterial invasion
B. opening of dentin tubules
C. increased depolarisation
D. increased hyperpolarisation

2. Usual diameter of dentin tubules:
A. 0.2-1 μm
B. 20-100 μm
C. 0.2-1 nm
D. 2-10 μm

3. Dentin hypersensitivity can be diminished by ….. of dentin tubules
A. drying
B. cleaning
C. closure
D. opening

5. 1.5. Amelogenesis – Gabor Varga

Tooth enamel is the hardest tissue of the body: the matured enamel consisting of 96% hydroxyapatite crystals and containing only a few percent of protein and water. It perhaps surprising that enamel is secreted by epithelia. However, it is very different from other epithelial products like the fluid-rich secretions of salivary glands, pancreas and liver.

Figure 1.64. Figure 1. – The arrangement of ameloblasts during enamel formation

The secretion of enamel by ameloblasts is a two-stage process. The first step is the building of a slightly mineralized matrix structure and the second step is the remodelling of this matrix to a highly mineral-rich structure.

Figure 1.65. Figure 2. – Amelogenesis
Ameloblasts originate from the inner enamel epithelium and have several forms according to their functional state during their life cycle. These are the morphogenic, inductive, early secretory, late secretory, maturation ruffled-ended, maturation smooth-ended and protective forms.

Figure 1.66. Figure 3. – Formal and structural changes of ameloblasts during enamel formation

These ameloblast forms change according to a strict time schedule and each has a specific functional role in particular phases of amelogenesis.

Figure 1.67. Figure 4.
In the secretory phase of amelogenesis ameloblasts are tall, columnar cells rich in mitochondria, endoplasmic reticulum and Golgi apparatus according to their active transport processes. Ameloblasts can be clearly distinguished from odontoblasts by their tight junctions. Ameloblasts have characteristic epithelial tight junctions throughout their lifetime which close the intracellular space and separate the apical and basolateral surfaces of the cell. These tight junctions allow the maintenance of extreme concentration gradients between the apical and basolateral extracellular spaces. The terminal bars prevent the entry of organelles into the Tomes process. The Tomes process of the ameloblast is a short, lance-shaped structure that provides the surface for the secretory transport processes. Calcium and phosphate ions (necessary for mineralization) are actively transported into the mineralization space in a basolateral to apical direction. The molecular mechanism of this mineral transport process is only partially understood at present.

Figure 1.68. Figure 5. – Secretory ameloblasts – formation of prismatic enamel (PE) and interprismatic enamel (IPE)
The enamel matrix is not homogeneous. During enamel development central and lateral (also called prismatic and interprismatic) crystal rods are formed. Central rods are formed right below the Tomes process and have higher density. Interprismatic rods are not directly below the processes but are lateral to them, therefore the mineralization efficiency is lower there and less dense crystal is formed than in the prismatic area.

After the secretory phase the enamel is 30% mineralized. In this phase the mineral content is condensed into thin, parallel crystal ribbons while the space between the crystals is filled by matrix with high amelogenin content.

**Figure 1.69. Figure 6. – Parallel running crystallites (Kr) in the early phase of enamel development**

![Image of parallel running crystallites](image)

**The enamel maturation.** During the maturation phase ameloblasts change their morphology and cyclically transform between ruffle-ended and smooth-ended forms. The result of this cyclical modulation is that ameloblasts have a double function in this phase: they have to secrete calcium and phosphate and neutralize the protons liberated during hydroxyapatite crystal growth while they also have to reabsorb and degrade amelogenin cleaved by kallikrein-4 and MMP-20.

**Figure 1.70. Figure 7. – Maturation ameloblast phenotypes**

Ruffle-ended and smooth-ended maturation ameloblasts cycle back and forth during the maturation phase. Cycling of the two phenotypes involves extensive remodeling of the distal cytoplasm and junctional complexes at both ends of the cells. The Golgi complexes (G) and the lysosomal (L) apparatus are well developed in both cell configurations. Zonula adherens (Za) and zonula occludens (Zo) shift from distal position in the ruffle-ended ameloblasts to a proximal position in the smooth-ended ameloblasts. Mitochondria (M) are located primarily in the distal cytoplasm. Endosomes (E) containing enamel matrix are present both in the ruffle-ended and smooth-ended ameloblasts. The ruffle ended surface primarily supports electrolyte exchange while the smooth ended form is for cell recovery and protein absorption.

The essence of the process is that the protein matrix with high amelogenin content is degraded while in parallel the crystals expand in thickness until the whole matrix is eliminated and replaced by the tightly packed and practically impermeable crystal structure. The smooth-ended ameloblasts produce and secrete enzymes to
In the smooth-ended form, electrolyte transport is the primary function. But its mechanism and control is not known in detail. The papillary cells above the ameloblasts may have an important role in supporting the transport activity of the ameloblast cells and the removal of unnecessary materials. **The ruffle- and smooth-ended forms interconvert cyclically.**

**Figure 1.71.** Hypothetic model for pH regulation by ruffle ended ameloblasts to neutralize liberated H⁺

The result of amelogenesis is an almost completely impermeable structure. There is some density difference between the crystal rods and the matrix but the whole enamel is 96% mineralized and it is the hardest tissue in the body.

**Figure 1.72.** Cross sectional arrangement of enamel cristal rods (prisms)

Cross-sectional arrangement of the prisms in human enamel. The position of each ameloblast in relation to the prism outline is represented by the superimposed boundary lines (BL). Each arcade-shaped prism is surrounded by interprismatic enamel (IPE), which is contributed by the secretions of seven ameloblasts. Note the offset arrangement of the horizontal rows of arcades. PE: Prismatic enamel; S: Sheath region.

**Figure 1.73.** Structure of the matured enamel
After the maturation phase, the ameloblasts de-differentiate, becoming short and cuboid rather than tall and columnar, and make a protective layer until eruption.

Secretory ameloblasts secrete numerous proteins whose functions are not completely clear but are essential for the construction and remodelling of enamel.

**Figure 1.74. Figure 11. – Amelogenesis – list of enamel proteins**

<table>
<thead>
<tr>
<th>Amelogenesis – list of enamel proteins</th>
</tr>
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<tbody>
<tr>
<td>- Amelogenin</td>
</tr>
<tr>
<td>- Enamelin</td>
</tr>
<tr>
<td>- Ameloblastin</td>
</tr>
<tr>
<td>- Amelotin</td>
</tr>
<tr>
<td>- Tuftelin</td>
</tr>
<tr>
<td>- Osterix</td>
</tr>
<tr>
<td>- Proteinases: enamelysin - MMP-20, kallikrein 4 – KLK4)</td>
</tr>
<tr>
<td>- Phosphatases</td>
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</tbody>
</table>

*Amelogenin* is coded by two genes, by AMELX and AMELY located on the X and Y chromosomes. Significantly more protein is produced from AMELX. Among the enamel proteins amelogenin is present in the largest quantity. Its exact role is unknown. It is a small, globular protein when produced, which then forms **nanospheres** during the self-assembly of the proteins. The nanospheres interact with each other and create grid-like sheets to control the formation and orientation of enamel crystals. During the early phase of maturation enamelysin, and later kallikrein-4, disrupt the grid structure and break down the nanospheres. The building blocks become degraded and can be taken up by the cells while the space left behind is filled with the thickening crystals. By the end of the maturation phase amelogenin has almost completely disappeared from the enamel.

**Figure 1.75. Figure 12. – Concept of the role of amelogenins in the mineralization of enamel**
Ameloblastin is the second most abundant protein in the enamel. Its functions are not completely established but one of them is to influence ameloblast function. Extracellular ameloblastin initiates cell differentiation via specific receptors. It also inhibits cell proliferation by the activation of tumor suppressor gene 21 and p27. In addition it supports amelogenin secretion by blocking the transcription factor MSX2. Overall it supports differentiation and matrix formation.

Figure 1.76. Figure 13. – Role of ameloblastin in the regulation of ameloblast function

Enamelin is secreted by the ameloblasts. Although its exact function is unknown, it is able to interact with amelogenin and other matrix proteins and regulates the growth of enamel crystals.

Enamelysin (MMP-20) is a protease produced in the early stage that is able to degrade the enamel matrix proteins. Kallikrein-4 (KLK-4), also called protease 2, is produced and secreted in the maturation phase. It eliminates proteins that were not degraded by enamelysin.

Mutations of the AMELX gene can cause amelogenesis imperfecta (AI). Mutations of AMELY are unknown. Deletion of the AMELX gene in mice results in the formation of a very thin enamel. Mutations in ameloblastin can also cause AI. In addition the thickness and prismatic organisation of enamel are found to decrease in enamelysin knock-out mice. Three different mutations of enamelin have been described so far, and active proteases can also cause the disease.

Figure 1.77. Figure 14. – Amelogenesis imperfecta
Figure 1.78. Figure 15. – Structure of the X-chromosomal copy of the human amelogenin gene

The bar segments represent the introns and the boxes (1 through 7) correspond to the exons. The nucleotide numbers are indicated below the exons. (Adapted from Simmer et al.)

5.1. Test – Amelogenesis (answers)

1. Cells forming enamel:
   A. osteoblasts
   B. odontoblasts
   C. cementoblasts
   D. ameloblasts

2. Percentual representation of amelogenin among proteins in non-maturated enamel:
   A. 5-10%
   B. 23-30%
C. 40-50%
D. 60-70%
E. 90%

3. Which protein gene mutations may lead to Amelogenesis imperfecta

A. collagen
B. kallikrein-4
C. proteoglycan
D. fosfophorin


6.1. Mineral components of dental enamel, dentin and cementum

Bone contains the highest amount of mineralized tissue in the human body.

Hydroxy(l) apatite is a main component of bones and teeth. Its two main types in the teeth are: hydroxy(l) apatite, Ca₁₀(PO₄)₆(OH)₂; and fluorapatite, Ca₁₀(PO₄)₆(F)₂. Hydroxil ion is substituted by fluoride ion in fluorapatite, making the enamel more resistant to acid exposure. This is why fluoride is included in toothpaste.

Figure 1.79. Table 1. – Hard tissue composition of teeth and bone. Data correspond to 100 g dry weight

<table>
<thead>
<tr>
<th></th>
<th>Enamel</th>
<th>Dentine</th>
<th>Cementum</th>
<th>Bone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total inorganic</td>
<td>97</td>
<td>70</td>
<td>61</td>
<td>65</td>
</tr>
<tr>
<td>Total organic</td>
<td>1.5</td>
<td>20</td>
<td>27</td>
<td>25</td>
</tr>
<tr>
<td>Water</td>
<td>1.5</td>
<td>10</td>
<td>12</td>
<td>10</td>
</tr>
<tr>
<td>Ash</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium*</td>
<td>36.5</td>
<td>35.1</td>
<td>35.2</td>
<td>34.8</td>
</tr>
<tr>
<td>Phosphorus*</td>
<td>17.7</td>
<td>16.9</td>
<td>16.1</td>
<td>15.2</td>
</tr>
<tr>
<td>Ca/P ratio</td>
<td>1.63</td>
<td>1.61</td>
<td>1.71</td>
<td>1.71</td>
</tr>
<tr>
<td>Sodium*</td>
<td>0.5</td>
<td>0.6</td>
<td>0.9</td>
<td></td>
</tr>
<tr>
<td>Magnesium*</td>
<td>0.44</td>
<td>1.23</td>
<td>0.73</td>
<td>0.72</td>
</tr>
<tr>
<td>Potassium*</td>
<td>0.08</td>
<td>0.05</td>
<td>0.03</td>
<td></td>
</tr>
<tr>
<td>Fluoride*</td>
<td>0.01</td>
<td>0.06</td>
<td>&lt;0.05</td>
<td>0.03</td>
</tr>
<tr>
<td>Chloride*</td>
<td>0.3</td>
<td>0.01</td>
<td>0.13</td>
<td></td>
</tr>
<tr>
<td>Unashed (dry tissue):</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbonate</td>
<td>3.5</td>
<td>5.6</td>
<td>5.5</td>
<td>7.4</td>
</tr>
<tr>
<td>Pyrophosphate</td>
<td>0.022</td>
<td>0.1</td>
<td>0.07</td>
<td></td>
</tr>
</tbody>
</table>

**Enamel** is hardest and best mineralized tissue in the human body. It contains 96% anorganic substances, 1 to 2% organic substances while the remaining 1 to 2% is water. Its density is 3 g/ml. Mature enamel contains no collagen, in contrast to dentin and cementum wherein collagen is the major protein component. Mature enamel contains enamelin, while immature enamel also contains amelogenin.
Dentin contains 70% anorganic components, 20% organic components and 10% water. Its density is 2.1 g/ml.

Cement contains 61% anorganic material, 27% organic material and 12% water. Its density is 2.1 g/ml.

Organic components may serve two types of function:

1. Organic components with nucleator function
   - sulfated acidic glycoproteins,
   - phosphoproteins.

2. Matrix proteins – they can bind preformed small crystals, which can grow on the surface of the matrix
   - immature enamel: amelogenin, enamelin,
   - mature enamel: enamelin, TRAP (tyrosin rich protein – low molecular weight degradation product of amelogenin, as amelogenin is degraded during maturation),
   - dentin and cementum: collagen.

3. Regulatory proteins.

Stability of apatite crystals is greatly dependent on the size and ionic radius of incorporated ions.

**Figure 1.80. Table 2. – Ionic radius. Ionic radius of ions incorporated into apatite crystals**

<table>
<thead>
<tr>
<th>Substituents</th>
<th>Ionic radius (Å)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ca²⁺</td>
<td>0.99</td>
</tr>
<tr>
<td>Si⁴⁺</td>
<td>1.12</td>
</tr>
<tr>
<td>Pb²⁺</td>
<td>1.2</td>
</tr>
<tr>
<td>Ba²⁺</td>
<td>1.33</td>
</tr>
<tr>
<td>OH⁻</td>
<td>1.53</td>
</tr>
<tr>
<td>F⁻</td>
<td>1.36</td>
</tr>
<tr>
<td>Cl⁻</td>
<td>1.81</td>
</tr>
</tbody>
</table>

**Figure 1.81. Figure 1. – Structure of hydroxil-, fluoro- and chlorapatite.**
The figure shows the locations of hydroxid-, fluoride- and chloride ions between Ca²⁺ ions in the apatite crystals. It can be observed that F⁻, an anion with smaller ionic radius, is easily incorporated between Ca²⁺ ions, and may thus lead to a more stable crystal structure. Several favorable properties of fluoro-apatite are explained by more stable crystal structure (see in preventive dentistry).
Structurally ortho (PO$_4^{3-}$), pyro (P$_2$O$_7^{4-}$) and poly ((PO$_3^{n-}$)$_n$) phosphates are distinguished. In pathological calcification (calcium deposition) often pyrophosphates are also present in addition to orthophosphates. In addition to hydroxyapatite, intact enamel and dentin contain small amounts of other apatite substituents such as fluorapatite and chlorapatite.

**Figure 1.82. Table 3.** – Types of calcium phosphates. In the table, naturally occurring types of calcium phosphates are shown along with an indication of their occurrence. The different types are likely to interconvert into each other through a maturation process. Calculus contains almost all calcium phosphate compounds in addition to the amorphous form.

<table>
<thead>
<tr>
<th>Name</th>
<th>Formula</th>
<th>Abbreviation</th>
<th>Ca:P ratio</th>
<th>Occurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>brushite - dicalcium phosphate hydrate</td>
<td>CaHPO$_4$·2H$_2$O</td>
<td>DCPD</td>
<td>1.0</td>
<td>pathological calcifications; calculus, chondrocalcinosis, urinary stones</td>
</tr>
<tr>
<td>monoxide - dicalcium phosphate anhydrite</td>
<td>CaHPO$_4$</td>
<td>DCPA</td>
<td>1.0</td>
<td>calcium-phosphate cement, nutritional supplements, tabletting aids, toothpaste components</td>
</tr>
<tr>
<td>octacalcium phosphate</td>
<td>Ca$_8$(HPO$_4$)$_4$(PO$_4$)$_2$·5H$_2$O</td>
<td>OCP</td>
<td>1.33</td>
<td>dental and urinary calculi</td>
</tr>
<tr>
<td>whitlockite</td>
<td>Ca$_9$(Mg$_2$Fe$_3$)(PO$_4$)$_3$(HPO$_4$)</td>
<td>WH</td>
<td>1.41</td>
<td>dental calculus, dentine caries, urinary stones</td>
</tr>
<tr>
<td>β-tricalcium phosphate</td>
<td>β-Ca$_3$(PO$_4$)$_2$</td>
<td>β-TCP</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>amorphus calcium phosphate</td>
<td>Ca$_3$(PO$_4$)$_2$·nH$_2$O</td>
<td>ACP</td>
<td>1.2–2.2</td>
<td>calcification sites, mitochondria</td>
</tr>
<tr>
<td>hydroxyapatite</td>
<td>Ca$_5$(PO$_4$)$_3$(OH)$_2$</td>
<td>HA</td>
<td>1.67</td>
<td>bone, enamel, dentine, calculus</td>
</tr>
</tbody>
</table>

**Biological apatites, also known as dahllites** are poorly crystallized forms of hydroxyapatites containing non-stoichiometric amounts of sodium, magnesium and carbonate.

### 6.2. Features of crystallization

The Gibbs-Kelvin principle states that crystal stability is increased with decreasing standard free energy and entropy. Both are at their minimum when the particles are as close as possible to one another. It is associated with maximal volume filling, which is defined by the ionic radius and charge of the components.

**Crystallization** starts easily in a **supersaturated solution**. It can start simultaneously at several points. **Cluster formation** is defined as the merging of small initial crystals. The relative surface area of crystals decreases with merging. The dissolution at crystal surfaces will therefore decrease, leading to crystal growth. Faults in crystal growth or the incorporation of impurity ions results in the disturbance of stability and ultimately leads to dissolution of the crystals. **Fault location** is a missing lattice point or an increased distance between two lattice points.
6.2.1. Homogeneous nucleation

Homogeneous nucleation: **there is no other substance is present but the anorganic constituents of the crystal.** High energy is required for such a crystallization. The typical process is seeding.

**Seeding (nuclei formation):** preformed crystals are put in a saturated solution to start the crystallization process, resulting in a homogeneous crystal growing on the surface of the substrate.

Seeding can occur in vivo when, for example, primary enamel is formed on the surface of pre-formed dentin crystals.

Types of homogenous crystallization: **mononuclear** - crystal surface grows layer by layer and **polynuclear** - multiple crystallization nuclei are formed simultaneously in highly supersaturated solutions.

**Epitaxy** means deposition of a crystalline overlayer on a crystalline substrate.

6.2.2. Heterogenous nucleation

Heterogenous nucleation occurs when nuclei forming organic substances are present (acidic glycoproteins, phosphoproteins, possibly phospholipids) and the material of crystallization nuclei is not identical to that of the resulting crystal. It requires less energy, so crystallization usually occurs by this process in living organisms.

**Booster effect:** incorporation of heterogenous materials that increase crystal stability.

6.2.3. Factors influencing crystallization

Factors crucially important to crystallization are in the following table.

**Figure 1.83. Table 4. – Most important influencing factors of crystallization**

<table>
<thead>
<tr>
<th>Table 4. Most important influencing factors of crystallization:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Quality of calcium phosphate chelates or compounds</td>
</tr>
<tr>
<td>2. Concentration of calcium phosphate compound in the solution (super saturation)</td>
</tr>
<tr>
<td>3. Temperature - increase reduces crystallization (see melting), reduction increases crystallization (see freezing, solidification)</td>
</tr>
<tr>
<td>4. pH – dissociation of calcium phosphates is increased in acidic pH, which helps Ca deposition (crystallization)</td>
</tr>
<tr>
<td>5. Nucleation Homogenous seeding – presence of preformed crystals in the solutions</td>
</tr>
<tr>
<td>Heterogenous nucleation – role of various protein nuclei as a center for crystal formation</td>
</tr>
<tr>
<td>6. Presence of matrix forming materials - collagen</td>
</tr>
<tr>
<td>7. Effect of trace elements – Coulomb’s law in the crystals, perfect and imperfect crystals</td>
</tr>
<tr>
<td>8. Inhibitors: eg. Mg²⁺ - it prevents the crystallization of amorphous calcium phosphate in mitochondria, completion of crystal formation, inhibition of further growth</td>
</tr>
<tr>
<td>9. Substances reacting with calcium or phosphates</td>
</tr>
</tbody>
</table>

6.3. Spatial structure of apatite crystals
The calcium and orthophosphate groups of apatite are positioned for maximum space filling, while satisfying the law of electroneutrality. Most of the space is occupied by the large orthophosphate ions in the lattice, separated from each other by calcium ions that surround the orthophosphate ions. The calcium to phosphate ratio is 1.67, with 10 calcium ions for every 6 phosphate anions, such that one calcium ion belongs to several orthophosphates. The majority of the calcium ions are located in a triangle relative to one another in the crystal lattice. Spatial representation of calcium and hydroxyl ions reveals the structure of the unit cell.

**Figure 1.84. 6. ábra**

![Figure 1.84. 6. ábra](image)

**Figure 1.85. Figure 3. – Structure of the unit cell. The unit cell is the basic building block of the apatite crystals. It has a characteristic flattened cubic shape and the base plate is rhomb**

![Figure 1.85. Figure 3. – Structure of the unit cell. The unit cell is the basic building block of the apatite crystals. It has a characteristic flattened cubic shape and the base plate is rhomb](image)

The basic unit of enamel is prism. The crystal structure of enamel and dentin is built up of regular hexagonal grids. The crystal size of enamel is much larger than that of dentin. This can be explained by the fact that the amount of heterogeneous nucleator is less in enamel crystals. On the other hand, enamel becomes more dense during maturation as it loses most of its organic material and water content.
6.4. Trace element effects on apatite crystals

Cariogenic trace elements: Al – exerts its effects through binding to the crystal surface, Hg, Cd, Pb, Cu, Se.

Carioprotective micronutrients: F, Sr, Sn – incorporated into the crystal, Mo, V.

6.5. Test - Mineral composition of enamel and dentine.

Bioapatites (answers)

1. Which matrix proteins are typical in the immature enamel?
   A. enamelin, TRAP
   B. amelogenin, TRAP
   C. amelogenin, collagen
   D. enamelin, collagen
   E. amelogenin, enamelin

2. Which are the carioprotective trace elements?
   A. Al, Sn, Mo
   B. Al, Cd, Sr
   C. F, V, Se
   D. Sr, Sn, Mo
   E. Sr, Sn, Cu

3. What does it mean that the calcium to phosphate ratio is typically 1.6 in apatite crystals?
   A. 10 Ca$^{2+}$ to 6 PO$_4^{3-}$
   B. 6 Ca$^{2+}$ to 10 PO$_4^{3-}$
   C. 3 Ca$^{2+}$ to 1 PO$_4^{3-}$
   D. 3 Ca$^{2+}$ to 1 OH
   E. 3 Ca$^{2+}$ to 1 F

References


S.V. Dorozhkin, M. Epple: Biological and Medical Significance of Calcium Phosphates Angew. Chem. Int. Ed. 41: 3130-3146, 2002


David B. Ferguson: Calcified tissues - Chapter 2 pp. 24-46; Calcification – Chapter 3pp. 47-50 in: Ferguson: Oral bioscience, 1999
7.1.7. Calcium homeostasis. Dental aspects of calcium and phosphate metabolism disorders – Dezso Szombath

Calcium and phosphate homeostasis is predominantly under hormonal control, modulated by locally produced cytokines.

From a dental aspect, childhood or geriatric vitamin D deficiency, and congenital alkaline phosphatase deficiency (hypoposphatasia) with rickets-like bone deformities and abnormal dentition are of interest.

7.1. Hyper- and hypocalcemia

Both increasing and decreasing serum calcium levels have deleterious consequences.

Bilateral recurrent nephrolithiasis is a leading complication in hypercalcemia. In addition, gastrointestinal, cardiovascular and neurologic symptoms are typical.

Figure 1.86. Table 1. – Consequences of hypercalcemia

<table>
<thead>
<tr>
<th>Table 1. Consequences of hypercalcemia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gastrointestinal</strong></td>
</tr>
<tr>
<td>• obstipation, nausea, vomitus: ileus, abdominal pain</td>
</tr>
<tr>
<td>• peptic ulcer, pancreatitis, anorexia</td>
</tr>
<tr>
<td>• polydypsia</td>
</tr>
<tr>
<td><strong>Renal</strong></td>
</tr>
<tr>
<td>• hypercalciuria, polyuria (Na and K loss), nycturia, albuminuria</td>
</tr>
<tr>
<td>• nephrolithiasis, nephrocalcinosis, azotaemia, renal failure</td>
</tr>
<tr>
<td><strong>Nervous system</strong></td>
</tr>
<tr>
<td>• emotional lability, delirium, psychosis</td>
</tr>
<tr>
<td>• neuromuscular disorders, muscle weakness</td>
</tr>
<tr>
<td><strong>Circulation</strong></td>
</tr>
<tr>
<td>• hypertension, short QT, pacemaking and conduction disorders</td>
</tr>
</tbody>
</table>

Major causes include primary hyperparathyroidism (PHP).

Figure 1.87. Table 2. – Causes of hypercalcemia
Hyperfunctioning solitary adenoma is the underlying cause of PHP in 80% of the cases.

**Figure 1.88. Table 3. – Primary hyperparathyroidism (1‰ frequency)**

<table>
<thead>
<tr>
<th><strong>Table 3. Primary hyperparathyroidism (1‰ frequency)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hyperfunction: solitary adenoma</strong></td>
</tr>
<tr>
<td>Parathyroid gland hyperplasia</td>
</tr>
<tr>
<td>Hyperparathyreosis based on regulatory disorder</td>
</tr>
<tr>
<td>As part of multiple endocrine neoplasia</td>
</tr>
<tr>
<td>Parathyroid carcinoma</td>
</tr>
</tbody>
</table>

In addition to hypercalcemia, hypophosphatemia, hyperchloremia, hypercalciuria and renal tubular acidosis develops in PHP.
Primary hyperparathyroidism leads to alterations in the oral cavity owing to bone resorption and hypercalcemia. Cystic lesions develop in mandibles with the resulting bone cavities filled by granulomatous tissue (epulis, brown tumor of the maxilla). Lamina dura is absent and the periodontal space is widened. Hypercalcemia leads to elevated calcium levels in both basal and stimulated saliva, resulting in increased calculus formation.

The symptoms of acute hypocalcemia are dramatic: increased nerve and muscle excitability results in muscle cramps all over the body. Carpopedal/laryngospasm (tetany) often occurs, with no medical intervention it may lead to unconsciousness.

Acute hypocalcemia occurs most often in response to acute increases in blood pH (hyperventilation respiratory alkalosis). This is because alkalization of plasma proteins increases their calcium binding affinity, hence reducing free ionized calcium levels in the blood. For every 0.1 decrease in serum pH serum calcium will increase by 0.1 mmol/L. Intensive splitting of fat into fatty acids and glycerol during fat necrosis in severe cases of acute pancreatitis is characterized by the formation of calcium soaps. This so-called steatonecrosis can remove a considerable amount of calcium from blood in a short time. Hypocalcemia is the hallmark of this process.

Chronic hypocalcemia results in muscle spasticity all over the body. Patients complain of fatigue and muscle pain and are emotionally labile.

In hypoparathyroidism (PTH dependent hypocalcemia) serum phosphate concentration is high in addition to low serum calcium.

Figure 1.89. Table 4. – Pathogenesis of hypoparathyroidism

<table>
<thead>
<tr>
<th>Pathogenesis of hypoparathyroidism</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary</strong></td>
</tr>
<tr>
<td>• surgery</td>
</tr>
<tr>
<td>• autoimmune process / polyglandular infiltrative process</td>
</tr>
<tr>
<td>• congenital</td>
</tr>
<tr>
<td><strong>Secondary</strong></td>
</tr>
<tr>
<td>• magnesium deficiency</td>
</tr>
</tbody>
</table>

Figure 1.90. Figure 1. – Patomechanism of hypoparathyreosis

Patients suffer from dental hypoplasia, X-ray images clearly show thickening of the lamina dura.

In PTH independent forms of hypocalcemia serum phosphate levels are low due to secondary hyperparathyroidism.
7.2. Dental aspects of vitamin D metabolism

Severe vitamin D deficiency may cause disorders in both enamel and dentin formation. Enamel may become thin, undermineralized with an irregular surface. Widening of predentin, an uneven boundary between mineralized dentin and predentin, a thin dentin layer and the development of interglobular spaces in dentin are all hallmarks of disorders in dentin matrix formation.

Hypervitaminosis D during tooth development may result in enamel hypoplasia, presumably due to decreased PTH levels and function.

However, controlled vitamin D supplementation may prevent or slow the progression of caries, and in an advanced age it may protect from periodontal diseases.

In chronic renal dysfunction gradually increasing phosphate retention inhibits the activity of residual 1-alpha-hydroxylase. The loss of vitamin D function leads to hypocalcemia, which is continuously compensated by increased PTH secretion (secondary and then tertiary hyperparathyroidism). The constant elevation of PTH levels induces bone alterations characteristic of hyperparathyroidism in addition to hypocalcemia and osteomalacia symptoms (uremic, renal and bone dystrophy). High phosphate concentration accompanying normalized serum calcium levels results in parenchymal calcification, further deteriorating renal function.

Figure 1.91. Table 5. – Conditions that lead to hypocalcemia

<table>
<thead>
<tr>
<th>Table 5. Conditions that lead to hypocalcemia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PTH dependent</strong></td>
</tr>
<tr>
<td>• Hypoparathyroidism</td>
</tr>
<tr>
<td><strong>PTH independent</strong></td>
</tr>
<tr>
<td>• vitamin D and / or calcium deficiency</td>
</tr>
<tr>
<td>• malabsorption, hypoproteinemia</td>
</tr>
<tr>
<td>• uremia</td>
</tr>
<tr>
<td>• EDTA, citrate and oxalate toxicity</td>
</tr>
<tr>
<td>• acute pancreatitis</td>
</tr>
<tr>
<td>• hypercalcitoninaemia (in some medullary thyroid cancers)</td>
</tr>
</tbody>
</table>

Figure 1.92. Figure 2. – Patomechanism of renal osteodystrophy
Owing to high salivary phosphate concentration, the rate of calculus formation and the frequency of consequent dental complications increases in patients with kidney disease.

### 7.3. Congenital hypophosphatasia

Congenital hypofunction of bone, liver and kidney alkaline phosphatase results in a mineralization disorder similar to rickets: short stature, bending and fragile bones, short limbs and premature loss of deciduous teeth. In less severe cases (odontohypophosphatasia) dental symptoms draw the attention to the disease: deciduous teeth are lost before the completion of resorption due to insufficient radicular cementogenesis. Usually canines are lost first, with no accompanying periodontal alterations or inflammation. Diphyodont tooth replacement is delayed, with hypoplastic enamel, slow rate of dentinogenesis and widened pulp chamber and root canal. While the level of alkaline phosphatase is low, serum levels of calcium and phosphate are in the upper third of the normal range or above.

To our current knowledge there is no cure for this disorder. The intake of phosphate and calcium should be limited to prevent tissue calcinosis (by preventing a high calcium–phosphate product value).

### 7.4. Test – Calcium homeostasis (answers)

1. **Complications of hypercalcaemia:**
   A. pancreatitis, portal hypertension
   B. obstipation, nephrolithiasis
C. emotional lability, nephrocalcinosis
D. peptic ulcer, ophthalmopathy
E. intense calculus formation, cardiac rhythm abnormalities

2. May be a complication of primary hyperparathyroidism:
A. mandibular cystic lesions
B. epulis
C. gingival sulcus disappears
D. lack of lamina dura
E. dental hypoplasia

3. May be a complication of congenital hypophosphatasia:
A. widened pulp chamber and root canal
B. disturbed dentinogenesis
C. retarded dentition
D. insufficient cementogenesis
E. loss of deciduous teeth before resorption is completed

References


8. 1.8. Formation of hard tissues, mineralization, bone resorption and osteoclasts – Gábor Varga

Bone remodeling and resorption are closely related processes. Bone remodeling is the result of a balance between osteoblast and osteoclast function, while osteoclasts play the major role during bone resorption.

The integrated action of these two complementary cell types ensures bone development and plasticity, and regeneration after a traumatic injury. An imbalance leads to the development of an abnormally dense bone (osteopetrosis) or an abnormally porous, low density bone (osteoporosis).

Figure 1.93. Figure 1. – Major cell types of bone
Osteoclasts are specialized phagocytosing cells with a diameter of 50 to 100 µm that are formed by the fusing of precursors from the monocyte-macrophage lineage. Inside the sealing zone which is located on the apical surface of the cells to facilitate attachment and to seal the resorption site from its surroundings, osteoclasts form a specialized cell membrane with increased surface area called ruffled border to meet the increased electrolyte transport requirements associated with bone resorption. Osteoclasts are characterized by a granulated, strongly basophilic cytoplasm with endoplasmic reticulum and a large amount of vesicles, vacuoles, Golgi complex and mitochondria. The surface of the basolateral membrane is unruffled.

**Figure 1.94. Figure 2. – Schematic representation of osteoblasts and osteoclasts**

Decreasing serum free calcium levels lead to a release of parathyroid hormone, which activates both osteoclast differentiation and migration. High serum free calcium in turn results in the release of calcitonin, which, acting on its receptor, will directly inhibit osteoclast activity. The development of activated osteoclasts requires RANKL (receptor activator of nuclear factor κβ ligand) and M-CSF (macrophage colony stimulating factor), which are released from neighboring stromal cells and osteoblasts.

**Figure 1.95. Figure 3. – Osteoclast differentiation and interaction with osteoblasts**
The adhesion of the attachment site (sealing zone) is mediated by specialized adhesion structures called podosomes. Adhesion is mediated by integrin receptors and osteopontin which contains RGD (Arg-Gly-Asp) motifs that facilitate adhesion. Osteoclasts secrete protons into the sealed resorption site against the gradient created by vacuolar ATPase and in turn absorb bicarbonate through a chloride-bicarbonate exchange mechanism. This is complemented by a carboanhydrase activity. The overall result is a strongly acidic environment within the sealed compartment, which leads to the dissolution of calcium and phosphate ions and a small amount of carbonic acid. In addition, osteoclasts secrete hydrolytic enzymes into the apical space. Osteoclasts are rich in lysosomal enzymes that they exocytose into the resorption bay. The most important enzymes are cathepsin K, which has a collagenase activity, other cathepsins including B, C, D, E, G, and L, and several phosphatases. The metalloproteases MMP-9 and MMP-13 are also very important in addition to other proteolytic enzymes.

**Figure 1.96. Figure 4. – Osteoclast structure**

Degradation products and dissolved ions are absorbed by osteoclasts. The absorption of calcium ions and their release through the secretory domain is especially important. Specific transporters and proteins involved in the temporary binding of intracellular calcium are crucial to this process. These mechanisms have not been entirely elucidated.

In addition to their bone resorbing activity, osteoclasts secrete different BMP and TGF type differentiation factors which directly effect osteoblast differentiation from mesenchymal precursors and the tissue regenerating activity of these cells.

**Figure 1.97. Figure 5. – Process of bone metabolism: Continuous rebuilding**
The basic mechanism of regulation is very simple. Calcium level is directly maintained by the balanced release of parathyroid hormone and calcitonin. Decreasing serum calcium levels lead to an immediate increase in the release of PTH, which stimulates both osteoclast differentiation and activity. This will mobilize calcium and eventually normalize its serum level. On the other hand, elevated serum calcium results in the release of calcitonin, which will increase the activity of osteoblasts and decrease that of osteoclasts, thus rapidly restoring the initial balance.

**Figure 1.98. Figure 6.** – The parathyroid and thyroid glands function to control the level of blood calcium

**Figure 1.99. Figure 7.** – Control of calcium balance: parathormone, kalcitonin, calcitriol (vitamin D)
The main activator of osteoclast activity is thus the reduction in circulating serum calcium level. However, this process is multifactorial, as, in addition to direct hormonal effects, calcium metabolism and calcium absorption from food in the small intestine play key roles in it. This is, however, crucially influenced by the function of active vitamin D, which affects calcium absorption acting on steroid receptors on intestinal epithelial cells. Receptor activation increases the expression of two proteins. The calcium/proton exchanger on the basolateral side of the cells increases calcium extrusion toward the bloodstream at the expense of cations accumulated by the cells. The other critical factor is calbindin, a calcium binding protein, which binds absorbed calcium and thus decreases the intracellular levels of free calcium, which could otherwise prevent calcium transport.

Figure 1.100. Figure 8. – Effect of active calcitriol (vitamin D) on epithelial cells resulting enhanced calcium absorption
Figure 1.101. Figure 9. – Summary of major hormonal mechanisms controlling plasma calcium

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Target organ</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parathyroid</td>
<td>Bone</td>
<td>Ca²⁺ resorption ↑</td>
</tr>
<tr>
<td>hormone</td>
<td>Kidney</td>
<td>Ca²⁺ reabsorption ↑, Phosphate reabsorption ↓ → phosphate excretion ↑, Synthesis of activated Vitamin D ↑</td>
</tr>
<tr>
<td></td>
<td>Small intestine</td>
<td>Indirect effect from increased synthesis of 1,25-D</td>
</tr>
<tr>
<td>Calcitonin</td>
<td>Bone</td>
<td>Osteoclastic activity ↓ → Ca²⁺ resorption ↓</td>
</tr>
<tr>
<td></td>
<td>Kidney</td>
<td>Minimal effect</td>
</tr>
<tr>
<td></td>
<td>Small intestine</td>
<td>No known effect in humans</td>
</tr>
<tr>
<td>1,25(OH)₂ D₃</td>
<td>Bone</td>
<td>Augments action of PTH to plasma Ca²⁺ ↑</td>
</tr>
<tr>
<td>vitamin</td>
<td>Kidney</td>
<td>Ca²⁺ reabsorption ↑</td>
</tr>
<tr>
<td></td>
<td>Small intestine</td>
<td>Uptake of Ca²⁺ ↑ (via calbindins)</td>
</tr>
</tbody>
</table>

Figure 1.102. Figure 10. – Calcium homeostasis
Disruption of the balance between bone deposition by osteoblasts and bone resorption by osteoclasts leads to osteoporosis. Interestingly, the result of such an imbalance is a decrease in bone density rather than bone size. Reduced number and thickness of trabeculae is one of the most apparent signs of osteoporosis. Depending on the extent of imbalance, it is the concentration of calcium and organic material which decreases, rather than bone volume.

Figure 1.103. Figure 11. – The trabecular structure in osteoporosis

Figure 1.104. Figure 12. – Changes in proportion of mineral (Ca) matrix and water/air phases
8.1. Test – Formation of hard tissues, mineralization, bone resorption and osteoclasts (answers)

1. *Average daily calcium excretion of the kidney in healthy adults:*
   
   A. 1,75 mg  
   B. 17,5 mg  
   C. 175 mg  
   D. 1750 mg

2. *Directly stimulates the calcium uptake of intestinal epithelial cells:*
   
   A. parathyroid hormone  
   B. calcitonin  
   C. active vitamin D  
   D. active vitamin E

3. *Cells that are not characteristic components of tooth formation*
   
   A. odontoblasts  
   B. cementoblasts  
   C. ameloblasts  
   D. osteoclasts

9. 1.9. Cementogenesis – Balint Molnar

9.1. Acellular fibrillar cementum

Covers the coronal two-third of the root surface. Acellular fibrillar cementum is a thin, translucent, non-cellular mineralized tissue. Histologically, a characteristic lamellar structure (parallel to the root surface) can be seen after decalcination and staining because of the appositional development. These lines of apposition show the cyclic, slow apposition of the cementum. This is a slow process, cells synthesizing the matrix stay on the outer surface. After eruption, the thickness of cementum increases over the years, up to a maximum of 60 to 70 microns. Collagen fibers are surrounded by a fine, granulated amorphous matrix. Intrinsic and extrinsic fibers are found in the cementum, most running perpendicular to the root surface. These are the sections of extrinsic fibers running through the cementum.

*Figure 1.105. Figure 1. – Intrinsic-extrinsic fibers*
9.2. Cellular, fibrillar cementum

Cellular fibrillar cementum covers the apical third of the root surface and the furcations.

Cementocytes can be found in the calcified matrix. Appositional growth is irregular compared to cellular cementum. Appositioned layers of cementum are thicker and contain more Sharpey-fibers. The rate of cementogenesis and mineralization is considerably faster compared to acellular cementum. Cementoblasts are trapped in the matrix they produce to become cementocytes later on.

Figure 1.106. Figure 2. – Structure of the attachment apparatus

Figure 1.107. Figure 3. – Secondary cellular cement formation
9.3. Process of Cementogenesis

During life, cementogenesis proceeds along the entire length of the root. However, initially it only occurs at the leading edge of Hertwig’s epithelial root sheath. The inner layer of the epithelial sheath consists of modified ameloblast secreting an enamel-matrix-like protein layer (hyalin) onto the root surface. In the beginning, the proliferation of Hertwig’s epithelial sheath cells is not apically directed, but promotes the eruption of fully developed crown. Proliferation of these cells only turns apically after the crown has almost reached occlusion. Hertwig’s epithelial sheath is responsible for root morphology, it also separates the dental papilla from the dental follicle. The developing predentin is covered by a 10µm thick, hyalin-like layer. This hyalin-like layer is amelogenin, or enamel matrix protein, secreted by the reduced ameloblasts of the Hertwig’s epithelial sheath. Thereby the differentiation of mesenchymal cells of the dental follicle is initiated, which become cementoblasts and start to synthesize acellular cementum.

Figure 1.108. Figure 4. – Development of the attachment apparatus

9.4. Cemento-dentinal junction
The first mesenchymal cells of the dental follicle are fibroblast-like cells, attached to the non-mineralised dentin matrix. These cells secrete a collagen matrix around non-mineralised collagen fibers. Initial dentin mineralisation starts from the direction of the pulp and is only finished once the most superficial dentin-derived collagen fibers and the deepest dental follicle-derived collagen fibers have interdigitated. Thus a very strong dentin-cementum junction is created. This junction is covered by peripheric, intrinsic fiber containing cementum layers during subsequent stages of cementogenesis. Once the layer incorporating intrinsic parallel fibers reaches a thickness of 15 to 20 µm, it comes in contact with collagen fibers of the developing periodontal ligament. Later on these layers constitute the greatest proportion of the collagen matrix of acellular cementum (Figure 1).

### 9.5. Cementoneogenesis, periodontal regeneration

PDL is a pool of pluripotent mesenchymal cells capable to form cementum, Sharpey-fibers and alveolar bone. The regenerative potential of a previously damaged attachment apparatus is limited. Regeneration involves the migration of locally available progenitor cells to the defect site, their differentiation into PDL forming fibroblasts and the appearance of mineralizing cementoblasts and bone-forming osteoblasts. PDL stem cells under certain circumstances can differentiate into cells that can produce mineralized tissue in vitro and vivo. This can be induced by certain biological factors such as amelogenin (Emdogain), used during periodontal regenerative surgery. This may facilitate the attachment of the multipotent mesenchymal progenitor cells and their differentiation to cement matrix forming cells on the surface of the cementum during wound healing.

### 9.6. Test – Cementogenesis (answers)

1. **The coronal two-third of root surfaces is covered by this type of cementum:**
   - coronal cementum
   - cellular fibrillar cementum
   - acellular fibrillar cementum
   - MTA cement
   - acellular afibrillar cementum

2. **Multinuclear giant cells, responsible for cement resorption:**
   - osteoclasts
   - cementoblasts
   - cementocytes
   - osteoblasts
   - cementoclasts

3. **The main fiber component in the extracellular matrix of the fibrillar cementum is:**
   - type I collagen
   - oxytyalan fibers
   - type XII collagen
   - type III collagen
   - argyrophil fibers

4. **Proteins synthesized by the epithelial cells of the Hertwig’s root sheath that induce cementogenesis:**
   - osteopontins
• enamel matrix proteins
• tenascins
• fibronectins
• sialoproteins

5. Localization of progenitor cells regulating the regeneration of cementum, PDL and alveolar bone:
• gingival epithelium
• gingival connective tissue
• alveolar bone
• dentin
• periodontal ligament

10. 1.10. Pathomechanism of bleeding and its relation to dentistry – Katalin Varnai

Many dental procedures are associated with a risk of bleeding, which in the large majority of cases is self-limiting and non-problematic. Sometimes, however, complications may arise due to inherited or acquired bleeding disorders, hemostatic defects secondary to the underlying disease or medication. A routine dental surgery may sometimes reveal a bleeding abnormality.

Before a dental procedure, it is important to ask about personal and family bleeding history (extensive bruising, frequent nosebleeds, heavy menstrual bleeding, prolonged bleeding after surgery or an invasive dental procedure), diseases affecting hemostasis (liver or kidney disease, malignant tumors) and therapies (coumarine derivatives, anti-platelet drugs).

Hemostasis is a highly regulated process that maintains the fluidity of blood in the vessels, while limits the amount of blood loss after an injury and optimizes wound healing. To fulfill this task, a highly regulated interaction is necessary between the vessel wall, platelets, coagulation factors and the fibrinolytic system.

Figure 1.109. Figure 1. – Regulation of hemostasis
Vascular defects are rarely the cause of bleeding disorders and are usually associated with mild bleeding confined to skin or mucosa.

Platelet disorders can be hereditary or acquired and may be due to decreased production, excess consumption or altered function of platelets. The following bleeding symptoms may occur: mucocutaneous bleeding (petechiae, ecchymosis, suffusion), epistaxis, and menorrhagia. In the oral cavity, the most common clinical symptoms are petechiae, gingival hyperplasia, spontaneous gingival bleeding and ecchymosis.

Thrombocytopenia may be mild, moderate or severe. The minimum platelet level before dental procedures is 50 G/L, extensive surgery may require > 100 G/L. Inherited thrombocytopenia is rare and is often one component of a syndrome. Acquired thrombocytopenia is most commonly of immune origin such as in the case of idiopathic thrombocytopenic purpura (ITP).

Thrombocytopenia can be congenital or acquired. It is very rare in its congenital form, such as Glanzmann thrombasthenia, when mucocutaneous bleeding occurs at birth or early infancy.

Of the acquired platelet defects, the most common are drug induced in antiplatelet therapy.

During ASA therapy (100 mg/day), dental procedures can be carried out atraumatically with local hemostatic agents without cessation of the drug. Non-steroid anti-inflammatory drugs (NSAIDs) act like ASA. Two days before surgery a cessation of NSAID is proposed and the therapy can be continued a day after the operation. Thienopyridine (clopidrogel, ticlipodin), ASA+clopidogrel or ASA+dipyridamole combination may significantly increase the risk of bleeding. In this case, a haematological concilium is mandatory.

Figure 1.110. Figure 2. – Platelet inhibitors
Among the congenital coagulation defects, haemophilia A (HA), haemophilia B (HB) and von Willebrand Factor (vWF) deficiency are the most common. Symptoms may include prolonged bleeding, ecchymosis, deep hematomas, epistaxis, spontaneous gingival bleeding and hemarthrosis. 25% of the normal level usually provides satisfactory clotting. Patients with levels of less than 5% will have symptoms of abnormal bleeding such as easy bruising. When the FVIII level is less than 1%, the condition is classified as severe, with spontaneous bleeding.

Dental treatment of haemophilic patients can be carried out only at a dedicated facility. Management of HA patients undergoing dental surgery consists of increasing FVIII levels, replacing FVIII and inhibiting fibrinolysis. Normal factor level (50-100%) has to be maintained from surgery to wound healing. DDAVP (Desmopressin) is used to achieve a transient increase of endogenous FVIII and vWF in mild forms of HA and vWD.

Acquired coagulation defects are most often caused by antihtrombotic therapy. The use of oral anticoagulants (coumarin derivatives) inhibits the modification on the K-dependent clotting factors zymogen II, VII, IX and X. Minor dental procedures can be carried out with local hemostasis with INR within therapeutic range (2-3 obtained within 24 hours).

\[ INR = \left( \frac{PT_{\text{patient}}}{PT_{\text{normal}}} \right)^{ISI} \]

INR = International Normalised Ratio, PT = prothrombin Time, ISI = International Sensitivity Index.

If the procedure is invasive, extensive or carries a large risk of bleeding, the dose of coumarin can be reduced or changed to heparin (LMWH), for which an hemostatic consilium and institutional background is indispensable. Mild bleeding can be reduced by supplying vitamin K. In case of an emergency, it is necessary to use fresh frozen plasma (FFP), recombinant Factor VIIa (rFVIIa), and Prothrombin Complex Concentrate (PCC). Treatment of patients with bleeding disorders is based on guidelines and agreement between different medical specialties.
Figure 1.111. Figure 3. – Anticoagulant drugs

Figure 1.112. Figure 4. – Blood collection tube containing sodium citrate for coagulation tests
Figure 1.113. Figure 5. – Blood collection tube containing K2EDTA for laboratory tests in hematology
1. Oral biology

Figure 1.114. Figure 6. – Blood collection tube for the determination of erythrocyte sedimentation rate

10.1. Test – Pathomechanism of bleeding and its relation to dentistry (answers)

1. Which defect(s) is (are) X-chromosome-linked?

A. von Willebrand factor deficiency
B. Factor VIII and Factor IX deficiencies
C. Factor V deficiency
D. Factor XIII deficiency

2. How long before a simple dental intervention should INR be tested in a stable patient?

A. One week before
B. Several days before
C. Testing is not mandatory
D. <24 hours

3. Dental treatment of a haemophilic patient can be carried out

A. any time and at any dental clinic
B. after factor supplementation and haematologic consultation at an expert institute
C. at an expert institute
D. after haematologic consultation at any dental clinic

References


11. 1.11. Tooth eruption and tooth movement – Balint Nemes

11.1. Tooth eruption

Tooth eruption is a polarized process: bone is resorbed around the coronal part of the tooth while it is formed apically. Tooth eruption has three stages: preruptive, prefutional eruptive and functional eruptive (posteruptive phase). The onset of bone formation precedes eruption. During the eruption phase it acts reciprocally with root development and results in tooth movement along the eruption path prepared by bone resorption. The intraosseal speed of eruption is 1 to 10 µm/day, the extraosseal is 75 µm/day. It is locally regulated, and the dental follicle plays the main role in its control.

11.1.1. Cellular processes

The osteoclasts and osteoblasts are responsible for local bone resorption and formation, respectively. However, coronally both the dental follicle and the enamel epithelium, which separates the enamel from the dental follicle, are also necessary for tooth eruption. Coronally, at the location of bone resorption, the dental follicle regulates mononuclear cells eruption by first recruiting mononuclear cells which, based on enzymatic and ultrastructural properties, are considered preosteoclasts.

Figure 1.115. Figure 1. – Stages of eruption
11.1.2. Molecular background

Several studies showed that epidermal growth factor (EGF), transforming growth factor-beta (TGF-β), interleukin-1 (IL-1), colony-stimulating factor-1 (CSF-1) and two proteins derived from the dental follicle and enamel organ play important roles in the regulation of tooth eruption.

During tooth eruption collagenous and non-collagenous protein composition of the dental follicle changes, with the collagen and proteoglycan content increasing. The most important follicle sialoprotein is DF-95. This follicle protein is concentrated in tonofibrils between ameloblasts in the enamel organ. Immediately before the eruption they are partially fragmentated by several proteases, initiating tooth eruption.

EGF, TGF-α, CSF and TGF-β1 induces follicular cells to differentiate into periodontal cells.

Injection of EGF can induce premature tooth eruption. EGF increases IL-1α expression. The presence of IL-1 in stellate reticulum (SR) is remarkable as it increases CSF-1 expression in cultured dental follicular cells, which cannot be directly stimulated either by EGF or by TGF-β1. Thus, increased CSF-1 mRNA production is caused by EGF or TGF-β1 through IL-1 expression in SR. Another molecule, TGF-α, also binds to the EGF-receptor, promoting tooth eruption.

Colony-stimulating factor-1 (CSF-1) stimulates the development of monocytes, and if it is injected into osteopetrotic mice, it initiates tooth eruption. Without this injection these mice do not have bone resorption because of osteoclast deficiency, which prevents eruption.

TGF-β1 acts as a monocyte chemoattractant factor, and in SR it is found before the rate of monocyte infiltration is highest. This shows that TGF-β1 initiates the process of eruption. Furthermore, several studies have shown that TGF-β1 in vitro stimulates the secretion of extracellular matrix proteins such as type 1 collagen and fibronectin in dental follicular cells. Thus, TGF-β1 participates in the regulation of periodontal ligament development.

So far one molecule has been found to inhibit tooth eruption. A 167 kDa protein isolated from the dental follicle and from the SR, probably a soluble EGF-receptor, decreased the stimulatory effect of EGF on tooth eruption.

11.1.3. Eruption of the deciduous and permanent teeth

Figure 1.116. Table 1. – The rule of 6’s
11.1.4. Abnormalities of tooth eruption

Delayed teething: 1 to 2 teeth are affected, hereditary background suspected

External resorption: Primary tooth root is resorbed due to the eruption of not the underlying but the neighbouring permanent tooth. More frequent in upper jaw and in boys. Reason: limited space.

Systemic resorption disorders: Mostly hormonal, related to PTH hormone secretion or defective PTH receptors. Hereditary form is also known.

Ankylotic primary teeth: Happens often with the aplasia of the permanent tooth. Clinical presentation: primary tooth in infraocclusion, neighbouring teeth tipped above the primary tooth.

Aplasia, oligodontia, hypodontia: Pimary loss of certain teeth or tooth-groups (mostly last teeth of tooth groups are affected: Bolk’s terminal reduction theory). Prevalence: M3 10 to 25%, P2 3 to 4%, I2 2%. Shows familial aggregation.

Aplasia and persistent primary teeth: In case of primary loss of permanent tooth deciduous teeth remain in the mouth, often with varying root sizes. When in infraocclusion, prognosis is poor.

Eruption disorders: The most common cause of wisdom teeth impaction is the lack of space.

Figure 1.119. Figure 5. – External resorption
Figure 1.120. Figure 6. – Systemic eruptive disorder (Decker et al., 2008)

Figure 1.121. Figure 7. – Aplasia, decidous tooth in infraocclusion
11.2. Tooth movement

Equilibrium theory: A tooth will not move if the net sum of forces acting on it is zero. Shifting the balance will move the tooth.

Tooth movements: Physiological: Eruption. Pathological: Early primary or secondary tooth extraction, Tumor, Thumb sucking, Muscular dysfunction. Therapeutic: Orthodontics.

Figure 1.122. Figure 8.

11.2.1. Active tooth movement: Basic rules

Bone formation on the tension side of the root, bone resorption on the pressure side.

Overload of the periodontium will compress the capillaries, leading to hyalinisation and a decrease in bone metabolism, decreasing the effectiveness of tooth movement. Effective tooth movement is therefore not directly proportional to the magnitude of orthodontic force. Orthodontic force needs to reach a certain threshold but should not exceed a certain maximum limit.

The force loaded on the tooth must be proportional to the root surface. (Of note, periodontally affected teeth have a smaller root surface.)

3rd Law of Newton: Action = Reaction. Anchor teeth will also move if they are loaded. (Skeletal anchorage may be used to prevent this effect.)

Loading the tooth at its center of resistance is almost never possible. Therefore, lever arm and torque should always be taken into account.
Figure 1.123. Figure 9.

Angulation of a canine with a box-loop. The force loaded on the tooth is proportional to the length of the wire/spring.

11.3. Test – Tooth eruption and tooth movement (answers)

1. *What is the eruption mostly induced by?*
   A. Local effects: The follicle binds different GFs, which results in bone resorption in the coronal region.
   B. Hormonal effects, mostly PTH.
   C. Mechanical effects, proliferation in the apical area.
   D. Functional effects, tongue and buccal muscle activity.
   E. Neuronal effects, with neurotransmitters playing the main role.

2. *The eruption sequence of permanent teeth is:*
   A. 1-2-3-5-4
   B. 1-2-5-4-3
   C. 6-1-2-4-5-3
   D. 6-3-1-2-4-5
   E. 6-5-1-2-4-3

3. *Which statement is true?*
   A. We need great forces to move the teeth as tooth movement is proportional to the force acting on the tooth.
   B. We need small forces to move the teeth as tooth movement is inversely proportional to the force acting on the tooth.
   C. Bone resorption occurs at the surface where periodontal fibres are stretched whereas bone absorption occurs on the pressure side.
D. The longer the spring, the bigger the force acting.

E. Orthodontic miniscrews and extraoral appliances are not suitable tools for skeletal anchorage because they cannot withstand orthodontic forces.

12. 1.12. The morphology and function of salivary glands – Gabor Varga

When saliva is missing, it has a serious impact on the quality of life of the patient. These people have very dry lips and mouth. For them it is difficult to swallow, difficult to chew, difficult to speak and difficult to taste. Although none of these symptoms are life-threatening, they mean serious problems. This is especially important to realize since at present no real salivary gland reconstruction therapy exists.

Figure 1.124. Figure 1. – No saliva – consequences

<table>
<thead>
<tr>
<th>No saliva – consequences:</th>
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</thead>
<tbody>
<tr>
<td>Dry lips, dry mouth</td>
</tr>
<tr>
<td>Difficult to swallow</td>
</tr>
<tr>
<td>Difficult to chew</td>
</tr>
<tr>
<td>Difficult to speak</td>
</tr>
<tr>
<td>Difficult to taste</td>
</tr>
</tbody>
</table>

Saliva contains 98% water!

Today salivary glands are not in the high focus of research. But in the past two great scientists earned Nobel prize as a result of their scientific achievements done using exocrine glands of the upper gastrointestinal tract, the salivary glands, the stomach and the pancreas. First, in 1904, Pavlov received the medal for his fundamental work using dogs with both salivary, pancreatic and gastric cannulas. These studies established modern gastrointestinal and neurophysiology research.

Figure 1.125. Figure 2. – Nobel prize 1904 – Pavlov in vivo physiology of the digestive glands

Seventy years later George Palade received the Nobel prize for the discovery of the process of exocitosis. His brilliant cell physiological studies described the mechanism of regulated protein secretion, again in salivary, pancreatic and gastric secretions.

Figure 1.126. Figure 3. – Nobel prize 1974 – Palade exocytosis of secretory glands
Most of the fundamental work on nervous innervation of the salivary glands, stomach and pancreas came from the work of Pavlov and his students.

Three pairs of major glands, the parotid, the sublingual and the submandibular glands produce 95% of the secreted saliva. The remaining amount originates from the minor salivary glands which are scattered around the oral cavity. The minor salivary glands are located in the buccal, labial, palatal and lingual regions, including the base of the tongue (namely, the von Ebner’s glands). In humans, their number is between 600 and 900. No minor glands can be found, however, in the gingiva and in the hard palate anterior part.

Salivary glands
1. Oral biology

During embryonic life all glands develop through the proliferation of a cord of cells from the epithelium into the underlying mesenchyme, followed by a branching process to form the grape-like arrangements of the secretory lobes. The parotid gland and the submandibular/sublingual glands develop from different embryonic origins. The parotid gland is derived from the ectoderm, whereas the submandibular and sublingual glands are derived from the endoderm. Glands develop in a similar pattern of morphogenesis driven by cytokines, growth factors and extracellular matrix components. The parotid, submandibular and sublingual glands are each enclosed within a well-defined capsule of neural crest derived mesenchyme and interaction with the surrounding mesenchyme is essential for the initial budding of the salivary gland. In all mammals, gland development starts by a thickening of the primitive oral epithelium that grows into the first branchial arch mesenchyme to form the solid epithelial placode. This placode grows inwardly into the mesenchyme forming a solid mass of epithelial cells (initial bud) which is connected to the oral epithelium by a stalk of immature duct epithelial cells.
Epithelial-mesenchymal interaction leads to cleft formation which separates the primary bud into multiple buds. This process is repeated multiple times through **pseudoglandular, canalicular and terminal bud stages**. While the main duct undergoes lumenization, acini and lumen formation starts in end buds so that a continuous lumenized duct connecting the acini to the oral cavity forms. During the cell differentiation different types of fibroblast growth factors (FGF), especially FGF7 and FGF10 play an important role. Other signaling pathways like that involve ectodysplasin and its receptors are also important. Nerve and blood vessels develop in association with the branching epithelium. Cellular differentiation and branching morphogenesis occur at the same time. The undifferentiated cells of the embryonic gland differentiate into acini, myoepithelium, intercalated, striated and excretory ducts. Unlike major salivary glands, minor salivary glands lack a branching network of draining ducts. Instead, each salivary gland unit has its own simple duct.

**Figure 1.131. Figure 8. – Stages of salivary gland development – epithelial/mesenchymal interactions**

Schematic showing prebud, initial bud, pseudoglandular, canalicular and terminal bud stage of development in the SMG.

**Figure 1.132. Figure 9. – A model of how FGF7 and FGF10 signaling through FGFR2b regulates morphogenesis**

The model summarizes our findings, and the dotted lines show other potential mechanisms: MMP2 may regulate FGFR1 cleavage; FGF1 expression may stimulate both FGFR1 and FGFR2; cofactors or coreceptors may specify the localization of FGF binding and, therefore, where proliferation occurs.

**Figure 1.133. Figure 10. – Factors participating in the regulation of salivary epithelial differentiation**
Salivary glands renew every 2-4 months and therefore are considered as “slowly renewing organs”. Homeostasis of the developed salivary glands is maintained by proliferation and differentiation of stem cells and by proliferation of already differentiated cells. Therefore, normal salivary gland turnover is maintained by more than one cell population. Adult salivary gland studies indicate that intercalated ductal cells harbor a population of undifferentiated stem cells. These intercalated ductal cells can differentiate into both acinar and ductal cells suggesting that they can be a source of salivary renewal. Additionally, slowly dividing cells include acinar cells, ductal cells, myoepithelium and connective tissue cells suggesting the presence of multiple stem/progenitor cells in the adult salivary gland. To date, the characteristics of salivary gland stem cells have not been identified.

Acinar cells, ductal cells and myoepithelial cells are the three major specialized salivary gland cell types. Acinar cells have pyramidal shape and form glandular end pieces which are called acini. Parotid glands are made up by serous acini to secrete watery saliva. Submandibular glands consist of both serous and mucous acini to secrete moderately viscous saliva. Finally, sublingual glands contain mucous acini with serous demilunes to secrete highly viscous saliva. Acinar lumen continues in the lumen of intercalated, striated and excretory ducts. Acini and intercalated ducts are surrounded by myoepithelial cells to form a basket like arrangement. Myoepithelial cells compress the lumen to press the intraluminal fluid through the ductular systems into the oral cavity. Intercalated ductal cells are cuboidal in a single layer arrangement. Striated and excretory ductal cells are columnar cells having deep basolateral invaginations and also intercellular plasma membrane interdigitations with a large number of mitochondria. Large excretory ducts have multiple cell layers columnar epithelium laying over basal cells. In major glands ducts have multiple branching forming a real tree-like organization.

**Figure 1.134. Figure 11. – Tissue organization of salivary glands**
Regulation of salivary fluid and protein secretion

Salivary secretion is regulated by both the parasympathetic and the sympathetic part of the autonomic nervous system. Both parts stimulate salivary secretion, but in a very different way, and their complicated interactions vary gland by gland. The secretory activity of minor glands depends on central neuronal control at a much lower level. For this reason, these glands continuously secrete a tiny amount of fluid, not only during the day, but also at night sleep when the major glands are practically in full rest. This special lubricating, surface protecting feature of minor glands is important for normal oral health and comfort.

The main blood supply and also the nerve fibers enter the body of the salivary gland along the main duct and divide with ductal branches in parallel until reaching the acini. Parasympathetic stimulation through muscarinic cholinergic receptor activation induces the watery saliva secretion. On the contrary, sympathetic stimulation on β-adrenergic acinar receptors principally activates the discharge of protein-rich saliva.

Salivary muscarinic receptors are coupled to Gq/11 type intracellular G proteins. Their activation initiates the hydrolysis of phospholipase C-mediated hydrolysis of phosphatidylinositol 4,5 bisphosphate (PIP$_2$), inositol 1,4,5, trisphosphate (IP$_3$) and diacylglycerol (DAG). Ca$^{2+}$ release from the endoplasmic reticulum is induced by the water soluble IP$_3$, leading to the stimulation opening of Ca$^{2+}$ channels in the cell membrane. Intracellular Ca$^{2+}$ concentration then leads to the opening of Ca$^{2+}$-activated basolateral K$^+$ and apical Cl$^-$ channels, resulting in an
accelerated Cl⁻ secretion to the acinar lumen, followed by paracellular Na⁺, to compensate electrochemical difference and para- and transcellular water movement. Noradrenaline stimulation of β-adrenergic receptor results in intracellular Gs-type G protein activation. Subsequently, ATP is converted by of cyclic adenosine monophosphate (cAMP) due to the increased adenylate cyclase (AC) activity. Finally, this event leads to activation of enzymes such as protein kinase A, which regulate exocytosis of previously synthesized proteins into the acinar lumen.

**Figure 1.137. Figure 14. – Regulation of protein and electrolyte secretion of the salivary glands**

The above described two major pathways strongly interact and directly crosstalk within the acinar cells. Simultaneous sympathetic and parasympathetic activation results in a considerable potentiation of the secretion of both fluid and proteins. Although sympathetic stimulation highly increases protein discharge, a moderate volume of fluid is also secreted during stress situations. Additionally, parasympathetic stimulation induces protein secretion to a moderate degree as well, for example during food intake-induced muscarinic activation. Both sympathetic and parasympathetic activity causes myoepithelial cell contractions, evidently leading to the expulsion of secretory products by washing them out into the oral cavity.
Video 1. – Isolated saliva collection from the minor salivary glands

12.1. Test – The morphology and function of salivary glands (answers)

1. Fundamental importance for embryonic salivary development:
   A. proliferation
   B. apoptosis
   C. gastrulation
   D. all three
   E. none of them

2. Location of secretory protein (export protein) storage in salivary glands
   A. Golgi vesicle
   B. zymogen granule
   C. Golgi cysternae
   D. cell nucleus

3. Which one of these transmitters stimulates both salivary secretion, gastric acid secretion and pancreatic enzyme secretion?
   A. adrenaline
   B. acetylcholine
   C. histamine
   D. somatostatin

The most abundant and important component of saliva is water, about 98 % of the secreted quantity. The rest is made up of electrolytes, proteins, and a few minor components, lipids, glucose and urea. The water and electrolyte secretion is an energy consuming, active two-stage process. First the acini produce isotonic primary saliva. This fluid is then modified in the ductal system by electrolyte reabsorption to form a hypotonic secretion.

**Figure 1.138. Figure 1. – Salivation – two-stage hypothesis**

- Acinar cells produce isosmotic primary saliva
- Passing through the ductal system reabsorption of electrolytes happens without water movement resulting hypotonic fluid
- The composition of saliva depends on the rate of salivary secretion (flow rate)

**Figure 1.139. Figure 2. – Flow rate curves of saliva and the two-stage hypothesis 1**

**Figure 1.140. Figure 3. – Flow rate curves of saliva and the two-stage hypothesis 2**

In various secretory epithelia fluid can either be transported through the cells by transcellular transport or between the cells through junction complexes by paracellular transport. The transcellular transport pathway in
the salivary glands primarily requires aquaporin 5 (AQP5) water channels, localized to the luminal surface of acinar cells. The lack of AQP5 decreases transport not only through the plasma membrane but also through the tight junction complex by decreasing the expression of tight junction proteins such as claudins and occludins. The basolateral-to-apical water movement by acini is rather due to osmotic gradient initiated by electrolyte transport and apical discharge. Therefore, passive water movement is a consequence of preceeding acinar secretion, resulting an isotonic primary secretory fluid.

**Figure 1.141. Figure 4. – Main transporters-channels-pumps**

<table>
<thead>
<tr>
<th><strong>Main transporters-channels-pumps</strong></th>
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<tbody>
<tr>
<td><strong>Primary pumps</strong></td>
</tr>
<tr>
<td><strong>Facilitating transporters</strong></td>
</tr>
<tr>
<td><strong>Ion channels</strong></td>
</tr>
<tr>
<td><strong>Water channels</strong></td>
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</table>

Acinar water secretion is primarily driven by vectorial, transcellular Cl⁻ transport. The energy for Cl⁻ movement across the cell is provided from a highly active pump, the Na+/K+ ATPase that uses ATP to extrude 3 Na⁺ and to allow entry of 2 K⁺ into the cell. A consequence, Na⁺ gradient between the intracellular and the extracellular space becomes much higher, than that in K⁺ concentration difference. At the expense of the Na⁺ concentration gradient, the Na⁺ K⁺ 2 Cl⁻ cotransporter (NKCC1) brings 3 Na⁺, 3 K⁺ and 6 Cl⁻ ions into the acinar cell from the interstitium, so concentration of K⁺ and Cl⁻ in the intracellular space well above their equilibrium potential.

**Figure 1.142. Figure 5. – Acinar cell transporters**

<table>
<thead>
<tr>
<th><strong>Acinar cell transporters</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Basolateral side</strong></td>
</tr>
<tr>
<td>Na⁺/K⁺-ATPase</td>
</tr>
<tr>
<td>Na⁺/K⁺/Cl⁻ cotransporters</td>
</tr>
<tr>
<td>Na⁺/Cl⁻ cotransporter</td>
</tr>
<tr>
<td>Na⁺/Cl⁻ cotransporter</td>
</tr>
<tr>
<td>Na⁺/H⁺ cotransporter</td>
</tr>
<tr>
<td>unknown substrate specificity transport</td>
</tr>
<tr>
<td>Cl⁻-HCO₃⁻ cotransporter</td>
</tr>
<tr>
<td>Na⁺/H⁺ exchanger (NHE)</td>
</tr>
<tr>
<td>Na⁺/Ca²⁺-activated K⁺- channels</td>
</tr>
</tbody>
</table>

In response to muscarinic receptor stimulation intracellular Ca²⁺ becomes elevated, Cl⁻ ions are released into the acinar lumen by Ca²⁺-activated chloride channels at the apical membrane and Ca²⁺-activated potassium channels open to release K⁺ into the interstitium at the basolateral membrane. Additionally, Na⁺ is driven through tight junctions from the interstitium into acinar luminal to compensate the developing electrochemical gradient.

The participating K⁺ and Cl⁻ channels have been recently identified. Two different K⁺ channels with somewhat different characteristics are involved. One of these is the named IK1 or SK4, a Ca²⁺-activated K⁺ channel of intermediate single channel conductance. The other one called maxi K or Slo is both Ca²⁺- and voltage-activated with a large single channel conductance. Fluid secretion is severely impaired when both IK1 and Slo channels are missing. The recycling of K⁺ ions into the extracellular space and hyperpolarizes the membrane and increases the drive for Cl⁻ to exit on the opposite side. The osmotic gradient, due to luminal NaCl accumulation then draws water through the acinar cells.
Figure 1.143. Figure 6. - Acini

There are two alternative mechanisms accounting about for about 30% of isotonic primary saliva secretion. The alternative Cl is achieved by the joint activities of basolateral Na⁺/H⁺ and Cl⁻/HCO₃⁻ exchangers. CO₂ diffuses through the basolateral membrane into the cell. In the cytosol carbonic anhydrase enzyme activity catalyzes H⁺ and HCO₃⁻ production from H₂O and CO₂. The newly synthetized HCO₃⁻ is exchanged to Cl⁻ by the Cl⁻/HCO₃⁻ exchanger while intracellular extra H⁺ is extruded by the Na⁺/H⁺ exchanger. Accumulated intracellular Cl then secreted apically. The secretory process can also be achieved to a certain degree even under Cl⁻-free conditions, since an apical anion conductance may also use HCO₃⁻ through the channels instead of Cl⁻ at somewhat lower level.

Formation of hypotonic saliva

Secondary saliva modifications by ductal cells

Among the ductal cells in salivary glands intercalated and striated ducts are intralobular, and excretory ducts are primarily extralobular. NaCl reabsorption happens both in intralobular and extralobular ducts. Ducts are impermeable for water, thus, ductal electrolyte reabsorption directly lead to hypotonic saliva formation. Final salivary electrolyte concentration highly depends on salivary flow rate. As basal flow rate is low, secretion goes through the ductal system at low speed. Thus, reabsorption is almost complete leading to extremely hypotonic solution. Food intake stimulated secretion is accelerated, so salivary fluid flushes through the ductal system at
high speed. In such conditions ductal reabsorption is incomplete, and the ionic strength of saliva reaching the mouth is very similar to the primary isotonic secretion of acini.

**Figure 1.144. Figure 7. – Salivary gland transporters**

The energy for reabsorbing electrolytes comes from the highly active Na\(^+\)/K\(^+\) ATPase producing extremely low Na\(^+\) level in the cell. ENaC, the epithelial Na\(^+\) channel, expressed in the apical ductal membrane clearly plays a crucial role in ductal Na\(^+\) reabsorption. Amiloride block of ENaC function severely impairs Na\(^+\) reabsorption, while two Na\(^+\)/H\(^+\) exchangers, NHE2 and NHE3 play a secondary, minor role in the process.

Cl\(^-\) is also reabsorbed by ducts. Apical Cl\(^-\) channels, namely the cAMP-dependent Cl\(^-\) channel CFTR and Cl\(^-\)/HCO\(_3\)\(^-\) exchangers are responsible for this process.

**Figure 1.145. Figure 8. – Ductal reabsorption mechanisms**

Salivary K\(^+\) concentration is higher than that found in blood plasma. K\(^+\) accumulation happens at the level of the intra- and extralobular salivary gland ducts. Apical K\(^+\)/H\(^+\) exchangers and K\(^+\)/HCO\(_3\)\(^-\) cotransporters may also be involved in this process.
Mechanism of protein secretion

Acinar salivary cells continuously synthesize the export proteins. Synthesized nascent amino acid chains undergo post-translational modifications in the Golgi apparatus, then stored in zymogen granules. Zymogen granules are transported and fused with the apical cell membrane in a process called exocytosis in response to appropriate stimulation. Various salivary glands produce proteins in different composition. The secretory products of each salivary gland are unique. The two types of acinar cells are serous and mucinous. Both serous and mucinous acinar cells form secretory acini. Serous acini discharge a watery fluid rich in α-amylase and additional enzymes, anti-viral and anti-microbial-bacterial proteins. α-amylase is the most abundant protein in serous saliva. About 70% of amylase is parotid product. Mucous acini secrete a thick, mucin-rich fluid. Seromucous acini produce both mucins and serous proteins. The parotid is made up mostly of serous acinar cells. The sublingual gland and most minor glands are mostly mucinous, while the submandibular gland is a mixed by serous and mucinous acini. Besides proteins, all salivary glands produce electrolytes and water for the protection of the oral soft tissues and teeth against acidic and demineralizing conditions.

Regulated and constitutive secretory pathways in salivary glands

13.1. Test – Salivary gland electrolyte, water and protein secretion (answers)

1. Major channel(s) for salivary acinar electrolyte secretion
   A. CFTR
   B. voltage gated K channels
   C. eNaC
   D. Ca** dependent Cl– channels

2. Major channel(s) for salivary ductal electrolyte transport
   A. CFTR
   B. voltage gated K channels
   C. eNaC
   D. Ca** dependent Cl– channels

3. Primary ion in salivary acinar vectorial electrolyte transport
1. Oral biology

A. Ca ion
B. Cl ion
C. Na ion
D. phosphate ion
E. none of them

14. Oral function and diagnostic role of secreted saliva – Gabor Varga

Normally about 1.0-1.5 liter of saliva is secreted each day; consisting mainly of water, electrolytes and proteins. This quantity greatly varies depending on age, gender, health state of the individual, the climate and environmental conditions. The submandibular gland produces about 65%, the parotid 25%, the sublingual gland about 5%, and the minor salivary glands also about 5% of daily saliva. During food intake the ratio changes, the parotid secretes the highest amount.

Protective saliva components

Under physiological conditions the oral cavity is continuously moistened, lubricated and buffered by saliva. Salivary components incorporate into hard tissue pellicle and the protective layer over the soft mucosa, while others serve as part of the immune system of the organism. All of these functions are quite significant for integrity of hard and soft tissue surfaces.

Figure 1.146. Figure 1. – Electrolyte concentrations in basal and stimulated mixed saliva

| Electrolyte concentrations in basal and stimulated mixed saliva |
|-------------------|-------------------|-------------------|
|                   | Plasma (mmol/l)   | Basal (mmol/l)    | Stimulated (mmol/l) |
| Na⁺               | 145              | 5                 | 0-20-80             |
| K⁺                | 4                | 12                | 20                 |
| Ca²⁺              | 2.2              | 1.4               | 1-4                |
| Cl⁻               | 129              | 15                | 30-100              |
| HCO₃⁻ (mmol/l)    | 25               | 5                 | 15-80               |
| phosphate (mmol/l)| 1.2              | 6                 | 4                  |
| Mg²⁺              | 1.2              | 0.2               | 0.2                |
| SCN⁻ (mmol/l)     | <0.2             | 2.5               | 2                  |
| NH₄⁺ (mmol/l)     | 0.05             | 6                 | 3                  |
| (NH₄)₂CO (mmol/l) | 2-7              | 3.3               | 2-4                |
| Protein (g/l)     | 70               | 3                 | 3                  |

Inorganic components for protection

Electrolytes are transported in large quantities by salivary glands. Active chloride, sodium and potassium transport primarily serves as an osmotic drive for moving a great amount of water into the oral cavity. However, secreted calcium, phosphate and bicarbonate both have important roles for maintaining normal oral conditions. The 1-2 mmol/L calcium and the 4-6 mmol/L phosphate in saliva serve for enamel remineralization. Half of calcium is in free ionic form while the other 50% is complexed with salivary proteins, mainly with proline-rich proteins and statherin. This important protective mechanism prevents the precipitation of calcium phosphate in a bicarbonate buffered environment where pH is nearly neutral.

The 4-40 mmol/L bicarbonate in saliva is the primary buffer in the mouth. Remineralization of tooth enamel happens when the pH is over 5.5. Enamel starts to dissolve when pH is under this value. Carbohydrate rich food components and low pH drinks such as most flavoured carbonated drinks, fruit juices and wine consistently reduce pH. Under these conditions calcium phosphate is dissolved meaning demineralization of the enamel. The bicarbonate of saliva is the main factor to neutralize this mechanism. Bicarbonate is also the primary balancing...
factor against tissue destruction when acid reflux from the stomach delivers gastric acid into the esophagus and the oral cavity.

**Figure 1.147. Figure 2. – No saliva - consequences**

![Table showing symptoms and consequences of no saliva](image)

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Background – electrolyte deficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rampant dental caries</td>
<td>Acid buffering</td>
</tr>
<tr>
<td>Erosion of Enamel</td>
<td>HCO₃⁻</td>
</tr>
<tr>
<td></td>
<td>NH₄⁺(forms and aminoacids)</td>
</tr>
<tr>
<td>No remineralization</td>
<td>Ca²⁺ and PO₄³⁻</td>
</tr>
<tr>
<td></td>
<td>Co-binding proteins:</td>
</tr>
<tr>
<td></td>
<td>- proline-rich proteins</td>
</tr>
<tr>
<td></td>
<td>- Statherin</td>
</tr>
<tr>
<td>Extensive and rapid dental caries</td>
<td>Antibacterial factors</td>
</tr>
<tr>
<td>Candida infections</td>
<td>Antistreptococcal IgA</td>
</tr>
<tr>
<td>Tissue damage</td>
<td>Streptococcus + SCN</td>
</tr>
<tr>
<td>Digestive problems</td>
<td>Lactoferrin</td>
</tr>
<tr>
<td></td>
<td>Histatins</td>
</tr>
<tr>
<td></td>
<td>Cystatins</td>
</tr>
<tr>
<td></td>
<td>EGF</td>
</tr>
<tr>
<td></td>
<td>Amilase</td>
</tr>
<tr>
<td></td>
<td>Lipase</td>
</tr>
</tbody>
</table>

**Organic components for protection**

Proline-rich proteins are relatively high, while statherin is a small size protein, they have both a dual role being negatively charged. First they prevent calcium phosphate precipitation into salivary stones while the juice is still in the ductal system. Second, these proteins have high affinity to bind to enamel surface in the mouth. Therefore, they form the dental pellicle, a biofilm covering the teeth. In addition they are also able to bind tannins and other bioactive food constituents which otherwise may damage the oral tissues.

**Figure 1.148. Figure 3. – Organic components of mixed saliva**

![Table showing organic components of mixed saliva](image)

<table>
<thead>
<tr>
<th>Organic components of mixed saliva</th>
<th>Quantity</th>
<th>Main function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full protein</td>
<td>1400-2000 mg/l</td>
<td>Caries protective</td>
</tr>
<tr>
<td>Proline-rich proteins</td>
<td>1000-1400 mg/l</td>
<td>Antimicrobial</td>
</tr>
<tr>
<td>Lysozyme</td>
<td>199 mg/l</td>
<td>Antimicrobial</td>
</tr>
<tr>
<td>Lactoferrin</td>
<td>na</td>
<td>Antimicrobial</td>
</tr>
<tr>
<td>Siloperoxidase</td>
<td>3 mg/l</td>
<td>Antimicrobial</td>
</tr>
<tr>
<td>Secretory IgA</td>
<td>19.6 mg/l</td>
<td>Antimicrobial</td>
</tr>
<tr>
<td>IgG</td>
<td>14 mg/l</td>
<td>Antimicrobial</td>
</tr>
<tr>
<td>IgM</td>
<td>2 mg/l</td>
<td>Antimicrobial</td>
</tr>
<tr>
<td>Statherin</td>
<td>na</td>
<td>Caries protective</td>
</tr>
<tr>
<td>Gustin</td>
<td>~42-60 mg/l</td>
<td>Taste sensation facilitation</td>
</tr>
<tr>
<td>Histatins</td>
<td>na</td>
<td>Antimicrobial</td>
</tr>
<tr>
<td>Cystatins</td>
<td>na</td>
<td>Tissue regeneration</td>
</tr>
<tr>
<td>Amylase</td>
<td>380 mg/l</td>
<td>Digestion</td>
</tr>
<tr>
<td>Lipase (lingual gland origin)</td>
<td>na</td>
<td>Digestion</td>
</tr>
<tr>
<td>Urea</td>
<td>2.6 mmol/l</td>
<td>Acid neutralization</td>
</tr>
<tr>
<td>Glucose</td>
<td>0.05 mmol/l</td>
<td>&quot;plaque feeding&quot;</td>
</tr>
<tr>
<td>Aminocids</td>
<td>1.2 mmol/l</td>
<td>?</td>
</tr>
<tr>
<td>Mucin</td>
<td>110-330 mg/l</td>
<td>Lubrication</td>
</tr>
</tbody>
</table>

The main functions of mucins, which cover mucosal surfaces is to lubricate food, to protect mucosal integrity and to support protection against viruses, bacteria and fungi. Mucins and other glycoproteins are able to bind and agglutinate bacteria, primarily through their carbohydrate groups. These glycoproteins often contain sialic acids, which serve as free radical scavengers and participate in the interactions of host ligands and microorganisms. The major mucins in the mouth are MUC5B (also named MG1) and MUC7 (also named MG2). The similarly structured glycoprotein 340 (Gp-340) is an important related glycoprotein, known as salivary agglutinin having particularly strong binding affinity to oral streptococci inhibiting bacterial colonization.
Saliva contains multiple adaptive components of the immune system in the form of immunoglobulins (IgA, IgE, IgG, IgM), Immunoglobulin A being the major form among them. Salivary secretory IgA is a dimer connected by a polypeptide (J chain) and covered by a proteolysis-resistant secretory component to ensure its long biological half life in the oral cavity.

Histatins are a family of relatively small histidine-rich cationic peptides. Their major role is to show toxic activities against various pathogenic fungi, especially against C. albicans. This is primarily achieved by causing disintegration of the fungal cell wall. Mitochondria also represent the main target for histatins. It causes the death of yeast cells by the interference with the cell respiration through the formation of reactive oxygen species and damage mitochondrial function.

Both lysozyme, lactoperoxidase and lactoferrin belong to the innate defense system. Lysozyme disintegrates gram-positive bacterial wall by muramidase enzymatic activity. Lactoperoxidase oxidizes thiocyanate, an actively secreted component of saliva into hypothiocyanate, which is a very potent antibacterial agent. Lactoferrin is an iron-binding glycoprotein, but additionally it has been shown to possess antimicrobial and immunomodulatory effects.

Because of its known wound healing effect, saliva is known as historically regarded as “fountain of life”. The major component of this activity is due to the mass production of epidermal growth factor (EGF) delivered by the parotid into the oral cavity. EGF has a marked proliferative and cell protective effect in the mouth, the esophagus, and also reaching lower parts of the gut. Other growth factors (TGF-α, TGF-β, FGF, IGF-I, IGF-II and NGF) are present in lower concentrations in saliva as well.

**Figure 1.149. Figure 4. – Protective effects of saliva**

<table>
<thead>
<tr>
<th>Protective effects of saliva</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="#" alt="Lubrication" /></td>
</tr>
<tr>
<td><img src="#" alt="Antimicrobial effect" /> (bacteriostatic, fungostatic)</td>
</tr>
<tr>
<td><img src="#" alt="Mucosal integrity clearance effect" /></td>
</tr>
<tr>
<td><img src="#" alt="Buffering capacity" /></td>
</tr>
<tr>
<td><img src="#" alt="Remineralization of tooth" /></td>
</tr>
<tr>
<td>• Mucins</td>
</tr>
<tr>
<td>• Proline-rich proteins (PRP)</td>
</tr>
<tr>
<td>• Glucoproteins</td>
</tr>
<tr>
<td>• Water</td>
</tr>
<tr>
<td>• Lysozyme</td>
</tr>
<tr>
<td>• Lactoferrin</td>
</tr>
<tr>
<td>• Lactoperoxidase</td>
</tr>
<tr>
<td>• Histatins</td>
</tr>
<tr>
<td>• Mucins</td>
</tr>
<tr>
<td>• Electrolytes</td>
</tr>
<tr>
<td>• Water</td>
</tr>
<tr>
<td>• EGF</td>
</tr>
<tr>
<td>• Cystatins</td>
</tr>
<tr>
<td>• Bicarbonate</td>
</tr>
<tr>
<td>• Protein buffering capacity</td>
</tr>
<tr>
<td>• Calcium</td>
</tr>
<tr>
<td>• Phosphate and proline-rich proteins (PRP)</td>
</tr>
<tr>
<td>• Salivins</td>
</tr>
</tbody>
</table>

**Digestive saliva components**

Saliva is important for the digestion of consumed food. Besides neuronal control mastication directly stimulates salivary secretion. For bolus formation water is inevitable. The water flow and elevated amylase discharge both facilitate clearance of food remnants and digestion. Salivary amylase is encoded by a different gene when compared to pancreatic amylase. It hydrolyses O-glycosidic linkages of starch. Since the optimal pH for its activity is around 6, it is inactivated in gastric acidic environment. However, salivary amylase may partially renaturate upon reaching the intestine when pH returns to neutral. Salivary lipase is also secreted by the parotid gland and by the lingual von Ebner’s glands, but only in small quantities. In early postnatal life, however, when pancreatic function is not mature as yet, breast milk fat is primarily digested by salivary lipase. Therefore, its real physiological role is during the first postnatal months, when the immature pancreas is unable to produce the small quantity of lipolytic activity necessary to digest the small amount of fat in milk. Salivary glands also produce gustin, which sensitizes lingual taste receptors.

**Figure 1.150. Figure 5. – Digestive and speech-related roles of saliva**
Saliva collection is noninvasive. It is an easily reachable body fluid that is suitable to detect diseases of non-salivary origin. There are different types of fluids that can be obtained from the oral cavity. Besides mixed, unstimulated and stimulated whole saliva, ductal saliva can be collected from individual glands. The gingival crevicular fluid and the mucosal transudate is taken from local surfaces by capillary application. Buccal swabs may provide information on cellular and genetic materials. Plaque collection is important for cariology research. Volatiles serve for analyzing vaporized or gassous materials.

**Figure 1.151. Figure 6. – Variations of oral sample collection**

<table>
<thead>
<tr>
<th>Variations of oral sample collection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole saliva (stimulated/unstimulated)</td>
</tr>
<tr>
<td>Duct saliva</td>
</tr>
<tr>
<td>Gingival Crevicular Fluid</td>
</tr>
<tr>
<td>Mucosal Transudate</td>
</tr>
<tr>
<td>Buccal Swabs</td>
</tr>
<tr>
<td>Plaque</td>
</tr>
<tr>
<td>Volatiles</td>
</tr>
</tbody>
</table>

Standard, classical test have long been used to identify salivary gland originated disorders. But recently a number of non-salivary laboratory applications have become available. Oral HIV test is applied for suspected AIDS patients. Multiple tests for drug and alcohol abuse can also be used. The throat swab culture is applied to detect germs especially streptococcus that may cause infection in the throat. Forensic studies as well as control of athletes are performed to monitor steroid and other hormone levels. Salivary DNA samples are is also used for identifying single nucleotide polymorphisms as determinants or risk factors of certain diseases.

**Figure 1.152. Figure 7. – Major Current Uses of Oral Tests**

<table>
<thead>
<tr>
<th>Major Current Uses of Oral Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral HIV test</td>
</tr>
<tr>
<td>Tests for drugs of abuse</td>
</tr>
<tr>
<td>Strep throat swabs</td>
</tr>
<tr>
<td>Forensic studies</td>
</tr>
<tr>
<td>Monitoring steroid hormone</td>
</tr>
<tr>
<td>Determination for blood alcohol levels</td>
</tr>
<tr>
<td>Genetic testing (SNPs)</td>
</tr>
</tbody>
</table>
A number of new directions are under development, such as tests for malaria, oral cancer and other cancers, pulmonary diseases, cardiovascular disease (CVD) marker identification. Immunoassays from salivary proteins may help to characterize periodontal diseases, the oral bacterial flora as well as other disorders.

**Figure 1.153. Figure 8. – Potential present targets for saliva as a diagnostic factor**

- Malaria
- Pulmonary diseases
- Oral cancer
- CVD markers
- Immunoassay SPR
- Periodontal diseases
- DNA microarray Oral bacteria
- Salivary Proteome

Current research focuses to develop new technologies that may simplify and miniaturize saliva analysis. The aim is to develop cheap and reliable non-invasive diagnostic methods. The success of this plan is already evident not only from scientific publications but also in actual laboratory test products. Saliva proteome analysis will result in early detection of multiple diseases in an inexpensive, fast and reproducible way.

**Figure 1.154. Figure 9. – Salivary research tool development**

New technologies will simplify and miniaturize saliva analyzing techniques reaching cheap and reliable non-invasive diagnostic methods. A prototype of such instruments is shown below that is suitable to read and analyze tiny quantities of salivary proteins.

**Figure 1.155. Figure 10. – Example for research directions**

Saliva proteome analysis may result in early detection of multiple diseases in an inexpensive, fast, noninvasive way.
At present 665 various proteins have been identified in saliva, but this number is increasing by the increase of sensitivity of the methods. Many of these, originating from the blood circulation, may serve as a marker of a certain disease. As it appears, these proteins are not only from salivary cell origin but virtually all proteins appear in saliva which is present in the plasma. This is important, because many proteins appear only in body fluids when a certain disease, such as cancer develops, partly because of the particles released from dead cells, partly because of the reaction of the immune system.

Figure 11. – Number of proteins identified in major gland secretions

Figure 12. – Salivary proteome: Functional categories

14.1. Test – Oral function and diagnostic role of secreted saliva (answers)

1. Main source of epidermal growth factor (EGF) in humans
   A. dental pulp
   B. pancreas
   C. duodenum
   D. parotid gland

2. The role of lysozyme in saliva:
   A. dissolves bacterial wall
   B. dissolves mammalian cell wall
C. breaks zymogen granules
D. all of them
E. none of them

3. Basic mechanism of the antibacterial action of lactoferrin
A. metal ion donor
B. metal ion binding
C. lactic acid liberation
D. all three
E. none of them

15. 1.15. Dental stem cells for dental research – Gabor Varga

Many of the outcomes of modern biomedical research have the potential for clinical application. From developmental biology research we increasingly recognize the molecular factors determining the number, position, and shape of the teeth. On this basis, it may be possible to regenerate or completely rebuild dental and periodontal tissue using the latest stem cell, molecular biology, and embryology results, using the normal growth and development of various tissues as a model.

Figure 1.158. Figure 1. – Sections of tooth undergoing development

The human body is capable of substantial regeneration. In certain tissues, such as blood cells or epithelial cells, cells continuously divide and regenerate throughout life while the cells of other tissues regenerate more slowly and this may start only with certain biological signals. This ability to regenerate depends on the varying degrees of commitment of stem cells. A stem cell is any cell which has a self-renewal capacity, and also can create differentiated progeny. However, this definition covers a heterogenous cell population in terms of their differentiation capabilities.

Figure 1.159. Figure 2. – Definition of stem cell
After conception, the fertilized ovum in the Fallopian tube migrates toward the uterus while dividing several times. The intercalating embryonic cells are totipotent. The embryonic stem cells that exist in this phase of ontogeny are, in principle, able to create all types of tissue. They therefore provide opportunities for research to identify the processes that take place during development and to develop new methods based on stem cells in tissue regeneration. However, in relation to stem cell biology, many fundamental questions are unanswered, as shown by the serious technical difficulties of controlling the differentiation of cells into certain tissue or eliminating an immune response during subsequent differentiation. In addition, the use of embryonic stem cells for clinical/research purposes raise many moral and ethical problems and the laws regulating their usage have not been solved. During the second week of fetal development these cells begin to specialize and, as a result, lose their totipotency. During fetal development, a small number of non-specialized stem cells remain, which can be obtained at birth from the blood of the umbilical cord.

**Ethical and immune problems**

It has long been known that the so-called adult, or postnatal, stem cells that are found in mature tissues have a role in the constant renewal and after-injury regeneration of tissues. Hematopoietic stem cells isolated from bone marrow are theoretically capable of creating each type of blood cell. Bone marrow stromal stem cells are responsible for injured bone reclamation and daily microfracture repairs. Recent results suggest that these tissue stem cells assume a much broader differentiation potential than had been expected. If it turns out to be true, the use of postnatal stem cells could by-pass the ethical problems related to the use of embryonic stem cells. These tissue stem cells may be useful in tissue differentiation research, can be used as a test system during the development of drugs, and may also serve as cell therapy for tissue regeneration.
Continuous, life-long growth of rodent incisors is well known and it may therefore be acceptable to everyone that they have a constantly renewing population of stem cells.

Figure 1.162. Figure 5. – Potential utilization of stem cells

Figure 1.163. Figure 6. – Stem cells are continuously present in the cervical loop of mouse incisors
Stem cells of dental origin that are similar to bone marrow stromal stem cells were described for the first time only about a decade ago. Since then, researchers have succeeded in identifying such stem cell populations from adult and deciduous tooth pulp, periodontal ligament and also from dental follicle. These are clonogenic cells with high proliferative activity and they express the signal proteins of mesenchymal stem cells.

**Figure 1.164.** Figure 7. – Colony forming of periodontal ligament derived stem cell cultures

The plated periodontium derived cells form colonies.
This photo shows 14 days old colonies.

These cells, enclosed in suitable capsules containing hydroxyapatite crystals, can be implanted under the skin of immunodeficient mice, and over several weeks dentin-like tissue is created. However, bone marrow stem cells transplanted to other animals created hard tissue with bone-like characteristics and blood-forming structures within the capsules. This clearly indicates the capacity of stem cells to construct new tissue but, on the other hand, clearly show the differences between the two cell types.

**Figure 1.165.** Figure 8. – Stem cells of dental and bone origin
These research results show that in a suitable extracellular medium, bone marrow stromal stem cells are capable of creating a bone tissue structure in which the cells are surrounded by the created mineral phase. Thus they may be applied mainly in the regeneration of bone.

**Figure 1.166. Figure 9. – 1 – Stem cells for bone regeneration**

However, in the case of the pulp-derived stem cells there is a hope for dentin-specific mineralization, in which the differentiating odontoblasts are arranged on the mineralized phase surface, and thus form a characteristic acellular mineralized dentin film, and cover the surface like mantle.

**Figure 1.167. Figure 10. – 2 – Pulp and dentin regeneration**
In the case of periodontal ligament stem cells, the use of similar conditions can give us hope for the renewal of entire periodontal structures. It is definitely more difficult than the reconstruction of bone and dentin, as in this case, the cement that covers dentin has to be created, and also collagen fibers containing a non-mineralized film that binds transversely both to the cementum and the bone.

**Figure 1.168. Figure 11. – 3 – Periodontal regeneration**

![Diagram](image1)

In addition to the above, the application of stem cells can give a new direction for implant-retaining osseointegration research as well. Currently the most important goal is to increase the speed and efficiency of the processes that promote ossification at the titanium implant surface. A more distant goal is to formulate a structure that is similar to normal periodontium, which would include the cement on the implant surface, the periodontal fibers that bind there and, at the other side, the insertion of the fibers in the bone tissue.

**Figure 1.169. Figure 12. – 4 – Support for implantation**

![Diagram](image2)

Rather than relying on artificial implants, regrowing missing teeth may be achieved within five years, said Paul Sharpe, a professor at King's College London, in a high-profile interview in 2004. To the best of our knowledge this prediction in humans has not yet become true, not least because not only stem cells are needed to develop a tooth properly. Additionally the topically timed release of bioactive growth and differentiation factors, and also a biocompatible and biodegradable carrier matrix is necessary for such an achievement.

**Figure 1.170. Figure 13. – 5 – Complete tooth regeneration**
Animal studies, however, indicate that for total tooth development we already have the technical aptitude. A Japanese research team has isolated separately the epithelial and mesenchymal cells from embryonic mouse tooth. After pre-culturing the cells, they embedded the two embryonic progenitor cell types in a collagen gel in two layers over each other. After a few days of incubation they implanted the organ culture in an adult mouse in place of an extracted molar. The study showed extraordinary success: in a few weeks a complete molar had developed from the initial artificial organ.

Figure 1.171. Figure 14. – Importance of cells, scaffold and bioactive molecules

Figure 1.172. Figure 15. – Process of tooth replacement in mouse
This work has an exceptional interest for studies concerned with partial or complete tooth regeneration. However, the direct translation of these studies to humans must be treated with great caution. First, we should not lose sight of the obvious differences between mouse and human tooth development. On the other hand, to isolate totally a human embryonic tooth germ and then implant it in another human is ethically unacceptable. In addition, the in vitro development of a human tooth germ has obviously not yet been achieved. This makes it much more realistic, and more promising for clinical dentistry to regenerate teeth and periodontal tissue. For this we have to proceed in the research of the stem cells, bioactive materials and other components used in implant research.
Figure 1.174. Figure 17. – Three key elements for regenerative dentistry

![Diagram showing scaffold, cells, and signals in regenerative dentistry.]

- **Scaffold**: Collagen, Fibronectin, Fibrin, Hyaluronic acid, Proteoglycan, Foams, fibers, Gels and membranes
- **Cells**: Adult Embryonic Marrow stroma PDL stem Dental pulp stem
- **Signals**:TGFRs/BMPs FGFs WNTs Hedgehogs

- **Regeneration**: Alveolar bone, Periodontal ligament, Cementum, Dentin, Dental pulp, Enamel

Figure 1.175. Figure 18. – Dental stem cells show neuronal morphology after a two weeks neurogenic differentiation protocol

![Image of neuronal morphology.]

Scale bar: 50 μm
Figure 1.176. Figure 19. – Due to osteogenic differentiation of dental stemcells, calcium deposits occur, and made visible with von Kossa staining

15.1. Test – Dental stem cells for dental research (answers)

1. The cells which form the pulp originate from the neuronal chrest, therefore we call them:
   A. neuromesenchymal
   B. ectomesenchymal
   C. neuromesodermal
   D. ectoneuronal

2. Fundamental characteristic(s) of stem cells:
   A. self-renewing capacity
   B. only those originate from early embryo
   C. cells without proliferation control
   D. cells which have the capacity to renew themselves, and additionally able to produce differentiated
   E. none of the above

3. The stem cells of dental origin are able to regenerate the following structure(s):
   A. exclusively dentin
   B. exclusively enamel
C. exclusively periodontal ligamentum
D. exclusively alveolar bone
E. none of the above

16. 1.16. Nutrition and oral health; Characterization of oral tissues and function in elderly – Gábor Varga

The consumption of an adequate quantity and quality of food is essential in maintaining the healthy functioning of the body. Healthy oral structures play an important role in food intake. For this, the intake and processing of an appropriate amount of food is essential. It is therefore clear that general health, oral health and an adequate supply of nutrients are closely interdependent.

Figure 1.177. Figure 1. – Triangle of the interrelationship between general and oral health and nutrition

This relationship is valid not only quantitatively but also qualitatively. For normal body function, not only macronutrients, including proteins, carbohydrates and fat, are strictly necessary, but also the micronutrients, such as vitamins, minerals, and other small quantities of components. Furthermore, insoluble, non-utilizable fibrous components, such as cellulose are also important, because they are necessary for the balanced function of the digestive tract. In addition to the above, with smoking and alcohol consumption, significant quantities of other substances get into the oral cavity and the gastrointestinal tract, that affect the biological processes. On one hand, ingested food components have direct effects on oral structures and digestive tract elements through direct physical and chemical routes. On the other hand, absorbed food components, once within the organism, can act either as a nutrient or as an agent that modifies or even damages the function of the gastrointestinal tract, including teeth and oral structures.

Figure 1.178. Figure 2. – Components of the diet

The various dietary components interact with each other. However, for didactic reasons we will cover the impact of individual components on oral health separately, even though their joint effect is an important issue as well. Protein-energy undernutrition (PEU), which occurs even in developed countries, is caused by a deficiency of all macronutrients including proteins, carbohydrates and fats, as well as of micronutrients, rather than that of a single component. Proteins provide about 10 to 15% of the total energy. The minimum daily protein intake is somewhat less than 1g/kg body weight, but this must include an appropriate balance of essential amino acids.
Protein-energy malnutrition is also important from the aspect of the oral cavity, as developing and renewable oral structures could be damaged as a result of malnutrition.

**Figure 1.179. Figure 3. – Protein necessities**

<table>
<thead>
<tr>
<th>Protein necessities</th>
<th>11-13% of total energy necessity</th>
</tr>
</thead>
<tbody>
<tr>
<td>□ The minimal necessary quantity of proteins that keeps nitrogen balance steady (1,0 g/kg b.w.).</td>
<td></td>
</tr>
<tr>
<td>□ Under balanced conditions, the uptake and loss of nitrogen is equal.</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 1.180. Figure 4. – Reasons for protein deficiency**

<table>
<thead>
<tr>
<th>Reasons for protein deficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>□ Quantitative deficiency</td>
</tr>
<tr>
<td>□ Qualitative deficiency (shortage of essential amino acids)</td>
</tr>
<tr>
<td>□ Shortage in energy uptake</td>
</tr>
<tr>
<td>□ Usually a combination: qualitative and quantitative deficiency, and also vitamin and mineral deficiency together: Protein-energy undernutrition - PEU</td>
</tr>
</tbody>
</table>

The largest share of the energy used by the body, 50 to 70%, is replaced by the intake of carbohydrates. So their intake is really important energetically, however, what really matters is what types of carbohydrate enter the mouth. Instead of the desired complex sugars such as starch, our eating is increasingly characterized by the consumption of simple sugars and disaccharides. However, these have a very strong cariogenic impact as they can be used directly as a substrate for the cariogenic anaerobic bacteria that accumulate during oral plaque formation. Simultaneously the protons released during bacterial glycolysis make oral pH acidic, which clearly favors the demineralization processes, such as formation of dental caries. Another important effect of the consumption of simple sugars and substantially pre-digested food is that the chewing activity is significantly reduced. If this loss of functional activity persists in childhood, it can lead to jaw size retardation, and thus malocclusion and congestion.

**Figure 1.181. Figure 5. – Harmful effects of high carbohydrate diet in the oral cavity**

<table>
<thead>
<tr>
<th>Harmful effects of high carbohydrate diet in the oral cavity</th>
</tr>
</thead>
<tbody>
<tr>
<td>□ Dental caries</td>
</tr>
<tr>
<td>• mainly sucrose, but other sugars and even starch</td>
</tr>
<tr>
<td>• can feed bacteria</td>
</tr>
<tr>
<td>□ Dental malocclusion</td>
</tr>
<tr>
<td>• reduction of mandibular in size, and functional</td>
</tr>
<tr>
<td>• inactivity of masticatory muscles</td>
</tr>
</tbody>
</table>

**Figure 1.182. Figure 6. – Balance of demineralisation and remineralisation**
About 20 to 40% of our energy is covered by the intake and degradation of fats. Excessive consumption of fats leads to obesity. However, this can occur even at a relatively low fat intake when there is excessive intake of carbohydrates or proteins, which shifts the balance toward the accumulation of fat. It is important to note that fat is essential for the dissolution and thus the absorption of fat-soluble vitamins and therefore a fat-poor diet can lead to a deficiency of such vitamins. Fats also have a favorable, cariostatic effect on teeth as they form a hydrophobic coating on the surface of teeth.

**Figure 1.183. Figure 7. – Function of fats**

<table>
<thead>
<tr>
<th>Function of fats</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Energy supply</td>
</tr>
<tr>
<td>☐ Nutrient store</td>
</tr>
<tr>
<td>☐ Carrier of vitamins (vit. A and D)</td>
</tr>
<tr>
<td>☐ Saturated vs unsaturated:</td>
</tr>
<tr>
<td>☐ unsaturated fats are favourable</td>
</tr>
<tr>
<td>☐ Omega-3 fatty acids – from fish</td>
</tr>
<tr>
<td>☐ Fats are – from a viewpoint of cariology – cariostatic</td>
</tr>
</tbody>
</table>

Hypovitaminosis and avitaminosis, namely the relative or complete lack of various vitamins, can lead to typical oral symptoms. The lack of vitamins A and E specifically lead to oral epithelial lesions and over-keratinisation. A reduced intake of B vitamins causes lip and oral epithelial lesions. This is accompanied by mouth angle cracking, inflammatory discoloration of the tongue, papillary atrophy, and a burning sensation.

**Figure 1.184. Figure 8. – Vitamin A deficiency**
Vitamin D deficiency primarily affects the organic and mineral deposits in bone tissue and affects the jaw. In addition, when it occurs in childhood, enamel and dentin formation is significantly impaired. A major and lasting lack of vitamin C can manifest in scurvy, and especially in gum atrophy. In the absence of vitamin K, the formation of gamma-carboxyl-glutamine groups of non-collagenous proteins involved in biomineralisation may not occur, but this has no significant negative effects.

Vitamin A deficiency
- Epithel cell hyperplasia
- Keratinization of epithelium, mucosa, gingiva, salivary gland ducts
- Inactivation of osteoclasts
  - thickening of bones
- Degeneration of ameloblasts
  - enamel hypoplasia
- Experimental withdrawal in rodents
  - dentin development disturbance
  - calcification of periodontal ligaments

**Figure 1.185. Figure 9. – Deficiency symptoms of B vitamins**

**Deficiency symptoms of B vitamins**

- **B2 Riboflavin**
  - Cheliosis angularis, atrophy of filiform papillae, enlarged fungiform papillae, shiny-red lips & tongue, painful tongue
- **B3 Niacin**
  - Cheliosis angularis, mucositis, stomatitis, pain & burning feeling in the mouth, glossitis, aching & swollen tongue, flushed tip of tongue, smooth & dry back of tongue, gingivitis ulcerosa
- **B6 Pyridoxin**
  - Cheliosis angularis, pain & burning in the mouth, glossitis, painful tongue
- **B12 Cyanocobalamin**
  - Cheliosis angularis, pain & burning in the mouth, mucositis, stomatitis, glossitis, gingivae bleeding, oral paresthesia (numbness & itching in the mouth), slower recovery of wounds, aphthous-like ulcers
- **Folic acid**
  - Cheliosis angularis, mucositis, stomatitis, pain & burning in the mouth, glossitis, increasing disposition for oral candidiasis, swollen tip and back of tongue, bare & pale back of tongue

**Figure 1.186. Figure 10. – Vitamin D deficiency in the oral cavity**

**Vitamin D deficiency in the oral cavity**

- Predentin widens, calcification time is elongated
- Dentin-predentin outline becomes irregular
- Enamel hypoplasia after long-term D vitamin deficiency (debatable, further proofs needed)
- No significant change in tooth calcification, because their calcification activity is stronger than that of bones; and because there is no remodeling in the dental minerals (vs. bone).

**Figure 1.187. Figure 11. – Other important micronutritional factors**
Overall, a balanced diet is a good basis for the maintenance of physical and also oral health. The two figures below provide guidelines for a balanced diet.

**Figure 1.188. Figure 12. – Direction towards healthy nutrition**

<table>
<thead>
<tr>
<th>Intake of saturates</th>
</tr>
</thead>
<tbody>
<tr>
<td>- milk- and meat-products, hard margarine</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Intake of trans fatty acids</th>
</tr>
</thead>
<tbody>
<tr>
<td>- margarine with partially hydrogenated fish oil, milk products</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ω-3 fatty acids</th>
</tr>
</thead>
<tbody>
<tr>
<td>- fatty fish, cod liver oil, fish oil</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vitamins D and E</th>
</tr>
</thead>
<tbody>
<tr>
<td>- fatty fish, cod liver/fish oil, grain, soya bean oil</td>
</tr>
</tbody>
</table>

**Figure 1.189. Figure 13. – Ratio of food components in healthy nutrition**
16.1. Test – Nutrition on oral health (answers)

1. Role of fat in caries formation
   A. cariostatic
   B. caries promotion
   C. caries initiation
   D. demineralization
   E. remineralization

2. Its deficiency leads to gingivitis and periodontitis
   A. vitamin A
   B. vitamin B
   C. vitamin C
   D. vitamin D
   E. vitamin E

3. The decrease of functional activity of chewing muscles may result in:
   A. erosion
   B. dehydration
   C. hypodontia
   D. malocclusion
   E. abrasion

17. 1.17. Pathophysiology of chewing – Mate Jasz

Movements of the mandibular joint
Human mandible is a limited free moving joint, because it is able to perform movements to a lesser or greater extent along all the three axes in space. The bony articular surfaces allow almost perfectly free movement, limited only by the joint capsule, ligaments, articular disc, mandibular muscles and the junction of antagonist teeth.\(^1\)

The temporomandibular joint (TMJ) can be divided into two parts regarding both topology and function. Within the common joint capsule, the discotemporal joint is situated cranially and the discomandibular joint caudally. In the discotemporal joint, the articular cartilage performs a translational (gliding) movement: on the lower surface of the tuberculum articulare it moves forward and descends, along an upwards and forwards differently convex path. In the discotemporal joint, the most important basic movement is the discus translation parallel to the sagittal plane. This is the only movement in the discotemporal joint during any jaw movement that lacks the sideward excursion from the midline. Such jaw movements include mouth opening or protruding the chin. With normal TMJ function, these movements are not associated with sideward excursion from the midline, because motions in the two joints are approximately identical.\(^2,3,4\)

![Figure 1.190. Figure 1.](image_url)

![Figure 1.191. Figure 2.](image_url)

During laterotrusion, ie. sideward excursion of the jaw, condylar heads on the working versus the balancing side perform different movements. On the balancing side, a medial excursion accompanies the basic translational motion. The possible extent of moving toward the midline increases advancing forward from the posterior jaw position, ie. moving forward in the sagittal plane, the range of motion in the frontal plane increases, forming a V shape. This defines an upward and forward concave, forward broadening, C-shaped surface that contains the points where the mandibular condyle can be located. In addition to this path, the condyle on the working side follows an irregular, often loop-shaped deviating path of a few millimeters lateral and/or posterior to the CR position during lateral excursions. In contrast to sagittal translational movement, medial and lateral excursions are never symmetric. Therefore, the Bennett angle, which describes the medial excursion of the condyle, and the side shift and shift angle, which describe lateral excursion, are often very different between opposite sides in the same patient, but it is not necessarily pathologic.\(^5,6\)

![Figure 1.192. Figure 3.](image_url)
In the more caudally located discomandibular joint, condyles perform a rotating movement. The rotation can occur along the horizontal, frontal and sagittal axes – one of the reasons why TMJ is called a free moving joint. Turning around the horizontal (transverse) axis is, the most important and most prominent one of the rotational movements. The horizontal axis passes through the two condylar heads, the location of which constantly changes due to translation during different mouth movements: it moves forward, as previously mentioned, along a forward and upward concave path.

Various combinations of two kinds of each translational and rotational movements in both mandibular joints yield the complex three dimensional movements of the jaw, such as those observed during mastication.

1. video – Mastication.
2. video – Mastication with chewing gum.
3. video – Mandibular movements: slide-open-close.
4. video – Mandibular movements: open-close-slide.

17.1. Test – Chewing (answers)

1. While doing a mouth opening:
   A. Condyles move backwards
   B. Condyles perform different movements on the left and right side
   C. Only rotational movement occurs
   D. The left and right condyles move symmetrically along a curved path forward and downwards, while a rotational movement also occurs
   E. First the right, then the left condyle perform a translational movement

2. Under normal circumstances the left and right condyles perform different movements during:
   A. Mouth opening
   B. Protrusive movement
   C. „Drawing” the sagital Posselt diagram
   D. Chewing
   E. Talking

3. Which one belongs to the temporomandibular joint?
   A. Condylus mandibulae
   B. Discus articularis
C. Fossa articularis
D. Capsula articularis
E. All of them

References
3. Friedman M. H., Weisberg J.: Temporomandibular Joint Disorders: Diagnosis and Treatment. Quintessence, Chicago

18. 1.18. Pathophysiology of inflammation – Beata Keremi

18.1. Inflammation

Inflammation is a defensive reaction of the tissues to eliminate harmful stimuli. It is a multi-step process: (1) elimination of the initial cause of cell injury - eg. diluting, destroying and neutralizing the harmful agents, (2) removal of the damaged tissue, (3) generation of new tissue.

Immune reactions, injuries and ischemic damages are involved in the induction of inflammatory response.

Figure 1.193. Table 1. – Causes of inflammation

<table>
<thead>
<tr>
<th>Table 1. Causes of inflammation</th>
<th>Endogenous:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exogenous:</td>
<td></td>
</tr>
<tr>
<td>• mechanical</td>
<td>• circulatory disorders, hypoxia</td>
</tr>
<tr>
<td>- foreign body</td>
<td>• fragmented tumor cells</td>
</tr>
<tr>
<td>- cut, stab, scratch</td>
<td>• extravascular blood</td>
</tr>
<tr>
<td>• physical</td>
<td>• endogenous protease release</td>
</tr>
<tr>
<td>- frostbite, scald</td>
<td></td>
</tr>
<tr>
<td>- radiation (UV, X-ray, radioactivity)</td>
<td></td>
</tr>
<tr>
<td>• chemical</td>
<td>• immune complex formation</td>
</tr>
<tr>
<td>- acid and alkali</td>
<td>• autoimmune reactions</td>
</tr>
<tr>
<td>• biological</td>
<td>• crystals of precipitated substances in the body</td>
</tr>
<tr>
<td>- microorganisms</td>
<td>- uric acid, calcium oxalate, calcium phosphate, cholesterol</td>
</tr>
<tr>
<td>- foreign body (foreign proteins)</td>
<td></td>
</tr>
</tbody>
</table>

Figure 1.194. Figure 1. – Classic signs of inflammation. The first four signs were described by Celsus (30 BC – 38 AD) and the fifth by Galen (129 AD – cca. 200-216 AD)
Repair mechanisms in the body include the thrombotic and fibrinolytic system, inflammation, immune response and oral defense. Common mechanisms in all systems include (1) the serum protease-antiprotease system, (2) a redox system with free radicals, (3) activation of the complement system, and (4) phagocytosis.

18.2. Features of acute inflammation

Figure 1.195. Table 2. – Classification of inflammation

<table>
<thead>
<tr>
<th>Table 2. Classification of inflammation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute</strong></td>
</tr>
<tr>
<td><strong>Cause</strong></td>
</tr>
<tr>
<td><strong>Duration</strong></td>
</tr>
<tr>
<td><strong>Specific symptom</strong></td>
</tr>
<tr>
<td><strong>Main components in the process</strong></td>
</tr>
<tr>
<td><strong>Reactions connected</strong></td>
</tr>
</tbody>
</table>

18.2.1. Cells involved in acute inflammation

Figure 1.196. Table 3. – Cells involved in acute inflammation and their functions
18.2.2. Two main phases of acute inflammation

Vascular phase

- leads to an increase in blood flow
- changes in the small blood vessels of the microcirculation are characterized by a tripleresponse:
  1. momentary vasoconstriction (seconds)
  2. vasodilation – arterioles, venules (minutes)
  3. increased capillary permeability and its consequences:
     - swelling
     - mediator release
       - histamine
       - nitric oxide (NO)
     - increased viscosity
• increased blood clotting (hours)

**Figure 1.197. Figure 2. – The vascular phase of inflammation is characterized by the triple response: 1. vasoconstriction, 2. vasodilation, 3. elevated capillary permeability, increased exsudate and consequently oedema formation**

![Diagram of vascular phases](image)

**Cellular phase**

• begins with changes in the vascular endothelial cell layer,
• leads to leukocytes exiting from the bloodstream,
• phagocytic leukocytes migrate to the site of infection or injury
• harmful factors are destroyed by their activation.

**Main steps:**

1. rolling,
2. margination
   • vasodilation– stasis
   • RBC forms coils in the middle of blood vessels
   • granulocytes at the walls of blood vessels – upregulation of adhesion molecules (selectin, integrins: ICAM-1, VCAM)
3. adhesion
   • granulocytes adhere to the epithelium
   • function of adhesive proteins
   • pavementing
4. emigration (diapedesis)
   • matrix changes in the cytoplasm of granulocytes
   • extravasation (leaving the blood vessel) through the pores
5. chemotaxis – migration
   • migration to the target cell
   • chemotactic agents (see below)
A variety of inflammatory mediators are released in inflammatory processes which can be vasoactive and chemotactic substances. Vasoactive substances affect local blood flow (rubor, calor), exudate formation (tumor) and platelet aggregation, and thus indirectly wound closure. Chemotactic substances promote the migration of cells involved in tissue defense.

**Figure 1.199. Table 4. – Inflammatory mediators in acute inflammation**
Figure 1.200. Table 5. – Grouping of inflammatory mediators based on function

<table>
<thead>
<tr>
<th>Cascade</th>
<th>Vasodilation</th>
<th>Oedema</th>
<th>Chemotaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mast cells</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thrombocyte</td>
<td>Histamine</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Serotonin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plasma components</td>
<td>Bradykinin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complement system</td>
<td>C5a/3a</td>
<td>C5a/3a</td>
<td>C5a/3a</td>
</tr>
<tr>
<td>Cytokines</td>
<td></td>
<td>IL-1</td>
<td>IL-8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TNF</td>
<td></td>
</tr>
<tr>
<td>Lipid mediators</td>
<td>LTC₄/D₄</td>
<td>LTC₄/B₄</td>
<td>LTB₄</td>
</tr>
<tr>
<td></td>
<td>PGE₂/I₂</td>
<td>PAF</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PAF</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### 18.2.3. Cytokines

Cytokines are small glycoprotein molecules produced by the cells involved in immune response and inflammation. They play roles in the transduction of information and in the regulation of immune response.

Cytokines may be functionally grouped based on whether they play a role in:

I. Natural immunity and inflammation.

II. Regulating lymphocyte activation and differentiation.

III. Maturation of immune cells.

<table>
<thead>
<tr>
<th>Table 5. Grouping of inflammatory mediators based on function:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Vasoactive and smooth muscle contracting properties</td>
</tr>
<tr>
<td>• histamine, serotonin</td>
</tr>
<tr>
<td>• arachidonic acid metabolites (originated from membrane phospholipids)</td>
</tr>
<tr>
<td>- phospholipase A&lt;sub&gt;2&lt;/sub&gt; – starting substrate for other inflammatory mediators</td>
</tr>
<tr>
<td>- prostaglandins</td>
</tr>
<tr>
<td>- thromboxane</td>
</tr>
<tr>
<td>- leukotrienes—lipoxygenase pathway</td>
</tr>
<tr>
<td>- thromboxyte activating factor (PAF)</td>
</tr>
<tr>
<td>- other inflammatory and hemostatic mediators</td>
</tr>
</tbody>
</table>

2A. Plasma proteases

- complement system
  - vasodilation, increasing of vascular permeability and phagocyte activation
  - its activation:
    - normal pathway — bacterial or viral infection
    - alternative pathway — antigen-antibody complex formation
  - C3a, C4a, C5a - anaphylaxin
- kininogen – bradykinin release
  - smooth muscle constriction and vessel dilation, increasing vascular permeability

2B. Coagulation factors

- Hageman factor (factor XII)

2C. Vasoactive peptides

3. Chemotactic factors

4. Reactive molecules and cytokines – release from leukocytes

- preformed mediators
- newly synthesized mediators
  - nitric oxide, superoxide derivatives
  - cytokines

5. Adhesive proteins

- selectin group

6. Antibodies

7. Neurogenic mediators

- substance P, calcitonin gene related peptide (CGRP)

8. Other factors

- growth factors (GFs)
- colony stimulating factors (CSFs)
18.2.4. Chemokines

Chemoattractants, promote and direct the migration of immune and inflammatory cells to sites of injury and / or pathogen entry.

They are classified into two groups:

- **inflammatory chemokines**
  - produced in response to bacterial toxins and inflammatory cytokines

- **homing chemokines**
  - continuous expression
  - up-regulated during inflammatory reactions and immune responses
They are proteins with a molecular weight of 8 to 10 kDa which share a 20 to 70% amino acid sequence homology. Subgroups are defined based on the location of the cysteine (Cys) residues within the molecule:

1. α-chemokines: contain four Cys residues with one amino acid (x) between the first two cysteines: a -C-x-C- structure.

This subgroup has been further divided into subgroups:

1. Before the -C-x-C- group, near the N-terminus, a Glu-Leu-Arg sequence is located. Substances of this subgroup have chemotactic effects on neutrophils.

2. Chemokines in this subgroup do not contain the motif mentioned in subgroup 1.1. Substances of this subgroup are chemotactic on lymphocytes.

2. β-chemokines: the first two Cys residues are adjacent, a -C-C- structure.

3. γ-chemokines: only have two Cys residues, one is N-terminal, the other is further away, a -C- structure.

4. fractalkines: have three amino acid residues between the two cysteine residues, a -C-x*-x*-C- structure.

Chemokine receptors are seven transmembrane G protein coupled receptors. Certain chemokine receptors are cell type specific while others are non-specific.

The two most important classes of chemokine receptors are:

1. α-chemokine receptors: this group includes eight currently known members that recognize CXC chemokines with an ELR amino acid motif next to their CXC motif.

2. β-chemokine receptors: they recognize cytokines of the CC chemokine family. Currently ten members of this group have been identified.

3. γ-chemokine receptors: currently one receptor known (-x-C-R1 motif).

4. CX3C-receptor: currently one receptor known (-C-x*-x*-C-R1 motif).

Figure 1.202. Figure 4. – Structures of chemokines. Figure reproduced by permission from László Köhidai

18.2.5. Oral aspects of inflammation

The characteristics described for inflammation in general apply to inflammatory conditions in the mouth as well. Some aspects, however, make these conditions special: mouth is the first section in the alimentary canal, and, importantly, a boundary between the inner and outer environment. Typical inflammatory conditions in the oral cavity are: pulpitis, gingivitis, periodontitis and mucositis. More information is provided on oral aspects of inflammation in the individual chapters on specific inflammatory conditions and on oral defense mechanisms.
1. Oral biology

18.3. Test – Pathophysiology of inflammation (answers)

1. What is the consequence of increased capillary permeability?
   A. vasoconstriction due to NO release
   B. platelet aggregation and thrombus formation in a few minutes
   C. pure fluid outflow from blood vessels
   D. oedema formation
   E. pos formation

2. Which of the following proteins are required for granulocyte adhesion?
   A. chemokines
   B. VEGF
   C. lysosome
   D. integrin
   E. histamine

3. Which is true for inflammatory cytokines?
   A. only chemoattractant properties
   B. have a role in the regulation of immune response
   C. produced by endothelial cells
   D. all of the above
   E. none of the above

References


19. 1.19. Structural and functional characteristic of dental pulp, blood supply to the oral tissues, pulpal pain and inflammation – Gábor Varga

The dental pulp is a non-mineralized loose-fibrous connective tissue that fills the pulp chamber. What determines its structure is the fact that its loose-fibrous texture is surrounded by dentin forming an inflexible structure which is unable to dilate. Nerves and vessels pass through the apical foramen located at the apex of the root and provide a connection between the pulp and the systemic environment. Occasionally narrower secondary
canals/channels can be discovered running laterally from main canal. The main function of the pulp is to supply the odontoblast layer. The dental pulp is also able to repair the dentin after a partial injury. In addition it has a sensory function for the protection of the dentin. The pulp is fundamentally different from the surrounding dentin. Nevertheless, due to their developmental and functional connection, the pulpo-dentinal complex forms a virtual unit.

**Figure 1.203. Figure 1. – What is the dental pulp?**

Pulp is separated from dentin by the praeedentin layer and above this a monolayer of odontoblasts form a sheet on the periphery of the pulp. Inside from the odontoblast layer the so-called *cell-free zone* (the zone of Weil) is located, with the nerve-rich subodontoblastic plexus. Under this is located a *cell-rich zone* with fibroblasts and other undifferentiated mesenchymal/blastoid cells and a rich capillary network. The arterioles and venules supplying the pulp run in the central zone which is located in the middle of the pulp chamber.

**Figure 1.204. Figure 2. – Fine structure of the pulp**

25% of the pulp is organic material, 75% is water. The basal substance has a gel structure. Among its organic components collagen is found, in the largest amount mostly type III, but types I and V can also be identified. Elastin can only be found around the larger vessels. The gel-like consistency of the pulp is mainly due to the proteoglycans which consist of polypeptide chains and long chains of glycosaminoglycans. These hold the water in a gel-like form and inhibit mineralisation. Of the other structural proteins, fibronectin has an important role in making the cell-matrix connection.

**Figure 1.205. Figure 3. – Constitutes of the pulp**
Functionally, dentin-producing, columnar-shaped odontoblasts are crucially important. The odontoblasts persist during the entire life of the tooth. Their function is to provide interstitial fluid, the fluid supply of dentin tubules. In addition, their tight contacts with the nerve endings, similar to a synapse, indicate that they participate in formulating and forwarding the local pressure and pain sensation. The synthesis of matrix components for pulp formation is the primary function of fibroblasts. In addition, fibroblasts are capable of secreting cytokines and growth factors, thus playing a role also in the development of pathological processes. Undifferentiated mesenchymal cells are also found in the pulp. These progenitor / stem cells are involved in repair and regeneration processes. Dendritic cells, macrophages and T lymphocytes can also be detected in small numbers as part of the healthy dental pulp immune system. The number of these cells greatly increases during the inflammatory response. In addition, blood and lymph vessels as well as nerve cells can be identified in the pulp.
1. Oral biology

**Blood supply of the pulp**

- It comes from branches of inferior and superior alveolar artery and vein.
- It is organized into larger central vessels (large venule and 1-2 arterioles) with a rich superficial plexus of capillaries around periphery in the crown.
- It is important for maintaining living cells (especially for odontoblasts) and for regulating fluid content.
- Excess matrix fluid is removed by lymphatics.

**Figure 1.209. Figure 7. – Blood supply of the pulp**

The capillaries do not reach the dentin channels. The capillary pressure in the pulp is much higher (30-40 mmHg) than elsewhere in the body.

**Figure 1.210. Figure 8. – Capillary network in the pulp**

**Arteriovenous anastomoses** (AVA) are present in large numbers in the pulp, making it possible to maintain the circulation when there is hypertension resulting from developing inflammatory processes. The venous part is as simple as the arterial system. Recent data prove the existence of lymphatic vessels. However, their capacity is limited because during inflammation they collapse first, preventing the escape of interstitial fluid.

The **innervation of the dental pulp** is rich, the fibers running in the tooth crown, branched densely to form the Raschow plexus. From there the fibers run to odontoblasts or terminate in the predentin or run into the dentinal...
tubules. **Adrenergic** sympathetic nerves control the arteriolar vasoconstrictor effect. The neuropeptides released from sensory nerve endings, calcitonin gene-related peptide (CGRP), substance P (SP) and neuropeptide Y (NPY) are the primary mediators of vasodilatation. In addition, nitric oxide (NO), which has vasodilator effects, is synthesized by nitric oxide synthase and released from the wall of the vessel causing hyperaemia.

**Figure 1.211. Figure 9. – Nerve supply of the pulp**

The sensory nerves of **trigeminal nerve (cranial nerve V)** and gl. superior cervical sympathetic nerve fibers supply the innervation. The majority of **myelin-sheathed fibers** are Aδ type; their average diameter is 1-6 µm, while only a few have a larger diameter Aβ fiber type. The C-type non-myelinated fibers are smaller in diameter. The Aδ fibers play role in the sensation of sharp, localized pain, while the **C fibers** play a role in the sensation of blunt, less well localized pain. The onset of action potential spreads to the center, but through branching returns to the periphery (antidromic effect) and results in release of bioactive peptides (CGRP and substance P) and secondary nitric oxide elevation. The **sympathetic efferent** fibers are primarily responsible for vasoconstriction. Their excitation leads to norepinephrine and neuropeptide Y (NPY) release. However, there is a lack of parasympathetic innervation of the pulp.

**Figure 1.212. Figure 10. – Current concepts of the generation of dentinal pain**

**Figure 1.213. Figure 11. – Sensation from tooth to brain**
The pressure in the pulp is determined by the forces of the Starling equilibrium, which determines the capillary fluid movement. The interstitial fluid hydrostatic pressure and plasma colloid osmotic pressure keep the balance with the capillary hydrostatic pressure, which may be combined with an interstitial fluid colloid osmotic pressure. During inflammatory processes (induced by mechanical damage and chemical stimuli, such as deep caries, a bacterial infection, intensive tooth grinding, traumatic occlusion and direct chemical stimuli by filling materials) the increased blood flow and hyperpermeability of the vessels leads to extravasation of plasma proteins, increasing the amount and the volume of blood and interstitial fluid. However, the pulp chamber volume is limited, therefore the pressure rapidly increases. The opening of arteriovenous anastomoses (AVA) in response to increasing pressure reduces the spread of inflammation. In addition, net absorption increases in the surrounding inflammation free area, and initially the lymphatic drainage increases as well. These mechanisms act against generalized inflammation of the pulp.

Figure 1.214. Figure 12. – Effect of drilling on pulpal blood flow

Increased inflammation can cause a generalized reaction leading to necrosis of the pulp tissue. Due to the increased level of inflammatory mediators, the resistance of the vessels becomes lower which leads to vasodilation. Both intravascular and capillary pressure gets higher and so does the vascular permeability, thus protein and fluid filtration is increased. The pulp becomes edematous. Tissue pressure rises above the intravenous pressure in the pulp chamber and because of the lack of dilation, veins become compressed. This process leads to decreased blood flow caused by increased venous resistance. Venous stasis provokes RBC (red blood cell) aggregation and increased blood viscosity. The vicious circle of pulpal inflammation further leads to tissue hypoxia, Tissue CO₂ increases and at the same time tissue pH and partial oxygen pressure decreases.
These processes increase the level of inflammation and the blood inflow. The bottom line is that vasodilation followed by venous resistance growth lead to further vasodilation and resistance growth. According to this positive feedback effect, chronically persisting inflammation may lead to a local then total necrosis of the pulp.

Figure 1.215. Figure 13. – Effect of increased intrapulpal pressure

Figure 1.216. Figure 14. – Vicious circle of pulp inflammation

As time goes by, the pulp chamber becomes gradually narrower due to the continuous but very slow odontoblast activity. In old age, sclerotic changes in the pulp tissue can be observed. During this process, calcification of the arterial wall may occur, and pulp stones (denticles) can appear in the pulp. True denticles are irregularly-structured dentin tissues produced by odontoblasts. False denticles are the result of spontaneous calcification.
1. Oral biology

Figure 1.217. Figure 15. – Regressive changes in pulp

<table>
<thead>
<tr>
<th>Regressive changes in pulp</th>
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<tbody>
<tr>
<td>□ It progresses gradually and continuously with age</td>
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<tr>
<td>• volume of pulp chamber decreases</td>
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<tr>
<td>□ Sclerotic alterations (blood vessel wall calcification)</td>
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<tr>
<td>□ Denticuli (pulp stones)</td>
</tr>
<tr>
<td>• denticuli (produced by odontoblasts)</td>
</tr>
<tr>
<td>• false denticuli (spontaneous calcification)</td>
</tr>
</tbody>
</table>

19.1. Test – Structural and functional characteristics of dental pulp, blood supply to the oral tissues, pulpal pain and inflammation (answers)

1. Main protein component of pulp matrix
   A. amelogenin
   B. proteoglycan
   C. phosphophorin
   D. enamelin

2. Localization of nerve fibres of cold induced pain in tooth
   A. in pulp
   B. in enamel
   C. in periodontal ligament
   D. in gingiva

3. Mediator liberated in the pulp during pulp inflammation:
   A. cholecystokinin (CCK)
   B. secretin
   C. calcitonin gene-relate peptide (CGRP)
   D. gastrin

20. 1.20. Radiation, oral symptoms associated with radiotherapy – Kristof Kadar

20.1. Biological effect of ionizing radiation

Ionizing radiation consists of subatomic particles or electromagnetic waves that are energetic enough to detach electrons from atoms or molecules, ionizing them. \((A\rightarrow A^+ + e^-)\)

Types of ionizing radiation:
• Particle: α, β, n (indirectly, its interaction with the nucleus results in ionizing particles).

• Electromagnetic radiation: γ, X-ray, far UV (λ < ~125 nm).

The biological effect of ionizing radiation is based on its absorption in biological material. This absorption is the consequence of physical interaction. Regarding the oral effects, therapeutic X-ray and γ irradiation have the most importance in the clinical setting. The physical interaction of X-ray and γ radiation with matter are considered similar. For the physical interactions of different types of ionizing radiation see the figure below. The dominant type of interaction is determined by the energy of the radiation and the effective atomic number of the absorbing matter.

Figure 1.218. Figure 1. – Interaction of ionizing radiation with matter

Ionizing radiation exerts its chemical/biological effects (1) directly, interacting with chemical bonds of biomolecules and (2) indirectly, by forming free radicals. In the latter case these free radicals interact with biomolecules and biochemical reactions. The interaction with the biochemical processes and biomolecules (chemical/biochemical effect) provides the basis for the cellular/tissue/organ level biological effect.

Figure 1.219. Figure 2. – Interaction of ionizing radiation with matter

<table>
<thead>
<tr>
<th>Interaction of ionizing radiation with matter</th>
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</thead>
<tbody>
<tr>
<td>Phase</td>
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<tr>
<td>physical</td>
</tr>
<tr>
<td>chemical</td>
</tr>
<tr>
<td>biochemical</td>
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<tr>
<td>biological</td>
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</table>

Figure 1.220. Figure 3. – Free radical formation
Of the biological macromolecules DNA and RNA are the most sensitive to radiation damage. DNA/RNA damage may result in either apoptosis or genetic damage (mutations) (see details on the figure below).

**Free radical formation**

1. \( \text{H}_2\text{O} \rightarrow \text{H}_2\text{O}^+ + \text{e}^- \)
2. \( \text{H}_2\text{O} \rightarrow \text{H}^+ + \text{OH}^- + \text{e}^- \)
3. \( \text{H}_2\text{O}^+ + \text{H}_2\text{O} \rightarrow \text{H}_3\text{O}^+ + \cdot \text{OH} \)
4. \( \text{H}_2\text{O} + \text{e}^- \rightarrow \text{H}_2 + \cdot \text{OH} \)
5. \( \text{H}^+ + \text{H}^+ \rightarrow \text{H}_2 \)
6. \( \cdot \text{OH} + \cdot \text{OH} \rightarrow \text{H}_2\text{O}_2 \)
7. \( \text{H}^+ + \cdot \text{OH} \rightarrow \text{H}_2\text{O} \)

Figure 1.221. **Figure 4. – Ionizing radiation induced DNA damage**

- Radiation sensitivity: DNA > RNA > protein > lipids
- DNA damage
  - Base damage
  - Single strand breaks
  - Double strand breaks
- Cross-link formation
  - DNA-DNA cross-links
  - DNA-protein cross-links
  - Protein-protein cross-links
- Chromosome aberrations
  - Deletion
  - Translocation
  - Dicentric and ring chromosomes

The actual DNA damage is also dependent on the activity of repair mechanisms. This provides explanation for the phenomenon that the same dose given over a longer time period or in fractions (dose fractionation) causes less damage to healthy tissues. Cellular components differ in their radiation sensitivity, with the cell membrane being the most and the cytoplasm the least sensitive.

Several other factors influence the biological effects, including the type of radiation, temperature, tissue oxygenization, tissue type and water content, the presence of free radical forming and scavenging (antioxidant) molecules.

**Figure 1.222. Figure 5. – Factors influencing the biological effect**

- Type of radiation
- Linear Energy Transfer – LET: higher LET, more damage
- Temperature
- Water content
- Oxygen
- Nitroimidazole compounds (ie. metronidazole)
- Sulfhydryl (SH) compounds (thiols)
- Tissue type
  - Radiosensitivity is different in different cell cycle stages: S phase < G1-G2 phase < M (Bergonie-Tribondeau principle) → dividing cells are the most sensitive
  - Radiosensitivity increases with increasing proportion of immature (not terminally differentiated) cells in the tissue
Different tissues and organs have different radiosensitivity, depending on the proportion of dividing cells, and (in organs) also on the ratio of parenchyma and connective tissue.

**Figure 1.22. Figure 6. – Radiosensitivity of different tissues**

![Radiosensitivity of different tissues](image)

Ionizing radiation can exert its effect on the body in a deterministic and a stochastic manner. **Deterministic effect** means acute radiation injury, typically occurring with higher radiation doses. There is a threshold dose for the injury and the damage is in a direct relationship with the dose. Lower doses can cause **stochastic effects**. There is no threshold dose and the dose is proportional with the **probability** of the radiation damage.

20.2. **Oral effects of therapeutic irradiation**

Therapeutic irradiation is one of mainstays of modern oncology. Oral side-effects of therapeutic irradiation of the head and neck region can strongly influence the quality of life and in certain cases the survival of patients.

20.2.1. **Osteoradionecrosis**

Osteoradionecrosis is a bone regeneration problem of people who received high doses of radiation, particularly to the jaw. It typically occurs related to dentoalveolar surgery, months, even years after the therapeutic irradiation. The exact pathomechanism is unknown, however, multiple etiological factors have been indentified.

**Etiological factors:**

- Mandible is an **end-artery system** supplied by the inferior alveolar artery, with negligible collaterals.
- Both mandible and maxilla are exposed directly to the external environment through the gingiva. Any breaches in the integrity of these tissues directly influence bone healing.
- After irradiation, minor insults such as periodontal disease, pulpal infections and surgical procedures (ie. dental extraction) can result in delayed healing and in some cases develop into osteoradionecrosis.
- In the relevant population, patients are typically elderly with co-morbidity such as diabetes or hypertension present (microcirculation problems), and are from a poorer socio-economic class with added risk factors such as smoking and alcohol consumption.

**Risk factor:**

- irradiation:
  - dose: increased risk over 50 Gy,
  - time course of the dose: the same dose delivered over a longer time provides more time for the regeneration of non-tumorous healthy tissues and therefore decreases the probability and severity of radiation damage,
  - mode of delivery: more localized forms of delivery as brachytherapy limits radiation damage of the surrounding tissue Also, optimal planning such as avoiding to deliver high dose to the less vascularized part of the mandible, decreases the risk of osteoradionecrosis.
• inadequate oral hygiene,
• surgical procedure.

**Pathomechanism**

According to the classic hypothesis (Marx 1983) radiation causes an endarteritis that results in tissue hypoxia, hypocellularity, and hypovascularity, which in turn causes tissue breakdown and chronic non-healing wounds. Histological examination revealed endothelial death, hyalinisation, and thrombosis of the vessels. Osteoblasts and osteoclasts were deficient. The overall finding described a composite tissue, characterized by hypovascularity, hypocellularity, also found to be hypoxic compared to non-irradiated tissue by direct measurement.

However, more recent data revealed that osteoclasts suffer radiation effects before vascular changes occur, and the loss of vitality of osteocytes is already observed before the manifestation of osteoradionecrosis.

In a more recent hypothesis, endothelial damage, caused directly by the irradiation and indirectly by the subsequent free radical formation, induce cytokine release and an inflammatory response, leading to further increased formation of free radicals. Endothelial injury also leads to thrombosis and necrosis of the microvessels. The released cytokines (TNFα, TGFβ, FGFβ, IL1, IL4, IL6, PDGF etc.) induce uncontrolled fibroblast proliferation, with these newly formed myofibroblasts being characterised by high rates of proliferation, secretion of abnormal extracellular matrix components and a reduced ability to degrade such components. During remodelling the low activity of osteocytes results in bone being replaced with an atrophic, fibrous scar tissue.

**20.2.2. Salivary dysfunction after radiotherapy**

The loss of salivary function starts early during treatment: in the first week, a 50% to 60% decrease in salivary flow is observed, which increases further, leaving a salivary flow of approximately 20% of the original the after 7 weeks of conventional radiotherapy. The salivary function continues to decline for several months after radiotherapy. Depending on the dose received by the salivary glands and the gland volume involved in the irradiated field some recovery may occur by 12 to 18 months after RT. Isolated data show that salivary output could still recover many years after RT, with an approximately 32% increase in salivary flow 1 to 5 years after treatment.

**Pathomechanism**

Histology findings show loss of secretory granules and vacuolisation in the acinar cells. Later p53 dependent apoptosis of acinar cells is observed, with ductal cells being intact. Finally the glands become fibrotic.

The exact pathomechanism is unknown. The chronic functional loss may be explained by the classic theory of radiation injury:

loss of (dividing) progenitor cells → decreased cell replacement and tissue regeneration → loss of function

To explain the acute functional loss, the current hypothesis proposes that selective membrane damage of acinar cell causes acute disorders of water secretion. Observations show that the early secretory response to a partial agonist of muscarinic receptors differs from the response to a full agonist after irradiation, suggesting a receptor mediated mechanism. Moreover, in animal models an irreversible dysfunction of water secretion and the loss of aquaporin 5 channels were found in acinar cells.

**20.2.3. Oral mucositis**

Oral mucositis is one of the most frequent and debilitating side effects of radio-/chemotherapy, that may even influence survival. The inflammed, loosened, ulcerated mucosa is not only extremely painful, but also loses its barrier function in the oral cavity, an already infected environment. The increased risk of systemic infection is especially dangerous in these patients, as both the malignant disease and chemo-/radiotherapy result in immunosuppression.

According to the classic theory of radiation injury, the mucositis is simply the consequence of clonogenic cell death:
loss of (dividing) progenitor cells → clonogenic cell death → imbalance of epithelial loss and replenishment → ulceration

New evidences help to refine this theory. Recent data show that subepithelial damage precedes epithelial injury. Mucosal damage is the result of a complex pathomechanism, involving cytokines and apoptosis as key factors.

**Figure 1.224.** Figure 7. – New evidences in the pathomechanism of oral mucositis

<table>
<thead>
<tr>
<th>New evidences in the pathomechanism of oral mucositis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>early tissue damage</strong></td>
</tr>
<tr>
<td>▶ subepithelial damage (fibroblast apoptosis, microvascular endothelial injury) before the clinical symptoms</td>
</tr>
<tr>
<td>▶ endothelial cells produce KGF (keratinocyte growth factor) and important growth signals for epithelial cells</td>
</tr>
<tr>
<td><strong>role of cytokines</strong></td>
</tr>
<tr>
<td>▶ tumor necrosis factor (TNF), interleukin-6 (IL-6), and interleukin-1 beta (IL-1β) levels increase before the damage</td>
</tr>
<tr>
<td>▶ severity of the mucositis correlates with the intensity of cytokine release</td>
</tr>
<tr>
<td>▶ platelet activating factor increases with the severity of mucositis, while inhibiting thrombocyte aggregation reduces the severity of mucositis</td>
</tr>
<tr>
<td><strong>role of apoptosis</strong></td>
</tr>
<tr>
<td>▶ apoptosis is responsible for much of the tissue damage in mucositis</td>
</tr>
<tr>
<td>▶ in certain cases inhibiting apoptosis may prevent mucositis</td>
</tr>
<tr>
<td>▶ the ceramide pathway of apoptosis may play an important role in mucositis; attenuators of ceramide pathway in the submucosa decrease the severity of mucositis</td>
</tr>
<tr>
<td>▶ genetic conditions affecting apoptosis also affect mucositis – in psoriasis (decreased apoptosis) the risk of mucositis is decreased; in Addison's disease (increased apoptosis) the risk of mucositis is increased</td>
</tr>
</tbody>
</table>

Recently Sonis (Sonis et al. 2007) proposed a new, five-step model for the pathomechanism of epithelial damage in oral mucositis.

**Figure 1.225.** Figure 8. – A potential model for the pathomechanism of oral mucositis
A potential model for the pathomechanism of oral mucositis

- **Initiation**
  - irradiation/chemotherapy → DNA damage, cell death
  - reactive oxygen species (ROS) formation:
    - further DNA and tissue damage
    - macrophage stimulation
    - SP1-related retinoblastoma control protein, p53, NF-κB (X-ray), ceramide pathway (chemotherapy) activation

- **Primary damage response**
  - NF-κB activated TNF, IL-6, IL-1β increase
  - NF-κB induced apoptosis through the activation of BCL2 family of genes
  - activation of ceramide pathway
  - AP1 activation → MMP (matrix metalloprotease) activation

- **Signal amplification**
  - previously activated signaling pathways perpetuate the activation of damage response pathways, creating positive feedback loops:
    - TNF activation → NF-κB activation → activation of the other members of TNF family → sphingomyelinase activation
    - ceramide pathway and NF-κB activation → MAPK → c-Jun → AP1 → MMP activation

Figure 1.226. Figure 9. – A potential model for the pathomechanism of oral mucositis

A potential model for the pathomechanism of oral mucositis

- **Ulceration**
  - submucosal cell death decreases the production of epithelial trophic factors such as KGF
  - ECM degradation by MMPs
  - ECM swelling (fluid) weakens the attachment between the submucosa and epithelium
  - inflammatory elements and activation
  - pseudomembrane formation
  - secondary colonisation → bacterial wall products → further activation of cytokines and MMPs

- **Healing**
  - usually 2-3 weeks after radio-/chemotherapy
  - COX-2 activation → angiogenesis
  - RM 2/3 macrophages downregulate inflammatory response
  - epithelial cell proliferation and migration to the ulcer
  - submucosal cells regenerate
  - the structure of the new mucosa will differ from the original

Figure 1.227. Figure 10. – Signaling pathways involved in the development of mucositis
20.3. Test – Ionizing radiation and the oral effects therapeutic irradiation (answers)

1. The most radiosensitive tissue of the followings is:
   A. blood/bone marrow
   B. salivary gland
   C. muscles
   D. lungs
   E. CNS

2. In typical salivary dysfunction related to radiotherapy the salivary flow after seven weeks is:
   A. about 90% of normal
   B. about 50% of normal
   C. about 20% of normal
   D. about 5% of normal
   E. about 30-40% of normal

3. Which of the following radiations is NOT considered ionizing?
   A. X-ray
   B. β-radiation
   C. α-radiation
D. γ-radiation

E. Infrared (IR) radiation

**Literature:**


Nowadays classical genetics and gene diagnostics cover a narrow area in the dental research field. This includes investigation of cystic fibrosis, which affects the salivary glands, and the examination of genetic mutation-based oral diseases: osteogenesis imperfecta, dentinogenesis imperfecta, amelogenesis imperfecta and hypohydrotic ectodermal dysplasia. In addition to these rare genetic diseases the increasing numbers of oral cancers justify the use of molecular diagnostics. However, the application of molecular techniques will be expanded by two novel developments: genomics (derived from human genome project) and gene therapy.

**Figure 1.228.** Figure 1. – Examples of disorders in relation to molecular diagnostics

<table>
<thead>
<tr>
<th>Examples of disorders in relation to molecular diagnostics</th>
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<tbody>
<tr>
<td>☐ Cystic fibrosis</td>
</tr>
<tr>
<td>☐ Osteogenesis imperfecta</td>
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<tr>
<td>☐ Dentinogenesis imperfecta</td>
</tr>
<tr>
<td>☐ Amelogenesis imperfecta</td>
</tr>
<tr>
<td>☐ Hypohydrotic ectodermal dysplasia</td>
</tr>
<tr>
<td>☐ Head and neck cancers</td>
</tr>
<tr>
<td>☐ Gene therapy</td>
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<tr>
<td>☐ Human genome project</td>
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</table>

Classical genetic research turned into a completely new direction once the human genome had been mapped. Nucleotide triplets – each triplet coding one amino acid - store the genetic code. The genetic code is degenerate because one amino acid can be coded by different triplets. This is important because a nucleotide alteration when translated can cause either a dramatic change, e.g. the complete loss of a protein, or result in no change.

**Figure 1.229.** Figure 2. – The genetic code of amino acids
The development of two gene sequence-detecting techniques allowed the implementation of the human genome project. These techniques are the polymerase chain reaction (PCR) and automated sequencing.

**Figure 1.230. Figure 3. – Polymerase chain reaction (PCR)**

- What for? – PCR is suitable to multiply a chosen DNA section from the complete mixture of DNA molecules – also for cDNA analysis (reversed from RNA by reverse transcription).

- What is it good for? – Examples for the fields of application:
  - detection of leukemias, lymphomas, chromosome-translocations, causative agents, monoclonality
  - in justice (VNTR PCR: murder, rape, father trials)
  - Also for evaluating expressional profile

- How is it possible? – PCR uses some features of DNA replication, and of DNA-polymerases.

**Figure 1.231. Figure 4. – An example of sequencing results**
After the human genome project had been completed, genetics took a new direction: research turned toward the analysis of genes with different sequences, so-called genetic polymorphisms. This approach is called genomics rather than genetics.

**Figure 1.232. Figure 5. – Genomics**

**Genomics**

- The genome:
  - Out of a diploid cell, haploid DNA content + mitochondrial DNA

- Genomics:
  - Analysis of the function, structure and interactions of the genome and the included methods. It also includes analysis of proteins, bioinformatics, "Systems Biology" and so on.

- Genomics for example
  - Structural genomics
  - Comparative genomics
  - Functional genomics
  - Human genomics
  - Pharmacogenomics
  - Medical genomics

Genomic analysis showed that only a small fraction of the human genome consists of coding sequences; the role of the remaining part is still under intensive research.

**Figure 1.233. Figure 6. – Characteristics of the human genome (a few examples)**

**Characteristics of the human genome (a few examples)**

- 2003 April: the sequencing of the human genome was completed
- Only 1.2%-a of euchromatic regions code proteins, (exon)
- At SNP-level only 1/1000 is the difference between two human individuals (99.9% identity)
- Average exon number: 12.2 exon/gene
- Most usual intron size: 87 bp; exon size: 145 bp
- Most exons in titin gene: 309 pieces
The vast majority of the variability comes from single nucleotide polymorphisms (SNP). An SNP is defined as a DNA sequence variation occurring when a single nucleotide is changed in the genome (e.g. GCCTA changed to GCTTA) and at least 1% of the population have this variation. Those nucleotide variations that cause immediate, serious phenotype change (mostly diseases) are called mutations. Therefore they are strongly negative selection factors and their incidence is generally below 1%. The difference between the genomes of two humans, considering every genetic variation, is only 0.1%.

**Figure 1.234. Figure 7. – What is SNP (Single Nucleotide Polymorphism)?**

<table>
<thead>
<tr>
<th>What is SNP (Single Nucleotide Polymorphism)?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Difference in a single nucleotide basis between two individuals of the same species, frequency &gt;1%</td>
</tr>
</tbody>
</table>

ATGGTAAAGCTGAGCTGACTTAGCGT
ATGGTAAAACCTGAGTTGACTTAGCGT

- SNP's appear as replication error or DNA damage.

**Figure 1.235. Figure 8. – Mutation and polymorphism**

<table>
<thead>
<tr>
<th>Mutation and polymorphism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polymorphism</td>
</tr>
<tr>
<td>Frequency</td>
</tr>
<tr>
<td>Effect</td>
</tr>
</tbody>
</table>

- Single nucleotide variations/ SNP ("snips")
  - 90% of known differences/variations in most SNPs only two allelic variation exists
- Length polymorphisms
  - STR (short tandem repeats)
  - VNTR (variable number of tandem repeats)

**Figure 1.236. Figure 9. – Genetic variability in humans**

The difference between two people in gene sequences is about 0,1%

Genomics is important not only in basic research but is also a remarkable step toward personalized medicine both in terms of diagnostics and therapy.
The genetic basis of two frequent dental disorders, tooth agenesis and periodontitis, has already been partially recognized. Among congenital dental disorders, hypodontia occurs most frequently. Hypodontia is by definition the absence of one or more permanent teeth while oligodontia is a subcategory where six or more permanent teeth are absent. Hypodontia affects approximately 20% of the population worldwide. It occurs in 16% of the Hungarian population. The lack of teeth is often coupled with other disorders such as ectodermal dysplasia.

Single nucleotide polymorphisms of transcription factors Msx1 and Pax 1 have been proven to contribute the absence of teeth in humans. Their role is probably related to the regulation of TGF-α expression and function.
Gingivitis and periodontitis are common diseases: more than 50% of the adult population have gingivitis and approximately 20% have destructive periodontal disease. Several risk factors of the disease have been suggested on the basis of epidemiological, clinical and experimental research. These include poor oral hygiene, local plaque retention factors, occlusal overload, smoking, emotional stress, age and gender, immunosuppressive primary diseases, nutrition deficiencies, osteoporosis, genetic/genomic factors and even ethnicity.

**Figure 1.240. Figure 13. – Risk factors in periodontitis**

<table>
<thead>
<tr>
<th>Risk factors in periodontitis</th>
<th>Diseases weakening the immune system</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bad oral hygiene</td>
<td>Nutritional problems</td>
</tr>
<tr>
<td>Local plaque retention factors</td>
<td>Osteoporosis</td>
</tr>
<tr>
<td>Occlusal overload</td>
<td>Earlier periodontitis</td>
</tr>
<tr>
<td>Smoking</td>
<td>Genetic factors</td>
</tr>
<tr>
<td>Emotional stress</td>
<td>Ethnical group</td>
</tr>
<tr>
<td>Age and gender</td>
<td></td>
</tr>
</tbody>
</table>

There are probably dozens of genes in which polymorphisms act as predisposing factors in periodontitis. Their actual manifestation is dependent also on the environmental factors affecting the population (gene-environment interaction). The involvement of polymorphisms in interleukin-1 (IL-1) and tumor necrosis factor-α (TNF-α) genes have been clearly proven in periodontitis.

**Figure 1.241. Figure 14. – Periodontitis**
In addition to the examples mentioned above there may yet be numerous other possible applications of genomics in dentistry.

**Figure 1.242. Figure 15. – Potential application of molecular genomics in dentistry**

<table>
<thead>
<tr>
<th>Potential application of molecular genomics in dentistry</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tooth development</td>
</tr>
<tr>
<td>Caries formation</td>
</tr>
<tr>
<td>Tooth erosion</td>
</tr>
<tr>
<td>Gingivitis</td>
</tr>
<tr>
<td>Periodontitis</td>
</tr>
<tr>
<td>Genomics of pathogenic bacteria</td>
</tr>
<tr>
<td>Salivary gland diseases</td>
</tr>
</tbody>
</table>

Discovery of the genetic basis of congenital oral and dentition disorders provides a basis for future treatments using gene therapeutic tools. The aim of **gene therapy** is to correct a cell function by delivering a DNA sequence into it by a suitable vector whose sequence is then transcribed and translated in the cell to replace a missing or a malfunctioning protein. Also the correction of other cell functions is possible when the produced protein is secreted.

**Figure 1.243. Figure 16. – Gene Therapy Principle**
The first success of gene therapy was the treatment of Severe Combined Immunodeficiency Disease (SCID). Briefly, the disease was caused by a mutation of the gene coding adenosine deaminase that leads to serious disorders in T cell maturation and function. This makes the immune system unable to defend against infections.

During the procedure leukocytes were isolated from the patient's blood and treated *ex vivo* by retroviral gene therapy (functioning genes were delivered into the cells by retroviruses) and the modified cells were given back by infusion into the body. The result was the restoration of normal immune function which meant that the patients were able to live a normal life instead of requiring complete sterile isolation.

**Figure 1.244. Figure 17. – Gene therapy – SCID for example**

![Diagram of gene therapy process](image)

**Figure 1.245. Figure 18. – Gene therapy – SCID for example**

![Diagram of SCID treatment](image)

The most appropriate method for targeted gene therapy of salivary glands is the retrograde injection of the vector into the ductal system that is connected to the apical side of practically every acinar and ductal cell. Some vectors like adenoviruses are able to bind both acinar and ductal cells while others like adeno-associated viruses only transduce ductal cells.

**Figure 1.246. Figure 19. – Salivary glands as targets of gene therapy**

![Diagram of salivary glands](image)
The primary pathway of protein secretion in salivary glands is regulated secretion by which proteins like amylase are stored in secretory granules and secreted in a luminal direction into the saliva after stimulation. Proteins secreted via the constitutive pathway (continuous secretion in the absence of stimulation) are not stored, they are secreted immediately after synthesis predominantly through the basal and lateral membrane of the cell into the interstitium or the bloodstream. The physiological role of this latter pathway in salivary glands has not been elucidated but this is the main pathway for secretion in the case of liver enzymes.

**Figure 1.247. Figure 20. – Salivary glands as targets of gene therapy 1.**

The most obvious aim of salivary gland gene therapy is to restore the function of the gland. Acinar parenchymal destruction occurs due to Sjögren's syndrome, or irradiation for head and neck cancer, and the ducts are not able to replace acinar function as without water permeability they only able to reabsorb electrolytes. The introduction of the gene for a highly selective water channel, aquaporin-1 (AQP1), by adenoviruses into the ducts results in the transduction of the ductal cells, which become water permeable and can secrete water and produce saliva.

**Figure 1.248. Figure 21. – Salivary glands as targets of gene therapy 2.**

There are numerous diseases caused by decreased protein level or the absence of the protein in the systemic circulation. Such pathological conditions include the reduced insulin production in diabetes, the decrease in erythropoietin synthesis during the progression of chronic renal failure or the deficiency of growth or other hormones due to endocrine hypofunction of pituitary gland caused by genetic or other disorders. This initiated research which aims to introduce growth hormone, erythropoietin or other protein coding genes into the salivary...
glands. According to recent results constitutively secreted erythropoietin goes primarily to the bloodstream while growth hormone is secreted in the regulated pathway into the saliva. Thus it is important to discover how to guide the latter to the blood in further research. In addition, by salivary gland gene therapy, it is possible to continuously produce and secrete bioactive proteins into the oral cavity to influence periodontal inflammation or the division of cancer cells.

**Figure 1.249. Figure 22. – Salivary glands as targets of gene therapy 3.**

Squamous cell carcinomas in the head and neck region provide another gene therapeutic example. Researchers have made a modified adenovirus for human testing, which is able to infect any epithelial cells but replicates only in p53 deficient cells. By this method, oral cancer cells in which this tumor suppressor gene function is missing because of mutations can be selectively destroyed.

**Figure 1.250. Figure 23. – Treatment of oral-head-neck cancers by application of Onyx-015**
21.1. Test – Gene therapy and gene polymorphism in dentistry (answers)

1. In the majority of cases, how many proteins are coded by an individual gene?
   A. one protein
   B. two proteins
   C. three proteins
   D. four proteins
   E. five proteins

2. Which disease can be cured by gene therapy of the mutation of the ADA gene?
   A. AIDS
   B. ADAI
   C. ADIS
   D. HIVD
   E. SCID

3. Secretory pathway of synthetized salivary proteins
   A. constitutive
   B. regulated
   C. constitutive or regulated depending on the actual protein
   D. none of them
   E. salivary glands do not secrete proteins
22. 1.22. Pathomechanisms in oral cancer – Gábor Varga

To understand the mechanism of tumor formation we must consider the basic mechanisms that regulate cell proliferation, differentiation, function and programmed cell death. Only a small fraction of our cells are in the cell cycle generating daughter cells in a regulated way. Most of the cells are in resting state, arrested in the G0 phase of cell cycle. They are differentiated and are fulfilling a certain function. Other mechanisms are responsible for programmed cell death (apoptosis) to provide the balance between cell number gain and loss.

Figure 1.251. Figure 1. – Most cells are in resting state, while a small percent of the cells are in the process of cell division

Some of these processes are based on the self-regulatory mechanisms of the cells. However, extrinsic factors, physiological conditions, injuries and the matrix secreted by other cells also affect cell fate through the pathways of differentiation, apoptosis, metabolism and motion.

Figure 1.252. Figure 2. – Cell response to external stimuli
Tumor formation occurs when normal regulation of the cell cycle is disrupted. It is generally caused by multiple mutations of somatic cells that lead to unregulated proliferation and other cell functions. Important consequences of this insufficient regulation are unlimited proliferation, the ability to invade into other tissues and to form metastases, insensitivity to growth inhibitor signals, evasion of apoptosis and continuous angiogenesis.

**Figure 1.253. Figure 3. – What is cancer?**

- Cancer is a complex multifactorial genetic disorder of somatic cells that leads to the loss of regulated cell proliferation.
- Cancer cells proliferate more frequently.
- Cancers cell do not exhibit contact inhibition and produce tumor.
- Cancer cells invade other tissues (metastasis).
- Cancer originates from a single cell.

Tumors may develop in every organ, but they are heterogeneous with respect to their incidence and outcome. There are significant differences between genders as well.

**Figure 1.254. Figure 4. – 1996 estimated cancer incidence by site and sex**
Environmental carcinogens have a crucial role in tumor formation as they increase the risk of mutations which then lead to the development of the disease. Cancer itself is not a hereditary disease. However, genetic
predisposition can be an important factor in its development. Mutations carried by gametes do not cause tumor formation alone but may severely increase the risk.

**Figure 1.256. Figure 6. – Environmental Carcinogens**

<table>
<thead>
<tr>
<th>Environmental Carcinogens</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Drugs: mutagens, immune suppressing, etc.</td>
</tr>
<tr>
<td>- Organic chemicals: Insecticides, herbicides, aromatic hydrocarbons, etc.</td>
</tr>
<tr>
<td>- Cigarette Smoke</td>
</tr>
<tr>
<td>- Ethanol</td>
</tr>
<tr>
<td>- Heavy Metals</td>
</tr>
<tr>
<td>- Sexually transmitted viruses: HTLV-I, Herpes simplex, Human papilloma virus</td>
</tr>
<tr>
<td>- Radiation: Ultraviolet light and radioactivity</td>
</tr>
</tbody>
</table>

**Figure 1.257. Figure 7. – Heredity and Cancer**

<table>
<thead>
<tr>
<th>Heredity and Cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Hereditary predisposition</td>
</tr>
<tr>
<td>- Clustering of environmentally induced cancers in families.</td>
</tr>
<tr>
<td>- Close relatives of cancer patients have three times greater risk of developing the same neoplasm.</td>
</tr>
<tr>
<td>- Close relatives of patients with breast, colon, or endocrine cancers have greater than three times risk for developing the same neoplasm.</td>
</tr>
<tr>
<td>- Increased cancer risk with inherited mutations of cancer suppressor genes such as Rb and p53.</td>
</tr>
</tbody>
</table>

Carcinogenesis is the consequence of consecutive mutations. During the process certain mutations provide selection advantages for the carrier cell, and these cells will overgrow the others. Thus, the genetic instability will be developed in multiple steps and carried by all daughter cells. As a consequence, the vast majority of the cells in a tumor are monoclonal.

**Figure 1.258. Figure 8. – The multi-step process of carcinogenesis**
Cancer cells also show functional heterogeneity. When the cells are exposed to a toxic agent, for example during chemotherapy, resistant cells will be selected by their survival, and later the whole tumor will become resistant to the treatment.

**Figure 1.259. Figure 9. – Cancer progression: Clonal selection due to therapy**

During carcinogenesis **four major groups** of genes have been identified to have a significant effect on its progression. These are the proto-oncogenes, the tumor suppressor genes, the genes coding DNA repair proteins and those genes that have a role in apoptosis.

**Figure 1.260. Figure 10. – Genes involved in malignant transformation**

<table>
<thead>
<tr>
<th>Genes involved in malignant transformation</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Proto-oncogenes</td>
</tr>
<tr>
<td>- Tumor suppressor genes</td>
</tr>
<tr>
<td>- DNS repair genes</td>
</tr>
<tr>
<td>- Genes involved in apoptotic cell death</td>
</tr>
</tbody>
</table>
In normal conditions proto-oncogenes enhance proliferation; but after mutation their activity increases and their mutation became dominant over the normal allele. Tumor suppressor genes in physiological conditions inhibit cell division or trigger apoptosis. Their mutations are recessive. Therefore, only the functional loss of both copies of the gene leads to pathological processes.

**Figure 1.261. Figure 11. – Comparsion of proto-oncogenes and tumor suppressor genes**

![Diagram of proto-oncogenes and tumor suppressor genes]

Proto-oncogenes can be activated in four different ways. In the case of point mutations the gene becomes constitutively active. In the case of chromosome translocation a cross-recombination of chromosomes occurs during replication. When a gene has increased copy number, it is called gene amplification. Epigenetic mechanisms involve the change of DNA methylation, thereby significantly modifying the response of a gene to activation. Usually the DNA of cancer cells has a lower level of methylation than that of normal cells.

**Figure 1.262. Figure 12. – Activation of proto-oncogenes**

<table>
<thead>
<tr>
<th>Activation of proto-oncogenes</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Point Mutations</strong></td>
<td>The ras gene is an oncogene that becomes activated by a point mutation.</td>
</tr>
<tr>
<td><strong>Chromosomal Translocations</strong></td>
<td>Translocation of chromosome 9 and 22 in CML creating a fusion gene that produces an activated tyrosine kinase (Philadelphia-chromosome).</td>
</tr>
<tr>
<td><strong>Gene Amplification</strong></td>
<td>Specific oncogenes such as N-myc and C-neu are amplified in neuroblastoma and breast cancer respectively.</td>
</tr>
<tr>
<td><strong>Epigenetic mechanisms</strong></td>
<td>A gene control mechanism which is not coded in the DNA sequence. Such is e.g. parental imprinting (gene expression depending on the parent’s sex). The mechanism of imprinting is selective methylation of genes. (Methylated genes are not expressed.) Most malignant tumors seem to have less methylated genes, than healthy cells.</td>
</tr>
</tbody>
</table>

The mutation of k-Ras proto-oncogene provides the best example of proto-oncogene activation. The mutation leads to constitutive activation of k-Ras protein as it loses its GTPase evoked degradability but not its signaling function. Normally when GTP binds to the active centre of the protein it is active (switched on) for a short period but then inactivated (switched off) by cleaving the GTP. In the mutated k-Ras the GTP is not able to
dissociate from the active centre, therefore, the protein stays switched on and the MAP kinase cascade sends continuous proliferation enhancing signals.

**Figure 1.263. Figure 13. – Activation of proto-oncogenes: K-RAS mutation**

Missense mutations in codons 12, 13 and 61 alter gene product activity

![Diagram showing normal and mutant RAS proteins](image1)

The Philadelphia chromosome is the result of the translocation of chromosomes 9 and 22 in chronic myeloid leukemia. This translocation results in a fusion gene whose product is a hybrid of ABL and BRC genes. The newly formed gene continuously activates the tyrosine kinase cascade thus sending an endless proliferation signal to the nucleus.

**Figure 1.264. Figure 14. – The Philadelphia chromosome**

<table>
<thead>
<tr>
<th>The Philadelphia chromosome</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Genes affected by the reciprocal translocation:</td>
</tr>
<tr>
<td>- ABL (9q34.1) Abelson leukemia proto-oncogene</td>
</tr>
<tr>
<td>- BCR (22q11) breakpoint cluster region gene</td>
</tr>
</tbody>
</table>

- The result is a new, abnormal, fusion gene on chromosome 9, which is translated into a protein with tyrosine kinase enzyme activity. This is specifically inhibited by the drug called Gleevec. Very good results have been achieved in CML and some other malignancies using this novel drug.

Viral oncogenes are actually analogues of mammalian genes, coded by viruses. HPV (human papilloma virus) is one of the oncogene viruses having a crucial role in the development of oral cancer.

**Figure 1.265. Figure 15. – Another group of oncogenes: DNA viral oncogenes**

<table>
<thead>
<tr>
<th>Another group of oncogenes: DNA viral oncogenes</th>
</tr>
</thead>
<tbody>
<tr>
<td>- True, viral encoded oncogenes</td>
</tr>
<tr>
<td>- Not analogues of mammalian genes</td>
</tr>
<tr>
<td>- Responsible for a few subtypes of human cancer</td>
</tr>
<tr>
<td>- Example: Human papillomavirus (HPV) in cervical and oral carcinoma</td>
</tr>
<tr>
<td>- E6: Inactivates p53 tumor suppressor gene</td>
</tr>
<tr>
<td>- E7: Inactivates Rb tumor suppressor gene</td>
</tr>
<tr>
<td>- Also, SV40, JC, polyoma, EBV</td>
</tr>
</tbody>
</table>

Tumor suppressor genes inhibit proliferation in normal conditions. Their inactivation means that proliferation is liberated from inhibition. Since the defective allele is inactive, the loss of both alleles (by mutation and/or
deletion) is necessary for functional inactivation. The most important members of the group are the retinoblastoma (Rb) and p53 genes.

**Figure 1.266. Figure 16. – Tumor Suppressor Genes**

<table>
<thead>
<tr>
<th>Tumor Suppressor Genes</th>
</tr>
</thead>
<tbody>
<tr>
<td>A class of genes that normally suppress cell proliferation. Examples are p53 and Rb.</td>
</tr>
<tr>
<td>Mutations that inactivate the tumor suppressor gene products can release cells from growth suppression and lead to hyperproliferation</td>
</tr>
<tr>
<td>Both alleles of the tumor suppressor gene must be inactivated by mutation for hyperproliferation to occur.</td>
</tr>
</tbody>
</table>

The most important function of the protein coded by the retinoblastoma gene is to inhibit the cell cycle in a phosphorylation-dependent manner. Rb is an extremely important regulatory gene therefore when the gametes carry its mutation (hereditary form) tumor formation occurs often before age of one year. The non-hereditary, sporadic form of Rb inactivation needs the loss of function of both alleles thus it appears later and the chance of secondary tumor formation is low.

**Figure 1.267. Figure 17. – Non-heritable vs. Heritable Retinoblastoma**

<table>
<thead>
<tr>
<th>Non-heritable vs. Heritable Retinoblastoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feature</td>
</tr>
<tr>
<td>Tumor</td>
</tr>
<tr>
<td>Family history</td>
</tr>
<tr>
<td>Average age at dx</td>
</tr>
<tr>
<td>Increased risk of second primaries</td>
</tr>
</tbody>
</table>

The other key tumor suppressor is the p53 gene. The protein coded by this gene has various functions. Its main function is to act as molecular chaperone. If p53 detects the synthesis of defective proteins it will stop the cell cycle to allow time for DNA repair by specific enzymes. If the synthesis of defective proteins continues the p53 will activate an apoptotic cascade leading to the death of the altered cell. The loss of functional alleles of p53 is a frequent, important step in carcinogenesis. However, it does not cause cancer alone. Significant change in the function of 4-10 proteins is needed for tumor formation in the vast majority of cases for cancer development.

**Figure 1.268. Figure 18. – p53 activity in normal and mutated cells**
The oral or ‘head and neck’ cancers (HNC) have the general features of cancer in many respects. Their development is often preceded by precancerous conditions such as oral leukoplakia that increases the risk of actual carcinogenesis. Oral cancers are amongst the most frequently occurring cancers in the body.

Figure 1.269. Figure 19. – p53: guardian of the genome

DNA damage (radiation, chemotherapy) sensor

Wild type p53

Cell cycle

G1

S

M

High DNA damage
Die by apoptosis

Low DNA Damage
Repair damage and survive

The oral or ‘head and neck’ cancers (HNC) have the general features of cancer in many respects. Their development is often preceded by precancerous conditions such as oral leukoplakia that increases the risk of actual carcinogenesis. Oral cancers are amongst the most frequently occurring cancers in the body.

Figure 1.270. Figure 20. – Incidence of cancers in Hungary in 2001
Figure 1.271. Figure 21. – Genetic, environmental and lifestyle factors play important role of the development of oral cancer

Figure 1.272. Figure 22. – Leukoplakia (precancerous conditions) and oral cancer
Oral cancers occur predominantly in men. HNCs are heterogeneous and can be divided into subgroups according to their underlying causes. One form of oral cancer is caused by HPV infection; it usually occurs at young age and mostly cannot be characterized by poor oral hygiene. Other forms usually appear in older age and are coupled with bad oral hygiene. Among them one group can be characterized by the loss of p53 alleles, another by the deletion of a given sequence of the chromosome 9 and the last group by the increased expression of epidermal growth factor receptor (EGFR) that activates the tyrosine kinase proliferation pathway.

**Figure 1.273. Figure 23. – Genetic types or oral cancer – four distinct categories**

<table>
<thead>
<tr>
<th>Genetic marker</th>
<th>HNC1</th>
<th>HNC2</th>
<th>HNC3</th>
<th>HNC4</th>
</tr>
</thead>
<tbody>
<tr>
<td>HPV</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>P53 mutation</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>9. Chromosome deletion (ARF1/INK4A/B)</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>EGFR</td>
<td>(+)</td>
<td>+++</td>
<td>++</td>
<td>++</td>
</tr>
</tbody>
</table>

HPV is a frequent cause of cervical and oral cancers. Essentially, after infection the viral proteins enhance cell proliferation thus driving forward the malignant transformation.

**Figure 1.274. Figure 24. – HPV and cervical tumorigenesis (similar event can induce oral carcinoma)**
HPV produces two key proteins that induce the loss of function of Rb and p53, a condition very similar to loss-of-function mutations. The HPV E7 gene binds to the Rb and blocks its function while the HPV E6 gene neutralizes the inhibitory effect of p53.

**Figure 1.275. Figure 25. – HPV oncogenes**

**Figure 1.276. Figure 26. – HPV is teaching us about tumor suppressor proteins p53 and pRb**
In addition to the mechanisms mentioned above it is worth mentioning the role of inactivation of another tumor suppressor gene in the development of head and neck cancers. The loss of p16 gene function is rarely caused by mutation. But more often the gene is blocked epigenetically by methylation and/or the loss of heterozygocity results in the loss of p16 function.

**Figure 1.277.** P16 tumor suppressor gene inactivation in oral cancer

Nowadays a high percentage of cancers patients can be cured. The key factor is the early recognition of the disease. Also the proper combination of modern surgical techniques and irradiation in the case of oral cancer, or chemotherapy in the case of various other cancers, has a crucial role in the treatment. However, to improve the efficiency of medical interventions, new approaches are required in addition to the existing technologies. These new approaches include inhibition of angiogenesis since it is known that tumors also need adequate blood supply as any cell can survive and divide only in a distance not more than 1 mm from the blood vessels. Another promising approach is gene therapy. Its main promise is directly targeting of the cancer cells to deliver toxic agents into them. The combination of all these therapies will hopefully lead to more efficient treatments in the future than those available today.

**Figure 1.278.** Inhibition of angiogenesis
1. Basic event(s) leading to tumor formation: ()

A. successive mutations of somatic cells
B. successive mutations of gamete cells
C. b successive mutations of any kind of cells
D. d.deformation of somatic cells
E. deformation of gamete cells

2. Protooncogen, its mutation frequently play an imprrtant rola int he development of oral cancer

A. p53
B. retinoblastoma
C. k-ras
D. Philadelphia
E. none of them

3. Etiologic factor for oral cancer

A. ICV virus
B. ABD virus
C. HPV virus
D. AAV virus
E. lentivirus

23. 1.23. Oral sensation: Taste and smelling – Jozsef Blazsek

Rapid and accurate recognition of chemical substances improves the chances for survival of organisms in nature. In addition to feeding, smells help with orientation, predation, escaping predators and finding mates: occasionally a couple of molecules are sufficient for identification. Tasting, in conjunction with smelling, is central to survival. These two senses „prescreen‖ food and drinks prior to ingestion to avoid harmful effects such as poisoning. Tastes and smells can be considered chemical or biological messages to the organism and therefore it makes sense to classify tastes based on whether they indicate harmful or beneficial substances, rather than using the conventional classification (sweet, bitter, sour, salty). In general, sweet indicates an energy source such as a carbohydrate. Electrolytes crucial for the homeostasis taste salty (NaCl, KCl). Harmful molecules such or poisons are often bitter or sour and evoke a defensive response such as strong salivation. Dissolved minerals and gases give water the taste enjoyed by many people, the taste often called „water taste‖. The presence of monosodium glutamate (and sodium aspartate) are responsible for the savory „salty-sweet‖ taste „umami‖.

Smells are much easier to describe. There are unpleasant (penetrating, pungent, rotting, etc.) and pleasant smells such as that of herbal volatile oils, smells associated with food or beverages, etc. Interestingly, even though smelling and tasting can mean life or death, they may still require cortical learning, even with preexisting molecular mechanisms.

Figure 1.280. Figure 1.

Figure 1 is, with NaCl as the example, a schematic representation of the sensing of different molecules by receptors or ion channels. These sensors, upon activation, depolarize the basal membrane of the taste receptor cells. Depolarization is then transmitted to nerve fibers. Not all receptors cells are directly connected to nerve fibers, some of them are indirectly via neighboring cells.

Figure 1.281. Figure 2. – Taste receptors
1. Oral biology

Figure 2 shows the sensors for the different basic tastes, which are ion channels or G protein-coupled receptors. Taste buds are sensitive to all tastes but individual gustatory cells are selective for one particular basic taste and we learn to differentiate between individual basic tastes on the basis of cortical representation. Figure 3 shows taste buds with taste receptor cells (modified epithelial cells specialized for gustatory sensing). Taste buds are located on the tongue on protrusions called papillae, four kinds of which exist: circumvallate, foliate, fungiform and filiform papillae. The latter are mechanical and not involved in gustation. In addition to the tongue, papillae are found on the soft palate, upper esophagus and epiglottis.

Figure 1.282. Figure 3. – The types of taste buds

Figure 4 shows that taste buds are innervated by cranial nerves VII, IX and X. The innervating neuronal bodies are in the ganglia geniculatum (VII), petrosum (IX) and nodosa (X). Synapsing occurs in nucleus tractus solitarius to afferent cranial nerves VII, IX and X, among other fibers, to fibers of chorda tympani (VII) and nervus petrosus (VII).

Figure 1.283. Figure 4. – The anatomy of innervation
Ventrobasal thalamus and gyrus postcentralis in the parietal cortex are involved in gustation. However, gustatory system is not the only one involved in sensing all the soluble molecules that accessed the oral cavity. In addition to taste, the extent of irritation (such as when tasting red hot peppers, black peppers and chili), consistency and temperature all add to the recognition of different substances to avoid the harmful ones. In this chemosensory process free nerve endings in tissues play important roles, with the signal conducted to the brain most often via the trigeminal nerve (V). Distinct nerve bundles run from the tongue, gingiva, teeth, nasal mucosa and cornea to carry information on pungent, burning, hot, corrosive, sharp and painful insults, on the extent and location of these. This altogether is chemical sensing, ie. chemesthesis.

Figure 1.284. Figure 5. – What is flavor?

The epithelial origin of taste receptor cells enables their high regenerative potential. It takes 10 to 11 days for a single cell to develop from the basal layer to the uppermost layer of the epithelium, but when it has emerged to the surface, it only functions for 3 hours. This is why when we burn our tongue with hot meal at lunch, we will be able to taste food again as soon as dinner comes. The innervation and the rapid regeneration of taste receptors demonstrate how important this sense is for survival.
The perception called 'taste' in everyday life comes from the concerted action of the gustatory and olfactory systems. It is easy to demonstrate by clamping our nose before tasting chocolate, fruit or other savory food, and then releasing the clamp: a new world of scents opens up.

In vertebrates, molecules of smell substances are trapped in the mucus of the olfactory epithelium. Receptors on olfactory neurons in the olfactory epithelium sense these molecules.

**Figure 1.285. Figure 6. – The olfactory's cell structure and location in the brain**

![Diagram of olfactory cell structure and location in the brain]

Figure 6 shows how olfactory sensory neurons are situated in the olfactory epithelium. Olfactory neurons project axons within the olfactory nerve through perforations in the cribriform plate to the olfactory bulb. Neurons in the olfactory bulb then send axons to other parts of the olfactory system. Strong impacts such as those occurring from car and bicycle accidents and from contact sports may damage nerve fibers passing through the cribriform plate, and, in severe cases, can result in loss of smelling. In 90% of cases, patients complaining of impaired tasting in fact suffer from impaired smelling. A schematic pathway of olfaction is shown in Figure 7.

**Figure 1.286. Figure 7. – Smelling**
Figure 8 compares the mechanisms of gustation and olfaction.

Figure 1.287. Figure 8. – Comparison of taste and smell

<table>
<thead>
<tr>
<th>Comparison of taste and smell</th>
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</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>Receptor number</td>
</tr>
<tr>
<td>Cell types</td>
</tr>
<tr>
<td>Turnover</td>
</tr>
<tr>
<td>Innervation</td>
</tr>
<tr>
<td>Excitation</td>
</tr>
<tr>
<td>Vulnerability</td>
</tr>
</tbody>
</table>

Disorders of taste and smell are increasingly prevalent due to increasing lifespan and the widespread use of chemicals and drugs. Combined with allergic rhinitis, the prevalence is 6 to 7%.

Figure 1.288. Figure 9. – The taste and smell diagnostic terminology
Over the age of 65, taste, and especially smell disorders is especially prevalent due to widespread use of prescription drugs and the resulting decreased receptor sensitivity and hyposalivation (also a frequent complication of removable dentures), the latter causing decreased solubility of tastants and odorants.

Figure 1.289. Figure 10. – Causes of taste disorders

Certain cardiovascular drugs are known for such side-effects: diltiazem causes impaired tasting and smelling (hypogeusia, hyposmia), while the use of nifepidin results in altered sense of taste and smell (dysgeusia, dysosmia). Taste and smell disorders may also be caused by, in addition to decreased secretion due to beta blockers, insufficient hydration leading to decreased surface dissolution of tastants and smellants. Diabetic patients often complain of impaired sweet sensation. The trace element zinc, in concert with the protein gustin/carboanhydrase VI, is indirectly involved in taste sensing. The complex of zinc and gustin is essential to taste sensing. Accordingly, impaired gustation and olfaction is a symptom of zinc deficiency. Zinc is important mainly for the formation and development of taste buds. The ever more prevalent allergic mucositis, flu and cold all cause impaired taste and smell sensing, even though, fortunately, just temporarily. The injury of chorda tympani (Bell’s palsy) causes a distinct tasting disorder, accounting for 2% of sensory nerve injuries. The treatment of head and neck and oral cavity cancers involves therapeutic irradiation (0.5%) and the use of cytostatics (5-FU, methotrexate, bleomycin), which have bitter/sour/metal dysgeusia side effects.

Figure 1.290. Figure 11. – Taste and sensory disturbances caused by drugs
Many agents used in dentistry have dysgeusia effects.

**Figure 1.291. Figure 12. – The effect of dental materials to the sense of taste**

Doxycycline and especially metronidazole have strong bitter dysgeusia effects. Chlorhexidine, a mouth rinse often recommended against mucositis, induces salt ageusia. SDS (sodium-dodecyl-sulfate) content of toothpastes causes temporary sweet ageusia. Tooth filling and bonding agents cause bitter dysgeusia that may last for days. Sour cements induce, due to their acid content, induce strong salivation. Analgesics such as lidocaine, tetracaine and benzocaine result in temporary ageusia. Patients frequently complain of metallic taste. Metallic taste is due to different metal alloys forming a galvanic cell, and is always a sign of corrosion. Taste is not determined by the identity of metal ions but by the hydroxide anions dissociating from the electrolytes. In addition to local toxicity, toxic materials may even cause systemic poisoning.

**In summary**, gustation is a robust sensory system, whereas olfaction can be exhausted and is more vulnerable. Chemosensory diseases are frequent, they affect the quality of life and their prevalence increases with advancing age.

**Figure 1.292. Figure 13. – Summary**

1. video – Tasting.
23.1. Test – Oral sensation: Taste and smelling (answers)

1. Which cranial nerve does not convey taste information?
   A. the third cranial nerve
   B. the seventh and fifth cranial nerves
   C. the seventh cranial nerve
   D. the ninth cranial nerve
   E. the tenth cranial nerve

2. What is not true for the mechanism of tasting?
   A. The sensing of each basic taste is localized to distinct areas of the tongue.
   B. Gustatory cells are of epithelial origin.
   C. Gustatory cells can function for three hours.
   D. Gustatory sensing regenerates rapidly.
   E. Gustatory sensing involves a so-called screening mechanism.

3. Which statement is false?
   A. Only one cranial nerve is involved in olfactory sensing.
   B. Three cranial nerves are involved in gustatory sensing.
   C. The olfactory system is vulnerable.
   D. The olfactory system is robust.
   E. Olfactory dysfunction is more frequent than gustatory dysfunction is.

4. Which one of the drugs below does not cause gustatory dysfunction?
   A. Klion
   B. Nifedipine
   C. Chlorhexidine
   D. Listerine
   E. Lidocaine


24.1. Gingival sulcus

Gingival sulcus (or crevice) is formed where the gum meets the tooth. It is a narrow groove surrounding the tooth neck. It communicates with the oral cavity to the occlusal direction.

Figure 1.293. Figure 1.
Depending on age, the inner wall is the enamel or, in elderly, the root cementum. The outer wall is the epithelium of the free gingiva facing the tooth and the bottom is the top of the junctional epithelial attachment. In clinically healthy gingiva it is only a virtual space or more frequently a less than 2 mm deep fissure around the teeth.

Teeth penetrate the gums. Joining of different layers can be problematic, especially when a sterile internal subgingival environment is to be isolated from the infected oral milieu. Although other sections of the gastrointestinal tract are also colonized, the oral cavity is unique in that solid tooth surface provides a firm basis for bacterial anchoring. When biofilm accumulation is undisturbed, inflammation of the gum (gingivitis) can develop. When genetic or acquired organic or behavioral risk factors are also present, destructive inflammation of the teeth supporting tissues occurs and their damage leads to apical proliferation of the epithelium and conversion of the gingival sulcus into a deep periodontal pocket. When the periodontal bone loss becomes significant, teeth loosen and may fall out at the end of the destructive process despite being completely healthy.

The first line of defense against invading bacteria is the gingival sulcus. Sophisticated defense mechanisms function to keep bacteria away from periodontal tissues. The sulcus epithelium from the gumline to the junctional epithelium is not just a passive barrier. Its structure is similar to that of the multi-layer stratified squamous epithelium covering the gum facing the oral cavity, but this part is non-keratinized. Furthermore, in contrast to the oral epithelium this segment is not entirely impermeable to fluids and cells. It has increased lysosomal activity. In addition, its cells produce polypeptides called defensines, which are part of the natural defense mechanisms.

At the base of the sulcus, the collar like modified epithelial ring around the neck of the teeth called junctional epithelium ensures a tight epithelial sealing. This functionally modified epithelium is non-keratinized as well, nor does it have separated layers as it does in other parts of the oral epithelium. Only 2 layers can be distinguished: stratum basale (cuboid cells) that faces the connective tissue and stratum suprabasale (flat cells with axis parallel to that of the tooth) that faces the tooth. The turnover of the basal dividing cells, i.e. the time until the cells of the stratum basale reach the surface and exfoliate, is approximately 1 week, just quarter of the turnover time in other oral epithelia. Epithelial cells remain in a dynamic connection with the tooth surface throughout their entire coronal migration. Another feature is that two basal laminae cover this part of the epithelium, the external providing connection to the connective tissue, while the inner to the tooth surface.

**Figure 1.294. Figure 2.**
1. Oral biology

Also typical of this zone is a high degree of transepithelial permeability (large intercellular gaps, with few intercellular connections), this is the main route of leukocytes towards the sulcus. In addition to the anatomical structure of the epithelial barrier, further antimicrobial protection is provided by the rapid, funnel-like migration of coronal cells, mechanically eliminating invading microbes. Furthermore, epithelial cells produce antimicrobial substances (defensins, lysosomal enzymes, MMPs), and various chemokines (IL, TNF, cytokines) to attract and activate professional immune cells (PMNs, lymphocytes).

24.2. Gingival crevicular fluid (GCF)

Fluid found in the sulcus is called gingival crevicular fluid (GCF). GCF is derived from the diffusion of the subepithelial transudate under normal conditions. The driving force for this is provided by capillary and connective tissue oncotic and hydrostatic pressure differences (transmigration of leukocytes is driven by chemotactic factors!). GCF provides supply and an adequate environment to epithelial cells. However, in the sulcus GCF mixes with saliva and is contaminated with bacteria, which can influence cellular functions, especially those of the most coronal dentally attached cells. In healthy sulcus the amount of GCF is small, its composition resembles that of the interstitial fluid and it contains epithelial cells, leukocytes and bacteria as well.

When the balance between biofilm bacteria and host defense is disrupted, both invading bacteria and excessive immune/inflammatory reaction can damage tooth supporting tissues and the sulcus deepens, forming a periodontal pocket. During inflammation the amount of GCF increases considerably, washing bacterial colonies and their metabolites out of the sulcus and by its components it plays an important role in the protection against the bacterial invasion. In the inflammatory exudate the elements of the classical inflammatory cascade (prostaglandins, cytokines, complement system, lysozyme, alkaline phosphatase, cathepsins, lactoferrin, white blood cells, etc.) appear along with bacteria and their metabolites/enzymes (endotoxins, hydrogen sulfide, collagenase, proteases, hyaluronidase, etc.), and different products of tissue breakdown (lactate dehydrogenase, polyamines, collagen peptides, etc.). Measuring the amount and composition of GCF can be used as a rapid diagnostic test or to monitor therapy.

24.3. Biological width

When the finishing line of the dental restoration is positioned apical to the sulcus (into or below the junctional epithelium), the biological width is compromised.

Figure 1.295. Figure 3.
The biological width (2 mm on average) is the distance between the bottom of the sulcus and top of the alveolar crest, which consists of the junctional epithelium (1 mm) and the connective tissue adherence (1 mm). When dental restorations violate biologic width, chronic inflammation of the periodontium and an unpredictable loss of alveolar bone will occur until biologic width is restored by tissue breakdown. Our ultimate goal is to maintain a healthy insulation barrier.

1. video – Sulcus fluid collection.

24.4. Test – Gingival sulcus and crevicular fluid (answers)

1. It is NOT the border of the gingival sulcus:
   A. tooth surface
   B. alveolar process
   C. sulcular epithelium
   D. junctional epithelium

2. Is is characteristic of the biological width:
   A. the distance between the bottom of the sulcus and the top of the alveolar crest
   B. average width is 2 mm
   C. when dental restorations violate the biologic width unpredictable bone loss occurs
   D. consists of the junctional epithelium and the connective tissue grip

3. Which statement is false:
   A. in the junctional epithelium only 2 layers can be distinguished
   B. in the junctional epithelium there is a high degree of transepithelial permeability
   C. the sulcus epithelium ensures the real epithelial seal
   D. the epithelium is able to produce antimicrobial substances
25. 1.25. Oral aspects of salt and water household disturbancies – Jozsef Blazsek

The water content of our body is 60 to 65% and our vital functions such as food absorption, metabolism and excretion occur in an aqueous environment. Moreover, the vitality and integrity of the defense functions of our surface tissues is also strongly determined by the hydration state of the tissues, their secretion capability and, especially as it is with mucous membranes, the presence of an outer lubricating layer. One of the requirements for the normal function of oral cavity is the presence of a saliva of normal quality and quantity. Secretion by the exocrine glands is strongly influenced by the hydration and electrolyte parameters of the organism. In dehydration, not only production of urine drops to minimal, but that of sweat, tear and saliva as well. On the long run, it can lead to chronic conditions decreasing the quality of life (corneal inflammation, dermatitis, disorders of food absorption and digestion, injuries and inflammation of oral mucosa, taste disorders, increased incidence of caries, etc.).

Hence, conditions affecting the body’s hydration and electrolyte balance, in addition to their influence on metabolism in general, also affect oral function.

The baseline value of water turnover of a healthy human in civilization is 40 ml/kg body weight/day, and may be modified by various factors including lifestyle, work and leisure.

**Figure 1.296. Figure 1. – Calculation of daily water requirement**

<table>
<thead>
<tr>
<th>Calculation of daily water requirement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal intake ≈ 40 ml/kg/day ≈ approximately 3 l fluid</td>
</tr>
<tr>
<td>Insensible perspiration = 24 x 1/2 body weight</td>
</tr>
<tr>
<td>Load increase (metabolic increase) may raise the level</td>
</tr>
<tr>
<td>Daily water requirement in fever =</td>
</tr>
<tr>
<td>+ 1 °C = + 10 % daily requirement</td>
</tr>
</tbody>
</table>

Daily water consumption can be heavily influenced by certain diseases. When there is a drastic increase in water turnover, diabetes mellitus and insipidus, acute and chronic renal disease, increased exudation due to inflammation or burns, fever, prolonged diarrhoea or vomiting, chronic fistulae or occult bleeding are often to be blamed.

**Daily excretion** can vary broadly, its main components are:

- urine (1 to 1.5 litres daily),
- saliva and digestive juice (5 to 9 litres daily) of which 1 to 2%, i.e. 100 to 200 ml daily remains in the intestines and becomes fecal water,
- pulmonary and dermal perspiration (insensible perspiration, 500 to 1,000 ml daily),
- sweat (sensible perspiration, 400 to 1,500 ml daily).

The amounts above add up to a total of 2 to 4.2 litres daily.

**Figure 1.297. Figure 2. – Surface water output**
In addition to these regular components, further factors may contribute fluid loss such as lacrimation, nasal discharge, sputum, increased sweating, lactation, vomiting, diarrhea, bleeding and thermal burn exudation.

To achieve a balance, our daily water consumption generally made up of the following components:

- 1 to 1.5 to 2.6 litres in a fluid form,
- 0.7 to 1 to 1.2 litres of water in solid food,
- 300 to 400 ml as metabolic water.

This is a total of 2 to 4.2 litres of water brought in daily.

With total water deprivation the expected survival is 8 to 10 days.
The optimal volume of the fluid compartment is regulated by two central mechanisms, baro and osmoreceptors. Excess fluid consumption leads to increased volume and decreased osmolality, sensed by osmoreceptors in the hypothalamus, which results in decreased secretion of antidiuretic hormone (vasopressin), which decreases water reabsorption in kidneys.

**Figure 1.300. Figure 5. – Maximum water loss**

- The maximum water loss that we can tolerate is 20% of the total water content, ca. 10-12 liters.
- Without water – with maximum kidney concentration level, and 0.1 l daily urine + 0.5 ml/hr/kg insensible perspiration – only 8-10 days survival is possible.
- Calculation of total individual amount of water deficit (if there is only water loss and no loss ion):

  \[
  0.5 \times \text{weight} \times \frac{(\text{normal plasma Na}^+ \text{ cc} - \text{actual plasma Na}^+ \text{ cc})}{\text{normal plasma Na}^+ \text{ cc}}
  \]

**Figure 1.301. Figure 6. – Kidney can compensate increased water intake with extra secretion**

If the water intake is higher than needed:

- Hypervolemia (hypoosmiosis)
  - Osmotic pressure ↓
  - Plasma volume ↑
  - Atrial pressure ↑
  - Hypothalamic osmorec. activity ↓
  - Baro- volume rec. activity ↓
  - ADH secretion ↓
  - Thirst ↓
  - Renin-angiotensin-aldosterone and sympathetic activity ↓
  - Water input ↓
  - Renal water secretion ↑

  Approximate to isoosmotic isovolemia

**Figure 1.302. Figure 7. – Reverse compensation is possible too, so can compensate the decreased water intake and/or increased water**

- Status: Hypovolemia or hypovolemic hyperosmosis
  - Hypothalamus
  - Thirst ↑
  - ADH secretion ↑
  - Water input ↑
  - Renal water secretion ↓
  - Water retention ↑

  Approximate to isoosmotic isovolemia
This leads to increased urine production, which decreases volume. On the other hand, extra volume increases blood pressure. In response to elevated atrial tension, production of ANP (atrial natriuretic peptide) will increase, which in turn increases sodium and water excretion by kidneys. Stimulation of baroreceptors in blood vessels such as aorta and a. carotis communis will decrease the secretion of mineralocorticoids and that of renin, angiotensin and aldosterone, resulting in decreased sodium reabsorption in renal tubules, and, with further salt dependent water excretion, helps to restore isovolemia.

In a contrary situation, during thirst or increased water need, the reverse of the above mechanisms increases ADH, aldosterone and mineralocorticoid levels, leading to water conservation to decrease water need.

Evidently, sodium (Na$^+$) plays an important role in the above mechanisms (58 mmol/kg body weight), the 95% of which is extracellular. Closely linked to sodium regulation is that of potassium (K$^+$; 54 mmol/kg body weight, 91% of which is intracellular), calcium (Ca$^{2+}$; 2.1 to 2.6 mmol/l), chloride (Cl$^-$; 33 mmol/kg body weight, 85% of which is extracellular) and hydrogen ions (H$^+$). Maintaining the electrolyte balance of the body is essential to the homeostasis of cells.

55% of the body fluid volume is intracellular. In the membrane enclosed compartments there are ionic and non-ionic osmotically active molecules with various functions.

**Figure 1.303. Figure 8. – Distribution of electrolytes in body fluid compartments**

<table>
<thead>
<tr>
<th>Distribution of electrolytes in body fluid compartments</th>
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</thead>
<tbody>
<tr>
<td><img src="image.png" alt="Table Image" /></td>
</tr>
</tbody>
</table>

Normally, the osmotic pressure maintained in living cells is 340 mOsmol (650 kPa), called isosmotic.

**Disorders of water balance: dehydration, hyperhydration and electrolyte disorders**

Changes in the volume and/or osmotic activity of fluid spaces may lead to abnormalities: iso, hyper and hyposmosis with normal volume, or the same with hyper or hypovolemia, a combined total of nine different disorders.

**Figure 1.304. Figure 9. – Relationship between the volume and the osmotic constitution of the body**
Most crucial to the cells of an organism is the isosmolality of their environment. Therefore, restoring isosmolality is the first priority of regulation. For example, in hyperosmotic hypervolemia hyperosmosis is decreased first, even if it requires a further increase in volume. Or, in hyposmotic hypovolemia, water is first excreted to restore osmotic balance and then follows fluid volume restoration. The above mentioned regulatory mechanisms are employed to restore the balance. Failure of these mechanisms underlies the development of water and electrolyte balance disorders.
Figure 1.307. Figure 12. – Water and salt abnormalities

<table>
<thead>
<tr>
<th>Water and salt abnormalities</th>
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</thead>
<tbody>
<tr>
<td><strong>Need</strong></td>
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<tr>
<td><strong>Input</strong></td>
</tr>
<tr>
<td><strong>Primer</strong></td>
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<tr>
<td><strong>Secunder</strong></td>
</tr>
</tbody>
</table>

For example, a deficit in ADH production or a receptor deficiency results in a significantly increased urine production, as high as 10 to 15 litres a day, and a corresponding increase in water intake in diabetes insipidus.

Figure 1.308. Figure 13. – Diabetes insipidus (DI)

<table>
<thead>
<tr>
<th>Diabetes insipidus (DI) frequency: 1:25000</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition:</strong></td>
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<td></td>
</tr>
<tr>
<td><strong>Two (+1) forms can be distinguished:</strong></td>
</tr>
<tr>
<td>1. Central Diabetes Insipidus = CDI. DI is caused by the absolute or relative deficit of vasopressin (VP)</td>
</tr>
<tr>
<td>2. Nephrogen Diabetes Insipidus = NDI. DI is caused by the kidney tubule insensitivity for VP (aquaprotein function loss)</td>
</tr>
<tr>
<td>(+1): Poisoning (medicine) inflammation caused damage in renal tubular cells, and because of that, water reabsorption disorder (which is, of course, nephrogenic)</td>
</tr>
</tbody>
</table>

Sodium is the main electrolyte. In reality, intake usually exceeds need, generating a volume load. Disorders of sodium balance involve either deficiency or excess.

Figure 1.309. Figure 14. – Primer Na⁺ metabolic disorders

<table>
<thead>
<tr>
<th>Primer Na⁺ metabolic disorders</th>
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</thead>
<tbody>
<tr>
<td><strong>Regulation disorders</strong></td>
</tr>
<tr>
<td>A⁺</td>
</tr>
<tr>
<td>Hypoaldosteronism</td>
</tr>
<tr>
<td>Addison’s disease</td>
</tr>
<tr>
<td>1-3 hydroxilase deficit or disorder</td>
</tr>
<tr>
<td>Primer Hyperaldosteronism</td>
</tr>
<tr>
<td>Conn syndrome</td>
</tr>
<tr>
<td>Cushing syndrome: Predation therapy</td>
</tr>
<tr>
<td>Secunder Hyperaldosteronism (renin-angiotensin-aldosteron)</td>
</tr>
<tr>
<td>Reno-vascular disease</td>
</tr>
</tbody>
</table>
Another main electrolyte is potassium, which is the major cation of the cytosol. The amount of potassium in the interstitial fluid is raised by increased secretion by cells or by simply increased escape from the cells such as when hyperkalemia results from tissue damage.

Figure 1.311. Figure 16. – K⁺ metabolic disorder HYPOKALEMIA – 1.

- **A** – Hypokalemia
  - Normal total body K⁺ content: 1. Alkalosis (with H⁺ ion retention)
  2. Insulin therapy

- **B** – Hypokalemia
  - Low total body K⁺ content:
    1. Decreased K⁺ intake by nutrition
       a. Alcoholism
    2. K⁺ loss through the GI tract
       a. Vomiting (pyloric stenosis)
       b. Diarrhea
    3. K⁺ loss via the kidneys, with urine
       a. Increased mineralocorticoid levels
       b. Osmotic diuresis (diabetes mellitus)
       c. diuretic therapy
       d. Renal - renal tubular acidosis (CA)
       e. K⁺ loss in kidney diseases

Figure 1.312. Figure 17. – K⁺ metabolic disorders: Development of hyperkalemia

- **Redistribution (kidney reabsorption)**
  - Acidosis (H⁺/K⁺pump: increased excretion of H⁺ with K⁺ accumulation)
  - Physical load (compensation of lactic acidosis in the kidney)
  - Insulin deficiency (ketoadiposis and compensation in the kidney)

- **Decreased excretion**
  - Acute / chronic renal failure
  - diuretic (K⁺- sparing types: spironolactone)
  - MNF failure (increased excretion of Na⁺ with K⁺ retention)
  - hipoaldosteronism, Addison’s disease
  - renal tubular injury: K⁺ retention (decreased function of the Na⁺/ K⁺ pump)

- **Increased K⁺-Intake**
  - Big dose of K⁺-intake (K⁺ salts, infusion, injection, ion substitution tablets)

- **Cell injury** (hemolysis, muscular injury - intracellular K⁺ release)
  - Endogenous increase
  - Pseudohyperkalemia – high level of K⁺ in the blood because of the Rbc hemolysis

Figure 1.313. Figure 18. – K⁺ metabolic disorders: consequences
**K⁺ metabolic disorders: consequences**

- **Cardiovascular disorders**
  - ECG changes (T-wave tall and narrow; membrane potential decreases, irritability decreases, bradycardia, conduction block, ventricular fibrillation)
  - Myocardial cell necrosis and fibrosis
  - Decreased blood pressure
  - Enhanced digital effect

- **Neuromuscular disorders**
  - Weakness, muscle paresis, paralysis, dysphagia, paralyses, paresis
  - Ileus (constipation)
  - Orthostatic hypotension

- **Renal consequences**
  - Na⁺ retention - edema (due to the water, reabsorbed with Na⁺)
  - Hypokalemic nephropathy (local, due to K⁺ from kidney tubule cells)
  - Metabolic acidosis (due to protons reabsorbed by H⁺:K⁺ pump)

- **Metabolic and hormonal consequences**
  - Glucose intolerance
  - Negative nitrogen balance
  - Decreased hormone secretion: decreased aldosterone secretion, decreased insulin secretion, growth hormone secretion

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**Figure 1.314.** Figure 19. – Silicone coated blood collection tube containing clot activator for serum separation used in laboratory tests for ions

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**Figure 1.315.** Figure 20. – Urine collection tube for urine analysis
Effects of water and salt balance on oral functions

Any condition that decreases the hydration and disrupts the electrolyte balance can initiate a pathologic process in the oral cavity. Hypovolemia leads to reduced blood and oxygen supply, which results in decreased epithelial regeneration and impaired oral defense due to immune hypofunction. Reduced saliva production results in decreased oral clearance, increased plaque formation and consequently gingivitis and caries formation. This is why parameters of water and electrolyte balance are of great importance in the dental anamnesis of patients.

25.1. Test – Oral aspects of salt and water household disturbances (answers)

1. Which of the following conditions will NOT lead to a drastically increased fluid consumption?
   
   A. Increased vasopressin (ADH) production
   B. Diabetes mellitus
   C. Diabetes insipidus
   D. Fever, inflammation
   E. occult bleeding

2. The maximum survival time with water deprivation is:
   
   A. 3 to 5 days
   B. 6 to 7 days
   C. 8 to 10 days
   D. 12 to 13 days
E. approximately 2 weeks

3. Expected after contusion, tissue injury:

A. increased plasma sodium
B. increased plasma potassium
C. decreased urine ammonia content
D. decreased salivary pH
E. increased chance for alkalosis


The gastrointestinal tract is usually illustrated in handbooks starting from the esophagus and down, even though food ingestion and processing begins in the oral cavity. The operation of oral structures has a close structural and functional relationship with other sections of the gastrointestinal tract. This chapter highlights gastric and pancreatic dysfunctions from digestive tract diseases because of their dental implications. Gastric motility and secretion react to the oral cavity both in health and disease, while the pancreas and the salivary glands function in synergy buffering acids and producing fluid and digestive enzymes to the gut.

The pH of the oral cavity and esophagus is basically set by the buffering capacity of salivary bicarbonate. Acidic fluid exposure immediately increases salivary secretion by reflex mechanisms. Intraesophageal pH meter measurements indicate that the intake of an acidic drink leads to an immediate decrease in pH, which is gradually increases back to neutral values by the ingestion of saliva.

Figure 1.316. Figure 1. – Esophageal luminal acid clearance mechanisms

Reflux esophagitis, also called gastric reflux esophage disease (GERD), is based on persistently increased gastric pressure. Because of this, when the lower esophageal sphincter (LES) opens, gastric acid moves upward and reaches the esophagus and the oral cavity implementing a harmful effect. The primary cause of the disease is a delay in gastric emptying due to enhanced neural activity caused by the elevated muscle tone primarily around pylorus. Thus, GERD is not based on increased acid production, but rather it is a motility disorder. One of its important consequence is tooth erosion. Dental erosion treatment management cannot be restricted to the local oral treatment, the underlying disorder should also be treated.
The GERD and the development of related esophageal and tooth erosions are multifactorial. It affects a wide population, especially in developed countries. As civilization hazards the accelerated, stress-rich lifestyle, and also alcohol consumption, smoking and spicy foods are considered as primary risk factors. In early phase of GERD development, appropriate lifestyle changes may serve as efficient treatment. In a more advanced stage, the use of prokinetic drugs is necessary to increase motility.

GERD can be clearly distinguished from peptic ulcer disease, which appears in the stomach or in the initial section of the small intestine. According to our present knowledge, it is not caused by acid overproduction, although gastric acid undoubtedly plays a role in the resulting damage of the stomach. Thus, previously the inhibition of acid secretion was its primary treatment.
Gastric acid itself causes ulcer only in the case of extreme overproduction such as in the extremely rare gastrin-producing endocrine tumor, Zollinger-Ellison syndrome. In terms of the pathomechanism of ulcer development, it is more important that under normal circumstances the integrity of the mucosal surface is protected by the secreted mucins and bicarbonate from the produced acid.

**Figure 1.320. Figure 5. – Mucous and bicarbonate protection of the mucosa**

The most common cause of gastric ulcer is a bacterium called *Helicobacter pylori* (HP) that is often detectable in the stomach. The essence of ulcer development is that the degradation byproduct of the bacterial cell wall, the so-called endotoxin or LPS is highly irritative. LPS passing through the mucosa initiates a local inflammatory process in lamina propria, which loosens the mucosa, that becomes available to the exposition of the gastric acid and pepsin, and the caustic process begins.

**Figure 1.321. Figure 6. – Helio bacter pylori – discovered in 1983 by Bery Marschall and Cristopher Warren**

### Treatment of peptic ulcer before 1985

<table>
<thead>
<tr>
<th>Treatment options</th>
<th>Average time period needed for healing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diet restrictions</td>
<td>4-8 weeks</td>
</tr>
<tr>
<td>Antacids</td>
<td>2-6 weeks</td>
</tr>
<tr>
<td>H₂-receptor antagonists</td>
<td>2-4 weeks</td>
</tr>
<tr>
<td>Proton-pump inhibitors</td>
<td>1-4 weeks</td>
</tr>
<tr>
<td>Surgery (vagotomy etc)</td>
<td>-</td>
</tr>
</tbody>
</table>
90% of duodenal ulcers, and 70% of gastric ulcers develops on the ground of HP infection. Removal of bacteria leads to the cessation of the ulcer. The other most common cause of ulcer is the use of non-steroidal anti-inflammatory drugs (NSAIDs). These compounds inhibit the activity of COX enzymes that play a key role in gastric protection by synthesizing prostaglandins.

**Figure 1.322. Figure 7. – Initiation of mucosal inflammation by Heliobacter pylori**

**Figure 1.323. Figure 8. – H. pylori is present in most peptic ulcers**
The pancreas has both exocrine and endocrine functions. Similar to salivary glands, essentially its exocrine function is to secrete water, bicarbonate and digestive enzymes to digestive tract. Its enzymes serve for proteolytic, amylolytic, lipolytic and nucleolytic activities.

**Figure 1.324. Figure 9. – Physiological function of the pancreas**

**Figure 1.325. Figure 10. – Pancreatic enzymes**

The pancreas is under complex hormonal and neuronal regulation. This includes both activatory and inhibitory components. The main activator of ductal fluid secretion is secretin. The protein secretion of acini is stimulated...
by acetylcholine (Ach) released from the vagus nerve. In many species cholecystokinin (CCK) receptor of acini is the most important enzyme secretion enhancer. In humans, however, it does not directly affect the pancreas, but rather through activation of neural mechanisms.

**Figure 1.326. Figure 11. – Neuro-hormonal control of pancreatic secretory activity**

<table>
<thead>
<tr>
<th>Neuro-humoral control of pancreatic secretory activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>□ Hormonal regulation</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Secretin</td>
</tr>
<tr>
<td>Cholecystokinin</td>
</tr>
<tr>
<td>Bombasin (GRP)</td>
</tr>
<tr>
<td>Neurotensin</td>
</tr>
<tr>
<td>Pancreas polypeptide (PP)</td>
</tr>
<tr>
<td>Glucagon</td>
</tr>
<tr>
<td>Peptide YY (PYY)</td>
</tr>
<tr>
<td>Enteroglucagon</td>
</tr>
<tr>
<td>Somatostatin</td>
</tr>
</tbody>
</table>

**Figure 1.327. Figure 12. – Neurohormonal control of digestive enzyme secretion**

To understand the events leading to pancreatic self-digestion, we need to know why this process does not occur all the time in a healthy gland. One of the defense mechanisms is that digestive enzymes are secreted in an inactive form as zymogens, and are activated only in the small intestine by proteolytic digestion initiated by enterokinase. Synthesized enzymes in acinar cells are stored separately, in the form of zymogen granules. The pancreas is protected by trypsin inhibitors from occasional activation. Finally, due to the pressure difference, produced pancreatic fluid immediately secreted into the duodenum via the ductal system.

**Figure 1.328. Figure 13. – Various protective mechanisms reduce the likelihood of premature activation of trypsinogen to trypsin within the pancreatic parenchyma and initiation of autodigestion of the pancreas**
Acute pancreatitis, formed within hours or days, has been described as an interaction of inflammatory and self-digestive processes. The exact mechanism is still unclear, but several factors are likely to play a role in it. The most common basic event is described by Opie’s two hypotheses. According to these, gallstones exit from the gallbladder through the bile duct, temporarily close the sphincter of Oddi at the joint section of the pancreatic and bile ducts. This creates an opportunity for the bile to access the pancreatic duct where it activates lipolytic processes. Another possibility is that the trapped stone causes stasis of the secreted pancreatic fluid, providing the opportunity for zymogens to transform into active digestive enzyme.

Figure 1.329. Figure 14. – Acute pancreatitis was described by Chiari a century ago as an autodigestion of the gland

Figure 1.330. Figure 15. – The two "Opie hypotheses"
Acute inflammation may start not only a single, but multiple initial steps. One of these is the early, abnormal activation of pancreatic enzymes. In this process, as the pressure increases in the ducts because of an obstruction, a random cleavage of the stagnant zymogens may result in an auto-activation cascade coupled to ductal wall injury, inflammation, edema or pancreatic necrosis. Finally these may lead to multiple organ failure (MODS = multiple organ dysfunction syndrome). In addition to the above, bile-activated phospholipase A-induced transformation of lecithin to lysolecithin is also an aggravating factor because of the membrane damaging effect of lysolecithin. The exact mechanism of the alcohol toxicity in acute pancreatitis is not entirely clear, but besides its direct metabolic effect, its role in the inhibition of fluid drainage by contracting sphincter of Oddi worsens the situation. All these are often accompanied by disturbances of the microcirculation, which leads to embolism and vasculitis. Late factors are the bacterial overinfection of the gland due to the decrease of intestinal barrier function, and the spread of local inflammation and the self-reinforcing process of the release of inflammatory mediators and free radicals.

**Figure 1.331. Figure 16. – Initiation of acute pancreatitis I.**

**Figure 1.332. Figure 17. – Initiation of acute pancreatitis II.**
As a consequence, the obstruction of the pancreatic ducts plays a key role at multiple levels in the development of disease. However, it is possible that the obstruction, such as gallstones inclusion, ceases, but the inflammatory process continues and becomes self-reinforcing.

Figure 1.333. Figure 18. – Possible role of secretory block in genesis of pancreatitis

75% of acute pancreatitis cases only reach the edematous level of inflammation and after regeneration pancreatic function completely normalizes. However, in 25% of the cases complications develop, and about 10% of severe cases lead to death by necrosis and by overinfections. The outcome mainly depends on the quantitative and qualitative composition of the inflammatory mediators released in the early and late phases of the disease.

Figure 1.334. Figure 19. – Candidate mediators for tissue ischemia in severe acute pancreatitis
Chronic pancreatitis develops over decades. The acinar tissue of the gland is slowly but gradually destructed and connective tissue takes over its place. Not only the exocrine, but also the endocrine function is damaged in captivity of the connective tissue. Chronic alcoholism and the related lifestyle and poor nutrition is the underlying cause in over 70% of cases. The two models describing the development of this disorder are called inflammatory and obstructive models. According to the obstruction model, alcohol increases protein and decreases fluid secretion, thus, the high protein and calcium concentration in ductal lumen results in precipitating protein plugs. They blockade of outflow of the pancreatic juice results in constant irritation, increased pressure and inflammation.

The other model suggests that alcohol causes constantly recurring local acute injuries that do not appear at the clinical level, but over time cause generalized tissue damage. In normal circumstances the myofibroblast-derived "Stellate cells" are inactive. However, they become activated in chronic pancreatitis, proliferate intensively and gradually replace the destroyed acinar tissue thereby creating fibrosis. In reality, the two models certainly prevail together.
Chronic pancreatitis is characterized by a strong, belt-like abdominal pain, which in advanced stage is difficult to be attenuated. The main reason of pain is that obstruction leads to increased pressure in the tissue, and this puts pressure on the nerve endings. In addition, signals from the small intestine stimulate the gland to increase secretion to replace digestive enzymes missing from the bowel. This triggers further pressure increase, pain, and as a consequence, inflammation, ischemia. The situation continues to deteriorate because of the development of pseudocysts and common bile duct stenosis.

Figure 1.338. Figure 23. – Causes of the pain in CP

<table>
<thead>
<tr>
<th>Causes of the pain in CP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ductular obstruction evoking hypertension in the duct</td>
</tr>
<tr>
<td>Increased intrapancreatic pressure with ischemia</td>
</tr>
<tr>
<td>Neural inflammation and scarring</td>
</tr>
<tr>
<td>Intra- and peripancreatic fluid collections</td>
</tr>
<tr>
<td>Pseudocysts</td>
</tr>
<tr>
<td>Common bile duct stenosis</td>
</tr>
<tr>
<td>Duodenal compression</td>
</tr>
<tr>
<td>Papillitis</td>
</tr>
</tbody>
</table>

The disease goes with increased physical impoverishment and in advanced stage with malabsorption and large amount of fatty stool. The underlying reason is that although the functional capacity of the pancreas is very large, less than 10% of the normal amount of lipase is not able to break down dietary fats any more. Thus, the level of undigested material rises sharply, which is a substrate for bacteria in the colon. The result is steatorrhea, fatty stools. When food intake is relatively low, as it is the case of most patients suffering from chronic pancreatitis, amylolytic activity can be maintained by salivary amylase, while proteolytic activity by gastric pepsin and small intestinal exopeptidases could still digest their respective components at reasonable efficiency.

Figure 1.339. Figure 24. – Steatorrhea and chronic pancreatitis
Cystic fibrosis develops due to a mutation of CFTR (cystic fibrosis transmembrane regulator). Its main manifestations are an extreme decrease in the pancreatic secretory function and inflammatory, fibrotic processes in the lung that in most cases lead to the early death of the patient. CFTR is a cAMP-dependent chloride channel, which plays a crucial role not only in the transport of chloride, but also in the consequential sodium and water secretion. In the absence of CFTR, these secretory activities are dramatically reduced.

Figure 1.340. Figure 25. – Failure of apical chloride transport by CFTR in cystic fibrosis patients

In normal pancreas, proteins and calcium ions secreted by acini are diluted by the large secreted volume of ductal fluid and quickly transported to the small intestine. In cystic fibrosis, because of the mutation in the CFTR channel, water and electrolyte secretion is low, the stagnation of secreted proteins and calcium in the ducts leads to the formation of protein plugs which then block the flow of secretion. The result is the destruction of the acinar parenchyma and the formation of fibrotic tissue similar to the process in chronic pancreatitis.

Figure 1.341. Figure 26. – Effect of cystic fibrosis
CFTR is a very large transmembrane protein, in its nucleotide sequence a large number of possible polymorphisms have been described. Some of these are missense mutations, like the most common cause of the disease the ΔF508 mutation, which is an "AA deletion", leads to a dramatic secretion decrease, while in the case of other mutations the function is partially preserved, and in some other cases sequence change is not accompanied by function loss at all.

Figure 1.342. Figure 27. – Gene mutations/polymorphisms in CF

26.1. Test – Oral aspects of gastric and pancreatic disorders (answers)

1. Usual consequence of gastroesophageal reflux disease:
   A. lesion of gastric mucosa
   B. dental erosion
   C. lesion of duodenal mucosa
   D. esophageal varices

2. The major cause of the gastric ulcer in the majority of cases:
   A. decreased acid neutralization capacity of the pancreas
1. Oral biology

B. bacterial infection
C. increased gastric acid production
D. decreased gastric acid production

3. Most common risk factor of chronic pancreatitis
A. hypertension
B. chronic decreased protein intake
C. hepatitis-B induced cholestasis
D. chronic alcoholism

27. 1.27. Oral aspects of acid-base regulation – Jozsef Blazsek

Our body maintains pH between 7.38 and 7.42 by regulatory mechanisms to provide an optimal environment for the function of biomolecules essential for vital functions. A change in pH would alter the conformation of biomolecules, preventing appropriate molecular interactions and thus their normal function.

ACID is a molecule that donates proton, while a BASE accepts proton. Some molecules are called amphoteric. They can, depending on the presence of more potent acids or bases, both donate and accept proton. Proteins are such molecules, which, due to the presence of functional groups, can either donate or accept protons at a given pH. Isoelectric point, characteristic for each particular protein, is the pH at which the molecule carries no net electric charge. Proteins behave either as acids or as bases at a given pH according to their isoelectric point.

Figure 1.343. Figure 1. – Acid-Base balance

<table>
<thead>
<tr>
<th>Acid-Base balance</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Acid = proton (H⁺) donor</td>
</tr>
<tr>
<td>☐ Base = proton (H⁺) acceptor</td>
</tr>
<tr>
<td>☐ Conjugated acid base pairs:</td>
</tr>
<tr>
<td>e.g.: acid: dissociate (cation) + base: (anion)</td>
</tr>
<tr>
<td>HCl = (H⁺) + Cl⁻</td>
</tr>
<tr>
<td>H₂CO₃ = (H⁺) + HCO₃⁻</td>
</tr>
<tr>
<td>H₃PO₄ = (H⁺) + H₂PO₄⁻</td>
</tr>
<tr>
<td>NH₃ = (H⁺) + NH₂⁻</td>
</tr>
<tr>
<td>Hprotein = (H⁺) + protein⁻</td>
</tr>
<tr>
<td>H₂O = (H⁺) + OH⁻</td>
</tr>
</tbody>
</table>

EC area (blood, tissue fluid) 6.8 < acidosis pH 7.4 < 7.8 isohydric alkalosis

Crucial to life on Earth is water as a solvent. Atmospheric carbon dioxyde dissolves in and reacts with water to form carbonic acid (H₂CO₃), which can in turn dissociate to form bicarbonate ions (HCO₃⁻) and protons (H⁺), with 20 times as many bicarbonate ions (26 mmol/l) as carbonic acid molecules (1.3 mmol/l) being present in the solution under equilibrium conditions. H⁺ concentration and pH can then be expressed from the equilibrium equation, yielding an equilibrium pH of 7.4 (Figure 2). Biomolecules have evolved such that they assume the conformation appropriate for their function at pH 7.4.

Figure 1.344. Figure 2. – Buffer-systems (Henderson–Hasselbach)
Weak acids can dissociate depending on the pH of their environment. When copresent with their base salts in solution, they form a so-called buffer system, capable of maintaining a stable pH in the solution.

An appropriate intra and extracellular pH is crucial. The intracellular pH is stabilized with the help of the soluble proteins, phosphate, bicarbonate and proton pumps (Na⁺/H⁺ pump, 36%; K⁺/H⁺ pump, 15%; other, 6%) of the cell. Carbon dioxide is the end product of cellular fat and carbohydrate metabolism, with a daily production of 12,000 to 13,000 mmol. Carbon dioxide is highly mobile, easily diffuses out of the cell where it reacts with water to form carbonic acid which subsequently dissociates. Protons produced in cells are absorbed by bicarbonate and basic proteins within seconds to restore pH to 7.4. Excess protons can be absorbed by bicarbonate produced by red blood cell carbonhydrase and by hemoglobin. Bound protons dissociate from hemoglobin in the lung, are absorbed by bicarbonate, and the resulting carbonic acid is converted by red blood cell carbonhydrase to water and carbon dioxide, which is exhaled. Carbonic acid can thus be eliminated by breathing, it is therefore called a volatile acid. Even though protons are not actually eliminated by this mechanism, they are effectively buffered so they do not cause systemic acidification. Therefore, breathing rapidly (within 2 to 15 minutes) and effectively regulates systemic pH. Cells also produce nonvolatile metabolic acids that cannot be eliminated by breathing. Acids are named after their anions: lactate is a product of anaerobic metabolism, phosphate is that of phospholipid catabolism, sulphate is that of amino acid catabolism, whereas acetoacetate and β-hydroxibutirate are liver ketones. These anions are excreted with urine, either after enzymatic processing or glucuronated.

A major aspect of the acid base balance is that proton excretion should balance daily acid load and production. Excess protons remaining after rapid buffering must be excreted (mostly) by kidneys with urine. Otherwise the reservoir of buffering bases will be exhausted and systemic pH will decrease, which on the long run will lead to apoptosis through the mitochondrial pathway (cytochrome-C, capases, A1F, SMAC). Alterations in pH in the extracellular environment can cause conformation alterations in essential proteins which may lead to fatal problems in regulation.

The essential steps in acid-base regulation are:

- decrease in intracellular pH;
- in situ intracellular buffering,
- extracellular buffering,
- transportation,
- excretion by the kidney.

Respiratory pH disorders are predominantly (99% of acidoses and 97% of alkaloses) buffered in the intracellular compartment in a carbonhydrase dependent manner. In tissues where carbonhydrase is present (red blood cells, kidneys, salivary glands) the rate of carbonic acid synthesis is increased a thousand fold. This enables these cells to make protons and bicarbonate and therefore they can participate in pH regulation.
Another import mechanism of long-term (hours-days) pH regulation based on the function of the proximal and distal tubules and collecting ducts of the kidney. Through carboanhydrase mediated carbonic acid synthesis proximal tubule cells (PTC) can exchange protons for sodium or potassium (via the sodium/proton exchangers). They transport three bicarbonate anions along with one sodium ion to the interstitial space via an electrogenic sodium/bicarbonate cotransporter. Additional sodium is transported to the interstitial space in exchange for potassium via the sodium/potassium ATPase. During increased acid load protons activate glutaminase in PTCs, which deaminates glutamine to form glutamate and ammonium ($\text{NH}_4^+$). Glutamate is then deaminated to α-ketoglutarate and another ammonium by glutamate dehydrogenase. Ammonium is transported to the luminal space via a sodium/proton exchanger or, after deprotonation, as $\text{NH}_3$ molecules by diffusion. Carbon dioxide produced by oxoglutarate dehydrogenase from α-ketoglutarate reacts to form bicarbonate, which is then transported to the interstitial via an electrogenic sodium/bicarbonate cotransporter, and, as a buffering base, decreases acidity. Protons remaining in the in PTCs are transported through a sodium/proton antiporter (NHE) to the luminal space where they may bind ammonia to be excreted as ammonium ions. Alternatively, they may be excreted along with phosphate or chloride.

Importantly, there is no single regulatory mechanism for systemic pH regulation. Respiratory pH regulation is based on carbon dioxide acting on the breathing center in the brain stem. Likewise, systemic pH is not a direct regulator of water and electrolyte metabolism in the kidney.

**Figure 1.345. Figure 3. – The acid-base balance disturbances: Forms and principal clinical signs**

<table>
<thead>
<tr>
<th>The acid-base balance disturbances: Forms and principal clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ACIDOSIS</strong></td>
</tr>
<tr>
<td>• Reduced sensitivity of blood vessels to the transmitters</td>
</tr>
<tr>
<td>- Impaired microcirculation</td>
</tr>
<tr>
<td>- Motility-movement disorder</td>
</tr>
<tr>
<td>• breathing: fast + deep (&quot;Kussmaul&quot;)</td>
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<td></td>
</tr>
</tbody>
</table>

Local acid-base imbalances in tissues may arise at any time, as pH may rapidly change with alterations in blood flow. Decreasing blood flow may cause hypoxia, which results in acidification that can lead to cell death by apoptosis. With increasing metabolism, such as that in conjunction with exercise, can also result in hypoxia when blood flow is not sufficiently increased.

Systemic acidosis or alkalosis occurs with changes in respiration, kidney excretion or metabolism. Generalized acidosis or alkalosis as such can occur due to respiratory or metabolic reasons.

**Respiratory acidosis**

Any condition that involves decreased respiration compared to needs (hypoventilation). Decreased respiration leads to increased systemic and tissue carbon dioxide levels. This results in an increase in the amount of carbonic acid and protons. This can be compensated by an increased formation of bicarbonate buffer base, given that kidneys can in turn increase the rate of proton excretion. This condition is therefore aggravated by hypernatremia and hyperkalemia. Conditions involving respiratory acidosis can be further aggravated by kidney diseases.

**Figure 1.346. Figure 4. – The conditions which lead to respiratory acidosis (hypoventilation)**
Respiratory alkalosis

Hyperventilation decreases systemic and tissue carbon dioxide levels which results in low carbonic acid and proton concentrations. As there is no normal carbon dioxide-generated bicarbonate production in the kidneys, this condition is rapidly compensated in the presence of normal kidney function. In addition, hyperventilation is decreased by CO₂ wash-out in the brain stem, which inhibits the breathing center. Conditions leading to respiratory alkalosis are summarized in Figure 5.

**Figure 1.347. Figure 5. – The conditions which lead to respiratory acidosis (hyperventilation)**

<table>
<thead>
<tr>
<th>The conditions which lead to respiratory acidosis (hypoventilation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Chest deformity (congenital or acquired)</td>
</tr>
<tr>
<td>☐ Lung Disease</td>
</tr>
<tr>
<td>• Obstructive: increased airway resistance</td>
</tr>
<tr>
<td>• Restrictive: decreased elasticity of the lung tissue</td>
</tr>
<tr>
<td>• Pulmonary blood flow disturbances; gas exchange disorder - VA shunts</td>
</tr>
<tr>
<td>☐ Nervous system disorders</td>
</tr>
<tr>
<td>• Medullary respiratory center damage: bleeding, thrombosis, inflammation, drug activity</td>
</tr>
<tr>
<td>• Transverse spinal lesion: anterior horn injury</td>
</tr>
<tr>
<td>• Guillain-Barre syndrome: spinal cord upward tracks after infection, paralysis</td>
</tr>
<tr>
<td>☐ Respiratory muscle weakness</td>
</tr>
<tr>
<td>• Myasthenia gravis</td>
</tr>
</tbody>
</table>

Metabolic acidosis

Most characteristic are the functional disorders of sodium/proton exchangers in the kidney (renal tubular acidosis), which result in decreased proton excretion and, further worsening the condition, decreased formation of bicarbonate buffer base. The underlying reason may be a decreased or absent function of the exchanger due to genetic, autoimmune reasons or resulting from an infection. Acidosis often arises from a systemic metabolic disease. For example, ketoacidosis in diabetes mellitus, combined with the kidney alterations in a more progressed disease, can cause serious acidotic disorders. Stomach resection, such as that performed to remove a tumor, may lead to metabolic acidosis, especially in combined forms.

**Figure 1.348. Figure 6. – The different background of metabolic acidosis**
Metabolic alkalosis

The underlying reason is most frequently a condition with severe vomiting, because of the resulting loss of acid. In addition to infectious diseases of the gastrointestinal tract, severe nervous system injuries resulting from concussion, viral diseases, brain tumors etc. can also lead to recurrent emesis and a resulting loss of acid. Disorders of proximal and distal tubule ion transporters may also cause serious disorders, especially when compensation is not possible due to the presence of the disorders in both parts.

Figure 1.349. Figure 7. – Metabolic alkalosis

The presence of a dysfunctional or decreased amounts of carboanhydrase (CA) due to a genetic deficiency can lead to various disorders of pH regulation. CA hypofunction in red blood cells results in respiratory acidosis, while that in the kidney causes metabolic acidosis. Importantly, organisms have mostly evolved for acidotic conditions, being more suited to compensate for acidosis rather than alkalosis.

The carboanhydrase content and function of saliva is crucial for regulating the pH of the oral cavity and therefore, by demineralizing or remineralizing the hard tissues, for the prevention of enamel, dentin and cement destruction and of caries. Thus, continuous production of an appropriate amount of saliva is crucial for the pH regulation of the oral region and of the esophagus. Often a disorder in saliva production may underlie the deterioration of the ever more prevalent reflux disease.

Figure 1.350. Figure 8. – Blood collection tube containing sodium heparin blood-gas analysis
27.1. Test – Oral aspects of acid-base regulation (answers)

1. The following condition will lead to metabolic acidosis:
   A. conditions with thoracic rigidity
   B. lead poisoning
   C. hyperventillation
   D. in disorders of kidney carboanhydrase function
   E. in kidney aquaporin-5 dysfunction

2. What is the amount of carbon dioxide produced daily by cellular respiration in a healthy adult human?
   A. 6500 to 500 mmol
   B. 8500 to 9500 mmol
   C. 12000 to 13000 mmol
   D. 16500 to 17500 mmol
   E. 18500 to 19500 mmol

3. Which of the following acids can be eliminated by respiration?
   A. lactate
   B. carbonate
   C. phosphate
D. hydrogen chloride
E. acetate

4. Which of the following anions are not found in urine as an anion partner of hydrogen ion?
A. ammonium chloride
B. phosphate
C. chloride
D. bicarbonate
E. hydroxyl anion


The most important function of the kidney is excretion, however, this is linked to many other vital functions. Maintaining osmotic conditions, salt and water balance regulation, acid-base balance, excretion of metabolic waste products and toxic substances, several endocrine functions, such as production of erythropoietin and active vitamin D, are all functions of the kidneys.

**Figure 1.351. Figure 1. – The most important functions of the kidney**

<table>
<thead>
<tr>
<th>The most important functions of the kidney</th>
</tr>
</thead>
<tbody>
<tr>
<td>To keep osmotic pressure</td>
</tr>
<tr>
<td>To regulate salt and water balance</td>
</tr>
<tr>
<td>To excrete metabolic byproducts</td>
</tr>
<tr>
<td>To excrete toxic compounds</td>
</tr>
<tr>
<td>Acid-base regulation</td>
</tr>
<tr>
<td>Endocrine function (erythropoietin, etc.)</td>
</tr>
<tr>
<td>Synthesis of enzymes and regulatory compounds</td>
</tr>
</tbody>
</table>

Renal function can be divided into three main elements: **glomerular filtration, tubular secretion and tubular reabsorption**. The structures responsible for these functions determine the functional and structural arrangement of the kidney.

**Figure 1.352. Figure 2. – Glomerular filtration and tubular secretion and reabsorption**
The initial step in renal excretory function is glomerular filtration, which results in an ultrafiltrate with a very similar composition to plasma, although it contains very little protein. A normal glomerular filtration rate (GFR) is about 180 liters/day, compared with about 1.5 liters of urine produced, the rest being reabsorbed along the nephron. GFR is defined by the arterial blood pressure, the intracapsular pressure and by the magnitude and permeability of the filtration surface.

**Figure 1.353. Figure 3. – Glomerular filtration (GFR)**

<table>
<thead>
<tr>
<th>Glomerular filtration (GFR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GFR = k. S. ((Pγ - Pτ) - (πγ -πτ))</td>
</tr>
</tbody>
</table>

k: diameter of pores (capillary permeability)
S: filtration surface
P: hydrostatic pressure
π: osmotic pressure
γ: at the glomerular side
τ: at the tubular side

There is a close relationship between renal blood flow and glomerular filtration and actual urine output. Below a critical blood pressure level, both glomerular filtration and urine output are terminated, with possible fatal consequences for the individual.

**Figure 1.354. Figure 4. – Blood pressure relationship to glomerular filtration rate (GFR), renal plasma flow (RPF) and urine output**
Acute renal failure (ARF) is characterized by a high level of GFR impairment, regardless of the cause. ARF develops rapidly, over days or weeks. Excretion of metabolites, electrolytes and water lessen due to the large decrease in filtration. Consequently, the urea and creatinine concentrations of blood plasma are significantly increased. The water, H⁺ and K⁺ secretion defects lead to edema, acidosis and hyperkalemia. ARF is often characterized by oliguria or anuria, however, sometimes urine output is not reduced.

**Figure 1.355. Figure 5. – Definition and characteristics of acute renal failure**

<table>
<thead>
<tr>
<th>Definition and characteristics of acute renal failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>- It develops in days or weeks</td>
</tr>
<tr>
<td>- It can be best characterized as a serious decrease in glomerular filtration (GFR)</td>
</tr>
<tr>
<td>- Plasma urea and creatinine conc. increases</td>
</tr>
<tr>
<td>- Water, H⁺ and K⁺ excretion decreases</td>
</tr>
<tr>
<td>(edema, acidosis, hyperkalaemia)</td>
</tr>
<tr>
<td>- Oliguria, anuria or non-oliguria</td>
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</tbody>
</table>

**Figure 1.356. Figure 6. – Clinical significance**

<table>
<thead>
<tr>
<th>Clinical significance</th>
</tr>
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<tbody>
<tr>
<td>- High mortality</td>
</tr>
<tr>
<td>- Current therapy is limited</td>
</tr>
<tr>
<td>supportive, preventive strategies, none of which have been definitively shown to alter mortality.</td>
</tr>
<tr>
<td>- Ischemic ARF - multiple organ failure and sepsis</td>
</tr>
<tr>
<td>- Independently carries a marked increase in mortality</td>
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</tbody>
</table>
The clinical significance of ARF stems from its relatively high incidence and high mortality rate. The therapeutic options are very limited, they based on preventing sepsis and mortality. On the basis of serum creatinine level, the glomerular filtration rate and the amount of excreted urine, the RIFLE classification provides a prognostic outcome for the disease. Usually, three stages are distinguished. The initial, formation stage is rapid and characterized by a rapid decline in GFR caused by harmful effects. During the second, existence phase the damage expands despite the cessation of harmful effects. This phase may take a few weeks to a few months. In the third, regeneration phase the tissue is regenerated, and the original function may be restored within weeks to months.

**Figure 1.357.** Figure 7. – Change in clinical parameters

**Figure 1.358.** Figure 8. – RIFLE classification of ARF

Acute renal failure is essentially due to three reasons: these are the prerenal, renal and postrenal causes. Prerenal causes are the disorders of systemic circulation, and diseases of the renal arteries. Renal (intrinsic) causes include hemodynamic or toxic factors, while postrenal causes of renal disease include the obstructed flow of urine.

**Figure 1.359.** Figure 9. – Etiology of acute renal failure
Hemodynamic factors that cause acute renal failure are disorders of afferent and efferent arterioles, diseases of glomeruli, systemic diseases, and diseases that cause acute tubular necrosis. Nephrotoxicity typically originate from acute tubular necrosis by endogenous or exogenous factors or from interstitial nephritis by endogenous nephrotoxins or infections.

**Figure 1.360. Figure 10. – Development of acute renal failure**

Vascular factors include constriction of the glomerular afferent arterioles, dilatation of efferent arterioles or decreased glomerular permeability. Tubular damage may derive from increased intratubular pressure due to tubular obstruction or decreased tubular pressure due to tubule wall injuries.

**Figure 1.361. Figure 11. – Role of vascular factors in acute renal failure**

**Figure 1.362. Figure 12. – Role of tubular factors in acute renal failure**
Owing to the direct physical link between the glomeruli and tubules, either glomerular or tubular damage may directly affect the operation of the glomeruli and cause dramatic decreases in the glomerular filtration rate. This feedback thereby serves to protect the kidney, because any damage to the excretory function immediately stops the flooding of damaged nephrons by filtrate.

Figure 1.363. Figure 13. – Tubulo-glomerular feedback

Renal blood flow decreases in the formation phase of acute renal failure, but this decrease in blood flow plays a less significant role in maintaining renal failure than it does in the formation period. The GFR during these phases is clearly low. Blood flow and glomerular filtration resolve in the regeneration phase, increased urinary excretion is observed until concentrating ability is restored.

Figure 1.364. Figure 14. – The regenerative phase of acute renal failure
Prerenal acute renal failure is substantially due to ischemic damage. It may be caused by decreased extracellular fluid volume, cardiac damage, peripheral vasodilatation, increased renal vascular resistance or blockage of renal arteries.

**Figure 1.365. Figure 15. – Prerenal causes of acute renal failure**

<table>
<thead>
<tr>
<th>Prerenal causes of acute renal failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic alterations due to disorders of a systemic circulation or diseases of renal arteries</td>
</tr>
<tr>
<td>- decreased effective extracellular fluid volume</td>
</tr>
<tr>
<td>- damaged heart function</td>
</tr>
<tr>
<td>- peripheral vasodilatation</td>
</tr>
<tr>
<td>- increased renal vascular resistance</td>
</tr>
<tr>
<td>- renal vascular obstruction</td>
</tr>
</tbody>
</table>

During the development of prerenal acute renal failure the systemic blood pressure drops below 60-70 mmHg, therefore the GFR decreases quickly. The urea-to-creatinine ratio of blood increases because of the backward diffusion of urea. Urinary sodium concentration decreases while urea, creatinine and potassium levels of the urine increase because of water retention.

**Figure 1.366. Figure 16. – Prerenal causes of acute renal failure**

<table>
<thead>
<tr>
<th>Prerenal causes of acute renal failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic pressure decreases to a value that is under the lower limit of autoregulation range (60-70 Hgmm)</td>
</tr>
<tr>
<td>GFR dramatically drops</td>
</tr>
<tr>
<td>- (it might happen at higher blood pressure due to other sensitizing reasons)</td>
</tr>
<tr>
<td>blood urea/creatinine ratio increases</td>
</tr>
<tr>
<td>urine Na⁺ concentration is low (reabsorption)</td>
</tr>
<tr>
<td>urine urea, creatinine, K⁺ high (tubular transport)</td>
</tr>
</tbody>
</table>

In the background of postrenal acute renal failure disorders of urine drainage can be found. It is caused by the deposition of renal compounds or by obstruction by other (not renal) causes. After complete obstruction the glomerular filtration continues at a decreasing rate and then stops.

**Figure 1.367. Figure 17. – Postrenal causes of acute renal failure**
Acute renal failure has numerous life-threatening consequences. Water and electrolyte excretion by the kidney is not balanced with their intake. Thirst increases. Na\(^+\) and Ca\(^{2+}\) levels decrease, K\(^+\), H\(^+\), Mg\(^{2+}\), urea and creatinine concentration increase in the plasma, and thrombosis, anemia and hemolysis occur.

**Figure 1.368. Figure 18. – Consequences of acute renal failure**

Slow, progressive destruction of the kidney tissue is called **chronic renal failure**. Usually it is a one-way process emerging over decades. Its most characteristic parameter is the glomerular filtration rate. Therefore, chronic renal failure can be divided into phases according to the GFR values. During the progression of the disease the excretory, synthesizing and hormonal functions of the kidney are continuously declining.

**Figure 1.369. Figure 19. – Definition and stages of chronic renal failure**

The kidney has a high level of reserve functional capacity and adaptive ability. This is based on the fact that the remaining nephrons take over the function of the destroyed ones. Consequently their operation is enhanced and their cells undergo hypertrophy. But this process leads to the destruction of other nephrons that in turn worsens the overall renal function.
In chronic renal failure many processes become pathological. Fluid drainage can be characterized by hyposthenuria, isosthenuria (asthenuria). The decrease of urine concentration ability is called hyposthenuria. When this condition worsens isosthenuria occurs: the kidney is unable to form urine more concentrated than plasma. Decreasing dilution ability is also called asthenuria. Sodium excretion is maintained for a long time, it only decreases in a late stage. Hyperkalemia arises progressively when GFR is less than 10%. The phosphate level of the plasma increases therefore the Ca\(^{2+}\) decreases and secretion of parathyroid hormone increases as the disease progresses. Disorders of acid-base balance cause metabolic acidosis that is accompanied by a decrease in ammonia synthesis and ammonium excretion. The level of urea is strongly increased when the GFR falls below 20%, however it also largely depends on the protein intake.

**Accompanying symptoms** of chronic renal failure are erythropoietin deficiency, hemolysis, thrombocyte dysfunction and anemia caused by acidosis. The cause of hypertension in addition to electrolyte and water retention, the disorder of the renin-angiotensin system and the vasopressor function of the kidney is the release of different vasoactive compounds. Hyperlipidemia and a decrease in sugar tolerance are also aggravating factors.
Uremia is the end stage of chronic renal failure. GFR drops below 10% of its normal value and consequently oliguria and then anuria occur. Generalized edema arises because of the salt and water retention. Metabolic acidosis occurs because of the disorder of proton excretion and the depletion of buffer systems. A further consequence of protein degradation and the excretory disorder coupled with acidosis is extracellular hyperkalemia. Anemia occurs because of erythropoietin production deficiency in the kidney. There is an increased risk for hypertension because of the salt and water retention and the disorder of the renin-angiotensin system. Decreased vitamin D synthesis causes osteomalacia and secondary hyperparathyroidism occurs. The direct cause of uremic coma is usually the acidosis, the high levels of Na⁺ and K⁺, and the accumulation of nitrogen containing metabolites. An important accompanying symptom of the process is the disorder of calcium homeostasis.

Figure 1.373. Figure 23. – Characteristics of uremia

<table>
<thead>
<tr>
<th>Characteristics of uremia</th>
</tr>
</thead>
<tbody>
<tr>
<td>GFR decreases below 10 % of normal (oliguria, anuria)</td>
</tr>
<tr>
<td>Generalized edema (salt and water retention)</td>
</tr>
<tr>
<td>Metabolic acidosis (H⁺ excretion, exhausted buffer capacities)</td>
</tr>
<tr>
<td>Extracellular hyperkalemia (protein breakdown, acidosis)</td>
</tr>
<tr>
<td>Plasma urea, creatinine levels increase</td>
</tr>
<tr>
<td>Anemia (erythropoietin decreases)</td>
</tr>
<tr>
<td>Hypertension (salt and water retention, renin-angiotensin system)</td>
</tr>
<tr>
<td>Osteomalacia (vitamin D metabolism damage)</td>
</tr>
<tr>
<td>Secondary hyperparathyroidism (increased PTH)</td>
</tr>
<tr>
<td>Uremic coma (acidosis, high K⁺, Na⁺, N and PTH conc.)</td>
</tr>
</tbody>
</table>

Figure 1.374. Figure 24. – Overview of the metabolic systems that maintain calcium homeostasis
Dental implications of kidney failure are important, especially since it is a decades-long process. Young-onset renal failure can lead to disruption of developing oral structures, with visible morphological features. Late-stage uremic symptoms are accompanied by a number of oral and gastrointestinal manifestations, including dry mouth, bad breath and other related symptoms.

**Figure 1.375. Figure 25. – Gastrointestinal effects of uremia**

<table>
<thead>
<tr>
<th>Gastrointestinal effects of uremia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Xerostomia</td>
</tr>
<tr>
<td>Oral malodor</td>
</tr>
<tr>
<td>Sialosis</td>
</tr>
<tr>
<td>Anorexia</td>
</tr>
<tr>
<td>Hiccups</td>
</tr>
<tr>
<td>Vomiting</td>
</tr>
<tr>
<td>Gastrointestinal bleeding</td>
</tr>
<tr>
<td>Constipation (due to perilesional dialysis)</td>
</tr>
<tr>
<td>Esophagitis, gastritis, duodenitis and peptic ulceration (in late CRF)</td>
</tr>
</tbody>
</table>

**Figure 1.376. Figure 26. – Orofacial features of renal failure**
1. How does protein concentration change usually in the urine when the glomerular function is impaired?

A. no or minimal amount of urine is present
B. increased
C. no detectable protein in the urine
D. not changed
E. decreased

2. In acute renal failure always happens:

A. increased tubular pressure due to tubular obstruction
B. tubular reabsorption is decreased
C. tubular secretion is decreased
D. glomerular filtration is decreased

3. Approximate starting GFR value for hemodialysis:

A. 50% of the normal GFR
B. 35% of the normal GFR
C. 23% of the normal GFR
D. 8% of the normal GFR
E. 2% of the normal GFR

29. 1.29. Pathophysiology of liver – Beata Keremi

Liver is the largest internal organ of the human body. It weighs 1.0–1.5 kg. It plays a major role in metabolism, functions include the synthesis of glucose (gluconeogenesis), bile acids, several serum proteins and coagulation factors. Liver is responsible for the breakdown and elimination of medications and hormones.

15% of cardiac output passes through the liver. It has a dual blood supply. 20% of its blood supply is provided by the hepatic artery, carrying arterial blood. On the other hand, hepatic portal veins account for 80% of the blood flow to the liver, carrying venous blood rich in nutrients from the gastrointestinal tract, and from the spleen. Blood from both the hepatic artery and from the portal vein flows into the sinusoids and reaches the hepatocytes which are arranged in single-cell thick plates separated by the sinusoids. Kupffer cells in the
sinusoids can remove old or defective red blood cells by phagocytosis. They also phagocytose enteral bacteria and other foreign substances, thus removing harmful substances from the blood.

**Figure 1.377. Figure 1. – Structure of a hepatic lobule**

![Structure of a hepatic lobule](image)

### 29.1. Major functions of the liver

**Figure 1.378. Table 1. – Main functions of the liver.** Mixed function microsomal oxidase plays a major role in detoxification. Glucuronate, glycine, taurine, sulphate and acetyl can be conjugating groups.
Figure 1.379. Figure 2. – Glucose metabolism in the liver. Liver stores excess glucose as glycogen. When fasting or during increased need it can generate glucose from amino acids, glycerol or lactate. It can convert excess carbohydrates to triglycerides, which will be then stored by adipose tissue.

<table>
<thead>
<tr>
<th>Table 1: Main functions of the liver</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A) Synthetic function</strong></td>
</tr>
<tr>
<td>➢ Protein synthesis (See details separately)</td>
</tr>
<tr>
<td>- albumin</td>
</tr>
<tr>
<td>- clotting factors</td>
</tr>
<tr>
<td>- transport proteins</td>
</tr>
<tr>
<td>- plasma proteins</td>
</tr>
<tr>
<td>- choline esterase</td>
</tr>
<tr>
<td>➢ Synthesis of bile acids (600-1200 mL/day)</td>
</tr>
<tr>
<td>➢ Regulation of energy metabolism</td>
</tr>
<tr>
<td>- gluconeogenesis (from amino acids, lactic acid, glycerol)</td>
</tr>
<tr>
<td>➢ Lipid metabolism</td>
</tr>
<tr>
<td>- lipoprotein synthesis</td>
</tr>
<tr>
<td>- synthesis of phospholipids</td>
</tr>
<tr>
<td>- formation of triglycerides from carbohydrates and proteins</td>
</tr>
<tr>
<td>- cholesterol production, recycling and removal</td>
</tr>
<tr>
<td><strong>B) Detoxification, oxidation, conjugation</strong></td>
</tr>
<tr>
<td>➢ Elimination of endogenous compounds</td>
</tr>
<tr>
<td>- steroid hormones – oxidation, conjugation</td>
</tr>
<tr>
<td>- sex hormones</td>
</tr>
<tr>
<td>- glucocorticoids</td>
</tr>
<tr>
<td>- aldosterone</td>
</tr>
<tr>
<td>- bilirubin – oxidation, glucuronide binding</td>
</tr>
<tr>
<td>- ammonia elimination – conversion to urea</td>
</tr>
<tr>
<td>➢ Detoxification of exogenous compounds</td>
</tr>
<tr>
<td>- elimination of drugs, eg. barbiturates</td>
</tr>
<tr>
<td><strong>C) Storage function</strong></td>
</tr>
<tr>
<td>➢ Glycogen</td>
</tr>
<tr>
<td>➢ Lipids</td>
</tr>
<tr>
<td>➢ Minerals</td>
</tr>
<tr>
<td>- iron, copper</td>
</tr>
<tr>
<td>➢ Vitamins – dissolved in lipid</td>
</tr>
<tr>
<td>➢ Blood</td>
</tr>
<tr>
<td><strong>D) One of the regulators of salt and water metabolism</strong></td>
</tr>
<tr>
<td>➢ Impaired function: sodium retention and hypokalaemia</td>
</tr>
<tr>
<td><strong>E) Elimination of bacteria</strong></td>
</tr>
</tbody>
</table>
Figure 1.380. Figure 3. – Protein metabolism in the liver. Transamination and deamination are the two major reactions used to interconvert amino acids. Transamination is the transfer of an amino group (NH$_2$) to an acceptor molecule. Thus amino acids can participate in the intermediate metabolism of carbohydrates and lipids. Most non-essential amino acids are synthesized in the liver by this mechanism. These biochemical reactions are catalyzed by aminotransferases. Deamination eliminates the amino group from an amino acid, converting it to keto acid and ammonia. Ammonia resulting from deamination is converted to urea in the liver and is later excreted in the kidney.

Figure 1.381. Figure 4. – Fat metabolism in the liver
1. Oral biology

Major proteins synthesized by the liver. “Identical” hepatocytes synthesize different proteins according to how far they are located from the capillaries of the hepatic artery and the portal vein. Hepatocytes that receive well oxygenated blood participate in energy consuming protein synthesis.

<table>
<thead>
<tr>
<th>Most important proteins produced by the liver</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumin</td>
</tr>
<tr>
<td>Alpha fetoprotein</td>
</tr>
<tr>
<td>Coagulation factors: II, V, VII, IX, X</td>
</tr>
<tr>
<td>Fibrinogen, haptoglobin, hemopexin</td>
</tr>
<tr>
<td>Prothrombin</td>
</tr>
<tr>
<td>Antithrombins (antithrombin III)</td>
</tr>
<tr>
<td>Hormone binding proteins (thyroxine, steroid)</td>
</tr>
<tr>
<td>Apolipoproteins</td>
</tr>
<tr>
<td>Transport proteins, enzymes: transferrin, ceruloplasmin</td>
</tr>
<tr>
<td>Angiotensinogen</td>
</tr>
<tr>
<td>Growth factors</td>
</tr>
<tr>
<td>Enzymes</td>
</tr>
</tbody>
</table>

29.2. Bilirubin metabolism

Bilirubin metabolism. Bilirubin is rapidly converted to free bilirubin. Free bilirubin is insoluble in plasma and is therefore transported bound to albumin. While passing through the liver, it dissociates from albumin, enters hepatocytes and is conjugated. Conjugated bilirubin is dissolved in bile and is secreted to the small intestine. There, bacteria convert bilirubin to urobilinogen, a highly water soluble compound.
Icteruses develop due to abnormally high, over 2 to 2.5 mg/dL serum bilirubin levels. One of the first symptoms is a yellow sclera, this is explained by the fact that bilirubin has a specific affinity to tissues containing high amounts of elastic fibers.

Figure 1.385. Table 4. – Most common causes of jaundice
29.4. Forms of hepatitis

Primary forms:

- hepatotropic viruses (HAV, HBV, HCV, HDV, HEV).

Secondary forms:

- autoimmune diseases,
- reactions to medications or toxins,
- infectious (mostly tropical) diseases.

29.4.1. Viral forms of hepatitis

<table>
<thead>
<tr>
<th>Table 4. Most common causes of jaundice:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prehepatic:</strong> <strong>(Excessive red blood cell destruction)</strong></td>
</tr>
<tr>
<td>Hemolysis</td>
</tr>
<tr>
<td>Hematoma</td>
</tr>
<tr>
<td>Hereditary red blood cell disorders</td>
</tr>
<tr>
<td>Acquired hemolytic disorders</td>
</tr>
<tr>
<td>Autoimmune hemolytic anemia</td>
</tr>
<tr>
<td>Icterus neonatorum</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Intrahepatic:</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital transport disorders (Gilbert’s disease)</td>
</tr>
<tr>
<td><strong>Decreased bilirubin uptake in the liver</strong></td>
</tr>
<tr>
<td><strong>Decreased conjugation of bilirubin</strong> (inhibition of glucuronyl-transferase)</td>
</tr>
<tr>
<td>Disruption of liver cells</td>
</tr>
<tr>
<td>Hepatitis</td>
</tr>
<tr>
<td>Cirrhosis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Posthepatic:</strong> <strong>(Obstruction of bile flow)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Structural bile duct disorders</td>
</tr>
<tr>
<td>Blockage of the bile ducts</td>
</tr>
<tr>
<td>(gall stone, tumor, inflammations)</td>
</tr>
</tbody>
</table>

The 4 most common causes are highlighted by **bold** letters.

**Figure 1.386. Table 5. – Change in bilirubin levels in different types of jaundice**

<table>
<thead>
<tr>
<th>Table 5. Change in bilirubin levels in different types of jaundice:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type:</strong></td>
</tr>
<tr>
<td>Typical cause:</td>
</tr>
<tr>
<td>Serum indirect bilirubin:</td>
</tr>
<tr>
<td>Serum direct bilirubin:</td>
</tr>
<tr>
<td>Urine direct bilirubin:</td>
</tr>
<tr>
<td>Urine uric acid:</td>
</tr>
<tr>
<td>Feces pigment:</td>
</tr>
<tr>
<td>Feces fat:</td>
</tr>
</tbody>
</table>

*: slight increase; ** remarkable increase
Hepatitis viruses differ in their route of transmission, incubation period, patomechanism, degree of inflammation, chronicity (duration) of damage and the time required to become a carrier.

The two main routes of liver damage in viral forms of hepatitis are:

• direct cellular damage,
• induction of an immune response against viral antigens.

The clinical presentation of liver damage may vary widely from asymptomatic infection to acute hepatitis, carrier status or chronic hepatitis, which can lead to cirrhosis or rapid-onset liver failure.

The course of acute hepatitis can be divided into three phases:

• prodromal or preicterus period,
• icterus period,
• healing period.

**Figure 1.387. Figure 6. – Structure of the Hepatitis B virus**

**Figure 1.388. Figure 7. – Pathogenesis of HBV**
29.4.2. Chronic hepatitis

A chronic inflammatory reaction in the liver which lasts for over 3 to 6 months. A chronic increased level of serum aminotransferase is characteristic. It usually develops in HBV, HCV and HDV infections, autoimmune and drug-induced hepatitis.
29.5. Cirrhosis

Figure 1.390. Figure 8. – Alterations in the space of Disse during liver cirrhosis

Cirrhosis is an irreversible, progressive process. It leads to the death of some hepatocytes, and to the inactivation of others. As a result of inactivation, the single-cell thick plate arrangement of hepatocytes is disrupted and instead they pile up on one another, shrinking down the space of Disse. Characteristic symptoms of cirrhosis are initial hepatomegaly and jaundice. Cirrhosis may co-occur with metabolic diseases wherein minerals may be deposited in the liver. The two most important such conditions are haemochromatosis (iron deposits) and Wilson’s disease (copper deposits).

Figure 1.391. Figure 9. – Development of liver cirrhosis

Cirrhosis results in decreased liver functions. For example, increased testosterone breakdown in men leads to a relative increase in female sexual hormones and a resultant feminization. Furthermore, portal hypertension and ascites develop. The late manifestation of cirrhosis is related to portal hypertension and hepatocyte dysfunction. The end stage of of cirrhosis is aggravated by further complications such as hemorrhages due to the lack of clotting factors and thrombocytopenia due to splenomegaly.

The development of reversible pathological alterations, such as fibrosis and fatty liver, precedes that of the irreversible cirrhosis.

29.6. Portal hypertension

In portal hypertension the vascular resistance increases in the portal vein system, leading to an increase of blood pressure to above 12 mmHg, the normal pressure in the portal circulation being 5 to 10 mmHg.

Reasons behind the development of portal hypertension:
• increased vascular resistance in the hepatic circulation,
• pre, intra or posthepatic obstruction.

**Figure 1.392. Table 7. – Causes of portal hypertension**

<table>
<thead>
<tr>
<th>Prehepatic causes:</th>
<th>Varicose veins</th>
<th>Ascites</th>
</tr>
</thead>
<tbody>
<tr>
<td>thrombosis of portal vein</td>
<td>often</td>
<td>rare</td>
</tr>
<tr>
<td>external compression of portal vein</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- tumor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- enlarged lymph nodes</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Intrahepatic causes:**

| narrowing/obstruction of blood flow in the liver | often | rare |
| alcoholic cirrhosis – structural remodeling in liver fibrosis | | |

**Posthepatic causes:**

| increased capillary pressure | |

- obstructed venous flow
- salt and water retention in the kidney:
- Decreased blood volume *(underfill theory)*
  - contraction in the effective blood volume → afferent signal → → salt and water retention in the kidney
  - loss of fluids into the peritoneal cavity
  - vasoconstriction due to the effects of circulating vasoconstricting substances

- Excess blood volume *(overfill theory)*
  - initial event: salt and water retention in the kidney
  - damage of aldosterone metabolism in the kidney

| decreased colloidal osmotic pressure | |

- decreased albumin synthesis

**29.6.1. Ascites**

Ascites develops when fluid builds up in peritoneal cavity due to increased lymph production by the liver. It usually occurs in late stages of liver cirrhosis and portal hypertension.

**Figure 1.393. Table 8. – Mechanisms of ascites development**

| increased capillary pressure | |

- obstructed venous flow
- salt and water retention in the kidney:
- Decreased blood volume *(underfill theory)*
  - contraction in the effective blood volume → afferent signal → → salt and water retention in the kidney
  - loss of fluids into the peritoneal cavity
  - vasoconstrictor due to the effect of circulating vasoconstricting substances

- Excess blood volume *(overfill theory)*
  - initial event: salt and water retention in the kidney
  - damage of aldosterone metabolism in the kidney

| decreased colloidal osmotic pressure | |

- decreased albumin synthesis

**Figure 1.394. Figure 10. – Development of ascites in cirrhosis**
Bacterial peritonitis is a frequent consequence of ascites. Stagnant peritoneal fluid is a fertile field for bacterial superinfection.

**29.6.2. Portocaval shunt**

Portal hypertension opens a connection between portal and systemic circulation due to the dilation of collateral veins.

- esophagus – esophagal varices,
- rectal veins,
- umbilical veins – caput medusae,
- portopulmonal shunt – blood oxygenization decreases due to mixing → cyanosis.

**Figure 1.395. Figure 11. – Consequences of portal hypertension**

**29.7. Liver failure**

Rapidly occurring, massive and progressive liver destruction. Characteristic of acute hepatitis and liver cirrhosis. 80 to 90% of the initial liver function is lost before the occurrence of liver failure.
Figure 1.396. Table 9. – Consequences of liver failure. A note on ammonia, the most important neurotoxin: under normal conditions, ammonium ion diffuses into the portal blood and is transported to the liver where it is converted to urea before getting to the systemic circulation

<table>
<thead>
<tr>
<th>Table 9. Consequences of liver failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematologic disorders:</td>
</tr>
<tr>
<td>&gt; anaemia</td>
</tr>
<tr>
<td>Causes:</td>
</tr>
<tr>
<td>▶ blood loss — excessive cell destructoton and impaired red blood cell formation</td>
</tr>
<tr>
<td>▶ folic acid deficiency—megalooblasticanaemia</td>
</tr>
<tr>
<td>▶ haemolysis — change in lipid components of red blood cell membrane</td>
</tr>
<tr>
<td>&gt; bleeding disorders (coagulation defects)</td>
</tr>
<tr>
<td>Causes:</td>
</tr>
<tr>
<td>▶ malabsorption of fat-soluble vitaminK → impaired synthesis of clotting factors</td>
</tr>
<tr>
<td>&gt; thrombocytopenia</td>
</tr>
<tr>
<td>&gt; leukopenia</td>
</tr>
</tbody>
</table>

| Endocrine disorders:                |
| > functional disturbances of gonads |
| Causes:                             |
| ▶ accompanying symptoms of cirrhosis and liver failure |
| > decreased aldosterone metabolism  |
| Causes:                             |
| ▶ increasedNa⁺ and water retention in the kidney |
| ▶ increasedK⁺ elimination → decreased serum K⁺ concentration |

| Skin disorders:                    |
| > telangiectases or spider nevi    |
| > palmar erythema                  |
| > jaundice                         |
| Hepatorenal syndrome:              |
| > progressive azoemia              |
| > elevated serum creatinine level  |
| > oliguria                         |
| Typical in terminal stages of liver failure with ascites |

Hepatic encephalopathy

✓ appears as a consequence of liver failure in the entire central nervous system
✓ neural disturbances – from lack of mental alertness to confusion, coma and convulsion
✓ accumulation of neurotoxins – decreased detoxification function of liver
- most important neurotoxin is ammonia

29.8. Liver tumors

Primary tumors rarely occur.

Figure 1.397. Table 10. – Types of primary liver cancer

<table>
<thead>
<tr>
<th>Table 10. Types of primary liver cancer:</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; hepatocellular carcinoma</td>
</tr>
<tr>
<td>- strongly related to HBV infection, but typically linked to cirrhosis in the western world</td>
</tr>
<tr>
<td>- aflatoxin poisoning</td>
</tr>
<tr>
<td>- arsenic poisoning</td>
</tr>
<tr>
<td>&gt; cholangiocarcinoma — primary tumor of bile duct cells</td>
</tr>
<tr>
<td>- less frequent than hepatocellular carcinoma</td>
</tr>
<tr>
<td>- typically not related to known risk factors, but long-time injury or inflammation of the epithelium may be a risk factor</td>
</tr>
</tbody>
</table>

Liver tumors typically occur as metastatic carcinomas. Tumors that metastasize to the liver are colorectal cancer, tumors of the breast, lung and the urogenital tract, and neuroendocrine tumors.

29.9. Pathomechanisms of liver diseases

Figure 1.398. Table 11. – Mechanisms that lead to the development of liver diseases
Liver function tests involve the measurement of enzymes released into the circulation as a result of hepatocyte destruction (aspartate aminotransferase, ASAT; alanine aminotransferase, ALAT; gamma glutamyl transferase, GGT), and of enzymes whose levels increase in response to bile duct obstruction (alkaline phosphatase).

ALAT is liver specific, while ASAT may also derive from organs other than the liver. Alkaline phosphatase, produced by bile duct epithelial cells and hepatocyte canalicular membrane, is discharged with bile. GGT plays a role in the transport of amino acids and peptides to hepatocytes. In addition, it is useful for the diagnosis of alcohol problems.

### Table 11. Mechanisms that lead to the development of liver diseases

<table>
<thead>
<tr>
<th>1. Edematous swelling of hepatocytes (inflammation)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Consequences:</strong></td>
</tr>
<tr>
<td>1. Decreased nutrient and oxygen uptake</td>
</tr>
<tr>
<td>2. Compressed bile canaliculi</td>
</tr>
<tr>
<td>3. Compressed diameter of sinusoids (elevated circulatory resistance)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>2. Hypoxia of hepatocytes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Consequences:</strong></td>
</tr>
<tr>
<td>1. Decreased energy production</td>
</tr>
<tr>
<td>2. Metabolic disorders</td>
</tr>
<tr>
<td>3. Increased permeability</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>3. Metabolic disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Consequences:</strong></td>
</tr>
<tr>
<td>1. Lack of endproducts</td>
</tr>
<tr>
<td>2. Accumulation of toxic intermediates</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>4. Increased permeability of hepatocytes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Consequences:</strong></td>
</tr>
<tr>
<td>1. Release of small molecules and proteins from cytoplasm</td>
</tr>
<tr>
<td>2. Disruption of hepatocytes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>5. Disruption of hepatocytes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Consequences:</strong></td>
</tr>
<tr>
<td>1. Reduced number of active cells (decreased synthetic and conjugating function)</td>
</tr>
<tr>
<td>2. Toxic substances accumulate (toxaemia)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6. Hepatic enzyme disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Consequences:</strong></td>
</tr>
<tr>
<td>1. Metabolism and energy production disorders</td>
</tr>
<tr>
<td>2. Decreased conjugation and detoxification</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>7. Obstruction or compression of bile capillaries</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Consequences:</strong></td>
</tr>
<tr>
<td>1. Increased intraductal pressure →</td>
</tr>
<tr>
<td>2. Increased passage of bile components to the blood</td>
</tr>
<tr>
<td>2. Decreased fat absorption</td>
</tr>
<tr>
<td>3. Retrograde hepatic damages</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>8. Increased storage function</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Consequences:</strong></td>
</tr>
<tr>
<td>1. Lipid accumulation in the liver</td>
</tr>
</tbody>
</table>

### 29.10. Liver function problems

Liver function tests involve the measurement of enzymes released into the circulation as a result of hepatocyte destruction (aspartate aminotransferase, ASAT; alanine aminotransferase, ALAT; gamma glutamyl transferase, GGT), and of enzymes whose levels increase in response to bile duct obstruction (alkaline phosphatase).

Blood clotting problems can occur when the synthesizing function of liver is damaged, because the elimination of endogenous compounds does not occur properly.

1. Blood clotting problems can occur when the synthesizing function of liver is damaged, because the elimination of endogenous compounds does not occur properly.

A. true-true, with connection
B. true-true, without connection
C. true-false
D. false-true
E. false-false
2. Which statement is true for the formation of conjugated bilirubin?

A. conjugated bilirubin binds to albumin
B. conjugation occurs in the serum
C. glucuronidation takes place
D. conjugated bilirubin is formed by breakdown of hemoglobin in the bone marrow or spleen
E. formation of conjugated bilirubin is increased in prehepatic jaundice

3. Which hepatitis virus can infect in the presence of another virus only?

A. HAV
B. HBV
C. HCV
D. HDV
E. HEV

Literatur


S. Silbernagl: Jaundice; Portal hypertension; Fibrosis and cirrhosis of the liver; Liver failure Ch. 6. Stomach, intestines, liver, pp 168-175. in: S. Silbernagl and F. Lang: Color atlas of Pathophysiology, Thieme, 2000


30. 1.30. Cardiac insufficiency and shock – oral aspects – Kristof Kadar

30.1. Heart failure (central circulatory failure)

Heart failure is a clinical syndrome characterized by the progressive failure of pump function of the heart, resulting in complex pathophysiological changes at a cellular, tissue, and systemic level. These processes lead to (further) myocardial cell damage, arrhythmias and finally death.

The healthy heart adapts in a wide range to a physiological and pathophysiological demand for increased blood flow in the body. In heart failure this ability is restricted and the heart may be unable to provide sufficient blood supply even in a resting state.

Figure 1.399. Figure 1. – Adaptation of the heart to the increased workload. The normal heart adapts to the increased workload through increasing contractility. In heart failure, the lack of contractility reserve results in increased EDV with the increased ventricular performance
Heart failure results in a decreased cardiac output. The observed symptoms are the consequence of (1) the congestion of the systemic/pulmonary vasculature (backward failure) (2) the inadequate blood supply of the systemic/pulmonary circulation (forward failure) (3) the compensation mechanism resulting from the decreased cardiac output.

Although left-sided or right-sided heart failure may initially occur isolated, both side will be damaged on the long term (interdependence), with a mixed presentation of symptoms (although symptom severity of the respective side may vary depending on the initiating event).

### 30.1.1. Symptoms of heart failure

Left side forward failure:

- decreased blood supply of the muscles → weakness, fatigue
- decreased renal blood supply → decreased urinary excretion in the daytime, circulatory redistribution by night (lying position) results in increased excretion → *nycturia* (frequent urination by night)
- decreased blood supply of the GI tract → hypoxia of the intestinal villi → absorption problems, *malabsorption*
- decreased skin circulation → decreased heat dissipation → *sweating*

Left heart backward failure:

- Pulmonary capillary pressure ↑ → interstitial oedema → *dyspnoe*, increasing with circulatory redistribution in lying position → *orthopnoe*, *paroxysmal nocturnal dyspnoe* in severe casesinterstitial pulmonary edema → leading to *alveolar pulmonary oedema* → hypoxia, hypercapnia

Right heart forward failure:

- decreased left ventricular filling → left ventricular (left heart) forward failure

Right heart backward failure:

- Venous congestion in the peripheral tissues → *peripheral oedema*, especially in the lower part of the body (ie. swelling of the legs), *hydrothorax, congestive hepatomegaly, ascites*

The symptoms of heart failure may manifest:

- As a consequence of temporary/sustained decrease of heart performance (cardiac output)
- As a consequence of temporary/sustained increase of peripheral demand
or the combination of thereof.

30.1.2. The underlying causes of heart failure

- Myocardial ischemia (acute/chronic),
- hypertension,
- valve problems,
- cardiomyopathy,
- other causes.

30.1.3. Aggravating factors in heart failure:

With increased workload:

- increased cardiac output demand:
  - increased metabolic demand (hyperthyroidism, fever etc.)
  - increased volume load (renal failure, high sodium intake)
- pressure overload (increased resistance in the outflow tract):
  - hypertension
  - pulmonary embolism.

With temporarily decreased ventricular performance:

- ischemia,
- decreased efficiency (arrhythmias),
- drug effect,
- inflammations (myocarditis, endocarditis).

30.1.4. The basic pathomechanisms of heart failure

Decreased inotropy (systolic dysfunction).

Decreased diastolic relaxation (diastolic dysfunction).

Figure 1.400. Figure 2. – Pressure-volume relationship in normal heart and in heart failure. (A) pressure-volume relationship in normal heart at a resting state (B) adaptation to physical exercise (black line – resting state; red line – physical exercise); effect of (C) decreased contractility (red line) and (D) decreased ventricular relaxation (red line) on the pressure-volume relationship. EDV: end-diastolic volume; ESV: end-systolic volume; P-V: pressure-volume
30.1.5. Neurohormonal response

Decreased cardiac output triggers a complex set of compensatory mechanisms.

Even though these mechanisms lead to short term favourable changes such as increased cardiac output (adaptive effects), on the long run they contribute to the progression of the disease and have adverse effects on survival (maladaptive effects).

**Figure 1.401. Figure 3. – Neurohormonal activation**

<table>
<thead>
<tr>
<th>Neurohormonal activation</th>
</tr>
</thead>
<tbody>
<tr>
<td>hemodynamic defense reaction</td>
</tr>
<tr>
<td>- salt and water retention</td>
</tr>
<tr>
<td>- vasoconstriction</td>
</tr>
<tr>
<td>- increased cardiac stimulation</td>
</tr>
<tr>
<td>inflammatory response</td>
</tr>
<tr>
<td>hypertrophic response</td>
</tr>
</tbody>
</table>

**Figure 1.402. Figure 4. – Hemodynamic defense reaction**
1. Oral biology

Hemodynamic defense reaction

- **Effect**
  - salt and water retention (ADH, aldosterone)
  - vasoconstriction (catecholamines, angiotensin II)
  - cardiac stimulation; contractility↑, faster relaxation, frequency↑ (catecholamines)
  - cell growth and proliferation (endothelin, angiotensin II)

- **Mediator**
  - catecholamines (central and peripheral effect)
  - angiotensin II
  - ADH
  - endothelin
  - ANP
  - NO
  - bradykinin
  - dopamine

**Figure 1.403. Figure 5.**

<table>
<thead>
<tr>
<th></th>
<th>Effect</th>
<th>adaptive (short term) consequences</th>
<th>maladaptive (long term) consequences</th>
</tr>
</thead>
<tbody>
<tr>
<td>salt and water retention</td>
<td>preload ↑</td>
<td>cardiac output ↑</td>
<td>peripheral oedema pulmonary oedema</td>
</tr>
<tr>
<td>vasoconstriction</td>
<td>afterload ↑</td>
<td>blood pressure ↑</td>
<td>cardiac output ↓ energy demand ↑</td>
</tr>
<tr>
<td>cardiac stimulation</td>
<td>contractility ↑</td>
<td>relaxation ↑</td>
<td>energy demand ↑ arrhythmias sudden death</td>
</tr>
</tbody>
</table>

**Figure 1.404. Figure 6. – Inflammatory response in heart failure**

**Inflammatory response in heart failure**

- **adaptive, short term consequences**
  - unknown (heat shock proteins?)

- **maladaptive, long term consequences**
  - cardiac cachexia
  - apoptosis
  - necrosis

**Figure 1.405. Figure 7. – Hypertrophic response in heart failure**

**Hypertrophic response in heart failure**

- **causes of gene expression changes in myocardial cell**
  - mechanical stress
  - hemodynamic-defense reaction
  - inflammatory response

- **growth factor changes**: TGF-β, IGF-1, FGF-β

  - adaptive hypertrophy
  - maladaptive hypertrophy

  - remodeling
  - energy consumption
  - cell death

- **depolarization abnormalities**: arrhythmias sudden cardiac death

**30.2. Circulatory shock (peripheral circulatory failure)**

Circulatory shock is a pathologic state of the regulation of systemic circulation, caused by and leading to a generalized tissue and organ level hypoxia, resulting in systemic damage. This is a self-sustaining process (positive feedback, “vicious circle”).
30.2.1. Types of shock

**Hypovolemic shock** – severe blood and/or fluid loss.

**Cardiogenic shock** – acute failure of the cardiac pump function.

**Distributive shocks** – acute change in the distribution of blood (ie. acute systemic vasodilation, sudden increase of vascular permeability) resulting from acute deaferentation (neurogenic shock), endotoxin effect (septic shock) or hypersensitivity reaction (anaphylactic shock).

**Obstructive shocks** – extravascular (ie. pericardial tamponade, pneumothorax) or intravascular obstruction (ie. pulmonary embolism) resulting in seriously decreased ventricular filling – severe decrease of cardiac output.

**Traumatic shock** – mixed form of shock, with hypovolemic and septic components.

**Burn shock** - mixed form of shock, with hypovolemic and occasionally septic components.

Figure 1.406. Figure 8. – Causes of hypovolemic shock

<table>
<thead>
<tr>
<th>Causes of hypovolemic shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood loss</td>
</tr>
<tr>
<td>- trauma, surgery, aneurysm rupture</td>
</tr>
<tr>
<td>- haemorrhage, haematoma</td>
</tr>
<tr>
<td>- haemophilia, anticoagulants, thrombolytics</td>
</tr>
<tr>
<td>Excessive fluid loss from GI tract</td>
</tr>
<tr>
<td>- vomiting, diarrhea - especially infants and children</td>
</tr>
<tr>
<td>Fluid loss through the urinary tract</td>
</tr>
<tr>
<td>- diabetes insipidus, diabetes mellitus, salt-wasting disorders, adrenocortical insufficiency, diuretics</td>
</tr>
<tr>
<td>Fluid loss through injured skin</td>
</tr>
<tr>
<td>- excessive burn, skin inflammation (generalized exfoliative dermatitis)</td>
</tr>
<tr>
<td>Internal sequestration of fluid</td>
</tr>
<tr>
<td>- Loss of volume into the interstitial space or body cavities</td>
</tr>
<tr>
<td>• Chronic liver disease (ascites), acute pancreatitis, angioedema</td>
</tr>
</tbody>
</table>

Figure 1.407. Figure 9. – Cardiogenic shock

<table>
<thead>
<tr>
<th>Cardiogenic shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute heart (pump) failure</td>
</tr>
<tr>
<td>• myocardial infarction</td>
</tr>
<tr>
<td>• asthma cardiac</td>
</tr>
<tr>
<td>• severe acidosis</td>
</tr>
<tr>
<td>• barbiturate intoxication</td>
</tr>
<tr>
<td>• effect of (end)toxias in septic shock</td>
</tr>
<tr>
<td>• acute valvular failure</td>
</tr>
<tr>
<td>• ruptured septum</td>
</tr>
</tbody>
</table>

Figure 1.408. Figure 10. – Distributive shocks

<table>
<thead>
<tr>
<th>Distributive shocks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurogenic shock</td>
</tr>
<tr>
<td>• spinal cord injury, toxic or drug effect, affecting the ability of the CNS to maintain the vasal tone → vasodilation</td>
</tr>
<tr>
<td>Anaphylactic shock</td>
</tr>
<tr>
<td>• generalized anaphylactic reaction, resulting in peripheral vasodilation and highly increased permeability → generalized oedema, intravascular fluid loss</td>
</tr>
<tr>
<td>Septic shock</td>
</tr>
<tr>
<td>• Generalized inflammatory reaction resulting from a systemic infection leading to generalized vasodilation</td>
</tr>
</tbody>
</table>
1. Oral biology

30.2.2. Pathomechanism of shock

The stages of shock are presented here through the development of hypovolemic shock.

Compensated stage:

Inadequate tissue perfusion induces compensatory mechanisms. These mechanisms are intended to maintain cardiac output and blood pressure to provide adequate tissue perfusion of vital organs (brain, heart) at the price of decreased perfusion of other organs („centralization” of the circulation).

As the probably occurring shock in the dental chair, anaphylactic shock is the most important form of shock in dentistry.

Figure 1.409. Figure 11. – Traumatic shock

<table>
<thead>
<tr>
<th>Traumatic shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed shock form, initially with hypovolemic mechanism, later may also include distributive shock components as a result of systemic inflammatory response.</td>
</tr>
</tbody>
</table>

- direct tissue injury \(\downarrow\) inflammatory reaction
- blood loss \(\downarrow\) hypovolemic shock
- tissue hypoxia \(\downarrow\) tissue injury
- infection \(\downarrow\) inflammatory reaction
- sepsis

Figure 1.410. Figure 12. – Compensatory mechanisms in hypovolemic shock

<table>
<thead>
<tr>
<th>Compensatory mechanisms in hypovolemic shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>decreased blood volume (\rightarrow) sympathetic nervous system activation (\rightarrow) adrenaline/noradrenaline release</td>
</tr>
</tbody>
</table>

- baroreceptor activation
- medullary ischemic reflex
- \(\beta_1\)-receptor activation: cardiac frequency \(\uparrow\), cardiac output \(\uparrow\)
- \(\beta_2\)-receptor activation: gluconeogenesis, glycolysis \(\uparrow\)
- \(\alpha\)-receptor activation: vasoconstriction (kidney, splanchnic, muscle, fat tissue)
- cortisol \(\uparrow\)
- renin-angiotensin-aldosterone system (RAAS) activation
- ADH/vasopressin \(\uparrow\)

As a result:

- maintained perfusion of brain and heart, blood pressure slightly decreased (70-90 Hgmm), stroke volume \(\uparrow\)
- decreased perfusion in other organs and tissues \(\rightarrow\) TPR \(\uparrow\)
  - skin – pale
  - kidney – decreasing urinary output
  - muscle – weakness
- metabolic changes:
  - cortisol \(\rightarrow\) antinsulin effect: gluconeogenesis, glycolysis \(\uparrow\), lipolysis \(\uparrow\) \(\rightarrow\) triglycerides, free fatty acids \(\uparrow\)
- salt and water retention (RAAS, cortisol, ADH), volumen \(\uparrow\)

Progressive stage:
Despite the compensatory mechanisms, tissue perfusion becomes inadequate and the redistribution of circulation (centralization) results in systemic microcirculatory dysfunction. Local hypoxia and the resulting acidosis and cellular damage lead to endothelial dysfunction and injury. This results in swelling of endothelial cells, intracapillary activation of the coagulation cascade (thrombus formation) and an inflammatory response, further increasing the microcirculatory dysfunction. Similar positive feedback pathophysiological loops ("vicious cycles"), occurring both at a tissue and a systemic level, contribute to progressive dysfunction and eventually to shock decompensation (see details on figure below). Tissue hypoxia and the local accumulation of metabolites will open precapillary arterioles and shunts, further decreasing tissue supply.

**Figure 1.411. Figure 13.**

![Diagram of pathophysiological loops](image)

**Irreversible stage:**

Toxic metabolites accumulated in hypoxic tissues get into the systemic circulation. The local accumulation of cytotoxic mediators due to shunt circulation, capillary and postcapillary thrombosis, increased capillary permeability and tissue hypoxia will result in tissue damage and a simultaneous failure of multiple vital organs (MODS – multi-organ dysfunction syndrome, MOF – multi-organ failure), eventually leading to death.

The first stage of distributive shocks differs from that described above. Here tissue hypoxia is the result of shunt circulation and **relative hypovolemia** (low blood volume compared to the volume of the vascular system), both caused by vasodilation. In contrast to hypovolemic shock, here cardiac output may even be higher than normal initially (**hyperdynamic stage**) with increased skin circulation in certain forms (e.g. septic shock). Subsequently, intravascular hypovolemia resulting from increased capillary permeability and tissue hypoxia as a consequence of shunt circulation will lead to pathophysiological changes as described for the hypovolemic shock above.

**Figure 1.412. Figure 14. – Hyperdynamic phase of septic shock**
30.2.3. Organ damage in shock – shock organs

Although the severity and pace of tissue/organ damage differ during shock progression, mainly due to the difference in organ perfusion, all organs and tissues are involved except the brain (until the very last stages).

The damage of certain organs (so called shock organs) dramatically influence survival. These include the gastrointestinal tract (guts), pancreas, lungs, kidneys and the fat tissue.

Figure 1.413. Figure 15. – Pathological changes of the lung is shock

Figure 1.414. Figure 16. – Pathological changes of the GI tract is shock

Figure 1.415. Figure 17. – Pathological changes of the kidney is shock
1. Oral biology

Pathological changes of the kidney in shock
- secondary shock organ, component of MOF
  - vasoconstriction
  - decreased ability to concentrate urine
  - decreased urinary output (ADH, aldosterone)

Acute (prerenal) kidney failure!
- hypoxia of the tubules → acute tubular necrosis, anuria, uraemia → dialysis required
- if the tubular damage is reversible, hypertensive polyuria during the healing process (polyuric phase) as the glomerular function is restored first

Figure 1.416. Figure 18. – Role of fat tissue in shock

Role of fat tissue in shock
- the mortality of shock is higher in obese patients!
- usually worse cardiovascular status
- centralised circulation in shock → decreased fat tissue perfusion
  - anaerobic metabolism
  - cell death → lactic acid, proteases
- large mass → many mediators and active immune cells released into capillaries in the late stages of shock

30.3. Test – Central and peripheral circulatory failure (answers)

1. When cardiac muscle inotropy is decreased:
   A. the stroke volume is decreased
   B. the end-systolic volume is decreased
   C. the contractility is increased
   D. the end-diastolic volume is decreased
   E. the afterload is increased

2. Symptoms of left ventricular forward failure include:
   A. nycturia
   B. orthopnoe
   C. fatigue, weakness
   D. peripheral oedema
   E. hepatomegaly

3. In the initial phase of hypovolemic shock there is a generalized vasodilation (A), and the resulting centralization of the circulation leads to a generalized tissue hypoxia in most of the organs (B). Right answer: d)
   A. true-true, with connection
   B. true-true, without connection
   C. true-false
   D. false-true
31. 1.31. Oral aspects of hypertension – Gabor Varga

Hypertension is frequently called the „silent killer‖. Without pain or warning signs, the rather aspecific symptoms of the disease would not alarm the patient to visit a doctor. However, hypertension and its complications are among the most frequent causes of mortality.

In healthy people diastolic and systolic pressure values vary in a wide range. Since blood pressure increases with age, higher values in young age predict hypertension in older ages.

**Figure 1.417. Figure 1. – Blood pressure: population level distribution**

Mean arterial pressure (MAP) is determined by the cardiac output (CO) and the total peripheral resistance (TPR) and can be expressed as their product. TPR can be calculated from the combination of the Hagen-Poiseuille equation and Ohm’s Law. Since vessel radius is on the fourth power in the equation, this clearly shows that constriction and dilation of the vessels are the most significant determinants of blood pressure.

**Figure 1.418. Figure 2. – Physiological regulation of BP**
The increase of blood pressure may result from the increase of TPR, CO or both. Cardiac output is the product of heart frequency and stroke volume, the latter being determined by circulating blood volume and the vascular tone of capacity veins, while the former by heart contractility.

**Figure 1.419. Figure 3. – Regulation of cardiac output (CO)**

<table>
<thead>
<tr>
<th>Regulation of cardiac output (CO)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CO</strong> = PP x HF</td>
</tr>
<tr>
<td>PP = Pulse pressure</td>
</tr>
<tr>
<td>HF = Heart frequency</td>
</tr>
</tbody>
</table>

- Central venous reflux
  - Circulating blood volume
  - Capacity of veins
- Contractility of cardiac muscle (inotropy)

The elements of blood pressure regulation are in a complex and dynamic relationship. The most important regulatory components include the pressure sensitive carotis and aortic receptors, the adrenergic tone of the sympathetic nervous system, the renin-angiotensin-aldosterone system (RAAS), the vasopressin (ADH), the glucocorticoids, the nitrogen-monoxide (NO), the intra- and extracellular Na\(^+\) and Ca\(^{2+}\) concentrations, the endothelin and the atrial natriuretic factor (ANP or ANF). This multifactorial regulation makes the treatment of high blood pressure a challenging task, as intervention on a single target may trigger compensatory mechanisms.

**Figure 1.420. Figure 4. – Regulation of total peripheral resistance (TPR)**

<table>
<thead>
<tr>
<th>Regulation of total peripheral resistance (TPR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baroreceptors</td>
</tr>
<tr>
<td>Adrenergic regulation</td>
</tr>
<tr>
<td>Renin - angiotensin II-alderosterone system (RAA)</td>
</tr>
<tr>
<td>Vasopressin (antidiuretic hormone – ADH)</td>
</tr>
<tr>
<td>Glucocorticoids</td>
</tr>
<tr>
<td>Nitric oxide (NO), other free radicals</td>
</tr>
<tr>
<td>Increase in intracellular Na(^+) and Ca(^{2+}) concentrations</td>
</tr>
<tr>
<td>Endothelin, ANF</td>
</tr>
</tbody>
</table>
The normal blood pressure range can be further divided into subcategories: (1) **optimal** (healthy) under 120/80 mmHg; (2) **normal** over 120/80 but under 130/85; (3) **high normal** or **prehypertension** over 130/85 but under 140/90. Blood pressure over 140/90 is regarded as hypertension. The risk greatly increases with the higher stages of hypertension. The presence of multiple risk factors exponentially increases the risk for cardiovascular events.

**Figure 1.421. Figure 5. – Normal and abnormally elevated blood pressure values**

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic (mm Hg)</th>
<th>Diastolic (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Optimal</strong></td>
<td>&lt; 120</td>
<td>&lt; 80</td>
</tr>
<tr>
<td><strong>Normal</strong></td>
<td>120-129</td>
<td>80-84</td>
</tr>
<tr>
<td><strong>High-normal (prehypertens.)</strong></td>
<td>130-130</td>
<td>85-89</td>
</tr>
<tr>
<td><strong>Hypertension:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 1 (mild)</td>
<td>140-159</td>
<td>90-99</td>
</tr>
<tr>
<td>Grade 2 (moderate)</td>
<td>160-179</td>
<td>100-109</td>
</tr>
<tr>
<td>Grade 3 (severe)</td>
<td>&gt;/= 180</td>
<td>&gt;/= 110</td>
</tr>
<tr>
<td>Isolated systolic hypertension</td>
<td>&gt;/= 140</td>
<td>&lt; 90</td>
</tr>
</tbody>
</table>

When a patient’s SBP and DBP levels fall into different categories, the higher category should apply.

**Figure 1.422. Figure 6. – Other Risk Factors and Disease History**

<table>
<thead>
<tr>
<th>Other Risk Factors and Disease History</th>
<th>Blood Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>High-normal</td>
</tr>
<tr>
<td>Grade 1 (mild)</td>
<td>Grade 2 (moderate)</td>
</tr>
<tr>
<td>Grade 3 (severe)</td>
<td>Grade 3 (very high)</td>
</tr>
</tbody>
</table>

ACC = associated clinical conditions (cerebrovascular disease, heart disease, renal disease, peripheral vascular disease, and advanced retinopathy)

TOD = target organ damage (left ventricular (LV) hypertrophy, arterial wall thickening, atherosclerotic plaque, slight increase in serum creatinine, and microalbuminuria).

Low: <15%, moderate 15-20%, high 20-30%, very high >30% (10-year risk of cardiovascular disease)

However, hypertension is not the only risk factor for cardiovascular diseases. Gender, age, smoking, dyslipidemia, familial genetic predisposition, obesity, high level of inflammatory mediators, stress, high sodium intake, glucose intolerance, and metabolic syndrome, which includes many of the factors mentioned above, create a synergistic network of risk factors.

**Figure 1.423. Figure 7. – The most common risk factors for cardiovascular disease**
Hypertension can be divided into two categories with respect to the underlying reason. In **primary or essential hypertension** a single causative factor cannot be identified, and therefore normal blood pressure cannot be permanently restored by targeting a single factor. On the contrary, **secondary hypertension** can be attributed to the dysfunction of a certain organ/tissue. Treatment of the dysfunction also restores normal blood pressure.

**Figure 1.4.24. Figure 8. – Classification of hypertension**

<table>
<thead>
<tr>
<th>Classification of hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary or essential hypertension (90%)</td>
</tr>
<tr>
<td>Secondary hypertension (10 %)</td>
</tr>
<tr>
<td>- Renal hypertension (5 %)</td>
</tr>
<tr>
<td>- Reno-vascular</td>
</tr>
<tr>
<td>- Reno-parenchymal</td>
</tr>
<tr>
<td>- Endocrine hypertension (3%)</td>
</tr>
<tr>
<td>- Pheochromocytoma (adrenal-medullary tumor)</td>
</tr>
<tr>
<td>- Hyperthyreosis</td>
</tr>
<tr>
<td>- Conn syndrome – (pr. Hyperaldosteronism)</td>
</tr>
<tr>
<td>- Cushing syndrome – (glucocorticoid overprod)</td>
</tr>
<tr>
<td>- Cardiovascular hypertension (1,5 %)</td>
</tr>
<tr>
<td>- Neurogenic hypertension (0,5 %)</td>
</tr>
<tr>
<td>- Drug induced (iatrogenic) hypertension</td>
</tr>
</tbody>
</table>

Half of secondary hypertension cases are kidney related, resulting from either parenchymal or vascular disorders. Several endocrine diseases also lead to secondary hypertension including pheochromocytoma (adrenaline secreting tumor), hyperthyreoidism, Conn’s-syndrome (aldosterone overproduction) or Cushing’s syndrome (cortisol overproduction). Less frequently, cardiovascular or neurogenic diseases, drug effects or gestation may also cause secondary hypertension.

**Figure 1.4.25. Figure 9. – Secondary hypertension**

<table>
<thead>
<tr>
<th>Secondary hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal hypertension (5 %)</td>
</tr>
<tr>
<td>- Reno-vascular</td>
</tr>
<tr>
<td>- Reno-parenchymal</td>
</tr>
<tr>
<td>Endocrine hypertension (3%)</td>
</tr>
<tr>
<td>- Pheochromocytoma (adrenal-medullary tumor)</td>
</tr>
<tr>
<td>- Hyperthyreosis</td>
</tr>
<tr>
<td>- Conn syndrome – (pr. Hyperaldosteronism)</td>
</tr>
<tr>
<td>- Cushing syndrome – (glucocorticoid overprod.)</td>
</tr>
<tr>
<td>Cardiovascular hypertension (1,5 %)</td>
</tr>
<tr>
<td>Coarctation of the aorta (children)</td>
</tr>
<tr>
<td>Neurogenic hypertension (0,5 %)</td>
</tr>
<tr>
<td>Drug induced (iatrogenic) hypertension</td>
</tr>
<tr>
<td>Sleep apnea</td>
</tr>
</tbody>
</table>
Essential hypertension results from a complex interaction of risk factors of genetic and environmental origin. These genetic (or rather genomic) factors are mostly unknown single nucleotide polymorphisms including those that are related to the increased function of the Na⁺-H⁺ exchanger, decreased Na⁺-K⁺ ATPase activity, increased renal response to salt load, or increased sensitivity of the adrenergic system. To make things more difficult, the clinical manifestation of the pathophysiological processes linked to certain SNP-s are also dependent on environmental factors such as daily sodium intake. Other environmental factors such as daily Na⁺, K⁺ and Ca²⁺ intake, or stress also increase the risk of hypertension.

**Figure 1.426. Figure 10. – Pathophysiology of essential hypertension**

<table>
<thead>
<tr>
<th>Pathophysiology of essential hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genetic versus environmental factors</td>
</tr>
<tr>
<td>☐ Multi-gene inheritance – mainly unknown</td>
</tr>
<tr>
<td>• Na⁺-H⁺ antiport (increased function)</td>
</tr>
<tr>
<td>• Na⁺-K⁺ ATPase (decreased function)</td>
</tr>
<tr>
<td>• Renal function (response to salt load)</td>
</tr>
<tr>
<td>• Increased sensitivity of adrenergic system</td>
</tr>
<tr>
<td>☐ Gene polymorphism studies to reveal more</td>
</tr>
<tr>
<td>☐ Environmental factors</td>
</tr>
<tr>
<td>• Salt load (mean value about 15 g/day instead of 5 or less)</td>
</tr>
<tr>
<td>• Low K⁺ and Ca²⁺ intake stress</td>
</tr>
</tbody>
</table>

**Figure 1.427. Figure 11. – Major factors in pathomechanism**

<table>
<thead>
<tr>
<th>Major factors in pathomechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Increase in peripheral resistance (TPR)-vasoconstriction</td>
</tr>
<tr>
<td>• Vasoactive compounds</td>
</tr>
<tr>
<td>Adrenergic regulation</td>
</tr>
<tr>
<td>Renin - angiotensin II - aldosterone</td>
</tr>
<tr>
<td>☐ Increase of cardiac output (CO)</td>
</tr>
<tr>
<td>• Usually CO increases first, then this is followed by the increase of TPR</td>
</tr>
<tr>
<td>☐ High carbohydrate intake</td>
</tr>
<tr>
<td>• Risk factor for diabetes mellitus, insulin indeed elevated renal tubular Na reabsorption vascular microinjuries</td>
</tr>
<tr>
<td>☐ Altered signal-transduction in vascular bed smooth muscle cells intracellular Ca²⁺ increase</td>
</tr>
</tbody>
</table>

As for the pathomechanism, it is clear that both increased peripheral resistance (TPR) (resulting from vasoconstriction) and increased cardiac output (CO) alone or in concert lead to hypertension. The actual increase of these parameters and the presence of effective autoregulatory mechanisms are individually different, highly depending on genetic and environmental risk factors.

**Figure 1.428. Figure 12. – Possible pathomechanism of primary hypertension**
Obesity is without doubt one of the most important risk factors of hypertension. High plasma cholesterol, a consequence of obesity, directly causes atherosclerosis and the loss of vessel wall elasticity. Increased food intake is also associated with high Na\(^+\) intake, leading to elevated extracellular fluid volume and thus increased CO. High carbohydrate consumption is a risk factor of diabetes mellitus, a disease that chronically impairs water and electrolyte excretion due to kidney dysfunction. Finally, increasing body weight decreases the kidney weight to body weight ratio and thus increases renal load.

**Figure 1.429. Figure 13. – Obesity**

<table>
<thead>
<tr>
<th>Obesity</th>
</tr>
</thead>
<tbody>
<tr>
<td>High plasma cholesterol level</td>
</tr>
<tr>
<td>• Risk factor for atherosclerosis</td>
</tr>
<tr>
<td>High food intake</td>
</tr>
<tr>
<td>• Usually accompanied by high Na(^+) intake and increased extracellular volume</td>
</tr>
<tr>
<td>High carbohydrate intake</td>
</tr>
<tr>
<td>• Risk factor for diabetes mellitus, insulin-induced elevated renal tubular Na(^+) reabsorption vascular microinjuries</td>
</tr>
<tr>
<td>Increased body weight</td>
</tr>
<tr>
<td>• Decrease in kidney weight/body weight ratio and reduced Na(^+) excretion</td>
</tr>
</tbody>
</table>

These factors altogether lead to a complex disease called metabolic syndrome. Obesity, insulin resistance, increase of abdominal fat, high blood glucose and triglyceride levels promote the development of hypertension, which in turn exacerbates metabolic syndrome.

**Figure 1.430. Figure 14. – Metabolic syndrome – also known as syndrome X or insulin resistance syndrome**

- may affect as many as 47 million Americans,
- insulin resistance and the presence of obesity, abdominal fat, high blood sugar and triglycerides, low HDL (good) blood cholesterol, and high blood pressure.

Consequences of hypertension include severe, sometimes life-threatening complications. It highly increases the risk of myocardial infarction or cerebrovascular events. Also, high blood pressure and atherosclerosis mutually increase each other. Hypertension and atherosclerosis lead to ventricular hypertrophy by increasing the
afterload. Hypertensive encephalopathy is a life-threatening syndrome caused by vasogenic oedema (extravasation of water and electrolytes). Hypertension (especially combined with diabetes mellitus) leads to visual impairment, by causing microvascular damage of the retina.

**Figure 1.431. Figure 15. – Consequences of hypertension**

<table>
<thead>
<tr>
<th>Consequences of hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image" alt="Consequences of hypertension" /></td>
</tr>
</tbody>
</table>

During a **dental procedure** pre- and postoperative management of anxiety, stress and pain are particularly important in hypertensive patients. The detailed knowledge of the interaction of administered drugs with the patient’s regular medication is also essential. Care must be taken for the psychological preparation of the patient.

**Figure 1.432. Figure 16. – For dentists: Dental surgery in hypertensive patient**

<table>
<thead>
<tr>
<th>For dentists:</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image" alt="For dentists: Dental surgery in hypertensive patient" /></td>
</tr>
</tbody>
</table>

The use of local anesthetics is especially important in moderate (Grade 2) or severe (Grade 3) uncontrolled hypertension. Vasoconstrictor drugs such as epinephrine or norepinephrine are in everyday use in the dental practice. In severe (Grade 3) hypertension however, vasoconstrictor amounts should be limited to 3,6 ml of local anesthetics containing 1:100,000 solution of epinephrine. Significant amounts of vasoconstrictors should be avoided in severe cases.

**Figure 1.433. Figure 17. – For dentists: Local anesthetic in poorly controlled Grade 2-3 hypertension**
Prevention of hypertension essentially means reducing risk factors. Therefore, avoiding obesity, high sodium intake, hypercholesterolemia, decreased physical activity, stress or untreated diabetes is very important. Hypertension develops slowly and remains asymptomatic for long. Therefore regular annual, or in high-risk groups, more frequent screening is advisable.

Figure 1.434. Figure 18. – Prevention of hypertension

The therapy of hypertension depends on its severity. Measures such as regular exercise or reducing the body weight, salt intake, alcohol consumption and smoking may be beneficial in mild hypertension. Moderate or severe hypertension require the application of drugs. Diuretics decrease blood volume, beta blockers and calcium channel blockers moderate the tone and excitability of blood vessels and cardiac muscle. Angiotensin convertase enzyme inhibitors (ACE inhibitors) and angiotensin antagonists minimize the spasm of blood vessels and water retention.

Figure 1.435. Figure 19. – Pathophysiological basis of therapy

Video 1. – Blood pressure measurement.
31.1. Test – Oral aspects of hypertension (answers)

1. Characteristic for essential hypertension:

A. incidence increases with age

B. risk can be diminished by increased NaCl intake

C. risk cannot be influenced by chronic stress

D. its development can be prevented by the induction of angiotensin convertase enzyme

2. Hyperthyreosis induces hypertension, since:

A. the patient usually gets tired soon

B. salt excretion of kidney is damaged

C. hyperthyroid hormones sensitize the arterioles to adrenergic compounds

D. bradycardia appears

E. chronic sleeping

3. It does NOT play role in blood pressure regulation:

A. aldosteron

B. renin

C. alkaline phosphatase

D. catecholamines

32. 1.32. Oral aspects of protein metabolism and energy balance – Gabor Varga

The energy requirement of a healthy adult is equal to the energy intake from nutrition that is required to cover the energy consumption of the body while body weight and composition stay constant. Its main components are basal metabolism and the energy needed for movement. The basal metabolism (basal metabolic rate, BMR) can be described to a good approximation by an equation of which most important variable is the body mass. There is a significant difference between genders: women are better energy utilizers than men. The energy requirement for movement and related activities can be represented as a multiplication of the BMR. Numerous conditions can affect the energy requirement such as physical development, pregnancy, lactation, and also fever. Pathologies, such as injuries and illnesses, can also significantly change the energy need.

Figure 1.436. Figure 1. – Basal Metabolic Rate (BMR)

<table>
<thead>
<tr>
<th>Basal Metabolic Rate (BMR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy requirements (ER)</td>
</tr>
<tr>
<td>can be described by linear equation, depends on age and gender.</td>
</tr>
<tr>
<td>18-30 years old man</td>
</tr>
<tr>
<td>ER (kJoul/day) = 64 x b.w. + 2640  b.w=body weight</td>
</tr>
<tr>
<td>18-30 years old woman</td>
</tr>
<tr>
<td>ER (kJoul/day) = 61.5 x b.w. + 2840  b.w=body weight</td>
</tr>
</tbody>
</table>
Proteins, lipids and carbohydrates are the energy sources that can be used by the body. There are big differences between their utilization, but all of them can serve as an energy source. The energetic or ‘caloric’ cycle can be divided into two main periods: the period following a meal is called postprandial and serves for anabolic processes, and the other period is the postabsorptive one in which catabolism is predominant.

The caloric cycle is controlled at both hormonal and substrate level. Among the main hormonal regulators, insulin is responsible for energy conservation while adrenaline, glucagon, cortisol and thyroid hormone can release energy.

An important factor in substrate control is the plasma concentration of amino acids and fatty acids. When their concentrations are high, gluconeogenesis is inhibited and when their concentrations drop, the inhibition ceases. The other key mechanism is Randle-type glucose-fatty acid cycle: high glucose levels inhibit the release of fat from the adipose tissue, while high plasma level of fatty acids significantly reduces glucose uptake by adipose tissue and muscle. The background to these regulatory cycles is that glucose must be utilized efficiently because the body can store only a small amount of this component as liver glycogen.
The complete absence of food intake rapidly leads to **starvation**. The body responds to starvation in several stages. The first stage is the postabsorptive phase that is the same as the second phase of the caloric cycle. This is followed by the early, non-adapted phase 3-4 days after termination of food intake. Then by the late, adapted phase develops 7-10 days after food intake stopped, and maintained until feeding.

### Figure 1.441. Figure 6. – Starvation

<table>
<thead>
<tr>
<th>Phases (start at):</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post absorptive phase (12-18 h)</td>
</tr>
<tr>
<td>Early, non-adapted phase (48-96 h)</td>
</tr>
<tr>
<td>Late, adapted phase (7-10 days)</td>
</tr>
</tbody>
</table>

In the early, non-adapted phase of starvation the primary aim is the maintenance of the blood glucose level by gluconeogenesis at the expense of the protein and fat reserves of the body. In the late, adapted phase the source of energy consumption is primarily based on burning of fatty acids and ketone bodies. This leads to a relative decrease in protein consumption and inhibition of gluconeogenesis.

### Figure 1.442. Figure 7. – Factors producing stable blood glucose level during the early, nonadapted phase of starvation
Death typically does not occur because of the depletion of energy stores. The body continuously needs at least a small amount of protein to function. The required amino acids derive primarily from the degradation of muscle proteins during starvation. But quantitative reduction in cardiac muscle often leads to circulatory collapse or arrhythmias associated with cardiac arrest.

After several weeks of starvation body mass decreases, but blood glucose level still remains relatively constant as the body switches to fatty acid utilization. The only exception is the brain; about 50% of its energy requirement is still covered by sugars even during long-term starvation.
Although the body is able to adapt to these circumstances well, prolonged starvation damages every tissue, including the oral structures. This damage is primarily manifest in rapidly renewable, continuously remodeling tissues such as the surface of the mucosa and tongue in the oral cavity.

Obesity is a global problem. It is based on extreme deposition of fat due to excessive energy intake. Several methods are available for its determination for example the comparison of the actual body mass with the ideal body mass for a certain height. According to statistics the ideal body weight results in minimal mortality. The body mass index (BMI) is the ratio of body mass to the square of height. Its normal value is 20-25 kg/m², 26-30 kg/m² means the person is overweight, and above 35 kg/m² they are severely obese.

**Figure 1.446. Figure 11. – Criteria for obesity**

<table>
<thead>
<tr>
<th>Criteria for obesity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relative body mass</td>
</tr>
<tr>
<td>= 100 x actual body weight/ideal body weight</td>
</tr>
<tr>
<td>optimal range: +/- 10% (obesity + 15-20%)</td>
</tr>
<tr>
<td>Quetelet-index (Body mass index, BMI)</td>
</tr>
<tr>
<td>= body weight/height² [kg/m²]</td>
</tr>
<tr>
<td>optimal value: 25 kg/m² (obesity &gt;30 kg/m²)</td>
</tr>
</tbody>
</table>

Obesity, according to histological classification, can be hypertrophic (cell size cells is increased in adipose tissue) or hyperplastic (cell number and often cell size is also increased). The former usually characterizes adult obesity, which is relatively easy to treat. The latter usually appears in a juvenile form that is genetically determined and more difficult to treat. According to anatomical classification, a visceral or android type (a more dangerous form because abdominal fat behaves as an inflammatory organ) and a gluteofemoral or gynoid type can be distinguished.

**Figure 1.447. Figure 12. – Classification of obesity**

<table>
<thead>
<tr>
<th>Classification of obesity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Histological classification</td>
</tr>
<tr>
<td>hyperplastic obesity</td>
</tr>
<tr>
<td>hypertrophic obesity</td>
</tr>
</tbody>
</table>

The main cause of obesity is the increase in the amount of stored energy due to the disruption of the balance between energy consumption and intake. In addition to excessive energy intake, the efficiency of energy
utilization can differ between individuals and the regulation of hunger and satiety may also be disturbed. Centrally the hunger and satiety centers of the brain are responsible for control. Peripherally numerous bioactive compounds play an important role in regulation. In response to an increase in the plasma level of different nutrients several compounds are secreted into the circulation such as ghrelin from the stomach, leptin from adipocytes, insulin from Langerhans islet cells, CCK (cholecystokinin) and GLP-1 (glucagon-like peptide) from specialized epithelial cells in the intestine. They primarily activate ascending neural pathways to transmit satiety and to reduce hunger. An interesting property of this multifactorial regulation is that after pharmacological inhibition of a certain component the others rapidly compensate and restore the original balance.

Figure 1.448. Figure 13. – Flow-chart for energy metabolism

![Flow-chart for energy metabolism](image)

Figure 1.449. Figure 14. – Traditional view on food intake control

![Traditional view on food intake control](image)

Figure 1.450. Figure 15. – Peripheral factors contributing to appetite
Obesity is a serious risk factor in the development of numerous diseases and it increases mortality. The risk increases sharply in correlation with the BMI increase. However, when BMI is too low, it also represent a risk factor not only for health but also for life under extreme circumstances.

The protein requirement of the body equals the protein intake required to maintain nitrogen balance and replace the degraded essential amino acids which cannot be synthesized by the body. The minimum protein requirement is 15g/day and the physiological protein requirement is 30-50g/day in a healthy adult.
Protein deficiency develops usually because of a combination of different factors: these include quantitatively or qualitatively insufficient protein intake, insufficient energy intake and the coupled reduction of vitamin and mineral intake. Together these factors are described as protein-energy undernutrition (PEU).

**Figure 1.454. Figure 19. – Reasons for protein deficiency**

<table>
<thead>
<tr>
<th>Reasons for protein deficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>quantitative deficiency</td>
</tr>
<tr>
<td>qualitative deficiency</td>
</tr>
<tr>
<td>(shortage of essential amino acids)</td>
</tr>
<tr>
<td>shortage in energy uptake</td>
</tr>
</tbody>
</table>

Usually a combination: qualitative and quantitative deficiency, and also vitamin and mineral deficiency together:
Protein-energy undernutrition - PEU

Numerous severe famines are known from world history and unfortunately they still kill the bulk of the people affected, mainly in Africa and Asia. A very different but also important and severe form of PEU is end stage of cancer i.e. cachexia (usually it is not related to financial matters, but rather to organism dysregulation reasons). The exact cause of this complex syndrome is unknown. But in end stage tumor patients, in addition to decreased nutrition, probably numerous other factors play a role, such as the dominance of inflammatory and catabolic processes.

**Figure 1.455. Figure 20. – Major famines of the world**

<table>
<thead>
<tr>
<th>Major famines of the world</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
</tr>
<tr>
<td>1845</td>
</tr>
<tr>
<td>1930s</td>
</tr>
<tr>
<td>1984-85</td>
</tr>
<tr>
<td>1996-97</td>
</tr>
<tr>
<td>?</td>
</tr>
</tbody>
</table>

**Figure 1.456. Figure 21. – The mechanism of cachexia caused by malignant tumors**
The two major forms of protein-energy undernutrition are **kwashiorkor** and **marasmus**. In kwashiorkor carbohydrate intake is relatively well maintained alongside an energy and protein deficiency. In contrast, marasmus means the massive deficiency of all nutrients. Therefore, the former can be characterized by hypoproteinaemia, hunger edema and hepatic steatosis (fatty liver), while in the latter one the muscle and the adipose tissue are severely reduced. The oral structures are severely affected in both disorders. Hormonal, metabolic, haematological, systemic neuronal and other damages are concomitant with both of them.

**Figure 1.457. Figure 22. – Clinical symptoms of PEU (kwas = kwashiorkor, mar = marasmus)**

**Clinical symptoms of PEU (kwas = kwashiorkor, mar = marasmus)**

- Decrease in body weight (-20% kwas, -40% mar)
- Negative nitrogen-balance
- Hypoproteinaemia (hypoaalubinaemia) (only kwas)
- Decreased serum amino acid conc. (only kwas)
- Starvation-edema (only kwas)
- Fatty liver (only kwas)
- Decrease in adipose and muscle tissues (more in mar)
- Anaemia, hypothermia
- Apathia, weakness
- Endocrine and neuro-psychiatric disorders

**Figure 1.458. Figure 23. – Disorders of protein metabolism (PEU)**

**Disorders of protein metabolism (PEU)**

- Hormonal changes
  - Insulin level decreases
  - Somatotropin level decreases
  - Adrenal function may change (increase in mar)
  - Thyroid function decreases (sexual hormone levels decrease)
- Metabolic changes
- Hematological changes
- Metabolism
- Starvation-edema (kwas)
- Immune system changes
- Psychological changes

Diseases of the liver, gastrointestinal tract, kidney and endocrine organs can develop as a result of **secondary protein metabolism disorders**. This includes quantitative and qualitative changes in plasma proteins.

**Figure 1.459. Figure 24. – Secondary protein metabolism disorders**
Amino acid metabolic disorders can be traced back to disorders of their transport, to disorders of their metabolism or to a combination of both. Transport disorders usually appear in specific organs, while a deficiency of enzymes catalyzing the degradation of certain amino acids manifests systemically often coupled with psychological disorders. From the dental aspect, the oral symptoms are atypical. Nevertheless, they still have to be considered during conventional dental interventions.
1. Oral biology

**Disorders of transport processes**

- Intestinal absorption disorders
  - Hartnup syndrome (tryptophan)
  - Methionine malabsorption
- Renal back filtration disorders
  - Cystinuria
  - L-mimosineurie

**Figure 1.463. Figure 28. – Metabolic amino acid disorders**

**Metabolic amino acid disorders**

- Because of the loss of enzyme(s) controlling amino acid metabolism, substrate concentration increases, product concentration decreases dramatically.

- Consequences: metabolite conc. increases, loss of essential amino acids, mental retardation, aminoaciduria, local precipitation of metabolites.

**Figure 1.464. Figure 29. – Metabolic amino acid disorders (missing key factors in brackets)**

**Metabolic amino acid disorders (missing key factors in brackets)**

- Phenylketonuria (phenylalanine-hydroxylase)
- Tyrosinosis (p-OH phenyl-pyruvic acid-oxidase)
- Alcaptonuria (Homogentisinate-oxidase)
- Albinism (tyrosinase)
- Hyperprolinenaemia (prolin breakdown)
- Oxalosis and hyperoxaluria (glycine)
- Maple-syrup disease (valin, leu, isoleu breakdown)

**32.1. Test – Oral aspects of protein metabolism and energy balance (answers)**

1. **Characteristic for the early, non-adapted phase of starvation:**
   
   A. increased gluconeogenesis
   
   B. decreased amino acid breakdown – protein conservation
   
   C. elevated plasma insulin level
   
   D. significant role of keton bodies in energy supply

2. **Calculation of Body Mass Index (BMI)**
   
   A. $\text{BMI} = 64 \text{T} + 2840$ where $\text{T}$ is the body mass
   
   B. $\text{BMI} = \frac{\text{actual body mass}}{\text{ideal body mass}} \times 100$
C. BMI = body mass (kg)/body height$^2$ (m$^2$)

D. BMI = body mass/(body height - 100)

3. Extreme low body weight is indicated by the following BMI value

A. 1.6
B. 16
C. 36
D. 106

33. 1.33. Oral aspects of carbohydrate metabolism and diabetes – Beata Keremi

Diabetes mellitus is the most common metabolic disorder. It affects carbohydrate, protein and fat metabolism as well. This results in an imbalance between the production of insulin and insulin needs. This can be manifested in the following ways: a complete lack of insulin, decreased insulin release from pancreatic beta cells, inadequate or defective insulin receptors, improper or inadequate insulin postreceptor regulation; production of inactive insulin and early degradation of insulin before it could reach its target.

33.1. Types

The disease earned its name because it causes sugar to appear in urine, making it sweet. Above 12 mmol/L systemic glucose concentration kidneys cannot reabsorb sugar, so it is excreted with urine.

33.1.1. Type 1 DM

It is caused by the destruction of pancreatic beta cells, leading to complete insulin deficiency. Beta cell destruction is due to autoimmune processes triggered by viral infection. In 80% of the patients antibody production is directed against glutamic acid decarboxylase expressed in beta cells. It usually occurs at a young age (in childhood), but may occur at an older age as well. In addition to the impairment of glucose metabolism, it is associated with increased fat and protein breakdown. Type 1 diabetics are prone to develop ketoacidosis.

2 subtypes are known: A) immune-mediated DM and B), idiopathic (non-immune mediated) DM.

33.1.2. Type 2 DM

Insulin resistance. The onset is slow and classic symptoms are often absent. Obesity is a major risk factor for type 2 DM, but genetic predisposition to decreased insulin sensitivity is an important factor. Insulin resistance, irregular insulin secretion and increased hepatic glucose production can lead to formation type 2 DM. In rare cases, insulin or insulin receptor synthesis, or occasionally insulin signaling is disturbed. Insulin levels in these patients may be either high, normal, or low. Insulin resistance initially stimulates insulin secretion, and higher than normal insulin levels can be measured. Increased insulin production for extended periods leads to beta cell exhaustion. This causes persistent high postprandial glucose levels and hepatic glucose production may also be increased.

Insulin resistance plays a role not only in the development of type 2 diabetes, but also in other metabolic diseases such as metabolic X syndrome. Insulin not only has an important role in regulating glucose metabolism, but it plays an important role in fat and protein metabolism as well.

Figure 1.465. Figure 1. – Blood glucose level changes in DM. Oral glucose tolerance test characteristic curves. In healthy subjects (normal), the fasting blood glucose level is 3.5 to 5.5 mM/L, followed by a post-meal rise to 7 to 9 mM/L in 30 to 60 minutes. Blood glucose level returns to below 8 mM/L by 2 hours after meal. IGT (impaired glucose tolerance); the fasting blood glucose level is at the upper limit of the normal range, i.e. 6 mM/L. After meal it rises above 12 mM/L in 30 to 60 minutes, but it returns to between
7. to 11.1 mM/L by 120 minutes after meal. DM (diabetes mellitus): the initial blood glucose value is already highly elevated to above 7.8 mM/L. After meal, the value remains persistently above 12 mM/L, with no return to below 11.1 mM/L by 2 h after meal.

![Graph showing glucose levels over time]

These patients are more susceptible to various infections, especially to anaerobic infections because of reduced oxygen diffusion through the capillary wall. Glucose is an osmotically active molecule. Therefore, elevated blood glucose concentration increases osmotic pressure. Thus, isosmotic neutrophils shrink in hyperosmotic environments, with a significant decrease in their function (impaired superoxide formation). Due to the suppression of neutrophils and bacterial synergism more severe infections will ensue. High glucose level promotes the growth of bacteria, leukocyte degradation and apoptosis in the affected areas. It inhibits normal macrophage functions (chemotaxis, phagocytosis and killing of bacteria). Hyperglycemia promotes the formation of glycosylated endproducts, such as the formation of glycosylated hemoglobin, which significantly reduces oxygen-carrying capacity compared to normal hemoglobin.

### 33.1.3. Secondary Diabetes Mellitus

It may develop as a consequence of pancreatitis when beta cells are destroyed by inflammation.

Increased levels of antagonistic hormones may also play a role in the development of DM. For example: somatotropin (acromegaly), glucocorticoids (Cushing’s disease, stress), adrenaline (stress), ACTH, thyroid hormone and glucagon. Severe infections increase the release of these hormones and thus lead to diabetes mellitus.

### 33.1.4. Gestational diabetes mellitus

It first occurs during pregnancy.

### 33.2. Symptoms

Typical symptoms are the “three polys”:
- polyuria (increased urination),
- polydypsia (permanent thirst),
- polyphagia (a large amount of food consumption).

### 33.3. Consequences

Acute consequences:
- diabetic ketoacidosis,
• hyperosmolar hyperglycemic state,
• hypoglycemia (weakness, sweating, mental confusion, lack of coordination, tremors).

Figure 1.466. Table 1. – Late consequences of diabetes mellitus (5+1)

| Retinopathy (cataracta, blindness) |
| Neuroopathy                     |
| Nephropathy                     |
| Micro- and macroangiopathy,     |
| Cardiovascular consequences,    |
| accelerated atherosclerosis      |
| Delayed wound healing           |
| Recurrent infections            |

+ Periodontal disease –
It has 2-fold increased prevalence in diabetic patients compared to the non-diabetic population. (Host inflammatory response). There is a close correlation between periodontal disease progression / severity, and not (properly) controlled blood sugar.

33.4. Oral manifestation

Figure 1.467. Table 2. – Oral manifestations of diabetes mellitus. Periodontitis induced bacteremia increases the levels of proinflammatory cytokines (TNF-α, IL-6, IL-1) and of reactive oxygen species in serum, which leads to increased insulin resistance

<table>
<thead>
<tr>
<th>Table 2:</th>
<th>Oral manifestation of diabetes mellitus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Candidiasis</td>
<td></td>
</tr>
<tr>
<td>Caries</td>
<td>decreased saliva secretion, increased carbohydrate levels in parotid saliva, oral yeast growth, elevated Str mutans and L. acidophilus numbers</td>
</tr>
<tr>
<td>Tooth loss</td>
<td></td>
</tr>
<tr>
<td>Gingivitis</td>
<td></td>
</tr>
<tr>
<td>Lichen planus</td>
<td></td>
</tr>
<tr>
<td>Neurosensory disorder</td>
<td>* burning mouth syndrome - unknown cause, usually on both sides * glossodynia</td>
</tr>
<tr>
<td>Periodontitis</td>
<td>changes in host response, the composition of subgingival microflora, collagen metabolism, periodontal vascularization and in the composition/amount of crevicular fluid</td>
</tr>
<tr>
<td>Saliva dysfunction</td>
<td>decreased saliva secretion and/or changes in saliva composition</td>
</tr>
<tr>
<td>Xerostomia</td>
<td></td>
</tr>
<tr>
<td>Dysgeusia</td>
<td>develops in conjunction with neuropathy, its relationship with obesity has been shown</td>
</tr>
<tr>
<td>Halitosis</td>
<td></td>
</tr>
<tr>
<td>Mouth disease</td>
<td>fissured tongue, irritation fibroma, traumatic ulcers, recurrent stomatitis aphictosa</td>
</tr>
</tbody>
</table>

Figure 1.468. Figure 2. – Elevated caries risk (with permission from Adam Gombos DMD)
Figure 1.469. Figure 3. – Severe periodontal lesions (with permission from Adam Gombos DMD)
33.5. Diagnosis

33.5.1. Blood glucose analysis – from fingertip capillary blood

Blood glucose determination is based on an enzyme reaction. This is the glucose oxidase method, based on the glucose oxidase mediated oxidation of glucose to gluconic acid. Glucose oxidase (GOD), peroxidase (POD) and a chromogenic substance is measured into the buffer, followed by the sample. GOD oxidizes the glucose to a peroxide derivative, which is degraded by peroxidase and the resulting nascent oxygen reacts with the chromogenic substance, an aromatic amine. The amount of the resulting colored product is proportional to the amount of glucose. This reaction is the basis of home blood glucose tests as well. The test can be quantitated by two methods:

- Photometric measurement of the colored endproduct using a photometer.
- Electrochemical – rather than using a color reagent, electrons from the oxidation of glucose are counted by a device called blood glucose meter.

The hexokinase / glucose-6-phosphate dehydrogenase / NAD method is a faster, more accurate but much more expensive method.

33.5.2. Measurement of glycosylated hemoglobin

It reflects average blood glucose levels over a prolonged period of time. The average lifespan of RBCs is 120 days. During this time, due the exposure to plasma glucose, hemoglobin in RBCs undergoes non-enzymatic glycation, which is irreversible. The higher the blood glucose level, the more sugar is bound to hemoglobin. Measuring the amount of glycated hemoglobin provides information on average blood glucose levels over a period of 2 to 3 months, indicating the effectiveness of therapy and patient compliance (diet, etc.). The amount of glycosylated hemoglobin is expressed as the percentage of total hemoglobin. The normal value is 4 to 6%, under 6.5% is acceptable in a well managed patient. This value may increase 2 to 3 fold of normal in diabetic patients.

33.6. Treatment

Type 1 DM:

- continuous, lifelong insulin replacement.

Type 2 DM:

- diet and lifestyle change,
- oral antidiabetic drugs,
- insulin replacement.

33.7. Metabolic X syndrome

Figure 1.470. Table 3. – Symptoms of metabolic X syndrome. The basis for the diagnosis is the co-occurrence of at least three symptoms. Obesity is typically of central type, a.k.a. visceral or abdominal
Adipocytes produce a number of important factors such as adiponectin and leptin.

In obesity larger adipocytes are present in the adipose tissue. Therefore the numbers of macrophages and apoptotic adipocytes are increased well. Cell necrosis induces inflammation, which triggers insulin resistance. In addition, obesity is associated with the activation of endothelial cells and the accumulation of free fatty acids. Free fatty acids also promote the production of proinflammatory adipokines and the activation of macrophages. Many pro-inflammatory adipokines produce chemokines which can bind macrophages and guide them to the adipose tissue; adhesion molecules are also produced which induce further recruitment. The development of metabolic syndrome is determined by the intake of main nutrients (total energy, total protein, saturated fats) between the ages of 6 and 14 years.

**Figure 1.471. Picture 4. – Blood collection tube containing NaF and K$_2$EDTA for glucose and lactate test**

1. video – Routine blood glucose measurement.

**33.8. Test – Oral aspects of carbohydrate metabolism and diabetes (answers)**
1. Can we measure high levels of insulin in II type DM patients?

A. no, diabetic patients always lack insulin
B. only normal or low levels of insulin can be measured in these patients due to insulin resistance
C. during the initial stage there may be high insulin levels due to insulin resistance
D. only high insulin levels can be measured because tissues do not respond to insulin
E. no, because depletion of β cells is characteristic of this disease

2. Which are the typical symptoms of metabolic X syndrome?

A. gluteal type obesity
B. low serum LDL levels
C. diabetes mellitus
D. periodontitis
E. hypertension

3. Patients with diabetes mellitus are more susceptible to infections because fewer neutrophils are formed and they cannot develop an effective protection.

A. true-true, with connection
B. true-true, without connection
C. true-false
D. false-true
E. false-false

References


F. Lang: Diabetes Mellitus Ch. 9, Hormones, pp 286-291. in: S. Silbernagl and F. Lang: Color atlas of Pathophysiology, Thieme, 2000

34. 1.34. Oral aspects of lipid metabolism – Beata Keremi

34.1. Classification of lipids

Lipids (except phospholipids) are nonpolar. They are poorly soluble or insoluble in water, but they can easily dissolve in nonpolar solvents.

**Figure 1.472.** Table 1. – Classification of lipids. The classification is based on chemical structure. Similar or identical biological functions are related to similar chemical structures

<table>
<thead>
<tr>
<th>Table 1: Classifications of lipids</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Free fatty acids (FFAs)</strong></td>
</tr>
<tr>
<td>o Non-esterified fatty acid</td>
</tr>
<tr>
<td>o Binding to albumin in the serum</td>
</tr>
<tr>
<td><strong>Triglycerides (TG)</strong></td>
</tr>
<tr>
<td>o Energy metabolism</td>
</tr>
<tr>
<td>o Structure: single glycerol molecule and 3 fatty acids</td>
</tr>
<tr>
<td><strong>Phospholipids</strong></td>
</tr>
<tr>
<td>o Contain a phosphatate group</td>
</tr>
<tr>
<td>o Important components of:</td>
</tr>
<tr>
<td>• cell membranes</td>
</tr>
<tr>
<td>• lipoproteins</td>
</tr>
<tr>
<td>• blood clotting components</td>
</tr>
<tr>
<td>• myelin sheath</td>
</tr>
<tr>
<td><strong>Cholesterol (Ch) and cholesteriesters</strong></td>
</tr>
<tr>
<td>o not composed of fatty acids</td>
</tr>
<tr>
<td>o Its steroid core is synthesised from fatty acids</td>
</tr>
<tr>
<td>o their chemical activity is similar to other lipids</td>
</tr>
<tr>
<td>• increased cholesterol level</td>
</tr>
<tr>
<td>• elevated risk of atherosclerosis</td>
</tr>
<tr>
<td>• elevated risk of haert attack and stroke t</td>
</tr>
</tbody>
</table>

34.2. Lipids in nutrition

Relative intake of nutrients:

• carbohydrates 45 to 50%,

• proteins 10 to 15%,

• lipids 35 to 40%.

Daily fat intake required: 50 to 150 g (important for the absorption of fat-soluble vitamins and essential fatty acids) !!!

Palmitic acid and saturated fatty acids increase the risk for cardiovascular diseases, while unsaturated and omega 3 fatty acids have a protective effect.

Reaction with free radicals can result in oxidative degradation of lipids, leading to the formation of lipid radicals and lipid peroxide.
34.3. Digestions and absorption of lipids

In oral cavity:
- lingual lipase - acting mostly in the stomach. Produced by minor salivary glands in tongue. Its role is most important in newborns.

In duodenum:
- steps of cholecystokinin (CCK) release,
- effect of CCK on the release of bile and pancreatic proteins,
- micelle formation and function,
- lipids dispersed in water,
- coagulation effect of bile acids,
- pancreatic lipase, phospholipase A2, lisophospholipase,
- cleavage of triglycerides in micelles into free fatty acids (FFA) and glycerol.

Intestinal absorption of FFA, glycerol and cholesterol
- Individual transporters for FFA with short, long, saturated and unsaturated chains and for cholesterol.

Role of intestinal epithelium:
- Synthesis of triglycerides in epithel cells.
- Apoprotein synthesis.
- Formation of macromolecules, ie. of native chylomicron.
- Newly formed apoproteins bind to nascent chylomicron in the blood and lymph.

34.4. Lipoproteins

34.4.1. Structure of lipoproteins

Figure 1.473. Figure 1. – Structure of lipoproteins. Cholesterol and triglycerides are insoluble in water. These hydrophobic molecules are transported in the blood within hydrophilic particles called lipoproteins, which consist of phospholipids and apoproteins
Roles of apoproteins:

- structural elements
- ligands for lipoprotein receptors in the membrane
  - apoB-100 – binds to LDL-receptor
  - apoCIII – inhibits VLDL uptake by the liver
  - apoE – promotes chylomicron remnant uptake by the liver
- enzyme activators and facilitate lipid efflux from lipoproteins
  - apoAI – activates lecithin cholesterol acyl-transferase (LCAT) enzyme
  - apoCII – activates lipoprotein lipase

34.4.2. Functions of lipoproteins

Transport of cholesterol and triglycerides to various tissues for:

- supplying energy,
- fat storage (lipid deposition),
- synthesis of steroids,
• bile acid formation.

Lipoproteins are dynamic structures that continuously change and are interconvertible.

### 34.4.3. Types of lipoproteins

**Figure 1.474. Table 2. – Classification of lipoproteins. A classification occurs based on the density of lipoproteins. The groups are separated by ultracentrifugation**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Composition:</th>
<th>Density:</th>
<th>Apoproteins:</th>
<th>Formation</th>
<th>Function:</th>
</tr>
</thead>
<tbody>
<tr>
<td>CH</td>
<td>Chylomicron</td>
<td>80 to 90%</td>
<td>3% cholesterol 2% protein</td>
<td>0.95 g/ml</td>
<td>small intestine</td>
</tr>
<tr>
<td></td>
<td>*start</td>
<td></td>
<td>apop8-49, apoe, apoaI, apocII, apocIII</td>
<td></td>
<td>transport TG from small intestine to periphery (muscles, fat tissues) activates lipoprotein lipase</td>
</tr>
<tr>
<td>VLDL</td>
<td>Very low density lipoprotein</td>
<td>55 to 65%</td>
<td>triglyceride 10 to 15%</td>
<td>1.006 g/ml</td>
<td>liver</td>
</tr>
<tr>
<td></td>
<td>*pre-β</td>
<td></td>
<td>cholesterol 5 to 10% protein</td>
<td></td>
<td>transport TG from liver to periphery (muscles, fat tissues) activates lipoprotein lipase</td>
</tr>
<tr>
<td>IDL</td>
<td>Intermediate density lipoprotein</td>
<td>(apoB-100,</td>
<td>apoE, apoaI, apocII, apocIII</td>
<td>from VLDL</td>
<td>from HDL</td>
</tr>
<tr>
<td></td>
<td>*</td>
<td>apoB-100, apoE, apoA, apocII, apocIII</td>
<td></td>
<td>from HDL</td>
<td>half transports lipids to liver other half is converted to LDL by lipoprotein lipase</td>
</tr>
<tr>
<td>LDL</td>
<td>Low density lipoprotein</td>
<td>10% triglyceride 50% cholesterol 25% protein</td>
<td>1.055 to 1.065 g/ml</td>
<td>apoB-100</td>
<td>from IDL</td>
</tr>
<tr>
<td></td>
<td>*β</td>
<td></td>
<td></td>
<td></td>
<td>delivers cholesterol from LDL to liver (70%) and to extrahepatic tissues (30%) LDL receptor binding pathway on periphery (adrenals, smooth muscle cells, endothelial cells, lymphoid cells hormone or membrane formation), or bind to scavenger receptors (macrophages – atherosclerosis)</td>
</tr>
<tr>
<td>HDL</td>
<td>High density lipoprotein</td>
<td>5% triglyceride 20% cholesterol 50% protein</td>
<td>1.210 g/ml</td>
<td>apoE, apoaI, apoaII, apoaIV, apoaIII, apoaD</td>
<td>Periphery</td>
</tr>
<tr>
<td></td>
<td>*β</td>
<td></td>
<td></td>
<td></td>
<td>apoprotein exchange with chylomicrons VLDL delivers cholesterol from periphery to IDL (into the liver) activates cholesterol esterifying plasma enzymes transports cholesterol and cholesterol ester to liver and steroid hormone synthesizing producing glands (ovaries, testicles, adrenals)</td>
</tr>
</tbody>
</table>

### 34.5. Steps of lipid metabolism

**Figure 1.475. Figure 2. – Exogenous lipid metabolism. Exogenous metabolism refers to the transport (and metabolism) of the absorbed lipids to the liver cells**
Figure 1.476. Figure 3. – Endogenous lipid metabolism. Endogenous pathway refers to the transport of the lipids, synthesised in the liver, to periphery. There, the free cholesterol is either utilized, or stored in an esterified form.
Reverse cholesterol transport. During the reverse cholesterol transport, the excess cholesterol is transported back from the periphery to the liver by the HDL.
34.6. Dyslipidemias

34.6.1. Types of hyperlipoproteinemias

- Elevated cholesterol level – hypercholesterolemia,
- elevated triglyceride level – hypertriglyceridemia,
- elevated levels both of cholesterol and triglyceride – combined hyperlipidemia.

Figure 1.478. Table 3. – Groups of hypercholesterinemias

<table>
<thead>
<tr>
<th>Type</th>
<th>Name</th>
<th>Lipoprotein abnormality</th>
<th>Known genetic defect</th>
<th>Clinical symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Exogenous dietary hypercholesterolemia</td>
<td>↑Chylomicrons and triglycerides</td>
<td>Mutations in lipoprotein lipase gene</td>
<td>Pancreatitis, hepatosplenomegaly, retinal hyperlipemia, xanthoma</td>
</tr>
<tr>
<td>2a</td>
<td>Familial hypercholesterolemia</td>
<td>↑LDL cholesterol</td>
<td>Mutations in LDL receptor gene or apoproteinB gene</td>
<td>Cornea, tendons, arteriosclerosis Early xanthelasma Xanthomas in the tendons</td>
</tr>
<tr>
<td>2b</td>
<td>Combined hyperlipidemia</td>
<td>↑LDL, VLDL, triglycerides</td>
<td>Mutations in LDL receptor gene or apoproteinB gene</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Remnant hyperlipidemia</td>
<td>↑Remnants (from chylomicrons), IDL, triglycerides, cholesterol Familial dysbetalipoproteinemia predisposes to it</td>
<td>Mutation in apoproteinE gene</td>
<td>Xanthoma in palms Early atherosclerosis</td>
</tr>
<tr>
<td>4</td>
<td>Endogenous or primary hyperlipidemia</td>
<td>↑VLDL, triglycerides</td>
<td>Unknown</td>
<td>Atherosclerosis</td>
</tr>
<tr>
<td>5</td>
<td>Mixed hyperlipidemia</td>
<td>↑VLDL, chylomicron, cholesterol, ↑triglycerides</td>
<td>Mutations in apoproteinCI gene</td>
<td>Atherosclerosis</td>
</tr>
</tbody>
</table>

34.6.1.1. Hypercholesterolemia

Figure 1.479. Table 4. – Classification of different cholesterol levels. Data are from the website of the American Heart Association:
http://www.heart.org/HEARTORG/Conditions/Cholesterol/AboutCholesterol/What-Your-Cholesterol-Levels-Mean_UCM_305562_Article.jsp

• total cholesterol above 240 mg/dl,
• risk factor for:
  • heart attack,
  • stroke,
  • cardiovascular events,
  • atherosclerosis.
• Caused by:
  • nutrition,
  • genetics,
  • medications,
  • comorbid conditions,
  • metabolic diseases,

34.7. Therapy of lipid metabolism disturbances

A) Dietary:
To reduce
• excess calorie intake,
• consumption of saturated and trans fatty acids,
• exogenous cholesterol intake.

B) Lifestyle changes:
• healthy food intake,
• increasing physical activity.

When the above are unsuccessful, pharmacological treatment may be necessary.

C) Pharmacological treatments – statins
• decreased cholesterol production,
• decreased cholesterol absorption from intestine,
• removing cholesterol from the bloodstream.

Figure 1.480. Figure 5. – Blood collection tube containing clot activator and gel for serum separation used in laboratory tests for lipids

34.8. Test – Oral aspects of lipid metabolism (answers)

1. Which apoprotein is responsible for the activation of lipoprotein lipase?

A. ApoAI
B. ApoB_{100}
C. ApoCII
D. ApoD
E. ApoE

2. Which lipoprotein/s transport/s triglycerides to the periphery to be used for energy?
   A. chylomicron and VLDL
   B. HDL and LDL
   C. LDL – LDL receptor binding pathway
   D. chylomicron remnant
   E. LDL – scavenger pathway

3. What is the essence of reverse cholesterol transport?
   A. transport of cholesterol ingested with food to tissues
   B. transport of cholesterol synthesized by the liver to the cells that synthesize steroid hormones
   C. transport of lipids produced by the liver to tissues
   D. transport of excess cholesterol from the periphery to the liver
   E. cholesterol breakdown in cells and transport of the components back to the liver

References


35. 1.35. Oral aspects of atherosclerosis – Beata Keremi

35.1. Atherosclerosis

Age-related thickening and decreasing elasticity of the arterial wall is called arteriosclerosis. The atherosclerotic plaque formation is atherosclerosis.

Atherosclerosis develops on large elastic arteries and medium-sized elastomuscular arteries. Typical occurrence in order of frequency is:

• abdominal aorta,
• coronary arteries,
• popliteal arteries,
• cerebral circulus arteriosus.

Figure 1.481. Figure 1. – Sclerotic plaque in the carotid arteries. CT and angiography (with permission from Marta Keresztes MD)
Figure 1.482. Figure 2. – Sclerotic plaque in the abdominal aorta and iliac arteries. CT and angiography (with permission from Marta Keresztes MD)
Figure 1.483. Figure 3. – Sclerotic plaque in the femoral and tibial arteries. CT and angiography (with permission from Marta Keresztes MD)
Atheromas occur mainly in areas where the turbulence of blood flow changes, or at sites of vascular damage. These are typically locations of vasculature with high mechanical stress, for example at sites of vascular branching.

Atherosclerosis is a slowly progressing disease whose first symptoms occur in early childhood. Nowadays clinical symptoms are first observed at an ever younger age. The disease is widespread in developed "Western" countries, being one of the main causes of death in North America and Europe.

Figure 1.484. Figure 4. – Stages and clinical symptoms of atherosclerosis. Development of lumen narrowing and occlusion: 1. fat deposits (fatty streak), 2. plaque formation, 3. lesions with complication. Symptoms begin to develop when the affected vessel lumen narrows down to 25% of its original cross section. Symptoms: 1.) Vessel calcification;
2. Ischemia – (myocardial) infarct, ischemic coronary disease; 3. Aneurism formation – rupture; 4. Thrombus formation – embolism, thrombosis; Clinical manifestations: Coronary Heart Disease (CHD): angina pectoris, myocardial infarction, sudden cardiac death; Cerebro Vascular Disease (CVD): transient ischemic attacks, stroke; Peripheral vascular disease: intermittent claudication, gangrene.

35.1.1. Risk factors in the development of atherosclerosis

Figure 1.485. Table 1. – Risk factors of atherosclerosis
Table 1. Risk factors of atherosclerosis

- High LDL-cholesterol levels
  - under 130 mg/dl – low risk
  - 130 to 180 mg/dl – moderate risk
  - over 180 mg/dl – high risk
- Low LDL-cholesterol level
  - under 40 mg/dl – high risk
- Obesity, especially abdominal or visceral type
- Nutrition
- Lack of physical activity
- Hypertension
- Genetic factors
- Smoking
- Diabetes mellitus
- Hyperhomocysteinemia
  - over 14 μg/l
  - missing methylene tetrahydrofolate reductase enzyme
- Environmental factors
- Chlamydia pneumoniae infections
- Deep (≥ 6 mm), untreated periodontal pockets!!!

Figure 1.486. Figure 5. – Cumulative risk factors. The figure shows the most important factors of atherosclerosis. When multiple risk factors are simultaneously present, odds of atherosclerosis do not simply add up but the risk factors potentiate each other’s effect

35.1.2. Development of atherosclerotic plaque
1. **Fatty streak lesion**

   - LDL derived cholesterol ester and other plasma proteins are deposited in the intima, they are found both intra- and extracellularly.

   - Monocytes adhere to and penetrate through the endothelial layer in the early stage of plaque formation. This process is promoted by adhesion molecules, chemokines and pro-inflammatory cytokines (see chapter on inflammation).

2. **Plaque formation**

   - By engulfing oxidized LDL, monocytes are converted into **foam cells**.

   - Monocytes are converted into macrophages that will play a key role in plaque development by releasing enzymes and inflammatory cytokines. The release of hydrolytic enzymes, cytokines (IL-1, IL-6 and TNF), chemokines, and growth factors contributes to localized necrosis, intramural thrombosis and eventually to plaque enlargement. Proinflammatory cytokines inhibit the function of lipoprotein lipase, upregulate adhesion molecules and stimulate mitogenesis and fibrinogen production.

   - This leads to smooth muscle cell proliferation, which promotes the formation of a fibromuscular cap beneath the endothelium. The cap covers the necrotic area, and later it will be responsible for the development of ischemia.

3. **Lesion with complications**

   - Calcification,

   - Ulceration.

**Figure 1.487.** Figure 6. – Development of atherosclerosis. Atherogenic lipoproteins accumulate in the intima in the first phase of the development of atherosclerosis (Figure 2), and this leads to an increase in the number of macrophages (Figure 3), which are converted to foam cells by engulfing LDL (Figure 4). Subsequently, the migration of smooth muscle cells from the media to the intima and the accumulation of foam cells leads to the formation of a fatty streak (Figure 5). This can start as early as during the first decade and may result in the formation of the first lipid deposits. Abnormal accumulation of lipids in the intima continues during the next two to three decades (Figure 6), and eventually leads to the formation of a fibrotic plaque, also known as atheroma, in the fifth decade of life (Figure 7)
Figure 1.488. Figure 7. — The fibrotic core has a necrotic lipid core, separated from the lumen by a thin, fragile fibrotic (connective tissue) cap (Figure 7). This damage can lead to thrombus formation. With advancing age, high amounts of calcium may deposit in the lipid core, further reducing the elasticity of blood vessels. IEL—internal elastic lamina, E—endothelial layer, LDL—low density lipoprotein, ox-LDL—oxidized LDL.

35.2. Theories for atherosclerosis development

Several simultaneously occurring events participate in the formation of atherosclerosis. There are several different hypotheses which are true themselves, however, each of them explains only part of what actually happens.
Figure 1.489. Table 2. – Theories on atherosclerosis development. According to non-lipid theories, the morpho-functional state of the vessel wall is altered which allows the infiltration and deposition of lipoproteins in the intima
### 1. Oral biology

<table>
<thead>
<tr>
<th>Lipid theories</th>
<th>Non lipid theories</th>
</tr>
</thead>
<tbody>
<tr>
<td>✓ Relationship between atherosclerosis and cholesterol</td>
<td>✓ Thrombogenic theory</td>
</tr>
<tr>
<td>• Chemical composition of plaque</td>
<td>• Classic thrombogenic theory – microinjuries on intima – microthrombi</td>
</tr>
<tr>
<td>• Hypercholesterinaemia</td>
<td>• Modern thrombogenic theories – hemostatic system is shifted to enhanced thrombus formation</td>
</tr>
<tr>
<td>✓ „Cellular“ lipid theory</td>
<td>✓ Mesenchymal theory</td>
</tr>
<tr>
<td>• High LDL-cholesterol level in the blood due to increased consumption</td>
<td>• Matrix remodelling (intima and media) promotes deposition of lipids (hypoxia, ageing)</td>
</tr>
<tr>
<td>• Atherogenic lipoproteins</td>
<td></td>
</tr>
<tr>
<td>✓ Atherosclerosis and abnormal levels of serum lipoproteins</td>
<td>✓ Response to injury theory</td>
</tr>
<tr>
<td>• Endothelial injuries ( \Rightarrow ) endothelial dysfunction</td>
<td></td>
</tr>
<tr>
<td>• Chronic inflammation</td>
<td></td>
</tr>
<tr>
<td>• Smooth muscle cell migration from media to intima</td>
<td></td>
</tr>
<tr>
<td>• Smooth muscle cell proliferation in intima</td>
<td></td>
</tr>
<tr>
<td>• Extracellular matrix accumulation</td>
<td></td>
</tr>
<tr>
<td>• Lipid deposition</td>
<td></td>
</tr>
<tr>
<td>✓ Free radical theory</td>
<td></td>
</tr>
<tr>
<td>• Oxidative stress ( \Rightarrow ) free radicals</td>
<td></td>
</tr>
<tr>
<td>• React with collagen and oxidize other components of the vessel wall</td>
<td></td>
</tr>
<tr>
<td>✓ Retention, perfusion theory</td>
<td></td>
</tr>
<tr>
<td>✓ Immunity theory</td>
<td></td>
</tr>
<tr>
<td>• A) Antibodies are formed against vessel wall proteins – autoimmune process (?)</td>
<td></td>
</tr>
<tr>
<td>• B) alterations in LDL molecular structure or certain structures of the aortic wall, triggering the production of antibodies</td>
<td></td>
</tr>
<tr>
<td>• Atherosclerotic plaques always contain gamma-globulins</td>
<td></td>
</tr>
<tr>
<td>• Immune complexes form on the surface of the vessel wall</td>
<td></td>
</tr>
<tr>
<td>✓ Mutagenic (transformation) theory</td>
<td></td>
</tr>
<tr>
<td>✓ Monoclonal theory</td>
<td></td>
</tr>
<tr>
<td>• Inflammation ( \Rightarrow ) inflammatory cascade activation ( \Rightarrow ) atherosclerosis/ tumor (eg. Helicobacter pylori)</td>
<td></td>
</tr>
<tr>
<td>• Angiogenesis</td>
<td></td>
</tr>
<tr>
<td>• Diet (positive / negative effect)</td>
<td></td>
</tr>
<tr>
<td>✓ Infection theory</td>
<td></td>
</tr>
<tr>
<td>• Some pathogens occur preferentially and induce either persistent infection or antibody response in the host</td>
<td></td>
</tr>
<tr>
<td>• Role of CRP (C reactive protein)</td>
<td></td>
</tr>
<tr>
<td>✓ Progenitor cell theory</td>
<td></td>
</tr>
<tr>
<td>✓ „Unifying“ theory</td>
<td></td>
</tr>
<tr>
<td>• Endothelial dysfunction and combined role of LDL</td>
<td></td>
</tr>
</tbody>
</table>
35.3. Oral implications of atherosclerosis – or what is common in dental and atherosclerotic plaques?

Periodontitis and atherosclerosis have complex etiological, genetic and gender predisposing factors, many of which considered as possible risk factors (for example, smoking). The pathomechanisms of these two disorders have much in common: (1) the infection has a direct effect on both atheroma and periodontal plaque formation, (2) the infection induces indirect or host-mediated effects as well in both diseases, (3) common genetic predisposing factors to atherosclerosis and periodontitis, (4) common risk factors, for example lifestyle.

(1) Direct effects include: S. sanguis and P. gingivalis induce platelet aggregation, which is related to thrombus formation. P. gingivalis and other periodontopathogenic strains contribute to extracellular matrix remodeling in the atherosclerotic plaque. Furthermore, serum lipid levels are elevated, which promotes the oxidation of LDL, foam cell formation and eventually atherosclerotic plaque rupture.

(2) Indirect or host-mediated effects: acute phase protein levels are increased in the systemic circulation (C-reactive protein and fibrinogen are both independent risk factors for coronary heart disease). Lipopolysaccharide (LPS) dependent response and monocyte hyperresponsive phenomenon. LPS binds via an LPS binding protein to CD14 receptor, which is found both in a soluble form and on endothelial cells, monocytes and macrophages. This leads to cellular activation. Adhesion molecules, chemokines and cytokines are released during cellular activation. Periodontal microflora promotes atherosclerotic and thromboembolic events by LPS or by inducing the release of inflammatory cytokines (IL-1, TNF-α, IL-6).

35.4. Test – Oral aspects of atherosclerosis (answers)

1. What is not characteristic of atherosclerosis?

A. vessel wall thickening on small blood vessels  
B. decreases oxygenation of the affected tissues  
C. structural changes in the intima  
D. increases the probability of thrombus formation  
E. aneurism rupture

2. What plays a role in the development of atherosclerotic plaque?

A. monocyte migration to the endothelial injury  
B. hypoxia, endothelial dysfunction  
C. free radicals  
D. none of them  
E. all of the above

3. Which are the risk factors for atherosclerosis?

A. decreased homocysteine level  
B. high HDL cholesterol level  
C. high oxLDL level  
D. deep, untreated periodontal pockets  
E. Chlamydia trachomatis infection

References


### 36. 1.36. Pain sensation – oral aspects – Gabor Varga

A precise definition of pain is difficult to find as both objective and subjective, emotional elements may be involved in its perception. According to the International Association for the Study of Pain, pain is defined as “an unpleasant sensory and emotional experience arising from actual or potential tissue damage or described in terms of such damage.”

#### Figure 1.490. Figure 1. – Pain

Pain, according to its origin, can be divided into four groups. Nociceptive pain is initiated in the primary afferent nerve fibers in response to mechanic, thermal or chemical stimuli. According to the location of the stimulus, somatic and visceral pain can be distinguished. The latter is hard to localize and often presents as a perception of somatic pain. Neuropathic pain usually occurs in response to tissue and organ lesions and central nervous system disorders, and can be attributed to disorders in sensory functions. Psychogenic pain is not attributable to periphery or to lower levels of the sensory system, it results from the activity of higher brain centers and the cortex.

#### Figure 1.491. Figure 2. – Classification of pain types
According to duration, pain can be divided into two groups. Acute pain is generally well localized and proportional to the duration and extent of the underlying trauma. It is usually well treatable and ceases with the elimination of the noxious agent. On the contrary, chronic pain is usually prolonged and a well-defined noxious agent is not necessarily identifiable. Therefore, it is harder to treat and often refractory to treatment. Sports injuries often underly acute pain while persistent inflammatory conditions such as rheumatic pain are good examples for chronic pain.

**Figure 1.492. Figure 3. – Acute Pain vs. Chronic Pain**

<table>
<thead>
<tr>
<th>Acute Pain</th>
<th>Chronic Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usually sudden, self-limiting, less than 6 months</td>
<td>May be sudden or gradually developing, with periods of remission, longer than 6 months</td>
</tr>
<tr>
<td>Precipitating event</td>
<td>May not be associated with injury</td>
</tr>
<tr>
<td>Resolves with treatment</td>
<td>Difficult to treat</td>
</tr>
<tr>
<td>Restless, anxious, crying</td>
<td>Depressed, withdrawn</td>
</tr>
</tbody>
</table>

**Figure 1.493. Figure 4. – Acute Pain vs. Chronic Pain**

The perception of external impacts is facilitated by several complex sensory systems in the skin, where the activation of nerve endings is facilitated by sensory structures. This is not true for pain, however, which is initiated in free nerve endings that lack complex sensory structures.

**Figure 1.494. Figure 5. – Description of skin receptors**
Two types of nerve fibers are involved in nociception. Aδ fibers mediate acute pain. They are myelinated with a moderate conduction velocity, and use glutamate as the neurotransmitter. Type C fibers are unmyelinated with a slow conduction velocity. They convey diffuse pain sensations using glutamate and substance P as neurotransmitters. They mostly mediate blunt, chronic pain.

**Figure 1.495. Figure 6. – Characterization of nociceptors**

<table>
<thead>
<tr>
<th>Stimulus</th>
<th>Afferent fiber</th>
<th>Drive speed</th>
<th>Transmitter</th>
<th>Modality</th>
</tr>
</thead>
<tbody>
<tr>
<td>strong, mechanical, thermo</td>
<td>Aδ</td>
<td>fast (12-30 m/s)</td>
<td>glutamate</td>
<td>localized, acute pain</td>
</tr>
<tr>
<td>at cell damage K⁺, bradykinin, histamine, serotonin</td>
<td>C</td>
<td>slow (0.5-2 m/s)</td>
<td>glutamate and substance P</td>
<td>diffuse, chronic pain</td>
</tr>
</tbody>
</table>

Direct activators and bioactive substances potentiating the effects of activators are involved in the activation of nociceptors. The former group includes potassium released upon cell death, serotonin, histamine and bradykinin. The latter category includes prostaglandins, leukotrienes, substance P (SP) and calcitonin gene-related peptide (CGRP).

**Figure 1.496. Figure 7. – Activation and sensitization of pain receptors**

<table>
<thead>
<tr>
<th>Substance</th>
<th>Origin</th>
<th>Substance</th>
<th>Origin</th>
</tr>
</thead>
<tbody>
<tr>
<td>K⁺</td>
<td>Damaged cells</td>
<td>Prostaglandins</td>
<td>Damaged cells</td>
</tr>
<tr>
<td>5-HT</td>
<td>Activated thrombocytes</td>
<td>Leukotrienes</td>
<td>Damaged cells</td>
</tr>
<tr>
<td>Histamine</td>
<td>Mast cells</td>
<td>Substance P</td>
<td>Polymodal primary afferents</td>
</tr>
<tr>
<td>Bradykinin, bradykinin</td>
<td>Plasma kininogen (effect of kallikrein)</td>
<td>CGRP</td>
<td>Polymodal primary afferents</td>
</tr>
</tbody>
</table>

Identifying the source of the pain is often difficult. This may be caused by radiating pain. Primary afferent nerves from the surface of the body and from the viscera activate the same secondary sensory nerves after synapsing in the dorsal horn. Such areas on the surface of the body, corresponding to distinct visceral locations, are called Head zones.
Proprioceptive and nociceptive nerve pathways diverge at the spinal cord level. Fibers that sense pressure ascend the spinal cord ipsilaterally and decussate only after synapsing in the medullary area. Primary nociceptive fibers meanwhile send collaterals to dorsal horn nociceptive synapses, inhibiting neurotransmission there. This can be considered as inhibition from the periphery. Primary afferents synapse in the dorsal horn, secondary nerve fibers decussate, project to the thalamus where they synapse again, and peripheral information reaches the cortex via tertiary neurons. Pain is actually perceived in the cortex.

Descending analgesic pathways play an important role in the modulation of nociception. The opioid pathway descending from the periaqueductal gray matter synapses in the medulla, then in the dorsal horn synapses it inhibits neurotransmission both presynaptically by inhibiting transmitter release from primary sensory neurons and postsynaptically by inhibiting the action potential of secondary neurons.
The gate control theory is essentially the description of the modulatory effects of peripheric proprioceptive and descending analgesic pathways on transmission of afferent stimuli. Inhibitory systems can therefore significantly inhibit the perception of afferent stimuli.

**Figure 1.500. Figure 11. – Gate control theory for suppression of peripheral nociceptive signals**

Sensitization is essentially a left shifting of the neural response curve elicited by a stimulus. The sensitization of perception, i.e. an increased neural activity in response to the same stimulus is called **hyperesthesia**. Increased pain in response to the same stimulus is **hyperalgesia**. A decreased pain threshold is **allodynia**. On the other hand, decreased sensation is called **anesthesia**, while a decrease in the perception of pain in response to the same stimulus is called **analgesia**.

**Figure 1.501. Figure 12. – Sensitization of nociceptive stimuli and inhibitory mechanisms**
In dental practice it is highly relevant that the inflammatory reaction induced by tissue injury causes sensitization: even a light touch will cause severe pain in a patient with pulpitis.

Nociceptive activation will at the same time induce local release of CGRP and substance P, resulting in vasodilation, which will in turn increase plasma infiltration into the interstitial space. Mast cell activation initiates prostaglandin release, which will induce sensitization of nociception. This is accompanied by local changes in tissue gene expression and activation of the immune system.
From the above facts it follows that pain relievers target elements of sensitization or inhibition of nociception. These are peripheric analgesia and anesthesia, the inhibition of dorsal horn synaptic transmission by activating peripheric proprioception and opioid descending pathways, the inhibition of the transmission of ascending stimuli, and inhibition of the elements of nociception at thalamic and cortical level.

**Figure 1.504. Figure 15. – Pain relief – major targets**

![Diagram showing pain relief major targets](image)

Analgesics exert their effect through mechanisms described above. One of the major targets is the inhibition of sensitization mechanisms by COX enzyme inhibition through the use of non-steroidal anti-inflammatory drugs (NSAIDs, non-selective COX inhibitors) or selective COX-2 inhibitors. The other option is opioid analgesia using non-addictive opioid analgesics.

**Figure 1.505. Figure 16. – Classification of analgesics**

<table>
<thead>
<tr>
<th>Classification of analgesics</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Non-opioid (non-narcotic) analgesics</strong></td>
</tr>
<tr>
<td>- Acetaminophen (Cox-3 enzyme inhibition)</td>
</tr>
<tr>
<td>- NSAIDS (non-steroid antiinflammatory drugs): Aspirin, Advil, Motrin, Naprosyn, Feldene, Toradol (Ketorolac)</td>
</tr>
<tr>
<td>- Cox – 2 Inhibitors (Vioxx &amp; Celebrex)</td>
</tr>
<tr>
<td>Side Effects: Gastric erosion, GI bleeding, fluid retention, Platelet dysfunction, &amp; renal insufficiencies</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Opioid Analgesics: Synthetic Narcotics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Commonly Used:</td>
</tr>
<tr>
<td>- Morphine Sulfate, Oxycodone</td>
</tr>
<tr>
<td>- Dilaudid (hydromorphone)</td>
</tr>
<tr>
<td>- Oxycodone (Percodan, Percocet, Oxycodin SR)</td>
</tr>
<tr>
<td>- Demerol (Mepiphenide)</td>
</tr>
<tr>
<td>- Fentanyl</td>
</tr>
<tr>
<td>- Codeine Plain</td>
</tr>
<tr>
<td>- Tylenol</td>
</tr>
<tr>
<td>- Vicodin (Hydromorphone)</td>
</tr>
</tbody>
</table>

COX-2 inhibitor analgesics are of special note. While NSAIDs inhibit the synthesis of prostaglandins and leukotrienes (key players in sensitization) by non-selective COX inhibition, COX-2 inhibitors selectively target the isoform that is induced locally during inflammation.
Dental practice and the concept of pain are often connected in popular thought and in public discourse. Frequently it is the pain that directs the patient to the dental office. On the other hand, fear of pain often keeps patients away until it is too late and they are left with the only option of tooth removal rather than conservative treatment.

The main causes of dental pain are caries, abscesses, gum disease and root irritation. In addition, severe pain may result from tooth fracture, temporo-mandibular disorders, an impacted wisdom tooth, but also the natural process of tooth eruption.

36.1. Test – Pain sensation – oral aspects (answers)

1. Primary afferent fibers involved in pain development:

A. A-alpha and A-beta fibers
1. Oral biology

B. A-delta and C fibers
C. A-epsylon fibers
D. all of the above
E. none of them

2. Peptide transmitter releasing from nociceptive sensory nerves
A. cholecystokinin
B. CGRP
C. secretin
D. gastrin
E. noradrenaline/norepinephrine

3. Meaning of „allodynia”:
A. Decreased threshold of pain activation
B. Increased pain response over threshold level
C. Inductive involvement of neighbouring nerve endings in neuronal transmission
D. Decrease of spontaneous activity
E. Increase of spontaneous activity

37. 1.37. Oral aspects of endocrine disorders – Dezso Szombath

Of the disorders of the hypothalamic-pituitary-target gland axes, disorders of growth hormone (GH-IGF-1) secretion is of the greatest interest to a dentist.

Figure 1.509. Figure 1. – Hypothalamus – pituitary gland – target gland axis

37.1. Growth hormone

Acromegaly, resulting from GH-IGF-1 overproduction, is a disease most frequently seen in middle-aged patients, with primary hormone secreting pituitary adenoma as the underlying cause. Permanent dysregulated hormone production leads to physical alterations gradually developing in the head and neck area as well:
circumference of the head will increase due, in part, to increased thickness of the skin and underlying tissues, and to an increased volume of the bony skull. The presence of a thick and oily facial skin results in deep nasolabial wrinkles, highlighted by thick lips. Facial features are further distorted by a prominent supraorbital ridge, a widened and thickened nose and prognathism. Also characteristic is distraction, a prominent diastema, temporomandibular arthritis and macroglossia.

Patients require increased dental care due to occlusion disorder and diabetes mellitus developing as a consequence of growth hormone overproduction.

Decreased height and delayed dentition or tooth transition may direct the attention to growth hormone deficiency in case it remains hidden initially.

Untreated congenital growth hormone deficiency results in gingival recession and pocket formation by adulthood due to the lack of trophic effects and decreased efficiency of the immune system.

A less frequent cause of growth hormone deficiency is hypothalamus injury.

**Figure 1.510. Table 1. – Symptoms of hypothalamic damage**

<table>
<thead>
<tr>
<th>Eating disorders</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Anorexia</td>
<td></td>
</tr>
<tr>
<td>Hyperphagia</td>
<td></td>
</tr>
<tr>
<td>Bulimia</td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td></td>
</tr>
<tr>
<td>Fluid intake</td>
<td></td>
</tr>
<tr>
<td>Absence of thirst</td>
<td></td>
</tr>
<tr>
<td>(adipsia, consequential hyponatremia)</td>
<td></td>
</tr>
<tr>
<td>Excessive thirst</td>
<td></td>
</tr>
<tr>
<td>(polydipsia)</td>
<td></td>
</tr>
<tr>
<td>Thermoregulation</td>
<td></td>
</tr>
<tr>
<td>Hypothermia</td>
<td></td>
</tr>
<tr>
<td>Hyperthermia</td>
<td></td>
</tr>
<tr>
<td>Disorders of consciousness and sleep-wake</td>
<td></td>
</tr>
<tr>
<td>Somnolence, coma</td>
<td></td>
</tr>
<tr>
<td>Inverted sleep-waking rhythm</td>
<td></td>
</tr>
<tr>
<td>Akinetic mutism</td>
<td></td>
</tr>
<tr>
<td>Psychiatric disorders</td>
<td></td>
</tr>
<tr>
<td>Emotional lability</td>
<td></td>
</tr>
<tr>
<td>Anger outburst</td>
<td></td>
</tr>
<tr>
<td>Behavioural disorders, inadequate emotional reaction</td>
<td></td>
</tr>
<tr>
<td>Hallucination</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
</tr>
<tr>
<td>Heart failure, arrhythmia</td>
<td></td>
</tr>
<tr>
<td>Lung oedema</td>
<td></td>
</tr>
<tr>
<td>Decreased tone of anal and urethral sphincters</td>
<td></td>
</tr>
</tbody>
</table>

Growth hormone releasing hormone hyposecretion results in symptoms characteristic of hypothalamic dysfunction in addition to alterations seen in growth hormone deficiency.

**Figure 1.511. Table 2. – Most common causes of hypothalamic dysfunction**
37.2. Adrenal cortex

Of the hypothalamic-pituitary-adrenal cortex axis dysfunctions oral symptoms of primary adrenal cortex deficiency and consequences of increased hypothalamus-pituitary-adrenal cortex activity induced by permanent emotional stress are of interest from a dentist's point of view.

Cronic primary adrenal cortex deficiency (Addison's disease) is a rare condition. The underlying harmful factor (autoimmune disease, tuberculosis, etc.) causes destruction leading to decreased hormone secretion in all three layers of adrenal cortex. Low peripheral cortisol results in constantly elevated ACTH secretion, at levels high enough to stimulate melanocytes. Hyperpigmentation occurs all over the body, including oral mucosa and tongue. Hyperpigmented patches are seen on the buccal mucosa, gingival, tongue, and even on lips.

Adrenal cortex hyperfunction (hypercortismus), when inducing predominantly increased cortisol secretion, can lead to dental alterations via secondary diabetes and osteoporosis.

Figure 1.512. Table 3. – Side effects of corticosteroid therapy on skeletal system (doses above 10 mg/day)

<table>
<thead>
<tr>
<th>Table 3. Side effects of corticosteroid therapy on skeletal system (doses above 10 mg/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• osteoblast activity is inhibited</td>
</tr>
<tr>
<td>• intestinal calcium absorption is inhibited (interaction with 1,25-(OH)₂-D₃ vitamine)</td>
</tr>
<tr>
<td>• increased calcium excretion in kidney</td>
</tr>
<tr>
<td>• secondary hyperparathyreosis</td>
</tr>
<tr>
<td>• increased osteoclast activity (via increased production of IL-1)</td>
</tr>
<tr>
<td>• hypogonadism</td>
</tr>
<tr>
<td><strong>Overall result:</strong> decreased rate of bone formation and trabecular bone density.</td>
</tr>
</tbody>
</table>

Hypercorticism is associated with a wide range of symptoms (Cushing’s syndrome), which can be caused by ACTH producing pituitary adenoma (Cushing’s disease), primary adrenocortical hyperplasia, or ectopic ACTH production (lung cancers).

Figure 1.513. Table 4. – Clinical symptoms of Cushing syndrome, in order of frequency
A moderate but prolonged increase of cortisol levels in emotional stress decreases the efficiency of the immune response. This involves a shifting of the immune response to a Th2 (humoral) direction with a decrease in Th1 cellular immunity. Stress-induced activation of the hypothalamic-pituitary-adrenal axis affects catecholamine release by the adrenal medulla, subjecting peripheral tissues to a dual, cortisol-catecholamine stress hormone effect. This reduces local cellular defense further, and, by degranulating tissue mast cells, can generate a local inflammatory reaction or reactivate persistent inflammatory foci. Stress hormones, by modulating local cytokine production, induce a shift toward inflammation (Th1↓, Th2↑ histamine release). These proinflammatory effects aggravate periodontal damage and result in a greater attachment loss in individuals suffering from stress.

Of note, cortisol and its analogs are important therapeutic tools. In pharmacological doses, they rapidly inhibit systemic inflammatory response and may save lives in a number of conditions such as asthma cardiale or laryngeal edema.

**Aldosteron** overproduction results in alterations in saliva ionic composition, i.e. a decrease in the sodium to potassium ratio due to increased ductal sodium reabsorption. This is in contrast with the increasing sodium to potassium ratio observed in serum, and may have a diagnostic relevance.

### 37.3. Thyroid

Thyroid hypofunction during tooth development leads to dental disorders. Low T3/T4 levels will delay tooth development with increased frequency of dentitio tarda and malocclusion. The most severe forms occur in cretenism (in areas with iodine deficiency).
Hypothyroidism in adults is a risk factor for the development of severe periodontitis leading to alveolar bone loss.

37.4. Test – Oral aspects of endocrine disorders (answers)

1. What is the most likely diagnosis based on the following symptoms: apparent diastema, temporomandibular arthritis, distraction, macroglossia?
A. congenital growth hormone deficiency
B. ectopic ACTH syndrome
C. GH-IGF-1 secretory dysfunction
D. hyperaldosteronism combined with vitamin D deficiency
E. adult hypothyroidism

2. Oral cavity alterations characteristic of Addison’s disease:
A. patchy pigmentation on the tongue
B. pigmentation all over the body including the gingiva
C. retarded dentition
D. prognathia
E. decreased salivation with canker sores

3. Retarded dentition with malocclusion is frequent in the following conditions:
A. hyperthyroidism, Cushing syndrome
B. Conn’s syndrome, acromegaly
C. congenital hypothyroidism, cretenism
D. congenital growth hormone deficiency, hypothalamic dysfunction
E. maternal chronic stress, primary adrenocortical hyperplasia

References
1.38. Disorders of respiratory functions – Beata Keremi

Diseases of the respiratory tract account for more visits with general practitioners than any other systems in the body. Increases in pollution, new industrial processes and the growing worldwide consumption of tobacco all have implications for the lungs.

The respiratory tract extends from the nose to the alveoli and includes not only the air-conducting passages but the blood supply as well. The left lung is divided into two lobes, an upper and a lower, while the right lung is divided into three lobes, the superior, middle and inferior lobes.

38.1. Lung defense

The lung is exposed to 6 litres of potentially infected and irritant-laden air every minute. There are, therefore, numerous defense mechanisms to ensure survival.

Figure 1.514. Table 1. – Defense mechanisms in the lung
38.2. Lung function

The function of the lung is to oxygenate the blood and to remove carbon dioxide. To achieve this, ventilation of the lungs is performed by the respiratory muscles under the control of the respiratory centre in the brain. The rhythm of breathing depends on various inhibitory and stimulatory mechanisms within the brainstem. These can be influenced voluntarily by higher centers and by the effect of chemoreceptors. The medullary or central chemoreceptors in the brainstem respond to changes in partial pressure of carbon dioxide in the blood ($P_{CO2}$). Chemoreceptors in the aortic and carotid body respond to low partial pressure of oxygen ($P_{O2}$) but only when this falls below 8 kPa. Thus, alteration in $P_{CO2}$ is the most important factor in respiratory control in health. The sensitivity of the medullary chemoreceptor to $P_{CO2}$ can be reset either upwards in prolonged ventilatory failure or downwards, as in patients placed on a mechanical ventilator. The first situation is most commonly seen in chronic airflow limitation (chronic obstructive lung disease) when patients may become dependent on hypoxic drive to maintain respiration. In the second situation, ‘weaning’ a patient away from a ventilator is difficult because the medullary centre demands a low $P_{CO2}$ that cannot be maintained by the patient unaided.

38.3. Assessing of respiratory function

Two fairly straightforward techniques, radiography and spirometry (the analysis of the volume of expired air over time), illustrate normality and help the physician to understand the abnormal. Some other tests also exist to measure static lung volume, ventilation or dynamic lung volume and gas exchange across the alveolar-capillary membrane.

Figure 1.515. Figure 1. – Normal x-ray view of the lung
38.3.1. Static lung volume

Figure 1.516. Figure 2. – Lung volumes. The inspiratory rate depends on chest wall distensibility and the elasticity of the lung tissue. *Total lung capacity* (TLC) is the largest inspiratory capacity, which depends on the elasticity of the lungs. TLC is reduced in fibrosis and in diseases associated with scarring of the lung tissue. TLC is increased in emphysema, asthma and chronic bronchitis. In the latter two cases, excessive lung expansion promotes airway dilation. *Functional residual capacity* is the volume of air present in the lungs after a passive exhalation when no more passive chest contraction is possible. It is the end of normal expiration. Further exhalation requires the active work of respiratory muscles. *Residual volume* (RV) is the air volume remaining in the lungs after maximal expiratory effort. RV is increased in emphysema because small airways lose their flexibility and they collapse during exhalation, and in chronic bronchitis which is characterized by the contraction of small airways or inflammation. This is because the increased pressure in the lung tissue helps keep the airway open so they will close later during expiration, thus reducing the residual volume. Vital capacity (VC) depends on the relative changes in RV and TLC, but is generally decreased in lung diseases
38.3.2. Gas exchange

Transfer factor (TF) is a measurement of gas transfer across the alveolar-capillary membrane. For technical reasons carbon monoxide is used as the test gas but oxygen behaves in a similar way. TF is reduced when there is destruction of the alveolar-capillary bed, as in emphysema, and also when there is a barrier to diffusion. This may occur when the alveolar-capillary membrane is thickened or where there is lack of homogeneity in the distribution of blood and air at the alveolar level. Both mechanisms are important in lung fibrosis. The TF will naturally be reduced if the lungs are small or if one has been removed (pneumonectomy). The transfer coefficient (KCO or DLCO divided by alveolar volume, calculated separately) is a more useful measurement because it reflects the true situation in the ventilated lung.

38.4. Lung disease

38.4.1. Dyspnoea

Most lung diseases will cause dyspnoea or difficulty in breathing. It may not be immediately clear whether patients refer to breathlessness or pain. If the complaint is in fact a pain then this may well be angina, which is in itself associated with breathlessness. Some patients with pleuritic pain complain of breathlessness, but what they really mean is that they are unable to take a deep breath because of pain.

38.4.1.1. Causes of breathlessness

Figure 1.517. Table 2. – Causes of breathlessness
1. Oral biology

<table>
<thead>
<tr>
<th>Table 2. Causes of breathlessness:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control and movement of the chest wall and pleura:</strong></td>
</tr>
<tr>
<td>➢ hyperventilation syndrome</td>
</tr>
<tr>
<td>➢ hypotalamic lesion</td>
</tr>
<tr>
<td>➢ neuromuscular disease</td>
</tr>
<tr>
<td>- spinal cord disease</td>
</tr>
<tr>
<td>- neuropathies (e.g. Guillain-Barré’s syndrome)</td>
</tr>
<tr>
<td>- myopathies</td>
</tr>
<tr>
<td>➢ kyphoscoliosis</td>
</tr>
<tr>
<td>➢ Bechterew’s disease</td>
</tr>
<tr>
<td>➢ pleural effusion and thickening</td>
</tr>
<tr>
<td>➢ bilateral diaphragm paralysis</td>
</tr>
<tr>
<td><strong>Lung diseases:</strong></td>
</tr>
<tr>
<td>➢ airway diseases:</td>
</tr>
<tr>
<td>• chronic bronchitis and emphysema</td>
</tr>
<tr>
<td>• asthma</td>
</tr>
<tr>
<td>• bronchiectasia</td>
</tr>
<tr>
<td>• cystic fibrosis</td>
</tr>
<tr>
<td>➢ parenchymal diseases:</td>
</tr>
<tr>
<td>• pneumonia</td>
</tr>
<tr>
<td>• cryptogenic fibrosing alveolitis</td>
</tr>
<tr>
<td>• extrinsic allergic alveolitis</td>
</tr>
<tr>
<td>• primary and secondary tumors</td>
</tr>
<tr>
<td>• sarcoidosis</td>
</tr>
<tr>
<td>• pneumothorax</td>
</tr>
<tr>
<td>• pulmonary oedema</td>
</tr>
<tr>
<td>➢ reduced blood supply:</td>
</tr>
<tr>
<td>• pulmonary embolism</td>
</tr>
<tr>
<td>• anemia</td>
</tr>
</tbody>
</table>

**Figure 1.518. Table 3. – Duration of breathlessness**

<table>
<thead>
<tr>
<th>Immediate: (minutes)</th>
<th>Short: (hours to days)</th>
<th>Long: (weeks to years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• pulmonary embolism</td>
<td>• pulmonary oedema</td>
<td>• chronic airflow limitation</td>
</tr>
<tr>
<td>• pneumothorax</td>
<td>• pneumonia</td>
<td>• cryptogenic fibrosing alveolitis</td>
</tr>
<tr>
<td>• pulmonary oedema</td>
<td>• asthma</td>
<td>• extrinsic allergic alveolitis</td>
</tr>
<tr>
<td>• asthma</td>
<td>• pleural effusion</td>
<td>• anemia</td>
</tr>
<tr>
<td></td>
<td>• anemia</td>
<td></td>
</tr>
</tbody>
</table>

**38.4.1.2. Asthma**

**Figure 1.519. Table 4. – Allergic and non-allergic factors in asthma**

<table>
<thead>
<tr>
<th>Table 4. Allergic and non-allergic factors in asthma:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allergic</td>
</tr>
<tr>
<td>• house dust mite</td>
</tr>
<tr>
<td>(especially cats)</td>
</tr>
<tr>
<td>• animals</td>
</tr>
<tr>
<td>(especially cats)</td>
</tr>
<tr>
<td>• pollens (especially grass)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

**38.4.1.3. Orthopnoea and paroxysmal nocturnal dyspnoea**

Orthopnoea and paroxysmal nocturnal dyspnoea need special consideration. Both are usually regarded as manifestations of left ventricular failure. Orthopnoea is defined as breathlessness lying flat which is relieved by sitting up. It is common in patients with severe fixed airways obstruction, as in some chronic bronchitics who may admit to not having slept flat for years. Normal people, when they lie flat, breathe more with the diaphragm and less with the chest wall. In patients with airways obstruction, the diaphragm is often flat and inefficient and may even draw the ribs inwards rather than out. Thus, when they lie down the diaphragm cannot provide the ventilation required. The term paroxysmal nocturnal dyspnoea is self explanatory and is a feature of...
pulmonary oedema from left ventricular failure. However, many asthmatics develop bronchoconstriction in the night and wake with wheeze and breathlessness very similar to the symptoms of left ventricular failure.

38.4.1.4. The hyperventilation syndrome

The hyperventilation syndrome is more common than is generally realised but produces a distinct pattern of symptoms. It is usually associated with anxiety and patients overbreathe inappropriately. The initial complaint is often, although not always, of breathlessness. The hyperventilation is the response to this sensation. Hyperventilation induces a reduction in the $P_{CO2}$, creating a variety of other symptoms.

Figure 1.520. Table 5. – Accompanying symptoms of hyperventilation

<table>
<thead>
<tr>
<th>Accompanying symptoms of hyperventilation:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• paraesthesiae in the fingers</td>
</tr>
<tr>
<td>• tingling around the lips</td>
</tr>
<tr>
<td>• dizziness</td>
</tr>
<tr>
<td>• lightheadedness</td>
</tr>
<tr>
<td>• frank tetany</td>
</tr>
</tbody>
</table>

The diagnosis can be confirmed by the presence of smoking-related airflow limitation for over a decade.

38.4.1.5. Dyspnoea and hypoxia

Many patients with airflow limitation from chronic bronchitis have hypoxia severe enough to cause right-sided heart failure, yet they have relatively little dyspnoea (blue bloaters). In contrast, some patients with emphysema seem to need to keep their blood gases normal by a heroic effort of breathing (pink puffers); they are very dyspnoeic.

38.4.2. Cough

Cough arises from the cough receptors in the pharynx, larynx and bronchi; cough, therefore, results from irritation of these receptors by infection, inflammation, tumor or a foreign body.

Figure 1.521. Table 6. – Causes of cough

<table>
<thead>
<tr>
<th>Causes of cough:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• asthma - particularly childhood asthma</td>
</tr>
<tr>
<td>may be accompanied by sneezing and nasal blockage</td>
</tr>
<tr>
<td>• postnasal drip from rhinitis</td>
</tr>
<tr>
<td>• laryngitis</td>
</tr>
<tr>
<td>• recurrent laryngeal nerve plasty</td>
</tr>
<tr>
<td>• tracheitis</td>
</tr>
<tr>
<td>• bronchitis</td>
</tr>
<tr>
<td>• bronchiectasis</td>
</tr>
<tr>
<td>• pneumonia</td>
</tr>
<tr>
<td>• carcinoma</td>
</tr>
<tr>
<td>• lung fibrosis</td>
</tr>
<tr>
<td>• increased bronchial responsiveness</td>
</tr>
<tr>
<td>• aspiration into the lungs from gastro-oesophageal reflux</td>
</tr>
<tr>
<td>• pharyngeal pouch</td>
</tr>
</tbody>
</table>

38.4.3. Sputum

It is the result of excessive bronchial secretion; itself being a manifestation of inflammation and infection. Sputum production is common in asthmatics and is occasionally the main complaint.

Figure 1.522. Table 7. – Characterization of sputum
38.4.4. Haemoptysis

The coughing up of blood is often a sign of serious lung disease. It is common in trivial respiratory infections. The blood in haemoptysis is usually bright red at first, then followed by progressively smaller and darker amounts.

Figure 1.523. Table 8. – Haemoptysis

<table>
<thead>
<tr>
<th>Table 8. Haemoptysis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Common</strong></td>
</tr>
<tr>
<td>infection including bronchiectasis</td>
</tr>
<tr>
<td>bronchial carcinoma</td>
</tr>
<tr>
<td>tuberculosis</td>
</tr>
<tr>
<td>pulmonary embolism and infarction</td>
</tr>
<tr>
<td>no cause found</td>
</tr>
</tbody>
</table>

38.4.5. Pain

The lungs and the visceral pleura are devoid of pain fibres, whereas the parietal pleura, chest wall and mediastinal structures are not.

Figure 1.524. Table 9. – Pleuritic pain
Although the lungs are insensitive to pain, the mediastinal structures are not. Cancer of the lung and other central lesions produce a dull, poorly localized pain, presumably from pressure on mediastinal structures.

### 38.4.6. Wheeze

It occurs both during aspiration and expiration but is always louder in the latter. It implies airway narrowing and is, therefore, common in asthma and chronic obstructive bronchitis. In asthma, the wheeze is episodic and clearly associated with shortness of breath. Nevertheless, some asthmatics may have little wheeze and acute severe attacks can be associated with a ‘silent chest’. In chronic obstructive bronchitis and emphysema, the associations are less clear-cut, with wheeze, shortness of breath, cough and sputum occurring in various proportions.

### 38.4.7. Stridor

Stridor is a harsh aspiratory and expiratory noise which can be imitated by adducting the vocal cords and breathing in and out.

### 38.5. Test – Disorders of respiratory functions (answers)
1. What is sensed by the brainstem chemoreceptors?

A. oxygen partial pressure in blood  
B. carbon dioxide partial pressure in blood  
C. oxygen partial pressure in the environment  
D. carbon dioxide partial pressure in the environment  
E. carbon monoxide partial pressure in blood

2. Which is correct?

A. total lung capacity = vital capacity + expiratory reserve  
B. vital capacity > tidal volume > residual volume  
C. vital capacity = functional residual capacity + tidal volume  
D. vital capacity = inspiratory + expiratory reserve volumes + tidal volume  
E. vital capacity > residual volume > functional residual capacity

3. Which are possible reasons for breathlessness?

A. hyperventilation syndrome  
B. neuro- and/or myopathies  
C. parenchymal diseases  
D. all of the above  
E. none of the above


Oral clearance is the sum of processes that shift the oral environment toward a non-cariogenic one. It includes all the physical and chemical processes that take place in the mouth after ingesting the food.

Materials in the oral cavity are diluted in freshly secreted saliva, and later while swallowed. This process is very similar to serial dilution. Elimination of food / nutrients from the saliva, and thus from the oral cavity, is called oral clearance.

This means that the rate of oral clearance determines how much fermentable substrate (sugar) is available to plaque bacteria.

39.1. Factors determining oral clearance rate

The most important factors are:

1. saliva secretion (flow rate),  
2. swallowing habit,  
3. breathing habit,  
4. general state of health,  
5. nutrition – food components.

39.1.1. Flow rate of saliva secretion
1. Oral biology

**Figure 1.525. Table 1.** – Flow rate and composition of unstimulated and stimulated saliva. Based on Fergusson (1999) and Whelton (2004). Stimulated saliva contains buffers (bicarbonate, phosphate, protein, and ammonia) in large quantities compared to resting saliva. The high buffering capacity of stimulated saliva helps to maintain a higher pH.

<table>
<thead>
<tr>
<th></th>
<th>Unstimulated saliva</th>
<th>Stimulated saliva</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flow rate (ml/min)</td>
<td>0.3-0.4</td>
<td>1.0-3.0</td>
</tr>
<tr>
<td>Borderline of xerostomia (ml/min)</td>
<td>&lt; 0.1</td>
<td>&lt; 0.7</td>
</tr>
<tr>
<td>pH</td>
<td>5.7-7.1</td>
<td>over 7.8</td>
</tr>
<tr>
<td>Bicarbonate (mmol/l)</td>
<td>0.1-8.0</td>
<td>4.0-40.0</td>
</tr>
<tr>
<td>Phosphate (mmol/l)</td>
<td>2.0-22.0</td>
<td>1.5-25.0</td>
</tr>
<tr>
<td>Protein (g/l)</td>
<td>1.7</td>
<td>1.0-6.4</td>
</tr>
<tr>
<td>Ammonia (mmol/l)</td>
<td></td>
<td>0.8-7.0</td>
</tr>
</tbody>
</table>

The effect of saliva on plaque pH is determined solely by its buffering capacity (bicarbonate concentration). Acid production by bacteria is pH dependent. The pH optimum of several plaque bacteria is lower than 5.0, which prefers lactic acid production. Minimum plaque pH is highly dependent on plaque thickness. pH is always lower at the inner plaque surface (i.e., at the interface between plaque and dental enamel).

When dental plaque is very thin, acids diffuse in easily into it before an increase in the quantity of saliva.

### 39.1.2. Swallowing habit

- Swallowing frequency and volume.
- Residual volume, maximal volume.

Swallowing frequency is determined by the amount of saliva in mouth. Therefore, the rate of saliva secretion is an important parameter that can change over a wide range. The greater the swallowing frequency, the faster the elimination of substances from the mouth.

Residual volume is that of the saliva remaining in the mouth after swallowing. Maximum volume is that in the oral cavity immediately before swallowing. Accumulation of this volume of saliva in the mouth automatically triggers the swallowing reflex.

Differences in residual volumes may be responsible for the observed individual differences in oral clearance. Those who are efficient in swallowing (low residual volume), the depletion of nutrients in the oral cavity will be very fast.

When a large amount of saliva does not accumulate in the oral cavity before swallowing, the maximum volume will be lower, the elimination will be thus much faster as swallowing occurs at a smaller volume.

A high residual volume is generally associated with a higher maximum volume.

### 39.1.3. Breathing habit

In adults, nasal breathing is typically normal. When it is obstructed, mouth breathing becomes predominant. Mouth breathing is associated with a number of disadvantages:

- dry mouth → microinjuries on the mucosa → increased susceptibility to infection,
- sensitivity to upper respiratory tract infections,
- gingivitis,
1. Oral biology

- dental abnormalities – for example, outward inclined teeth.

39.1.4. General state of health

- Pre-existing diseases

  They may contribute to changes in salivation and can significantly affect the amount of saliva – for example, Diabetes Mellitus.

- Medications:

  A number of widely used drugs affect the composition and amount of saliva. Examples include antidepressants, of the anti-hypertensive agents particularly beta blockers, and some medicines for allergy.

39.1.5. Food consumed

From a cariological aspect, the quality (i.e., whether simple or complex) rather than the quantity of carbohydrates consumed is important. Simple sugars are easier for bacteria to break down (see preventive dentistry).

Fat in the normal diet helps to remove the food from the mouth and therefore shortens the length of time during which oral bacteria can metabolize carbohydrates. In addition, it promotes oral clearance as it coats carbohydrates (mainly in chocolate) and prevents their breakdown by bacteria. Some fatty acids also have antimicrobial effects.

In areas of the oral cavity where the clearance mechanism is fast, less sugar can diffuse into the plaque, decreasing the rate of acid formation.

39.2. Modeling of oral clearance

Figure 1.526. Figure 1. – Dawes model of salivary clearance (a schematic drawing). Taste buds sense the changing concentration of sugar and acid in the oral cavity. This stimulates salivary glands through the central nervous system to secrete a large amount of watery saliva, which will mix with the saliva already present in the oral cavity. A swallow is initiated when the amount of saliva reaches a maximum volume (swallowing reflex). A residual volume of saliva is left in the mouth after a swallow. This amount of saliva is constantly present in the oral cavity and is responsible for lubricating the mucosal surfaces. The original model is suitable for measuring the speed of oral clearance and was created by Colin Dawes in 1983. CNS: central nervous system
39.3. Phases of oral clearance

Figure 1.527. Figure 2. – Phases of oral clearance. After a meal (especially when consuming fermentable carbohydrates, acidic liquids and sugar) pH rapidly and significantly drops in the oral cavity in a few minutes. When oral pH falls below the critical range (pH 5.2-5.7), enamel demineralization is initiated. The fall of oral pH significantly stimulates saliva secretion. The saliva thus secreted contains large amounts of sodium bicarbonate, making it slightly alkaline (pH 8) and affording a high buffering capacity. Therefore, stimulated saliva has two major effects: first, its substantial volume significantly dilutes the nutrients in the oral cavity, facilitating their clearance through increased frequency of swallowing. On the other hand, it neutralizes acids due to its buffering capacity. This is the first or rapid phase of oral clearance, its duration is approximately 20 minutes. During this period, the main goal is to increase the oral pH to above the critical level. Subsequently, the rising pH will stimulate saliva excretion less and less, so the amount of secreted saliva gradually returns to the resting level, while oral pH will also return to the initial level. This is the second or slow phase of oral clearance, which takes approximately 40 minutes. The figure is based on the Stephan’s curve which shows pH changes measured in the dental plaque after consuming a sugar solution. The curve was published by Robert Stephan in 1943. The dashed line indicates the critical pH value (5.5).
39.4. Alterations associated with ageing

Reduction of salivation and oral clearance are increased the possibility of colonization of pathogenic bacteria and fungi (especially in the elderly) on the oral area, thereby increasing the likelihood oral or upper respiratory tract infection.

39.5. Fluoride clearance

Elevated fluoride concentration in saliva and therefore in the plaque is sufficient to protect against caries when rapid pH changes occur in the oral cavity.

The elimination of orally administered fluoride occurs in three different ways with very different kinetics. Fluoride from toothpastes, fluoride tablets or chewing gum washes out rapidly, within 1 to 2 hours. The elimination of fluoride from fluoridated mouth rinses is moderately rapid (about 3 hours) and the elimination of fluoride from topically applied fluoride varnish takes long, at least a week.

39.6. Bacterial clearance

Freshly secreted saliva is sterile, but mouth bacterial count is over 10^9/ml. 250 to 350 species are present in normal mouth flora. Bacteria need to adhere to survive and reproduce. This can be achieved mainly on hard tissue surfaces. The least appropriate for this among soft tissue surfaces are mucosa surfaces as the surface epithelial layer is shed cca. every 3 hours mostly during chewing. About 1 to 3 grams of bacteria are ingested daily. Therefore clearance of bacteria from the oral cavity towards the digestive tract is an important factor in preventing overgrowth of oral bacteria that is common in patients who have hyposalivation. This is helped by agglutinin in the saliva.

39.7. Test – Practices: Oral-Clearance (answers)

1. What is characteristic of the first phase of oral clearance?

A. rapid fall in pH
1. Oral biology

B. stimulated saliva secretion
C. slow, extended response over time
D. dilution and buffering are the primary characteristics
E. pH reaches a critical value of 4.2 at the end

2. What is characteristic of stimulated saliva?
A. Its bicarbonate concentration is higher than that of the unstimulated saliva and therefore it is better buffered.
B. Its pH is lower than that of the unstimulated saliva and therefore it is better buffered.
C. Its composition is the same as that of the unstimulated saliva, but its amount is different, so it has a better diluting effect.
D. Its viscosity is higher than that of the unstimulated saliva and therefore it is more diluted.
E. Its buffering capacity is lower than that of the unstimulated saliva

3. What has a positive effect on oral clearance?
A. high swallowing frequency and small swallowing volume
B. large residual volume
C. high maximal volume
D. high swallowing frequency and large swallowing volume
E. decrease in stimulated saliva secretion

References


The internal environment of the body offers habitat, nutrients and breeding opportunity for microbes, protozoa, fungi and other parasites. Key to the homeostasis is to defend against these attacks, ie. to fend off the invasion of pathogens. Layers of defense mechanisms have evolved during the millions of years. These layers are interdependent but can also function by themselves. It is important to understand the biological, biochemical and physical structures and principles of defense. Its layers are structurally and functionally interrelated and form parts of an integrated system. For example, there is no such thing as oral defense in itself: inflammations or ulcers in the oral cavity are often secondary to other diseases in the body.

**Figure 1.528. Figure 1. – The oral clearance process**

<table>
<thead>
<tr>
<th>The oral clearance process</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Mechanical drifting of the fluid flow to the stomach or the outside world</td>
</tr>
<tr>
<td>▶ non-specific trapping phenomenon: viscous saliva medium (network-glycoproteins), ionic bonds between microbes</td>
</tr>
<tr>
<td>▶ specific trap mechanism: secretory IgA</td>
</tr>
<tr>
<td>☐ Reduction caused by natural killer-killing mechanisms</td>
</tr>
<tr>
<td>▶ Non-specific salivary fluid destructive factors (lysozyme cell wall breakdown, thiolesterase system, lactoferrin, hypochlorosis, pH)</td>
</tr>
<tr>
<td>▶ Specific alfa trap and PNN phagocytosis (oroleukocyte)</td>
</tr>
<tr>
<td>☐ Artificial mechanisms</td>
</tr>
<tr>
<td>▶ mouth rinsing with clean water or chemical solutions in the preventive or therapeutic treatment procedures</td>
</tr>
</tbody>
</table>

Exclusion is a key defense mechanism: a physical surface barrier prevents the entry of pathogens in such a way that absorption of nutrients and excretion of metabolic waste products can still occur. This outer barrier is the first line of defense. Once through this barrier, invading agents are isolated and eliminated by a second line of defense. This internal defense system can be subdivided into innate and adaptive components. On surfaces covered by mucosa, components of the first line of defense are complemented by certain, modified elements of the second line of defense. This addition is crucial to the defense of the oral cavity. Another key component of oral defense is called microbial antagonism, whereby certain microorganisms of the normal microbiota compete for nutrients and produce inhibitory substances, thus suppressing the growth of invading microorganisms. Components of the first and second lines of defense are summarized on Figure 2.

**Figure 1.529. Figure 2. – The oral defense shield components**

<table>
<thead>
<tr>
<th>The oral defense shield components</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Innate components</td>
</tr>
<tr>
<td><strong>Anatomical factors</strong></td>
</tr>
<tr>
<td>• Protective layer of epithelial cells</td>
</tr>
<tr>
<td>• rapid regeneration – EGF</td>
</tr>
<tr>
<td><strong>Temperature</strong></td>
</tr>
<tr>
<td><strong>H+-concentration</strong></td>
</tr>
<tr>
<td><strong>O2-tension</strong></td>
</tr>
<tr>
<td><strong>Salivary factors</strong></td>
</tr>
<tr>
<td>• washing effect: mucus – viscoety</td>
</tr>
<tr>
<td>• Lysosome</td>
</tr>
<tr>
<td>• Glycoprotein</td>
</tr>
<tr>
<td>• Thocyninate</td>
</tr>
<tr>
<td>• Lactoferrin</td>
</tr>
<tr>
<td>• Complement (?).</td>
</tr>
</tbody>
</table>

The human oral cavity is subjected to the recurrent effects of numerous physical, chemical and microbial factors due to its diverse functions (sensation, feeding, breathing, verbal communication, etc.), often resulting in injuries to the mucous membranes or dental tissues. Therefore, our body is at high microbial risk in the oral cavity. An effective, layered oral defense including the elements of the first and second layers is thus crucial.
A key component of the first line of oral defense is the tight barrier formed by the mucous membrane, where the tight barrier epithelial components (occludin, claudins, ZO-1, JAM-1, cingulin) make it impermeable to microbes.

**Figure 1.530.** Figure 3. – Anatomical factors of the oral defense

When the epithelium is breached, salivary epithelial growth factor (EGF) stimulates cell proliferation, followed by differentiation and regeneration, effectively decreasing the time frame for microbial invasion.

The anatomical structure and functional properties of the oral cavity determine the temperature and oxygen content of the salivary environment, that has a strong influence on the composition of oral microflora.

**Figure 1.531.** Figure 4. – The protective role of oral temperature

When the epithelium is breached, salivary epithelial growth factor (EGF) stimulates cell proliferation, followed by differentiation and regeneration, effectively decreasing the time frame for microbial invasion.

The anatomical structure and functional properties of the oral cavity determine the temperature and oxygen content of the salivary environment, that has a strong influence on the composition of oral microflora.

**Figure 1.532.** Figure 5. – Oral oxygen tension conditions
Both local and temporal variability of the oral cavity oxygen tension has to be mentioned.

- The O₂ tension in the mouth is generally low, substantially it means the oxygen dissolved in the saliva.
- The oxygen tension in the oral cavity is too low for the aerobic microbes.
- In the mouth milieu the facultative anaerobes & obligate anaerobes can proliferate.

**Figure 1.533. Figure 6. – Oral microflora**

<table>
<thead>
<tr>
<th>Oral microflora</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minutely changing, but the with microflora community can be kept balanced:</td>
</tr>
<tr>
<td>- Its composition is discussed by microbiology, and analyzed specially by anatome, periodontology and oral diagnostic.</td>
</tr>
<tr>
<td>- In the oral microflora sometimes and / or somewhere there is a PATHOGENIC overweight, which, in case of optimal situation, can be controlled by the increase of inhibitory factors and specific mechanisms.</td>
</tr>
<tr>
<td>- But the pathogens that colonize the hard tooth tissue surface can only be driven out of the harmful processes with mechanical and chemical influences and proper hygiene protocols.</td>
</tr>
</tbody>
</table>

Beyond these factors, saliva facilitates defense in several ways (Figure 2) due to its physical (viscous, lubricative) properties, chemical effects (dilution by volume, buffering capacity) and anti-microbial properties (specific and non-specific trapping effect, osmotic lysis).

**Figure 1.534. Figure 7. – Salivary factors I.: Buffer**

- Carboxy-anhydrase catalyser enzyme
- H₂O + CO₂ = H₂CO₃ (carbonate)
- Proton (H⁺) Bicarbonate (HCO₃⁻)
- pH 5.6-7.0
- The pH of the oral cavity is a crucial factor for development of oral microflora
- Acidophil bacteria => Caries
- Proteolytic bacteria => Periodontitis

**Figure 1.535. Figure 8. – Salivary factors II.: Washing and coating**
1. Oral biology

- Mechanical washing effect: a non-specific (non-selective) removal of microbes from the oral cavity
  - Efficient: a large amount of saliva, thick consistency (viscous, mucinous) saliva
  - Inefficient: a small amount of dilute (less viscous) saliva
- Flowing liquid film coating, “Fluid Film” (enamel pellicle)
  - Nonspecific “adhesion” to trap-glycoproteins
  - Specific “binding” trap to saliva antibodies
- PROS: high flow, density and much saliva – causes the microbes clumping from the enamel and thus high inertia swallowing. Thereby inhibiting the formation of dental plaque.
- CONS: low density and low salivary flow. The microbes bound, “colonize” to the enamel. It promotes the formation of dental plaque.

**Figure 1.536. Figure 9. – Salivary factors III.: Free iron reduction**

- **Lactoferrin**
  - 77 kD protein
  - Can bind 2 Fe-ions
  - Reduces the number of free Fe-ions in the oral milieu and also accumulates them in the body
  - Inhibits the development of microbes

**Figure 1.537. Figure 10. – Salivary factors IV.: Hydrogen peroxid blocking system**

- **Thiocyanate**
  - With salivary components it have higher efficiency of inhibiting microbial growth factors
    - Hydrogen peroxidase + peroxidase? (e.g. lacto peroxidase, thiocyanate)

**Thiocyanate inhibits the development and colonization of microbes**

**Figure 1.538. Figure 11. – The saliva functions (protective)**
A non-pathogenic symbiontic microflora further assists oral defense by occupying habitat, regulating the pH and chemical milieu and by producing bacteriocins, functions collectively called microbial antagonism.

**Figure 1.539. Figure 12. – Complement system**

Catabolic substances in saliva (lysozyme) can degrade the protein types of protective factors, such as complement-subunits as well.

**Figure 1.540. Figure 13. – „Microbiotic antagonism”**

C1-C9 complement work in gingival tissues. In the sulcus they break down in the saliva. The complement cascade can not be built.
Both non-specific removal by a steady flow of viscous saliva and targeted removal by pathogen-specific secretory IgA antibodies and by oral leukocytes contribute to maintaining a non-pathogenic microbial flora.

**Figure 1.541. Figure 14. – The development of the oral microflora balance I.**

<table>
<thead>
<tr>
<th>The development of the oral microflora balance I.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Microorganism invasion</strong></td>
</tr>
<tr>
<td>- Food, breath, etc.</td>
</tr>
<tr>
<td><strong>The first selection of microorganisms</strong></td>
</tr>
<tr>
<td>- Mechanical aspecific factors, e.g. swallowing, cough, flushing, etc.</td>
</tr>
<tr>
<td>- The microbe’s adaptation ability to temperature, pH, oxygen, nutrients</td>
</tr>
<tr>
<td><strong>The binding of microorganism to</strong></td>
</tr>
<tr>
<td>- saliva components (pl.mucin, glycoproteins, etc.)</td>
</tr>
<tr>
<td>- the mucosa epithel coated by saliva-mucin</td>
</tr>
<tr>
<td>- enamel, neck cementum and dentin surfaces</td>
</tr>
</tbody>
</table>

**Figure 1.542. Figure 15. – The development of the oral microflora balance II.**

<table>
<thead>
<tr>
<th>The development of the oral microflora balance II.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Further quantitative and / or qualitative activation of chemical and cellular defense mechanisms</strong></td>
</tr>
<tr>
<td>- E.g. Lactoferrin, lysosome, serylA, orodinocyte,</td>
</tr>
<tr>
<td><strong>Selective adhesion of some bacteria</strong></td>
</tr>
<tr>
<td>- Development of dental plaque: mucin-glycoprotein-serylA</td>
</tr>
<tr>
<td><strong>Selection according to the microbiotic antagonism effect</strong></td>
</tr>
<tr>
<td><strong>Elimination of some pathogenic agents by phagocytosis and immune complex formation</strong></td>
</tr>
</tbody>
</table>
The immune system is adapted to function in the internal milieu of organisms, hence the salivary environment of the oral cavity is suboptimal for its function. Antibodies released into the saliva from the gingival sulcus and through mucosal injuries are proteolytically digested by lysozyme and polymorphonuclear phagocytes (PMN) swell in the hypoosmotic salivary environment (“salivary bodies”). Therefore, accessory immune function can only be fulfilled by secretory antibodies (secIgA) specific to the oral environment and by oroleukocytes, specialized PMNs cells possessing a protective stable hydrate shell.

Figure 1.543. Figure 16. – The development of the oral microflora balance III.

The development of the oral microflora balance III.

- The proliferation of the selected microorganisms
- The combined effect of all these factors:
  - Invasion-selection-proliferation-extinction
  - Temporal onset of the constantly changing steady state

Figure 1.544. Figure 17. – AG-specific Ab-s (dimer-IgA) production in the oral region

Figure 1.545. Figure 18. – The dimer-IgA and the PMN-cells penetration to the ductus saliva through the salivary gland
Figure 1.546. Figure 19. – Proteolytic effect in the oral cavity: protected antibodies prolongate

Figure 1.547. Figure 20. – The effect of oral hypo osmosis: protected PMN cells „Oroleukocytes”
Figure 1.548. Figure 21. – The oral proteolytic effect: Unprotected antibodies decompose

- IgG, IgA, IgM, IgD antibodies in the saliva are degraded by proteolytic enzymes from the c-terminus.
- Tissue IgG, IgA, IgM, IgD, IgM: the IgM pentamer complex that is bound to the c-terminus complex is too large for diffusion.

Figure 1.549. Figure 22. – The effect of oral hypo osmosis: unprotected PMNs to become the “saliva body”

Unprotected immune components can only function deep down in the gingival sulcus within the crevicular fluid. Importantly, they do not necessarily play a beneficial role as persistent gingival inflammation may progress into periodontitis that leads to bone loss.

40.1. Oral specific immunity

The production of specific IgA antibodies starts by the transformation into plasma cells of B cells migrating into salivary gland parenchyme. Monomeric IgA molecules are joined at their Fc ends, facilitated by the simultaneously produced J-protein, to form dimers. J-protein can strongly bind specific receptors on the basolateral membranes of salivary gland cells, facilitating its transcytotic secretion to the luminal side. Whether or not they bind J-protein, the receptors dissociate from the membrane into the luminal space. Dissociated receptors, when not binding J-protein, are called secretory protein. The secretory IgA (secIgA) complexes comprising the receptors and bound J-protein/IgA dimers are washed into the oral cavity by saliva secretion. The Fc region of secIgA not being exposed, they are resistant to degradation by lysozyme. On the other hand, the four binding sites of the IgA dimer can efficiently aggregate microbes, which facilitates the elimination of pathogens by swallowing with saliva. Microbes already attached are eliminated by phagocytes that migrated through the salivary glands and are covered by salivary secretory glycoproteins. These glycoproteins are receptors shed from salivary gland cells and facilitate the formation of a stable hydrate shell on the surface of phagocytes, thus preventing the cell swelling effect of hypo-osmotic saliva. These long-lived PMN cells are called oroleukocytes. Figure 23 shows components of oral immune protection.

Figure 1.550. Figure 23. – Summary of oral immune defense mechanisms
A fundamental issue in immunology is the discrimination between pathogenic and non-pathogenic microbes. The basis of this discrimination is that throughout the lifetime of an individual, microbes encounter phagocytes of the immune system (mostly along the gastrointestinal tract, for example when in infancy antigen presenting cells of the gut associated lymphoid tissue encounter bacteria at the fenestrated intestinal mucosa). The increasing number of contacts leads to a broad spectrum of antigen-educated lymphocytes. These B cells then migrate to mucosal tissue or to gland tissues. Soon after pathogen invasion, memory B cells are activated to differentiate into plasma cells that produce a large amount of antibodies to fight infection. Obviously, memory cells from other lymphoid organs are also suitable for this function. A general outline of oral defense is shown in Figure 24.

Figure 1.551. Figure 24. – The main source of antigen information for organisms comes from nutrient proteins and micro-structures derived from fenestrated intestinal lumen after birth. Then these proteins and structures are processed and delivered to the lymphocyte memory cells by the PMN cells.
In light of the above, the degree of oral defense of each individual patient is crucial to know in the dental practice. When saliva is scarce and viscous, it fulfills only its trapping but not its flushing function, resulting in plaque accumulation at sites of predilection for caries and gingivitis. Abundant secretory IgA in the plaque aggravates the problem since it facilitates not elimination of bacteria but incorporation of them into the plaque. Therefore, measuring the production and viscosity and, occasionally, the secretory IgA content of saliva is part of a state-of-the-art dental treatment plan. Furthermore, complete oral irrigation by a suitable device to clean the entire gingival sulcus groove must be part of the recommended oral hygiene protocol.

40.2. Test – Practices: Oral defense mechanisms (answers)

1. Which is not a component of the oral defense barrier?
A. tight mucosa
B. thiocyanate
C. mucinous saliva
D. microbiotic antagonism
E. secretory IgA

2. Which non-secretory immunoglobulin can stay functional for an extended time in the oral salivary milieu?
A. IgG
B. IgA
C. IgM
D. IgD
E. IgE

3. Which is the most important in maintaining a normal oral microflora?
1. Oral biology

A. non-specific capture and elimination
B. phagocytosis
C. washing effect of saliva
D. secretory IgA mediated capture and elimination
E. proteolysis

4. Which is not directly related to the formation of secretory IgA and its transport to the oral cavity?
A. J protein
B. plasma cells developing from B cells
C. C4 complement
D. Transporter receptors on the basolateral membrane of salivary gland cells
E. The transporter secretory protein secreted at the apical side of salivary gland cells

41. 1.41. Practices: Analysis of dental plaque – Gabor Kiss

In the oral cavity there are natural and artificial non-shedding surfaces which make stable bacterial attachment possible. Dental plaque is the bacterial biofilm firmly adhering to these surfaces via the acquired pellicle.

41.1. Composition

Bacterial microcolonies occupy 20 to 25% of plaque volume dispersed in the extracellular matrix which constitutes the remaining 75 to 80%.

41.2. Location

Clinically we can differentiate between supragingival plaque which has a well-defined structure, and subgingival plaque which is looser and more heterogeneous.

Figure 1.552. Figure 1. – Structure of subgingival plaque

41.3. Mechanism of plaque formation
Plaque formation occurs in three distinct stages:

1. acquired pellicle,
2. initial colonization,
3. maturation of plaque.

**Figure 1.553. Figure 2. – Formation of dental plaque**

**Acquired pellicle** is a less than 1 µm thick layer of mostly salivary mucin glycoproteins and, to a smaller extent, of immunoglobulins and other molecules, adhering to the tooth surface. Pellicle binds to the negatively charged tooth surface by means of van der Waals and electrostatic forces.

**Initial colonization.** Initially bacteria bind to the surface reversibly by physicochemical forces, which are easily disrupted by shear forces acting in the oral cavity. Irreversible bond between functional groups in the pellicle and bacterial adhesins can form in areas where the effect of shear forces is small or negligible. The first colonizers are predominantly Gram-positive cocci. Metabolism and division of bacteria is upregulated after attachment and changes in the microenvironment enable the adherence of additional bacteria. These secondary colonizers are mainly Gram-positive and Gram-negative rods.

**Table 1. Characteristic bacteria in dental plaque**

<table>
<thead>
<tr>
<th>Pellicle</th>
<th>sterile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial colonizers</td>
<td>Gram-positive cocci (Streptococcus)</td>
</tr>
<tr>
<td>Secondary colonizers</td>
<td>Gram-positive rods (Actinomyces)</td>
</tr>
<tr>
<td></td>
<td>Gram-negative rods (Capnocytophaga)</td>
</tr>
<tr>
<td></td>
<td>Fusobacterium</td>
</tr>
<tr>
<td></td>
<td>Prevotella</td>
</tr>
<tr>
<td>Late colonizers</td>
<td>Porphyromonas gingivalis</td>
</tr>
<tr>
<td></td>
<td>Spirochaetae</td>
</tr>
</tbody>
</table>
Maturation of plaque. During thickening, physical and chemical characteristics of the plaque become heterogeneous. Oxygen and nutrients originating from saliva cannot penetrate to deeper parts of plaque. Therefore, aerobic and facultative anaerobic saccharolytic bacteria will colonize surface areas, whereas obligate anaerobic bacteria will occupy deeper, subgingival parts. These latter are proteolytic bacteria consuming microbial metabolites and proteins originating from crevicular fluid and blood.

Figure 1.556. Table 2. – Maturation of dental plaque

<table>
<thead>
<tr>
<th>Days</th>
<th>Dominant bacteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Steril pellicle</td>
</tr>
<tr>
<td>1</td>
<td>Gram-positive cocci</td>
</tr>
<tr>
<td>3</td>
<td>Gram-positive rods</td>
</tr>
<tr>
<td>6</td>
<td>Gram-negative bacteria</td>
</tr>
<tr>
<td>21</td>
<td>Anaerobes, Gram-negatives</td>
</tr>
</tbody>
</table>

41.4. Dental plaque as biofilm

Growth in a biofilm is more beneficial to bacteria over planktonic growth. It offers a substantially increased protection from host defense mechanisms, environmental toxins and antibiotics. Microorganisms do not merely coexist in biofilms but influence each other through complex interactions. The characteristics of biofilm are mostly due to the extracellular matrix produced by the bacteria, which has structural as well as nutrient storage functions.
Pathogenicity of plaque

Dental plaque is the main etiological factor of the two most common oral diseases:

1. caries,
2. periodontal diseases.

**Caries** is caused by demineralization of dental hard tissues by acids produced by saccharolytic bacteria if continuous remineralization cannot balance it.

**Gingivitis** is caused by plaque bacteria in direct contact with gingival tissues. It can be observed in each person in the presence of sufficient amount of plaque. In susceptible individuals, tissue breakdown in periodontitis is caused partly by osteoclasts activated by inflammatory factors and partly by proteolytic enzymes produced by anaerobic bacteria.

Dental calculus

Calculus is mineralized dental plaque.

**Supragingival calculus**

Initially it is a yellowish-whitish substance with chalk consistency which can become discoloured and very hard with time. Susceptibility sites for its formation are surfaces adjacent to duct openings of main salivary glands, lingual surface of lower front teeth and buccal surface of upper molars. The key factor in its mineralization is saliva.

**Subgingival calculus**

Subgingival plaque is mineralized by blood and crevicular fluid. It is dark and very hard. It can build up only in already developed periodontal pockets, therefore it is not the cause of periodontitis but plays a very important role in maintaining it.

Composition

Calculus is composed predominantly of inorganic material, two thirds of which is crystalline in structure.

### 41.5. Pathogenicity of plaque

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### 41.6. Dental calculus

Calculus is mineralized dental plaque.

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### Composition

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### Figure 1.557. Table 3. – Composition of extracellular matrix

<table>
<thead>
<tr>
<th>Type</th>
<th>Name</th>
<th>Source</th>
<th>Role</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate</td>
<td>Glucan (dextran, mutan)</td>
<td>Glucosyltransferase (extracellular bacterial enzyme)</td>
<td>Matrix structure</td>
</tr>
<tr>
<td></td>
<td>Fructan</td>
<td>Fructosyltransferase (extracellular bacterial enzyme)</td>
<td>Nutrient</td>
</tr>
<tr>
<td>Lipid</td>
<td>Lipopolysaccharide (LPS) (endotoxin)</td>
<td>Gram-negative bacteria</td>
<td>Induces severe inflammation</td>
</tr>
<tr>
<td>Protein</td>
<td>Proteolytic enzymes</td>
<td>Anaerobic bacteria</td>
<td>Periodontal tissue breakdown</td>
</tr>
</tbody>
</table>

### Figure 1.558. Table 4. – Composition of dental calculus
Varying ages and locations of calculus are associated with different characteristics due to differences in mineral content.

**Pathogenicity of calculus**

Its pathogenic potential is caused by the constant presence of dental plaque coating its surface, which cannot be removed from the rough surface. Therefore dental calculus maintains a continuous inflammation in the vicinity of periodontal tissues.

### 41.7. Test – Practices: Analysis of dental plaque (answers)

1. **What is the first step of dental plaque formation?**
   
   A. biofilm formation
   
   B. maturation of plaque
   
   C. synthesis of extracellular matrix
   
   D. acquired pellicle
   
   E. initial colonization

2. **What type of bacteria are the most frequent amongst the initial colonizers of dental plaque?**
   
   A. Gram-negative cocci
   
   B. Gram-negative rods
   
   C. Gram-positive cocci
   
   D. Spirochaetae
   
   E. Gram-positive rods

3. **What is the main ingredient of dental calculus?**
   
   A. sodium phosphate
B. calcium phosphate
C. calcium carbonate
D. sodium carbonate
E. magnesium phosphate

42. 1.42. Practices: Salivary secretion – Gabor Varga

During a thorough questioning of patients at the dental visit, they often come up with the complaint of dry mouth and decreased salivary function. This subjective feeling, called **xerostomia**, is not necessarily accompanied by the objectively detectable decreased salivary gland function, the so-called **hyposalivation**. Salivary dysfunction can be attributed to many reasons. These may range from the very common reduced fluid consumption to the really serious cancer and inflammatory diseases. In order to determine the actual cause of this condition, we can obtain valuable clues from patient history. Also, we can use various methods available to test salivary secretion.

Saliva is produced by three pairs of major salivary glands and several hundreds of minor salivary glands scattered around the oral cavity, in a ratio of about 92-95% and 5-8%, respectively. All this volume is supplemented with a small amount of plasma filtrate (also containing some leukocytes) from the gingival sulcus.

The saliva from the parotids is optically clear, less viscous; while the saliva from the submandibular, sublingual and the minor salivary glands is opalescent, highly viscous. During sleep, major salivary glands are at rest, and the low amount of secretion produced by the minor salivary glands essentially serves the purpose of covering, protecting oral surfaces.

The total daily amount of saliva varies between wide limits, but usually it is at least 600-700 ml. The secretion rate differs significantly during mealtime, at rest or during sleep. About 50% of the total daily amount of saliva is produced during the 1.5-2 hours long period of food intake, in which the parotid gland plays a crucial role. In the daytime, during those time periods when there is no food intake, saliva is predominantly coming from the submandibular and sublingual glands. During sleep, only 1-2% of the total daily amount is secreted, which is essentially the product of the minor salivary glands. During food intake the main functions of produced saliva are lubrication, bolus formation, gustation, digestion and washing-cleaning effects. At rest, the protective functions of saliva become important such as its acid buffering effect, its antimicrobial effect and its surface-coating features.

**Figure 1.560. Figure 1. – Amount of saliva in health**

<table>
<thead>
<tr>
<th>Amount of saliva in health</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image" alt="Table" /></td>
</tr>
</tbody>
</table>

For the sake of reproducibility of the data, salivary tests are usually executed in the morning hours, on an empty stomach in a laboratory environment with standardized conditions. Diurnal variation can cause significant differences. If the tests are done in a different time period of the day, then one should wait at least 2 hours after the last meal before the examination.

**Figure 1.561. Figure 2. – Sialometry determination of salivation rate**
Technically the less challenging method is the collection of mixed saliva. However, this has the disadvantage that the sample is not pure. It contains microorganisms from the oral flora, interstitial fluid and food residues. Yet, due to its simplicity, it is the most common method. It is suitable for the determination of the most important, easily measurable parameter, the amount of secreted saliva reaching the oral cavity. The amount of secretion is usually measured both at basal conditions and after stimulation.

Saliva collection and analysis from individual glands is more complex than mixed saliva collection. Formerly it was a frequently used method to insert a thin plastic cannula into the ducts of the major salivary glands. However, one may easily transmit infectious pathogens to the glands with this method. Because of the aforementioned complications, this method is rarely used today.

**Figure 1.562. Figure 3. – Classification of saliva collection**

**Classification of saliva collection**

- A
  - Collection of mixed saliva
  - Collection of pure saliva (from a given gland)
- B
  - Collection of basal secretion
  - Collection of stimulated secretion

**Figure 1.563. Figure 4. – Collection of saliva secretion**

**Collection of saliva secretion**

- Patient position: sitting, head slightly pushed ahead, open eyes: A) Passive draining method
- Collection for 5-6 min: B) Spitting method
- Results are given in ml/min: C) Aspiration method
- D) Absorption method

Mixed saliva can be collected by the passive, draining method, by spitting, by the aspiration technique, or by the absorption of the fluid on a pre-weighed filter paper. Values are given in ml/min, after 5-10 minutes long collection of saliva.

Applying the passive draining method, the saliva flows into a calibrated container at the lower lip of the patient. The amount of accumulated fluid is determined according to its volume. The density of saliva is very close to 1 g/ml, which is approximately equal to the density of water.

**Figure 1.564. Figure 5. – A. Passive, draining method**
The spitting method is similar to the previously described one. In every minute (or another determined time period) the examined person spits the accumulated fluid into a calibrated vial or tube, wherein the secretion rate can be determined. Unlike spitting, in the case of aspiration technique the fluid is continuously aspirated by a vacuum driven system.

**Figure 1.565. Figure 6. – Spitting and Aspiration method**

<table>
<thead>
<tr>
<th>Spitting and Aspiration method</th>
</tr>
</thead>
<tbody>
<tr>
<td>B) Spitting method</td>
</tr>
<tr>
<td>fluid produced into the mouth is spitte/d/ejected into a calibrated tube in each minute</td>
</tr>
<tr>
<td>C) Aspiration method</td>
</tr>
<tr>
<td>the produced saliva is collected by a vacuum driven system into calibrated tube</td>
</tr>
</tbody>
</table>

Using the absorption method, saliva taken up by a pre-weighted cotton tissue, filter paper or a piece of sponge, and the amounts are determined by weighing.

**Figure 1.566. Figure 7. – D. Absorption method**

<table>
<thead>
<tr>
<th>D. Absorption method</th>
</tr>
</thead>
<tbody>
<tr>
<td>saliva is collected by preweighted sponge or cotton tissue</td>
</tr>
<tr>
<td>repeated determination of the weight calculated from the amount of saliva collected</td>
</tr>
<tr>
<td>this is the least reliable method</td>
</tr>
</tbody>
</table>

During collection of saliva, the changes in the functional capacity of salivary glands are usually examined not only at basal secretion, but also in stimulated conditions. Increased secretion can be achieved by both masticatory stimulation and gustatory and olfactory activation or by the joint activation of both. The most common method is that, when the patient puts a small piece of standard volume paraffin piece in the mouth. Oral saliva should be completely swallowed before the patient starts chewing the piece of paraffin. Generally after two minutes, saliva is immediately drained into a collection vial, and this whole process is usually repeated for two times.

**Figure 1.567. Figure 8. – Collection of stimulated mixed saliva 1.**
Another classic way is the gustatory stimulation with the application of 2% citric acid solution. The solution is dropped on the tongue surface in 30 second intervals, and after 2 minutes the patient spits the produced saliva into a test tube. The test is typically repeated twice more, and the secretion rate of produced saliva per time unit is determined.

**Figure 1.568. Figure 9. – Collection of stimulated mixed saliva 2.**

A safe method to determine the secretion rate of an individual salivary gland is to use the Carlson-Crittenden device (Curby cup). In application, a suction disc is put over the opening of the Stenon-duct, with an outer vacuum ring. The device is kept on site by suctioning the air out from the outer ring thereby forming the vacuum.

**Figure 1.569. Figure 10. – Saliva collection from individual glands**

In numerous cases, the amount of mixed saliva cannot give enough information about the state of saliva secretion. Such case is the autoimmune Sjögren's syndrome. The disease is characterized by the continued destruction of acinar parenchyma due to auto-antibody production against salivary glands. However, it first becomes significant in the case of minor salivary glands, because of their high surface/weight ratio, their exposure to antibodies are significantly higher than that of the major salivary glands. Thus, the secretory activity of minor salivary glands has an important differential diagnostic significance. Previously, this was limited to inspecting the minor salivary glands of the internal surface of lips. During this procedure after wiping the
surface off dry, the reappearing saliva droplets were collected with a filter paper or glass capillary. This technique has an improved, more advanced, reliable version used in clinical practice: the Periotron device. The measurement starts as the investigated mucosal area is dried, and then the reappearing drops of saliva are soaked up with special filter strips developed for the instrument for 1-2 minutes. The Periotron device gives a definite value based on the conductivity change, from which the secretion rate of minor salivary glands can be determined after appropriate calibration.

**Figure 1.570. Figure 11. – Collection of secretion of minor salivary gland**

Secretion of mixed saliva varies within relatively wide limits even in healthy persons. At rest, basal value is about 0.4 ml/min. During activation by paraffin chewing, the value rises to 2 ml/min on average. The objectively measured state of decreased salivary function, called **hyposalivation** occurs when the basal value is less than 0.15 ml/min and the secretion volume obtained by chewing paraffin is less than 0.7 ml/min. It is important to note, that this is not the same phenomena as the subjective feeling of dry mouth, the **xerostomia**. Real hyposalivation often cannot be detected in patients complaining about dry mouth. However, this does not mean that there is no reduction in salivary secretion in the individual, but it just falls into the wide normal range.

**Figure 1.571. Figure 12. – Amount of saliva in health and disease**

Decreased salivary function occurs more often in women, especially after menopause. The frequency of the symptom increases with age. The decrease in secretion can be due to many reasons. One of the most obvious reasons is the direct damage of salivary glands, for example after irradiation of the head and neck area in tumor therapy, or in patients with Sjögren's syndrome as a result of acinar destruction. In addition, the dehydration of the body alone, even without any other contributing factors, leads to reduced saliva secretion. A wide range of possible causes are known. The insufficient fluid consumption is the easiest to treat. Diabetes mellitus could be another common reason behind reduced saliva flow, which can be connected to the reduction of extracellular fluid space, as a consequence of glycosuria. Several drugs used in a number of systemic diseases may also lead to side effects such as decreased salivary secretion. Among these must be highlighted high blood pressure. Its usual treatment methods can seriously interfere with saliva production through inhibition of adrenergic activity and reducing extracellular fluid space. The other most important classes of drugs are anti-depressants and
antipsychotic drugs, which inhibit saliva secretion through reducing either the sympathetic or parasympathetic activity.

Besides the so-called sialometry studies of salivary flow rate, saliva composition and salivary components can also be studied, this is called sialochemistry. These include saliva tests for immune composition and immune components (primarily IgA), enzyme activity tests (e.g. for amylase and lipase), as well as hormone level tests. The concentration of some components in saliva shows a very good correlation with their concentration in the blood plasma. Thus, saliva, a body fluid that can be obtained in a non-invasive manner, is increasingly gaining ground in laboratory diagnostics.

Figure 1.572. Figure 13. – Sialochemistry – saliva diagnostics

Video 1. – Resting saliva collection.
Video 2. – Resting mixed saliva collection with absorption.
Video 3. – Isolated saliva collection from the minor salivary glands.
Video 4. – Collection of the stimulated saliva.
Video 5. – Stimulated mixed saliva collection with absorption.

42.1. Test – Practices: Salivary secretion (answers)

1. Primary factor determining esophageal pH in humans:
   A. bicarbonate
   B. gastric acid
   C. amylase
   D. lysozyme

2. Instrument for measurement small salivary gland secretion
   A. Deriotron
   B. Beriotron
   C. Meriotron
   D. Periotron
   E. Metriometer
3. Xerostomy by definition:

A. decreased salivary secretion based on laboratory measurements

B. decreased values for stimulated salivary secretion

C. decreased salivary secretion based on the subjective feeling of the patient

D. joint appearance of laboratory and clinical parameters, only in Sjögren syndrome

E. decreased basal salivary secretory rate

F. red, cracked oral mucosa

43. 1.43. Practices: Chewing – Mate Jasz

Movements of the mandible are primarily influenced by two factors. Of these, mandibular joint movements are detailed in the presentation titled Mastication movements. In addition, the anterior leading trajectories are constituted by contacting opposing teeth during the movements associated with tooth contact. Movements defined solely by the joints, which allow significant excursions in all the three directions, are called free movements. Those also influenced by tooth contact are identified as articulations. Consequently, movements have only two determinants in the former situation, while at least three in the latter case; under all circumstances these are the two jaw joints, which are accompanied by one or more occlusions.

During protrusion in TMJs on both sides a symmetrical translation movement is performed, largely as described in the presentation titled Mastication movements. In addition, a rotation of greater or lesser extent occurs, depending on the degree of overbite and overjet. During the movement, the anterior midpoint begins to move forward and downward, then in the edge-to-edge bite position it proceeds horizontally and eventually it turns upwards (Fig. 1).

Figure 1.573. Figure 1.

Normal mouth opening is a variable combination of translational and rotational movement. Importantly, the pure rotational movement up to 1.5 to 2 cm mouth opening, described in earlier textbooks, can only be observed in an extreme posterior jaw position, on the posterior boundary curve of the sagittal Posselt diagram. In case of daily used comfortable mouth openings, translation starts after a few milimetres.
During **laterotrusion** the movements on the two sides are completely different. On the excursion side, the dominant movement of the working side condylar head is the turn along the vertical axis, the degree of which is characterised by the shift angle. This is complemented by a motion of 0 to 2.5 mm in the lateral direction. This massive lateral shift of the jaw is called Bennett movement after its first describer. The extent of the lateral excursion of the working side condylar head is described by the side-shift.

### 43.1. Backward movements of the mandible in the sagittal plane

The curve defined by the border movement boundaries of the anterior midpoint is the sagittal Posselt diagram (Fig. 2). Its „tracing“ should start from the farthest possible posterior joint position which is still accompanied by occlusion (retruded position – marked RKP in the pictures). From this spot, maintaining tooth contact, the chin must glide to the ICP position (marked IKP in the pictures) and then progress to the maximal protrusion position (P), continually concentrating on maintaining tooth contact. Mouth opening starts from this position and advances until the possible maximum (NY). From the maximum possible mouth opening position, mouth closure has to be driven to the retruded position (RKP) while pulling the chin backwards.

**Figure 1.574.** Figure 2.

Posselt’s sagittal diagram shows:

1. the condylar heads of the mandible shift forwards and, to a greater or lesser extent, upwards during the movement leading from RKP to IKP;

2. in maximal protrusion (P) the condylar head does not reach the maximum translational position, it arrives there only at the time of maximum mouth opening (NY),

3. during mouth closing in a forced posterior position, there is a sharp break (TP) in the curve showing the motion of the incisal point. On the rear curve defining the movement range boundaries (and exclusively here!!) translational and rotational movements are isolated from each other. In this position, mouth closure before the TP is translation, and that after the TP is rotation,

4. as opposed to protrusion performed with closed teeth, in case of mouth closure in the forced back position, translational movement may occur 1 to 2 mm more caudally. This can be explained by the reversible compression and distraction of the joint,

5. the movement range of protrusion – retraction decreases with the advance of mouth opening.

### 43.2. Backward movements of the mandible in the frontal plane

The frontal Posselt diagram is the curve defining the border movement boundaries of the anterior midpoint in the frontal plane. The excursions of the right hand side (green) and the left hand side joint in the sagittal plane are identical with those seen on the sagittal Posselt diagram. In the horizontal plane, the movement is completed with the excursions on the working as well as on the balancing side, as described under laterotrusion. The anterior centerpoint (blue) forms the characteristic shape of a coat of arms.

**Figure 1.575.** Figure 3.
Posselt’s frontal diagram shows:

1. The arc and slope of the midpoint’s upper excursion depend on the morphology of the grinding surface of the teeth contacting during lateral movements. In the present case a canine guidance associated with a vertical overbite can be seen.

2. Along with the advance of mouth opening, the movement range decreases in the frontal plane as well. At maximum mouth opening practically no excursion is possible in any direction.

43.3. The backward movements of the mandible in the horizontal plane

Among the border movements in the horizontal plane, the lateral and protrusion–retrusion shift of the anterior centerpoint is especially significant. This shape is called “arrowhead” and is used for determining the central joint position in prosthetic care (Fig. 4).

Figure 1.576. Figure 4.

The video shows the movements associated with the frontal and sagittal Posselt diagram. The motions of the mandible are represented by the excursions of the two condyles (right: green, left: red) as well as those of the anterior centerpoint (blue).

Video 1. – Posselt diagram.

43.4. Test – Practices: Chewing (answers)

1. Which one does not fit on the sagittal Posselt diagram?

A. Maximal opening position

B. Maximal protrusion position

C. ICP (Intercuspal position)
1. Oral biology

D. RCP (Retral contact position)
E. 2 cm mouth opening

2. Which one is not true for the Bennett movement?
A. It takes place during laterotrusive movements
B. The whole mandible moves sideward
C. It is usually more than 2 cm
D. It takes place in the direction of the working side

3. During translational movement:
A. The condyles move forward and downward
B. The condyles move along a curved path
C. The angle between the movement and the horizontal plane is called the sagittal condylar angle
D. Usually the left and right condyles move along a similar path
E. All of the above answers are true

References
4. Friedman M. H., Weisberg J.: Temporomandibular Joint Disorders: Diagnosis and Treatment. Quintessence, Chicago

44. 1.44. Practices: PCR technique in dental research – Erzsebet Bori

Molecular biology is the branch of biology that deals with the background of physiological/pathophysiological processes at the level of macromolecules such as nucleic acids and proteins. Central to its approach is the view that all biological functions are based on proteins, which are encoded by DNA and this information is expressed through the processes of transcription and translation. Therefore the investigation of these processes and macromolecules has a great importance in, for example, understanding the background of a disease or in the establishment of its therapy. After we have learnt the molecular basis of a disease the methods of molecular biology can be applied as diagnostic tools as well. The knowledge of our genetic code can even be utilized in personalized medicine or used for curing diseases on the genetic level by gene therapy. As the role of molecular biology is increasing both in research and diagnostics, the understanding of its basic methods is necessary.

Figure 1.577. Figure 1.
To isolate macromolecules from tissues or cells we have several methods available, such as the outdated CsCl gradient ultracentrifugation, the guanidine isothiocyanate-phenol-chloroform extraction or today’s most commonly applied techniques, the “column based” methods.

The amount of DNA/RNA/protein molecules in these preparations can be measured by spectrophotometer.

Figure 1.579. Figure 3.
After isolating a given type of macromolecules, it may be necessary to selectively detect or manipulate the individual molecule(s) of interest among many similar biomolecules. Biomolecules can be size-separated by gel electrophoresis. During this process, molecules migrate in an electric field through the pores of a gel. The direction and rate of migration depends on the charge, shape and size of the particular biomolecule.
Several options exist to separate certain molecules or parts of molecules based on their structure. Specific detection of proteins is based on the antibody-antigen interaction. A specific sequence in nucleic acids, on the other hand, may be identified using restriction enzymes or hybridization.

Figure 1.582. Figure 6.
The classic molecular biological methods combine size and structure-based separation techniques. Even though their significance has diminished, it is important to understand the principles underlying different blotting techniques. **Southern blot** is for the investigation of DNA, while **Northern blot** is for RNA and **Western blot** is for the detection of proteins. All of these blotting methods apply size separation by gel electrophoresis as the first step. Separated molecules are then transferred to a nitrocellulose membrane, a step called blotting. Finally, the membrane-bound nucleic acids are detected by hybridization probes while proteins by antibodies. Differences between blotting techniques are due to differences between the nature of target molecules.

**Figure 1.583.** Figure 7.
Polymerase chain reaction (PCR) is the most frequently used molecular biological method today. The essence of this technique is the amplification of DNA. Subsequently, the sequence of interest can be detected by gel electrophoresis using DNA dyes, without blotting and radioactive probes. PCR is a three-step process. During denaturation, the chains of DNA are separated by heat at around 95°C. Then, during annealing, sequence specific starter oligonucleotides called primers hybridize to the single stranded template DNA at 50 to 60°C and initiate (‘prime’) chain elongation. In the third extension step the heat-stable DNA polymerase extends the primer at around 72°C in the 5’ to 3’ direction, forming a nascent DNA strand that will serve as template in subsequent cycles. The above temperature cycles are repeated 20 to 30 times with the amount of product theoretically doubled in each cycle. In reality, amplification is exponential only during the early phase of cycling, when all reagents are available. Afterwards, as reagents are being used up, amplification first becomes linear and then reaches a plateau. The PCR is often used to detect the presence of viruses or genetic differences such as SNPs.

Figure 1.584. Figure 8.
<table>
<thead>
<tr>
<th></th>
<th>in vivo</th>
<th>in vitro</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>separation of DNA strands</td>
<td>enzyme</td>
</tr>
<tr>
<td>2</td>
<td>initiation</td>
<td>RNA primer, enzyme</td>
</tr>
<tr>
<td>3</td>
<td>elongation</td>
<td>enzyme</td>
</tr>
</tbody>
</table>

Figure 1.585. Figure 9.
Genes actually expressed in a tissue can also be investigated by PCR. Obviously, gene transcription yields RNA molecules that first need to be reverse transcribed into DNA to serve as template in the reaction. Retroviral enzymes called reverse transcriptases are used for this purpose. Thus, in this process called RT-PCR, DNA amplification is preceded by reverse transcription.

**Figure 1.586. Figure 10.**
In conventional PCR, products are detected after completion of the amplification. However, results obtained by such an endpoint detection are not reproducible as with reaching the linear and then the plateau phase, the efficiency of amplification becomes highly variable. Polymerase chain reaction can be made quantitative by real-time detection of the PCR products by fluorescent techniques. These include the use of fluorescent dyes such as SYBR Green which selectively bind to double-stranded DNA, or of product specific fluorescent probes such as those used with TaqMan chemistry.

Figure 1.587. Figure 11.
The determination of DNA sequence (sequencing) is also a PCR based method.

Figure 1.588. Figure 12.
1. Oral biology

Figure 1.589. Figure 13.
Nowadays the importance of high throughput screening is increasing. With these methods large number of samples can be tested or numerous questions can be answered using one sample. These kinds of examinations are possible by microarray methods such as the DNA chip. Just like Northern and Southern blotting, microarrays are based on nucleic acid hybridization. Unlike, however, in blotting techniques, it is the array of probes that are immobilized on microarrays and the sample molecules, not the probes, are labelled (with fluorescent dyes). These methods are suitable, for example, to study the expression of numerous genes simultaneously, and, when using different fluorescent dyes to label different samples, we can even compare their expression pattern (e.g. in normal vs. in cancer cells).

**Figure 1.590. Figure 14.**

![Diagram of microarray process](image)

**Figure 1.591. Figure 15.**
Figure 1.592. Figure 16.
Figure 1.593. Figure 17.
Figure 1.594. Figure 18.
Figure 1.595. Figure 19.
1. Oral biology
Figure 1.596. Figure 20.

Figure 1.597. Figure 21.
1. Oral biology
44.1. Test – Practices: PCR technique in dental research (answers)

1. You would like to separate longer and shorter PCR products (linear, double stranded DNA) by gel electrophoresis. During the separation...

A. shorter molecules migrate faster because their weight is less
B. shorter molecules migrate faster because they can move more easily through the pores of the gel
C. longer molecules migrate faster because they have more charge
D. longer and shorter molecules migrate with the same speed as all fragments are linear

2. Which statement is FALSE for the PCR reaction.

A. Cycles of PCR are driven by temperature change
B. The sequence of interest is amplified in the reaction to become easily detectable
C. The specificity of the reaction depends on the sequence of primers and the annealing temperature
D. DNA primase is used for the reaction

3. We perform quantitative (Real Time) PCR reaction using Taqman chemistry. Which statement is TRUE?

A. The Taqman probe binds between the strands of DNA and then its fluorescence intensity increases
B. Primers are not necessary when Taqman probe is used
C. The Taqman probe increases the specificity of the reaction because not only the primers but also the Taqman probe must bind to the sequence to be detected
D. The Taqman probe is labelled by a reporter and an interviewee dye

45. 1.45. Practices: Investigation methods of mineralised tissues – Kristof Kadar
A wide variety of methods are used for experimental and clinical hard tissue investigations. Without striving for completeness, this chapter focuses on techniques that help to diagnose and follow the disease processes of dental hard tissues (erosion, caries) either in an experimental or in a clinical setting.

45.1. Quantitative methods

45.1.1. Investigation of surface hardness

The general principle of surface hardness testing is to press a so called indenter (a tip, usually made of very hard material such as diamond) into the test material’s surface with a known force. The geometric parameters of the impression (depth, diameter, surface) are related to the hardness of the test material’s surface. Hardness measurement methods differ in the geometry and size of the indenter, as well as in the applied force and in the impression size/depth. **Static hardness measurement** methods measure the irreversible deformation of the test material. The more recently developed **depth-sensitive (dynamic) hardness measurement** methods became increasingly popular in dental hard tissue studies.

**Static hardness measurement**

The most frequently used hardness measurement methods in dental hard tissue investigations are the Vickers and the Knoop hardness tests, although the Rockwell and the Brinell methods are also used in certain studies. These methods require flattened polished surfaces and the test surface to be positioned perpendicularly to the long axis of the indenter, and are thus almost exclusively used in in vitro models.

**Figure 1.598. Figure 1. – Vickers hardness measurement**

**Figure 1.599. Figure 2. – Static hardness measurement methods**

<table>
<thead>
<tr>
<th>Static hardness measurement methods</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vickers hardness</td>
</tr>
<tr>
<td>➢ indenter: diamond, square pyramid with 136° between the faces</td>
</tr>
<tr>
<td>Knoop hardness</td>
</tr>
<tr>
<td>➢ indenter: diamond, rectangular pyramid apex angles 130°, and 172°30’</td>
</tr>
<tr>
<td>Rockwell hardness</td>
</tr>
<tr>
<td>➢ uses different indenters for hardness calculation:</td>
</tr>
<tr>
<td>➢ 1.50 mm (0.06”) diameter steel sphere (HRB)</td>
</tr>
<tr>
<td>➢ 120° diamond cone (HRA and HRC)</td>
</tr>
<tr>
<td>Brinell hardness</td>
</tr>
<tr>
<td>➢ indenter: steel ball (or tungsten carbide for harder materials), typically 10 mm diameter</td>
</tr>
</tbody>
</table>
Depth-sensitive (dynamic) hardness measurement (nanoindentation methods, nanohardness measurement)

In depth-sensitive (dynamic) hardness measurement methods, the size of the indenter and the impression are typically on the micro-/nanoscale. Most commonly, these methods use a Vickers (see above) or a trigonal pyramidal Berkovich diamond indenter. During the measurement, the indenter tip is pressed into the test material’s surface with a predefined loading and unloading profile, while both load (P) and displacement (h) is recorded. The resulting load-displacement curve reflects the mechanical properties of the test material; using the known geometry of the indenter tip several parameters including surface hardness and Young modulus can be calculated. As a further advantage, the investigation of very hard materials (such as enamel) is possible without microscopic measurement of the impression. These methods are also capable of measuring depth dependent variations of hardness, as well as the change of hardness under pressure.

Figure 1.600. Figure 3. – Schematic P-h curve of depth-sensitive hardness measurement. P_m, maximum impression force; h_m, maximum impression depth; h_o, permanent impression depth

45.1.2. Surface profilometry

Measurement of surface roughness (surface profilometry) is a useful tool for the investigation of material loss during erosion.

It involves contact (tactile) methods (direct contact, using a thin tip) and non-contact (optical) methods using laser or other light sources to scan the surface of the test material. The size and geometry of the tip are important determining factors of the horizontal resolution; therefore the careful selection of a proper tip is important. The vertical resolution for smooth surfaces can be as low as 0.1 nm under optimal conditions. The scanning probe (stylus) may damage softened hard tissue surfaces during scanning, thus distorting the results. The test surface can be scanned directly, in certain situations, however, the scanning of a high-quality replica is more appropriate.

Although optical (laser) scanning methods leave the test surface intact, transparency and colour of the dentin/enamel may influence the results. A possible solution is to substitute the test surface with a high-quality replica.

Several parameters are in use for the characterization of surface roughness. The arithmetic mean (R_a) or root mean square (R_q) of deviations from the surface centerline, are the most commonly used parameters in dental hard tissue research.

The figures below show some of the basic concepts of surface roughness measurement and most common amplitude parameters for surface roughness.

Figure 1.601. Figure 4.
45.1.3. Microradiography

Microradiography quantifies the changes in mineralisation during the erosion/caries process, by measuring the attenuation of X-rays transmitted through a thin section of the sample. The intensity of the emergent beam is recorded with a photographic emulsion, CCD camera etc. Quantification is usually performed using a simultaneously imaged reference aluminium step wedge. In transversal microradiography (TMR) the measuring beam is perpendicular to the direction of lesion progress, while in longitudinal microradiography (LMR) the beam is approximately parallel with it. The sample tissue section is typically 50-200μm thick in TMR, while in LMR it is usually thicker, with dentin samples as thick as 800μm. Microradiography is mostly suited for in vitro use.

45.1.4. Fluorescence based methods

Dental hard tissues show considerable autofluorescence in several wavelength ranges. The green fluorescence in response to blue light excitation is the physical basis of the QLF (quantitative light-induced fluorescence) method. The source of auto-fluorescence is thought to be the enamel dentinal junction (EDJ), removal of the underlying dentine abolishes the fluorescence. Fluorescence from an area with demineralised enamel is weaker probably due to the light scattering effect of lesioned enamel, which partially masks both the excitation light and the fluorescent signal. A light with a wavelength (λ) of 370 nm is used for excitation, the resultant emission is then detected by a small intra-oral camera fitted with a long-pass filter with a 540 nm cut-on wavelength to filter out the excitation light. The lesion is measured by analysing the captured image.

Using the same principle laser-based systems are also available. These methods are also used in clinical devices for the diagnosis of caries.

45.2. Qualitative and semiquantitative methods

Dental hard tissues can be also studied by other methods generally used for micro-/nanoscale cellular and histological investigations.
Light microscopy methods are used for classical histological investigations, to study mineralization structure (by polarized light microscopy), and to study surface topography and cellular structure (confocal laser scanning microscopy).

Transmission electron microscopy has been used to study subcellular structures. The ability to image segmented surfaces with a wide range of magnification (10x~1,000,000x) make scanning electron microscopy especially suited for dental hard tissue studies. One of the main disadvantages of the above methods is that the sample preparation required creates non-physiological conditions. Thus, extreme care is needed to preserve (ie. fixate) the original structures to be examined.

The large family of atomic force microscopy (scanning force microscopy) methods are suited to study the surface on the atomic scale. Moreover, some of these methods can be used under near-physiological conditions (ie. in an aqueous medium).

45.3. Test – Practices: Investigation methods of mineralised tissues (answers)

1. Which one is true for the quantitative light-induce fluorescence (QLF) method:
A. fluoresence from an area with deminarelised enamel is weaker
B. the excitation light is red
C. the method is useful in caries diagnostics
D. used only for research purposes
E. the fluorescence is determined only by the enamel

2. Which one is true for static hardness testing methods:
A. widely used in clinical diagnostics
B. require a prepared, flat surface
C. the size of the impression is typically on the nanoscale
D. suitable for the measurement of the Young modulus
E. R, is a commonly used parameter

3. Which of the following methods uses a ball (sphere)-shaped indenter?
A. Vickers hardness test
B. Rockwell hardness test
C. Brinell hardness test
D. Knoop hardness test
E. Young hardness test

Literature


46. 1.46. The structure and development of dental deposits. Dental plaque and calculus. Scaling, polishing, professional and home care oral hygiene – Balazs Sandor

46.1. The structure and development of dental deposits

Dental deposits

Definition: various materials accumulated on the surface of the teeth

Classifications according to consistence

- soft deposits: acquired pellicle, dental plaque, materia alba, debris,
- hard deposits: calculus (tartar).

46.1.1. Acquired pellicle (salivary pellicle)

A thin (0.1-0.8µm), initially transparent salivary film formed on the teeth surfaces (or dentures and other appliances in the mouth). The pellicle consist of absorbed proteins and macromolecules from the saliva and the crevicular fluid and components from bacterial host cells may also be incorporated into it. It has to be distinguished from the microbial biofilm (plaque). The pellicle has great importance in the protection of the enamel surface by controlling the de- and remineralization processes and protecting the tooth from abrasion, but it also facilitates bacterial absorption. Extrinsic discoloration of the teeth is usually caused by compounds incorporated into the pellicle (Table 1; Figure 1.)

Table 1.1. Table 1. – Extrinsic tooth discoloring compounds

<table>
<thead>
<tr>
<th>Extrinsic causes of tooth discoloration</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diet</td>
<td>Tea, coffee</td>
</tr>
<tr>
<td>Oral hygiene</td>
<td>Dental plaque, calculus</td>
</tr>
<tr>
<td>Habits</td>
<td>Smoking</td>
</tr>
<tr>
<td>Medications</td>
<td>Chlorhexidine</td>
</tr>
<tr>
<td>Occupation and environment</td>
<td>and Exposition to metals</td>
</tr>
</tbody>
</table>

Figure 1.603. Figure 1. – Extrinsic discoloration of the acquired pellicle caused by chromogenic bacteria
Acquired pellicle can be removed from the surfaces by professional cleaning (see later). It takes approximately thirty seconds to form again.

46.1.2. Dental plaque

A naturally acquired bacterial deposit that forms on the surface of the teeth, and is an etiological factor for dental caries and inflammatory disease of the periodontium. Caries, gingivitis and periodontitis are not caused by single pathogenic microorganisms, but by the whole community. Bacterial accumulation on the tooth surfaces covered by the acquired pellicle happens randomly. There are forces which facilitate bacterial adhesion (undisturbed environments: fissures, interproximal surfaces apically from the contact surface) and forces that remove them (mastication, tongue, toothbrushing). These forces determine more or less if plaque accumulates at a given site on a tooth. Initial plaque formation takes at least 2 hours. Isolated colonies attach to the irregularities of the tooth surface. The colonizing bacteria begin to multiply. By the 21 day plaque accumulation becomes relatively stable. With the thickening of the dental plaque oxygen diffusion will be limited, allowing facultative or obligate anaerobe bacteria accumulate.

Stages of supragingival plaque formation:

• acquired pellicle,

• initial colonization,

• secondary colonization–maturation.

**Initial colonization:** a few hours after cleaning the tooth surfaces, bacteria are found on the surface of the pellicle. Their attachment requires physicochemical interactions between molecules (electrostatic forces, hydrophobic bonding, calcium bridging), and adhesions with specific molecules called adhesins (Figure 2.) The initial bacteria on the tooth surface are gram-positive facultative anaerobe microorganisms (*Actinomyces viscosus, Streptococcus sanguine, Streptococcus mutans*).

**Figure 1.604. Figure 2. – Mechanisms of bacterial attachment to the acquired pellicle**
Secondary colonization- maturation: bacteria that are not capable of colonizing the clean tooth surface do that so by inter-microbial adherence (co-aggregation). The growth of the attached species leads to a oxygen-deprived environment causing a shift from gram-positive facultative bacteria to gram-negative anaerobic microorganisms (Prevotella intermedia, Prevotella loescheii, Capnocytophaga species, Fusobacterium nucleatum, Porphyromonas gingivalis). With the maturation of plaque intermicrobial metabolic associations develop. By the 21st day of plaque accumulation the microbial composition is relatively stable.

The localization of plaque:

• supragingival,
• subgingival.

Supragingival plaque is found coronally from the gingival margin. It can also be visualized by plaque disclosing agents. (Figure 3.) In the gingival sulcus plaque accumulates when the gingival inflammation begins. The deepening of the sulcus and edema (swelling) of the gingiva leads to the accumulation of the subgingival plaque. If the accumulation continues it may eventually result in periodontitis.

Subgingival plaque is found below the gingival margin, between the tooth and the gingival sulcular tissue. Tooth-associated and tissue-associated regions of subgingival plaque have been identified. The different regions of plaque are significant in the formation of periodontal diseases and dental caries. For example, marginal plaque has great importance in the formation of gingivitis.

Figure 1.605. Figure 3. – Visualization of plaque with plaque disclosing agent (Curaprox® Plaque Finder Tablets)
The role of plaque bacteria in the etiology of dental caries and periodontal disease

"Non-Specific Plaque Hypothesis" considers that disease is the outcome of the overall activity of the total plaque. This would not explain the different progression rate of caries and periodontal disease in different patients.

"Specific Plaque Hypothesis" proposes that, out of the diverse collection of microorganisms only a few species are actively involved in disease. This would not explain the presence of these specific bacteria in the healthy areas.

More recently the "Ecological Plaque Hypothesis" has been proposed which combines key elements of the earlier two hypotheses. The hypothesis holds that shifts in the relative number of organisms can be the trigger to the development of disease. These populational shifts can be caused by a change in environmental conditions (dietary intake, local or systemic host immune status).

46.1.3. Materia alba

Soft deposit on the tooth surfaces containing microorganisms, desquamated epithelial cells, proteins, leukocytes. It has no regular structure. It is usually yellow or grayish white. It can be easily removed by rinsing.

46.1.4. Debris (food debris)

Scattered remnants of food on the surface of the teeth. It is rapidly liquefied by the bacterial enzymes. Can be easily removed by rinsing.

46.1.5. Calculus (tartar)

Calculus is formed by the mineralization of plaque. It can be defined also as the last developmental stage of plaque formation. It is usually covered by a layer of unmineralized layer of dental plaque. Unmineralized layers of bacteria on the surface of the calculus are potentially pathogenic. It can be classified according to its localization to supra- or subgingival calculus. It can only be removed professionally (scaling), which in some cases may damage the tooth surface, especially on the roots.

Supragingival calculus: mineralized from the saliva, thus greater amounts are located adjacent to the excretion ducts of the large salivary glands (Figure 4; Figure 5.). Its color may vary from yellowish to dark brown.

Figure 1.606. Figure 4. – Supragingival calculus on the lingual surface of the lower incisors
Figure 1.607. Figure 5. – Supragingival calculus on the buccal side of the upper first molar

*Subgingival calculus* in mineralized from the crevicular fluid and blood. Its black color may sometimes be visible under the gingiva close to the gingival margin. Interproximal subgingival calculus can be visualized well on periapical and bite wing x-rays. It is harder than the supragingival calculus and is harder to remove.

Composition of calculus: consists of organic and inorganic components:

**Inorganic components:** calcium-phosphate, calcium-carbonate, magnesium phosphate. The main crystal forms are:

- Brushite,
- Octacalcium phosphate,
- Hydroxyapatite,
- Tricalcium phosphate,
- Magnesium whitlockite.
**Organic components:** mixture of protein-polysacharides, desquamated epithelial cells, leukocytes, and microorganisms.

### 46.2. Oral hygiene

**Oral hygiene** is the one of the four main pillars of primary dental prevention next to the use of fluorides, diet, and early treatments such as fissure sealing or orthodontic treatment.

Traditionally oral hygiene focuses on the control of the two most prevalent oral diseases, caries and periodontal disease. Basically it can be divided into professional and home care. The exact methods of keeping a good oral hygiene should be evaluated individually according to one's oral condition, personal preference, manual dexterity and lifestyle.

### 46.2.1. Professional oral hygienic care

Professional hygiene or also called professional cleaning includes scaling, root planning, curettage and polishing.

*Scaling* is the mechanical removal of calculus, dental plaque and stains from the tooth surfaces

*Root planning* is the removal of contaminated (bacteria, toxins) cementum or dentin, and leveling the irregularities, to make a smooth surface

*Curettage* is the removal of inflamed granulation tissues from the gingival pocket

*Polishing* is a procedure aiming to create a smooth tooth surface, to retard the rate of plaque accumulation.

#### 46.2.1.1. Scaling, root planning, and curettage

The tools of scaling (supra- and subgingival) can be classified as manual and mechanical instruments. For infectious patients (hepatitis, HIV, etc.) the use of manual instruments is favorable to reduce the chance of cross infection. Manual instruments are basically divided into three parts, the handle, the shank, and the blade. (Figure 6.)

![Basic parts of a manual instrument](figure6.png)

Manual instruments

- Scalers: sickle shape, hoe shape, chisel shape, files
- Curettes
  - Universal curettes
  - Area specific curettes.

Mechanical instruments:

- Sonic scalers
- Ultrasonic scalers
- Other special instruments (more details in periodontology).
There are several advantages and disadvantages for both mechanical and manual instruments, they complement each other well (Table 2.).

**Figure 1.609. Table 2. – Comparison of mechanical and manual scaling instruments**

<table>
<thead>
<tr>
<th>Hand instruments</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sickle scalers</td>
<td>Superior tactile sensation</td>
<td>Precise angulation of the blade is necessary</td>
</tr>
<tr>
<td></td>
<td>Good access to tight pockets</td>
<td>Frequent sharpening required</td>
</tr>
<tr>
<td></td>
<td>Good adaptation to different surfaces</td>
<td>Force is needed</td>
</tr>
<tr>
<td></td>
<td>No aerosol</td>
<td>Tiring the operator</td>
</tr>
<tr>
<td></td>
<td>No heat production</td>
<td>Takes more time</td>
</tr>
<tr>
<td>Hoe scalers</td>
<td>Little soft tissue damage</td>
<td>Poorer tactile sensation</td>
</tr>
<tr>
<td></td>
<td>Requires less time</td>
<td>Aerosol highly contaminated</td>
</tr>
<tr>
<td></td>
<td>No sharpening of tips needed</td>
<td>Not all handpieces can be sterilized</td>
</tr>
<tr>
<td></td>
<td>Less things are needed</td>
<td>Possible risk for patients with pacemakers (magnetostrictive ultrasonic scaler)</td>
</tr>
<tr>
<td></td>
<td>Contraindicated in infectious patients</td>
<td></td>
</tr>
</tbody>
</table>

*Sickle scalers:* (Figure 7.)
- Heavy instruments to remove calculus.
- Flat surface with two cutting edges that converge in a sharp tip.
- For supragingival use because of the design (third edge, size).
- Activated with a pulling movement.

**Figure 1.610. Figure 7. – The „toe view” of a sickle scaler**

*Hoe scaler:* (Figure 8.)
- Mainly for subgingival scaling.
- Blade angled 99° - 100° degrees to the shank.
1. Oral biology

- Activated with a pulling movement.

**Figure 1.611. Figure 8. – Schematic drawing of a hoe scaler**

*Chisel scaler:*
- Used for the scaling of the interproximal surfaces of teeth too close to each other.
- Usually double ended instruments (one strait and one curved end).
- Activated with a pushing movement.

*File scalers: (Figure 9.)*
- Series of blades on their surface.
- Used subgingivally to remove hard calculus.
- They can easily make a rough and hollowed surface.
- Nowadays curettes are used instead, which are also suitable for root planning and fine scaling.

**Figure 1.612. Figure 9. – File scaler**

*Curettes:*
- Designed for the removal of subgignival calculus, root planning, and removing the inflamed soft tissue lining of the gingival pocket (sulcus deeper than 3 mm).
• Only the cutting edges are sharp (without third edge), causing less trauma to the gingival, when being used in deep pockets.

• Semicircular convex base (Figure 10.).

• The blades are curved to fit the convexities of the teeth.

• Two basic types: universal and area specific (Gracey curette; Figure 11.).

**Figure 1.613.** Figure 10. – Universal curettes: face is perpendicular to the lower shank. Area specific curettes: Gracey curettes are designed to adapt specific anatomic areas. Only one working edge (lower). There are usually double ended instrument in the set of 7.

**Figure 1.614.** Figure 11. – Gracey curette (7/8). Double ended

*Ultrasonic scalers* (Figure 12):

• Suitable for the removal of calculus, plaque, or stains and for root planning.

• Two types: piezoelectric and magnetostrictive.

• The use of magnetostrictive ultrasonic scaler is contraindicated in patients with pacemaker.

• Alternate current generates oscillations.

• The frequency of the vibration ranges between 20,000–45,000 Hz.

• Specially designed tips for different indications and areas.

• Heat is produced by the ultrasonic vibrations, water spray is used for the cooling.

• Bubbles are formed inside the vapor, that collapse quickly, releasing energy (cavitation).

• Cavitation increases the effectiveness of the ultrasonic scalers.
Cavitation is only effective when water is vaporized totally (Figure 13.).

**Figure 1.615.** Desktop ultrasonic scaler

**Figure 1.616.** Appropriate water amount (left); inappropriate, too much water (right)

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**Sonic scalers:**

- Vibrations are generated with compressed air.
- Specially designed tips are available.
- Frequency of vibration ranges from 2,000-6,000 Hz.

Both sonic and ultrasonic mechanical instruments have advantages and disadvantages (Table 3.).

**Figure 1.617.** Comparison of mechanical scalers according to Perry DA, Beemsterboer P, Carranza FA: Techniques and Theory of Periodontal Instrumentation. Philadelphia, WB Saunders, 1990
46.2.1.2. Polishing

It can be divided into traditional polishing and selective polishing:

- *traditional polishing*: coronal polishing of every accessible tooth surface, to remove plaque and stains. According to its philosophy it is used prior to fluoridation to enhance the fluoride uptake, or after scaling to make a smooth surface. Some studies show that polishing does not increase fluoride uptake, what’s more it removes some of the fluoride-rich enamel surface. The opinions on polishing after scaling differ, the end product of scaling a root planning is emphasized to be a smooth surface that needs no further polishing.

- *selective polishing*: the concept is that plaque should be removed by the patient and polishing should only be done to remove extrinsic stains, this way reducing the side effects of polishing, such as:
  
  - the removal of fluoride rich layer,
  - inappropriate abrasive agent selection may increase the surface roughness,
  - Loss of tooth structure with increased dentinal sensitivity,
  - Increased gingival inflammation due to tissue manipulation, or chemical irritation.

Polishing with abrasive materials is a part of tribology, a discipline associated with physics and chemistry of materials science and surface contact engineering. Tooth polishing can be defined as two-body or three-body abrasion. In the case of two-body abrasion rubber cups impregnated with abrasive materials are used (two-body: two surfaces are in contact, the tooth surface and the rubber cup). Most frequently the three-body abrasion is used where the abrasive material is being applied to the tooth surface in a paste form with a polishing brush (three-body: polishing brush, polishing paste, tooth surface).

Factors influencing the efficiency of polishing:

- Speed (rpm),
- Pressure,
• Quantity of paste,
• Shape of abrasive particles,
• Size of abrasive particles,
• Hardness of abrasive particle (they should be harder than the surface being polished).

Tools of polishing
• Rubber cups,
• Polishing brushes (bristle brush),
• Dental tape (interproximal polishing strips),
• Air abrasion,
• EVA-system.

**Rubber cups** (Figure 14.) are hollow rubber shells. They are being used in the low-speed handpiece (RA bur shank) The soft rubber material may be impregnated with abrasive agents and fluoride. When using a non-impregnated cup, polishing pastes may be applied inside. This is suitable for gradual dosage of the polishing material. With light pressure the sides of the cup can access hidden areas without harming the soft tissues.

**Figure 1.618. Figure 14. – Rubber polishing cup**

![Rubber polishing cup](image)

**Polishing brushes** (Figure 15.) are available in conical and bell shape, with natural silicon and plastic bristles. They are used in a low speed handpiece. It may be impregnated with polishing material. Use of the brush should be confined to the crown to avoid injury of the soft tissues.

**Figure 1.619. Figure 15. – Polishing brush**

![Polishing brush](image)
Dental tapes (Figure 16.) are used in the interproximal areas that are inaccessible for the brushes and the cups. The tape is coated with abrasive material (aluminium-oxide).

Figure 1.620. Figure 16. – Dental tapes

Air abrasion (Figure 17.) is effective in removing staining and soft deposits. The results of studies about the abrasive effect of the air-powder polishing device on cementum and dentin show that tooth substance can be lost, its use on rerestorations may result in clinically relevant surface damage and material loss. Damage to gingival tissue is transient and insignificant clinically. The powder usually contains sodium- or calcium-bicarbonate. This may also reduce the tooth sensitivity. It is contraindicated in patients with low sodium diet, hypertension, respiratory illness, infectious disease, renal insufficiency, Addison's disease, Cushing’s disease, metabolic alkalosis, or certain medications, such as mineralocorticoid steroids, antidiuretics, or potassium supplements.

Figure 1.621. Figure 17. – Air abrasion handpiece

46.2.2. Home care oral hygiene
1. Oral biology

After professional cleaning soft microbial dental plaque continually reforms on the tooth surfaces, therefore, individual home oral hygiene cannot be replaced by any other methods. Plaque can be removed mechanically and chemically.

46.2.2.1. Mechanical plaque control

- Toothbrushes,
- Supplemental tools,
- Dentifrices.

The most important measure of home care mechanical plaque control is toothbrushing. The patients has to be taught individually, how to carry out toothbrushing according to ones age, gingival and dental conditions, lifestyles, and manual dexterity. This instructing includes choosing the optimal toothbrush, dentifrice and toothbrushing technique. Tooth brushing should be practiced at least two times a day for 3 minutes.

46.2.2.1.1. Toothbrush

There are two basic types of toothbrushes:

- Manual toothbrushes.
- Powered (electric) tooth brushes.

*Manual toothbrushes*

Manual toothbrushes may vary in size, form, and texture according to ones preference. The three main parts of the tooth brush are the handle, the neck (shank) and the head. The bristles are on the head of the toothbrush (Figure 18.).

The bristles are closely clustered, known as the tuft. The profile of the toothbrush may be convex, concave, multileveled or flat. Studies have shown the multilevel profiled toothbrush to be the most effective in plaque removal, especially in the interproximal areas.

*Figure 1.622. Figure 18. – Parts of the manual toothbrush*

![Figure 18. – Parts of the manual toothbrush](image)

The bristles of the toothbrush may be natural or nylon/polyester. Nowadays natural bristles are almost totally excluded from the market due to its disadvantages. Nylon or polyester bristles have uniform shape and size with rounded end to reduce the soft tissue damage (Table 4.)

*Figure 1.623. Table 4. – The comparison of natural and nylon/polyester toothbrush bristles*
The texture or firmness of the bristles is related to their composition, diameter, length and number. According to the firmness of the bristles, it may be graded as “soft”, “medium” or “hard”, although it is not standardized. The tufts of the toothbrush may be cross-angled to increase interproximal cleaning efficiency (Figure 19.).

**Figure 1.624. Figure 19. – Cross-angled bristles on OralB® Cross Action toothbrush**

The handle of the toothbrush may have different designs. For better grasp the sides may have indents for the fingers and a “thumb position” on the back of the handle. The toothbrush may be angled for better access around the mouth. (Figure 20.). For children usually the head is smaller, the texture is soft, and the handle is oversized for easier grip.

**Figure 1.625. Figure 20. – Toothbrush handle designs**

**Powered toothbrushes**

Toothbrushing with manual brushes requires good manual dexterity. This need led to the development of powered toothbrushes. The first electric toothbrushes were first introduced to the market in the US in the late 1960s. These toothbrushes were battery powered, with the vibrating movement of the head (first generation). Studies showed no significant difference in the cleaning efficiency when compared with manual brushes,
therefore, it was only beneficial for the handicapped. In the 1980s the second generation of electric toothbrushes was introduced with a small round head, and oscillating movement powered by rechargeable long-life batteries. Later the oscillating movement was combined with a perpendicular pulsation (3D) and a pressure control. Pressure control prevents damage of the oral tissues. The third generation of toothbrushes uses sonic vibrations. The heads of electric toothbrushes are usually smaller than of the manual ones, and replaceable. Although the second and third generations have been shown to be more efficient in plaque removal than the manual toothbrushes some studies have shown increased gingival recession and abrasion associated with the use of oscillating (second generation) electronic toothbrushes.

**Methods of toothbrushing**

There are four main goals of toothbrushing

- Removing plaque
- Removing debris and stains from the surface of the teeth
- Gingival stimulation
- Applying the toothpaste with special ingredients

Several toothbrushing techniques have been introduced in the past 50 years (Table 5., Figure 21.) Studies have shown no significant difference between the cleaning efficiency of these methods, so if the patient is managing effective tooth brushing without injuring the tissues, the brushing method should not be changed.

**Figure 1.626. Table 5. – Methods of toothbrushing**
<table>
<thead>
<tr>
<th>Method</th>
<th>Position and movement</th>
<th>Advantage/Disadvantage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roll method (Fones)</td>
<td>large, sweeping, scrubbing circles in occluded teeth, with the toothbrush held at</td>
<td>- easy to learn</td>
</tr>
<tr>
<td></td>
<td>perpendicularly to the surfaces.</td>
<td>- no subgingival plaque removal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- recommended for children</td>
</tr>
<tr>
<td>Horizontal “scrubbing”</td>
<td>Bristles held perpendicularly to the surface in occluded teeth</td>
<td>- Easy to learn</td>
</tr>
<tr>
<td>method</td>
<td></td>
<td>- Not efficient</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Recommended for small children</td>
</tr>
<tr>
<td>Bass method</td>
<td>Vibrating movement of the bristles placed in the sulcus with light pressure, angled in</td>
<td>- Efficient supra- and subgingival plaque removal</td>
</tr>
<tr>
<td></td>
<td>45° to the tooth surface</td>
<td>- May cause gingival injury</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Recommended for people with healthy periodontium</td>
</tr>
<tr>
<td>Modified Bass method</td>
<td>Same as Bass method combined with a sweeping movement</td>
<td>- Same as Bass method</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Sweeping increases efficiency of interproximal plaque removal</td>
</tr>
<tr>
<td>Stillman method</td>
<td>The bristles are placed on the gingival and the tooth surface in the same orientation</td>
<td>- Gingival stimulation</td>
</tr>
<tr>
<td></td>
<td>as the Bass method</td>
<td>- Effective subgingival plaque removal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Recommended for patients with advanced recession</td>
</tr>
<tr>
<td>Modified Stillman method</td>
<td>Same as Stillman’s method combined with a sweeping movement</td>
<td>Same as Stillman’s method</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Sweeping increases efficiency of interproximal plaque removal</td>
</tr>
<tr>
<td>Charters method</td>
<td>Bristles held towards the occlusal in 45° angle. Activated with a back- and forth</td>
<td>- Effective interdental cleaning</td>
</tr>
<tr>
<td></td>
<td>vibratory movement</td>
<td>- Hard to achieve</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- for patient after periodontal surgery during healing</td>
</tr>
</tbody>
</table>

Figure 1.627. Figure 21. – Schematic drawing of toothbrushing methods: (A) roll method, (B) horizontal technique, (C) Bass method, (D) modified Bass method, (E) Stillman’s method, (F) modified Stillman’s method, (G) Charters method
The sequence of toothbrushing:

- Maxillary teeth are the first ones to be cleaned, to avoid the deposition of loosened debris on the cleaned mandibular arch.

- Start in the molar region on vestibular surface around to the other side, then back around on the lingual side, then the occlusal surfaces.

- The mandibular arch, the same way as on the maxilla.

- Each brush placement should overlap each other.

46.2.2.1.2. **Supplemental tools**

It is necessary for thorough plaque removal. Toothbrushing is ineffective in the interproximal areas of the teeth. In special conditions (fixed prostheses, crown margins, root furcations, fixed orthodontic appliances) there are sites (tongue) where toothbrushing would not be effective without supplemental tools. Through plaque removal is important because remnant plaque increases the rate and growth of new plaque.

**Dental floss**

Recommended for healthy gingiva, where the papilla fills the interproximal space totally. A variety of flosses can be found in the market. The floss may be waxed or unwaxed. Studies have not shown any difference between the cleansing efficiency of waxed and unwaxed floss. Unwaxed floss may slip through tight contact areas, but may also tear and fray more easily. No wax residues have been found on the tooth surfaces where waxed floss was used, which makes it a good alternative, where the space is not restricted. The shape of the floss may be round in cross-section, or flat, tape shaped. The flat waxed type floss may be effective in tight contact areas. Polytetrafluoroethylene (Teflon) floss is more tear and shred resistant. The floss may be tufted for cleansing wide spaces. It may be flavoured, and colored. The color helps to visualize the removed plaque because of the contrast, or may be a help for visually impaired people.

When using floss it is important to control their movement in the interproximal area, to prevent gingival damage. Two basic methods can be applied.
• The *spool method* (finger wrap method) is recommended for people with good manual dexterity. About a 45 cm long floss is needed for this technique. The two sides of the floss are being rolled on the middle fingers of both hands. The two sides are then pulled apart, to taut the 15-20 cm floss left unrolled. Then the floss can be easily guided between the teeth by securing it with the index finger and the thumb (Figure 22.).

• The “loop method” is recommended for children or for those with less manual dexterity. In this technique the two ends of the 45 cm long floss is being tied together with a knot, to form a circle. Both hands, except the thumbs and index fingers are placed in the loop. For flossing in the upper arch the floss is guided with the thumbs, in the lower arch with the index fingers (Figure 23.).

Figure 1.628. Figure 22. – Methods of flossing: spool method

Figure 1.629. Figure 23. – Methods of flossing: loop method

Regardless the method being used the flossing procedure is the same in both methods.

• The floss is gently inserted between the teeth, with a seesaw motion to pass through the contact points.

• After passing the contact point the floss has to adapted to the interproximal surface being cleaned by forming a “C” shape.

• With up and down apical-coronal movement the surface is being cleaned. Seesaw movement may injure the gingiva. To avoid gingival damage, changing to the opposing interproximal surface may only be done in the contact area by creating a “C” form on the surface to be cleaned.
In some cases special variation or tool is needed for the application of the floss (fixed orthodontic appliances, fixed prosthesis).

- Floss threader: a plastic loop similar to the threading needle. Used for guiding the floss through tight areas, under pontics or orthodontic wires.

- Variable thickness floss (OralB® Super Floss®) has three parts (Figure 24.):
  - Stiff end makes it possible to thread under bridges, orthodontic wires.
  - Tufted part for cleaning wide spaces.
  - Normal floss.

Figure 1.630. Figure 24. – Variable thickness floss: (OralB® Super Floss®)

Special automated, vibrating appliances are available in the market to facilitate the use of dental floss.

Toothpicks

Toothpicks are used to dislodge debris and plaque from the interproximal area. The consistent use may result in gingival recession and the damage of the gingival tissues. To reduce the gingival injury triangular cross-section, softwood (balsawood) toothpicks are recommended.

Interdental brushes

Recommended in open interproximal areas, or for patients with fixed prosthesis and orthodontic appliances. May be effective in areas where the floss cannot be used (Figure 25.). Different sizes of interdental brushes are available in the market. To choose the right size Curaprox® company has developed a special tool (IAP-interdental acces probe) for measuring the size of the gap. The color-code of the probe determines the color and thereby the size of the interdental brush needed in the specific area (Figure 26.).

Figure 1.631. Figure 25. – On concave interproximal surfaces the dental floss is ineffective, in these spaces the use of interdental brush is recommended
Single tufted toothbrush

Only one bunch of bristles can be found on the head of the toothbrush (Figure 27.). Effective in the following areas:

- Mesially or distally from the teeth in open gaps, or in an edentulous area
- In the furcations of the teeth
- In wide embrasures, where there is no papilla
- Around the braces of the fixed orthodontic appliances, especially in the gingival third of the teeth.
Tongue cleaners

The irregular surface of the tongue may facilitate microorganisms and debris to accumulate on the surface. Deposits on the tongue may promote dental plaque formation and. Reduction of this debris, may decrease plaque accumulation and bad breath (halitosis). Some toothbrushes are equipped with a rough rubber surface on the back of the head for tongue cleaning. Other various tools are available in the market, but a “soft” toothbrush may also be suitable for tongue cleaning.

Irrigation devices

They are special home care devices, producing pulsating stream of fluid for the flushing of the mouth. Effective in removing loosely attached supra- and subgingival plaque. Recommended for patients with gingivitis, periodontal disease, and people with fixed orthodontic or prosthetic appliances.

46.2.2.1.3. Dentifrices

Cosmetic and therapeutic products are sold in the market. They aid in cleaning and polishing the tooth surface. Basically three types are available: powder, paste, gel.

Ingredients of toothpastes

- Abrasive materials 20-40%,
- Water 20-40%,
- Humectants 20-40%,
- Foaming agents 1-2 %,
- Binding agents 2%,
- Flavoring agents 2%,
- Sweetening agents 2%,
- Therapeutic agents 2%,
- Coloring and preservatives 1%.

Abrasives: abrasiveness of the toothpaste is dependent on the size and hardness of the abrasive particles. The basic abrasive property of the toothpaste may vary depending on the type of toothbrush (soft, medium, hard), and the toothbrushing technique used (pressure). The main types of abrasives found in toothpastes are: calcium-carbonate, sodium-bicarbonate, silicas, silicon-oxides, aluminum oxides.

Humectants and binding agents: they are added to the paste to maintain the moisture, to prevent the water from evaporating, and the hard particles to settle.

Detergents (foaming agents): facilitate the removal of loosened plaque. The main detergent used in toothpastes is sodium-lauryl-sulfate (SLS).

Flavoring and sweetening agents: added to improve the taste, and to ensure fresh aftertaste (spearmint, peppermint, wintergreen, cinnamon). Earlier honey and sugar was the only sweetening agent used in toothpastes. Nowadays sugar substitutes (saccharin, cyclamate) and sugar alcohols are used (sorbitol, mannitol, xylitol). Sorbitol and mannitol also have humectant effects. Glycerin as a humectant may also have sweetening effect. Xylitol is capable of facilitating remineralisation of the enamel.

Therapeutic ingredients of toothpastes:

- Fluorides: sodium fluoride, sodium-monofluorophosphate, stabilized, stannous fluoride, ammin-fluoride (See I-48 local fluoride administration).
- Plaque inhibiting agents: chlorhexidine, sanguinaria, lactoperoxidase, triclosan, zinc stabilized stannous-fluoride.
• Desensitizing agents: fluorides, strontium-chloride, potassium-nitrate, sodium-citrate.

• Tartar control agents: pyrophosphate system with a negative charge binds to positively charged calcium ions. Zinc system has a positive charge, which binds to the negatively charged phosphate ions.

• Whitening agents: removes stains with high abrasive content. They may also contain hydrogen-peroxide or carbamide-peroxide.

• Baking soda: therapeutic benefits have not been demonstrated in controlled clinical trials.

• Essential oils: clinical and laboratory tests suggest a benefit to gingival health and plaque reduction.

46.2.2.2. Mouthrinses and chemical plaque control

Mouthrinses can be classified as cosmetic, therapeutic and fluoride rinses. Chemical plaque control is the use of therapeutic and fluoride rinses. Chemical plaque control should be used in addition to mechanical plaque control, and not as a replacement.

46.2.2.2.1. Cosmetic mouthrinses

Mouthrinses against halithosis (bad breath), and xerostomia.

46.2.2.2.2. Therapeutic mouthrinses

According to their effect on plaque they can be categorized as:

• Antiadhesives: prevents bacterial attachment (Chlorhexidine, Delmopinol, Amine alcohol),

• Antimicrobial: Stops or slows down bacterial proliferation (Chlorhexidine),

• Antipathogenic: alters the pathogenicity of plaque (Chlorhexidine).

Chlorhexidine (CHX):

Most effective anti-plaque agent to date. CHX is a cationic compound that binds to the enamel, the acquired pellicle, the plaque and to the mucous membrane. It is slowly released in the following 12-24 hours. Different concentrations are available in the market. Therapeutic 0,2 % CHX solutions are only recommended for short-term use (2 weeks) after surgery or acute inflammations in the mouth. Its side effects are altered taste sensation, metallic taste, increased calculus formation, superficial desquamation of the soft tissues. The surfactants of the dentifrices inactivate CHX. It is important draw the attention of the patient no to use CHX thirty minutes before and after regular toothbrushing.

Essential oils:

Listerine® was the first antiseptic mouthrinse with essential oils. The main therapeutic ingredients are thymol, menthol, eucalyptol and methyl-salicylate. The original formula contains 26,9 % alcohol. It reduces plaque accumulation and the severity of gingivitis.

46.2.2.2.3. Fluoride rinses

(See I-48 local fluoride administration.)

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47. 1.47. The development of the carious lesion. Demineralization and remineralization – Balazs Sandor

47.1. Definition of dental caries (decay)

A progressive process in the hard tissues of the teeth, due to the disturbance in the balance of the demineralization-remineralization processes, caused by the acidic pH level induced by the bacterial deposit (plaque) on the surface of the teeth. This chronic process starts on the surface of the teeth covered by plaque, and in progresses into the deeper layers. The initial phase of caries formation is reversible, but in time irreversible destruction is formed. If chemical processes that cause demineralization prevail, it leads to the formation of caries: first the initial lesion is formed (white spot lesion, macula cretosa). In this phase the lesion is limited to the superficial layers of the enamel, the disease is reversible, and may even be remineralized from the saliva. In the case of decrease in the pH level of the oral cavity and the plaque, calcium and phosphate ions are being leached out from the enamel, causing demineralization. Demineralization is a reversible process, its dynamics is mainly determined by the current pH levels in the surroundings of the enamel. If the pH level decreases under a critical level (5,5), demineralization; above this critical level the process is turned, remineralization can be seen. Without intervention the white spot lesions will discolor, the enamel prisms collapse in the middle and the lesion progresses into the deeper layers of the teeth. From this point the process is irreversible, filling therapy is indicated.

According to these there are four main factors required for the formation of caries (Figure 1).

Figure 1.634. Figure 1. – Factors required in the formation of caries
47.2. Structure of the tooth enamel

Microscopically the tooth enamel is a lamellar structure, with a mass of organized crystals. It is the hardest substance in the human body. As it is formed by the active ameloblast during tooth development, after eruption of the tooth further enamel development is not possible. The basic unit of the enamel is the enamel prism (enamel rod) surrounded by interprismatic substance, their characteristic radial orientation is shown in Figure 2 and Figure 3.

Figure 1.635. Figure 2. – The orientation of the enamel prisms

Enamel prisms represent a characteristic orientation on the microscopic pictures of cross-section grindings, it is best compared to keyhole (Figure 4.).

Figure 1.637. Figure 4. – Schematic drawing of the microscopic picture of the enamel
47.3. Demineralization

47.3.1. The process of demineralization
Demineralization is the loss of minerals, which starts in the interprismatic region of the tooth enamel. The enamel crystals become thinner, intercrystal spaces broaden, the orientation and arrangement of the crystals are lost. The porosity of the enamel increases, it looses its transparency, causing the opaque, matt, “white spot” appearance. The superficial layer of the enamel (10-30 μm) is sound and hard. This is a periodic process characterized by the continuous alternation of de- and remineralization.

**Figure 1.640. Figure 7. – Superficial enamel demineralization, the interprismatic region is damaged**

Layers and characteristics of the demineralized area:

1. Relatively intact surface zone: it has a comparatively greater resistance due to the higher mineralization rate and fluoride concentration.

2. Body of the lesion: porous layer representing the greatest volume of the lesion, with a markedly homogeneous appearance.

3. Dark zone: zone of the demineralization.

4. Translucent zone: a represents the advancing front of the lesion, increased porosity compared to the normal enamel, this zone is not always present.

**Figure 1.641. Figure 8. – Layers of the demineralized enamel**

### 47.3.2. Reducing the chance of demineralization

1. Reducing the number of acidogenic bacteria.

2. Reducing the amount of acids in the oral cavity.

3. Reducing the amount of plaque.
4. Mechanical plaque control: toothbrushing, flossing, etc.
5. Antimicrobial function of the saliva.
6. Limiting the consumption of refined carbohydrates.
7. Increased salivary secretion.
8. Immunization may be considered, still under investigation.

47.4. Remineralization

47.4.1. The process of remineralization

Definition: The re-deposition of minerals into the demineralized areas of the hard tissues of the erupted tooth. The formation of apatite crystals through the deposition of calcium (Ca\(^{2+}\)), phosphate (PO\(_4^{3-}\)) and fluoride (F\(^{-}\)) ions from the supersaturated saliva of synthetic materials. Remineralization is achieved through the growing of the remnant apatite crystals, rather than the formation of new ones. Fluoride ions promotes apatite deposition, it precipitates in the form of fluoroapatite, and enhances crystal growth. The uptake of fluoride ions by the demineralized areas of the enamel is increased, compared to the intact enamel and it acts as a fluoride reservoir. The composition of the remineralized enamel differs from that of the intact enamel, as it is more compact and more resistant against acidic attacks. De- and remineralization are reversible processes, diffusion through the pores of the enamel, transport and precipitation of ions.

47.4.2. The use of remineralizing solutions

There were several attempts for remineralizing with solutions containing silver nitrate (AgNO\(_3\)), stannous fluoride (SnF\(_2\)), eugenol, phosphoric acids, but calcium phosphate was proven to be the most effective out all. The combination of calcium-phosphate with fluoride significantly accelerates remineralization.

Xylitol increases the pH level and the salivary flow rate, this way increasing the concentration of phosphate (PO\(_4^{3-}\)) calcium (Ca\(^{2+}\)) and bicarbonate (HCO\(_3^{-}\)) ions in the oral cavity, resulting in significant remineralization.

Too high concentration of ions may slows down the remineralization, as the calcium and phosphate ions precipitate in the form of insoluble particles and are withdrawn from the active chemical processes.

47.5. Caries

47.5.1. The histopathological progression of caries

Caused by strong acidic pH level

- Change and demolition in the structure of enamel prisms and the interprismatic substances.
- Progression into the deeper layers, with cavitation on the surface.
- Caries reaches the dentino-enamel junction, allowing rapid lateral spreading.
- Fast progression in the dentin may lead to penetration of the pulp.
- Pulp involvement which eventually may lead to the gangrene of the pulp, as a consequence of dental caries.

Clinically these changes can be classified in the following way:

- Incipient caries (caries incipientis).
- Superficial caries (caries superficialis).
- Moderate caries (caries media).
- Deep caries (caries profunda).
- Deep complicated caries (caries penetrans).
Figure 1.642. Figure 9. – Incipient caries (demineralization, enamel translucency decreases, clinically white spot is visible, not detectable with probing, the main characteristic of this lesion described earlier, is that there is no cavitation)

Figure 1.643. Figure 10. – Superficial caries (loss of enamel matrix, rough or cracked surface can be detected with probing, may be discolored)

Figure 1.644. Figure 11. – Moderate caries (Dentin is involved, but does not extend through more than half of the dentin, the cavity spreads along the dentino-enamel junction and the dentin tubules, the total collaps of the enamel layer is visible, usually brownish discoloration and clinical symptoms are present)

Figure 1.645. Figure 12. – Deep caries (involves almost the entire thickness of the dentin, usually causes clinical symptoms)

Figure 1.646. Figure 13. – Deep, complicated caries (characterized by inflammatory symptoms)
47.5.2. Main bacteria involved in the formation of dental caries

Streptococcus mutans: main bacteria responsible for enamel caries.

Lactobacillus casei and Lactobacillus acidophilus.

Actinomyces viscosus: usually causes root caries.

47.5.3. The role of saliva in the formation of caries

The mucin and mucoid content of saliva facilitates plaque formation and accumulation

Protective components against caries: B

- Bicarbonates: salivary buffer capacity,
- Immunoglobulines: (IgA, IgG, IgM) antibacterial effect.

Trace elements:
• cariogenic: copper, selenium, mercury,
• Anti-cariogenic: molybdenum, tin, vanadium, fluoride.

47.5.4. Clinical classification of caries

According to formation of caries:

• **Primary caries**: forms on the previously intact surfaces.
• **Secondary caries**: form next to the fillings and restorations.
• **Residual caries**: demineralized non-infected dental tissues under the fillings.

According to localization:

• **Coronal caries**: on smooth surfaces or in the pits and fissures,
• **Root** caries.

According to progression:

• **Active** caries showing the signs of progression.
• **Inactive** (arrested) caries showing no further signs of progression and deterioration.
• **Caries rapida (rapid)**: caries showing fast progression, usually present in children.
• **Caries tarda (slow)** caries showing slow progression, usually present in elderly.
• **Caries insistens**, caries showing no signs of progression.

The appearance of the coronal caries is dependent upon:

• time of appearance of the tooth in the oral cavity
• position of the tooth in the dental arch
• anatomical shape of the crown

According to these the predilection sites of coronal caries can be seen in the following figures.

**Figure 1.648. Figure 15. – Approximal surfaces under the contact points**

![Approximal surfaces under the contact points](image)

**Figure 1.649. Figure 16. – A vestibular and oral surfaces between the crest of curvature and the border of the gingiva**

![A vestibular and oral surfaces between the crest of curvature and the border of the gingiva](image)
Figure 1.650. Figure 17. – In the pits and fissures of the occlusal surfaces

47.5.4.1. Fissure caries

Usually forms in the pits and fissures of the occlusal surfaces of the teeth, which is the typical area of debris retention. The enamel layer is the thinnest in the area of the fissure; therefore, the involvement of the dentin by the progression of the caries occurs soon. As long as this pathological process is limited to the enamel the progression takes place along the enamel prisms.

Figure 1.651. Figure 18. – Blue line showing the original orientation of the enamel prisms

47.5.4.2. Dentin caries

Due to the anatomical structure microorganisms can spread along the dentin tubules of the dentin towards the pulp. As a defensive reaction from the pulp, a sclerotic dentin layer is formed. The calcification of the dentin tubules in this area prevents the spreading of bacteria into the pulp. This mechanism is essential in the case of defense against caries showing slow progression.

Figure 1.652. Figure 19. – Microscopic picture of dentinal sclerosis

After bacterial invasion reaches the DEJ. Histologically the lesion shows a typical triangular shape pointing towards the pulp. More and more dentin tubules are involved. This process results in a fast cavitation, with fast lateral progression of the caries along the DEJ, eventually leading to unsupported enamel structures.

Figure 1.653. Figure 20. – Histological picture of dentin caries
1. Oral biology

Figure 1.654. Figure 21. – Schematic drawing of the histology of dentin caries

1. Zone: showing fatty degeneration of Tomes’ fibers, dentin tubules became impermeable. This is a part of odontoblast degeneration, prior to the formation of dentinal sclerosis.

2. Zone showing dentinal sclerosis, characterized by the calcification of the dentin tubules.

3. Zone is the zone of demineralization in the dentin.

4. Zone of bacterial invasion.

5. Zone is the area of disintegrated dentinal layer due to the effect of acids and enzymes.

**47.5.4.3. Root caries**

Soft, progressive lesion, which can be formed on any denuded root surface. It can only occur on the surfaces exposed to cariogenic processes of the oral cavity.

Mainly caused by actinomyces strains (Actinomyces naeslundii, Actinomyces viscosus, Actinomyces israelii), but significant number streptococcus mutans colonies can also be found. Microorganisms invade the cementum along the Sharpey’s fibers, from where they spread laterally (due to the concentric lamellar arrangement of the cementum). After the demineralization of the cementum layer the progression of the caries in the dentin is identical to that of the coronal caries. The rate of progression is slower compared to the coronal caries, due to the lower numbers of dentin tubules.
The critical pH level is 6.7: higher than that of enamel caries. A smaller decline in the pH level may be harmful.

Figure 1.655. Figure 22. – Picture showing root caries

47.6. Acid etching

More and more adhesive materials are being used during our dental treatments, to assure micro retention of these materials acid etching of the surfaces is required. This is a short (15 seconds), aggressive exposure of the dental hard tissues to acid, to achieve a microscopically rough surface. Similarly to the carious processes it acts through the demineralization of the surface of the enamel prisms and destruction of the interprismatic substances. This takes place under controlled conditions, depending on the restoration technique underlining and pulp protection may be indicated.

Figure 1.656. Figure 23. – Etching of the prepared cavity

Figure 1.657. Figure 24. – Scanning electron microscopeic picture of the enamel after etching (SEM 402x)
The aim of etching: Roughening leads to an increase in the surface, resulting in a much broader area of micro retention between the restorative material and the enamel. In controlled circumstances etching results in 30 µm deep tubules orientated perpendicularly to the surface. Etching may also have clinical consequences (postoperative sensitivity, pulp irritation, etc.)

Figure 1.658. Figure 25. – Scanning electron microscopeic picture of the enamel in higher magnification

Figure 1.659. Figure 26. – Adhesive material on the surface of the etched enamel
48. 1.48. The effect of fluorides and calcium, toxic considerations. Local and systemic fluoride administration – Balazs Sandor

48.1. Features of fluoride

Fluorine is a pale yellow gas, the most electronegative element. As it is the most reactive element it binds to other materials rapidly. The dental significance of fluoride is that it increases the resistance of the tooth enamel against acids. In presence of 1ppm fluoride concentration the hydroxyl group of the hydroxyl-apatite is replaced by fluoride ions, and fluoro-apatite is formed. This facilitates the remineralization of demineralized areas, the resulting enamel will be harder, thus will be more resistant against caries. In the presence of higher fluoride concentration calcium fluoride (CaF₂) is formed.

In the preeruptive stage of tooth development it has an endogenous effect on the hard tissues of the teeth. In the posteruptive stage it has a local exogenous effect on the microenvironment and surface of the teeth, as it may accumulate in the dental plaque or is secreted by the saliva.

It influences the shape of the teeth during development: cusps, shallower fissure, less susceptible to caries

In 10ppm concentration in the plaque it inhibits bacterial polysaccharide synthesis.

It inhibits the adherence of proteins and bacteria to enamel surface as well as the growth of cariogenic microorganisms (e.g.: Streptococcus mutans).

It accelerates the recrystallization of calcium and minerals dissolved from the demineralized tissues, thereby increasing the microhardness of the enamel.

General effects of fluoride are also well known: The effects on cardiovascular diseases, is that it inhibits arteriosclerosis. It has a beneficial effect on osteoporosis, as sufficient fluoride intake reduces the rate of femoral neck fracture in elderly women.

Formation of fluoro-apatite:

As earlier described in the presence of optimal fluoride concentration hydroxyl-apatite in the enamel is transformed into fluoro-apatite, thereby decreasing acid-solubility, and improving the physical properties of the enamel.

Fluoro-apatite formation:

\[ \text{Ca}_{10} (PO_4)_6 (OH)_2 + 2F^- \rightarrow \text{Ca}_{10} (PO_4)_6 F_2 + 2OH^- \]

Next to fluoride other trace elements such as strontium, vanadium and molybdenum also have beneficial influence on the process.

In high concentration of local fluoride, by the dissolution hydroxyl-apatite, insoluble calcium-fluoride precipitate is formed.

\[ \text{Ca}_{10} (PO_4)_6 (OH)_2 + 20F^- \rightarrow 10\text{CaF}_2 + 6\text{HPO}_4^{2-} + 20\text{OH}^- \]
Calcium-fluoride is a strong conglomerate forming molecule, which “entrap”s fluoride and calcium ions in an insoluble precipitate form. The calcium and fluoride ions are inhibited from the participation in the dynamic balance of apatite formation.

Fluoride can be present in different forms depending on the product:

- Stannous fluoride,
- Mono-fluoro-phosphate (MFP),
- Sodium-fluoride,
- Amin-fluoride,
- Acidulated phosphate fluoride (APF).

### 48.2. Toxic effects of fluoride

#### 48.2.1. Acute fluoride intoxication

The skin or mucosal contact with concentrated fluoride results in the formation of the extremely strong hydrofluoric acid due to superficial tissue humidity which can severely damage living organisms.

In case of fluoride intoxication the consequential injury of cellular protoplasm manifests in the inhibition of different enzymatic systems.

The aggressive CaF₂ forms a precipitate which binds calcium ions needed for physiological processes. The reduction of calcium causes reactive hypercalaemia (high potassium level) resulting in muscle weakness and cardiotoxicity.

It is important to mention CaF₂ may act as a reservoir for calcium a fluoride ions, at low pH levels (under five) in the oral cavity the slow dissolution of the molecule provides free fluoride ions.

#### 48.2.1.1. Fluoride intoxication

1. **Symptoms of fluoride intoxication:** nausea, vomiting, muscle cramps (local, generalized), abdominal pain or cramp, head ache, excessive salivation and lacrimation, nasal discharge, sweating. The extensive secretory production is of differential diagnostic importance.

   Further symptoms occur in case of higher fluoride concentrations: diarrhea, weakness/fatigue, hypocalcaemia, hypercalcaemia. If the latter increases it leads to loss of consciousness, coma, cardiac arrhythmia and later to death within 4 hours. Survival is only within 4 hours possible!

2. **According to the fluoride intake intoxication is indicated by following abbreviations:**
   - STD (safely tolerated dose) – 1-5 mg /bwkg dose, there are no symptoms.
   - PLD (potentially lethal dose) – 5-15 mg /bwkg is the lowest dose where death can occur; according to another classification this is the same as PTD (probable tolerated dose).
   - CLD (certainly lethal dose) – 32-64 mg/bwkg, at this dose there is no chance for survival.

3. **Tasks in case of fluoride intoxication:**

   If the intake is less than 5 mg F-/bwkg, only Ca has to be given orally ( e.g. milk), as it slows the absorption of fluoride from the stomach. Later on 4 hours of medical observation is sufficient.

   In case of an intake between 5-15 mg F-/bwkg hospitalization is indispensable. Beside oral Ca administration, induce vomiting. A toxicologist or other experienced specialist should be involved into the treatment, with specialized drugs and instruments.

Table 1. shows how to count the amount of consumed fluoride according to the age, body weight and the amount of swallowed tooth paste. PTD is 5 mg/kg.
Table 1.2. Table 1. – Calculations according to the body weight, age and the amount of consumed tooth paste

<table>
<thead>
<tr>
<th>age</th>
<th>average body weight</th>
<th>PLD / body weight 5mg/kg</th>
<th>1000ppm fluoride containing paste the swallowed amount from a 100 gr tube</th>
<th>400ppm fluoride containing paste the swallowed amount from a 45 gr tube</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>weight</td>
<td>tube</td>
</tr>
<tr>
<td>2 years</td>
<td>12 kg</td>
<td>60 mg F</td>
<td>60 g</td>
<td>60%</td>
</tr>
<tr>
<td>4 years</td>
<td>15 kg</td>
<td>75 mg F</td>
<td>75 g</td>
<td>75%</td>
</tr>
<tr>
<td>6 years</td>
<td>20 kg</td>
<td>100 mg F</td>
<td>100 g</td>
<td>whole tube</td>
</tr>
</tbody>
</table>

48.2.2. Chronic fluoride intoxication

48.2.2.1. Dental fluorosis

There are many different clinical appearances of fluorosis from white opalescent patches, through brownish discolorations to porous enamel. Nowadays the two mostly accepted index is the 10 stage Thylstrup-Fejerskov scale, which not only defines the severity of the lesion, but also aids our treatment planning. (Table 2) There are also other staging methods like the one determining the grade of fluorosis according to the F⁻ concentration of drinking water:

- Mild fluorosis (2 ppm fluoride in the water)

**Figure 1.660. Figure 1. – Clinical picture of mild fluorosis**

- Moderate fluorosis (3-5 ppm fluoride in the water)

**Figure 1.661. Figure 2. – Clinical picture of moderate fluorosis**
• Severe fluorosis (5-6 ppm fluoride in the tapwater)

**Figure 1.662. Figure 3. – Clinical picture of severe fluorosis**

### 48.2.2.2. Skeletal fluorosis

Symptoms: osteosclerosis, calcification of tendons, formation of multiple exostosis.

### 48.3. Systemic fluoridation

#### 48.3.1. Fluoridation of water

The recommended, ideal level of fluoride in water is 1mg/liter concentration. Natural water contains optimal concentration of fluoride in relatively few geographical locations. Some countries have decided to fluoridate the tap water. There are some concerns regarding the fluoridation of the public water supply as the amount of water consumption changes individually, increased water consumption may lead to excessive fluoride intake (can be caused by warm climate or may be dependent on working conditions). It can only be used in places where tap water is available. It raises equality issues. Some environmental groups oppose it, for fluoridation is intervention with nature and some natural processes can adversely affected. According to other organizations the freedom of choice is being violated by forcing the people to drink fluoridated water. The cannot choose to drink fluoride free water if the public water supply is fluoridated. With the spreading of bottle water this controversy overshadowed.

**Figure 1.663. Figure 4. – The change of DMF-S and the occurrence of fluorosis depending on the concentration of fluoride in the water**
48.3.2. Salt fluoridation

Cheaper method than water fluoridation, the freedom of choice is not violated, collective use is available. The recommended fluoride concentration in salt is 250 mg/kg.

48.3.3. Milk fluoridation

Fluoridation of milk is also cheap method. Fluoridation has no effect on the taste of milk, thus it can be well integrated into daily living. It is recommended as an alternative method for fluoridation with a 2-5 ppm fluoride concentration. It is only effective where immediate consumption possible, as fluoride can become ineffective in a short period of time, as it may bind to the milk proteins.

Figure 1.664. Figure 5. – Results of milk fluoridation

48.3.4. Fluoride tablets

According to studies, nowadays fluoride tablets are only recommended to patients of the high caries risk group. They are:
• Children from low socioeconomic status families.
• Mentally retarded children.
• High caries rate in primary dentition.
• High caries prevalence in the family.
• Long term medical treatment.
• Children during orthodontic treatment.

The effect of fluoride tablets is dependent on:

• time of administration (postnatal, preeruptive stage, but at least from the age of two years leads to 50-80% caries reduction),
• duration of administration (using fluoride tablets from birth until the age of 7 leads to 39-80% reduction),
• frequency administration, organizing administration (recommended at least for 200 days/year),
• other dietary factors (other ways of fluoride intake),
• oral hygiene and types of local fluoridation used.

Fluoride tablets also have local effects as they are slowly dissolved in the mouth. As a posteruptive effect fluoride incorporates during maturation and remineralization of the enamel. After absorption it is secreted with the saliva, thereby raising the concentration of fluoride in the oral cavity.

48.3.4.1. Types of fluoride tablets

Dentocar® tablet (contains 0.23 mg fluoride).

Dentocar® forte tablet (contains 1.00 mg fluoride).

Zymafluor® tablet (contains 0.25 mg fluoride).

Recommended daily intake for fluoride is 0.03mg/bwkg.

<table>
<thead>
<tr>
<th>Age</th>
<th>Fluoride tablet dosage according to the amount of fluoride in the water</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–0.25 mg/l</td>
<td>0.25–0.50 mg/l</td>
</tr>
<tr>
<td>0–2 years</td>
<td>0.23 mg fluoride</td>
</tr>
<tr>
<td>2–4 years</td>
<td>0.46 mg fluoride</td>
</tr>
<tr>
<td>4–6 years</td>
<td>0.69 mg fluoride</td>
</tr>
<tr>
<td>above 6</td>
<td>1 mg fluoride</td>
</tr>
</tbody>
</table>

48.4. Local fluoridation

Local fluoridation is an intervention of very high importance as it is frequently and easily performed, available for everyone and is a good anti-cariogenic prevention method. However we often see that the population is not properly informed regarding fluoridation and it is the task/responsibility of the dentist to clear doubts and pass correct information and facts.

We can divide local fluoridation into two groups:
• home (performed by the patient himself according to professional recommendation),

• professional (performed by a dentist or an oral hygienist in the office).

The results of tooth brushing, and this way the amount of fluoride delivered on the surfaces of the teeth holds more significance than the systemic fluoride administration. Most dentifrices available in the market contain fluoride in greater or lesser amount. There are only few fluoride free toothpastes. These tooth pastes are recommended for children under the age of three, who are unable rinse and are more likely to swallow the toothpaste.

48.4.1. Materials of home local fluoridation

48.4.1.1. Tooth pastes

Also fluorid-free forms exist, but these are appropriate only for children age 2-3 years (who are not able to expectorate), later depending on the age tooth pastes containing 500-1500 ppm fluoride are recommended.

Ingredients of tooth pastes:

• **Abrasives materials** (20-40%): calcium carbonate, calcium phosphate, aluminium oxide, silicate. Their effect depends on the particle size and hardness. Sometimes oily polishing materials are added to increase the abrasive effect.

• **Humectant** (20-40%): glycerol, propylene alcohol.

• **Detergents** (1-2%): earlier soaps and fatty alcohol sulfonates were used, which decrease surface tension. Nowadays, due to the unfavorable side effects of soaps as bad, sharp taste, other kinds of detergents are used e.g. sodium-lauryl-sulfate. These substances penetrate the plaque, facilitate the mechanical removal and cause the foaming of the paste.

• **Stabilizers and bonding agents** (<2%): oils, jellies, alginate, stabilizing the consistence, providing the pasty feeling.

• **Flavouring** (<2%): the most used material is menthol (spearmint), freshening the breath, cooling oral cavity.

• **Sweeteners** (<2%): Earlier honey and sugar was the only sweetening agent used in toothpastes, nowadays these are not accepted instead sugar substitutes and sugar alcohols are used (saccharine, cyclamate, sorbitol, mannitol) in tooth pastes.

• **Colorants** (<1%).

• **Therapeutics, chemical agents** (<5%)

  • **Fluorides**: inorganic or organic fluoride (tooth pastes and gels contain 1000-1500 ppm fluoride with the maximum of 120 mg per tube, special therapeutic toothpastes may contain 2500 ppm fluoride. For children between the age of 2 and 6, the tooth paste contains 500 ppm fluoride.

  • **Disinfectants**: chlorhexidine (ineffective with fluoride), triclosan.

• **Whitening agents**: carbamide-peroxid or hydrogen-peroxide.

48.4.1.2. Special gels for home use

Next to toothpastes gels may also be used. They contain higher concentration of fluoride (12000 ppm), not recommended for everyday use. The use is indispensable for the moderate and high-risk group.

Ways of application: direct application with toothbrush, after the regular evening tooth brushing. Rinsing is not allowed, they may only expectorate, this was the high concentration of fluoride has an effect during the sleep. The use of fluoridating splints is easy, comfortable and effective.

**Figure 1.665. Figure 6. – Fluoridating splint made with deep vacuum technique**
48.4.1.3. Mouthrinses

Various products are available. Before purchasing a mouth rinse one must be aware that no all of them contain fluoride. Even if they contain fluoride it is in a low concentration, the fluoridating effect is not sufficient. Some mouthrinses may contain disinfectants that are no compatible with other fluoride containing products. According to these, the fluoridating effect of mouthrinses is limited

Characteristics and requirements of fluoride rinses:

• It supplements mechanical cleaning.
• Supplements the oral hygiene of mentally or physically retarded patients.
• permanently inhibits bacterial attachment and growth on the surface of the teeth.
• Selectivelly inhibits growth of bacteria that are responsible for caries and periodontal disease.
• Reaches areas, where mechanical cleaning is impractical.
• Long lasting effect, ensure total plaque removal used 2-3 times a day used 2-3 times a day.
• Mouthrinses may not be toxic.
• May not have any local or systemic adverse effect.
• May not cause imbalance in the ecology of the oral cavity.
• Prolonged use should not cause allergy, may not accumulate, and should not be irritant.

48.4.2. Professional treatment

Fluoride solutions: NaF, SnF or aminfluoride containing solutions. Their use is easy but the effect is short lasting.

Varnishes: a strongly adherent fluoride layer is formed on the surface of the tooth. The cooperation of the child is important.

Gels: contain fluoride in a very high concentration (20,000ppm), thus great care must be taken during application. The fluoride may be introduced into the surface layer of the teeth with iontophoresis. For these treatments usually acidulated phosphate fluoride (APF) is being used. Rarely used method in Europe.
49. 1.49. Fissure sealing – Balazs Sandor

The four basic pillars of primary caries-prevention (Figure 1.).

1. Regular oral hygiene mechanical and chemical plaque control (immunization).
2. Increasing the resistance of the tooth against caries (reinforcement of tooth structures), fluoridation.
3. Healthy diet, nutritional advice.

Figure 1666. Figure 1. – Basic pillars of fissure sealing

Caries is formed most frequently on different predilection surfaces of the teeth that are not self-cleaning, and mechanical cleaning is difficult. There is increased plaque accumulation on these surfaces. Due to these pits and fissures are primary areas of caries formation. The situation is worse in the case of young permanent teeth, as their maturation is still in progress.

Concerning the efficiency of cleaning we can distinguish favorable and unfavorable fissures

There are five different, genetically predestinated fissure patterns (Figure 2.):

- “U”-formed fissures (wide fissures),
- “V”-formed fissures,
- “I”-formed fissures (narrow fissure),
- “IK”-formed fissures,
- Inverted “Y”-formed fissures.

Figure 1667. Figure 2. – Pattern of fissures

Unfavorable fissure forms are I, IK, and the inverted Y form as they may inhibit the complete removal of plaque.
49.1. Epidemiology

Caries is one of the most common chronic diseases. Among children and adolescents the primary sites of decay are the pits and fissures of deciduous teeth, above the age of six years the first permanent molars and later second permanent molars are affected. Although pits and fissures represent only 3% of all tooth surfaces, 70% of the caries and fillings are found on these sites. (Brown et al. 1996). The high caries levels of the fissures are due to their morphology. Steiner et al. demonstrated that among 13-14 year old children with low caries-risk the first permanent molars are affected in 80% of the cases. The second permanent molars are the next most affected teeth. Occlusal surfaces alone account for well over 50% of the caries in children ages 6-18.

Pit and fissure sealing is an effective method for the reduction of caries. According to the American Dental Association (ADA) the reduction of caries incidence in children and adolescents after the placement of resin-based sealants ranged from 86%-at first year to 58.6% at 4 years.

49.2. Definition of pit and fissure sealing

Pit and fissure sealing is a surface-specific preventive method to obliterate fissures and pits of permanent in order to enable a thorough cleaning of the smoothened surface thereby lowering caries-risk without altering normal function. (Figure 3;4)

Figure 1.668. Figure 3. – The fissure of a lower molar tooth, before fissure sealing

Figure 1.669. Figure 4. – Clinical picture after fissure sealing

Advantages of pit and fissure sealing:

1. Effective preventive method,
2. Cheap (compared with other restorative methods/interventions),
3. Painless,
4. Fast and easy to perform.

What to seal?
a. caries susceptible fissures and pits of permanent molars and premolars,

b. pits of the lingual surface of permanent upper lateral incisors,

c. deciduous teeth may be sealed if there are already one or more primary molars affected by caries, or because of the fissure morphology even if there is no caries.

The effectiveness of sealants for caries prevention depends on long-term retention. It is recommended to use fissure sealing for prevention whenever the tooth is at risk of developing caries (unfavorable fissure morphology, structural abnormalities, newly erupted hypomineralized teeth, or the patient is in the moderate or high caries risk group. The susceptibility of the tooth and the caries group of the child may change annually, therefore, risk assessment is necessary yearly. The advantages of sealants are obvious at high caries risk, but information on the benefits of sealing specific to different caries risk is lacking. Non cavitated carious lesions in the fissures are also indications of fissure sealing, as it reduces the percentage of lesions that progress, although in this case it is rather a therapeutic treatment, than a preventive one.

49.3. Basis of indication for pit and fissure sealing

Before indicating pit and fissure sealing a thorough caries-diagnostic examination has to be performed.

The main examination methods for diagnosis of caries are:

- visual inspection after cleaning and drying the tooth surfaces,
- adjunct detection tools,
- radiographic examination.

Visual inspection is regarded as primary examination among many others used. The use of sharp explorers is unnecessary, as it may harm damage the tooth surface. It is important to note that stains and extrinsic stains are no equivalent to non-cavitated lesions. Obtaining radiographs for the decision on fissure sealing should be avoided. Before treatment the age, dental status and caries risk of the patient have to be taken into account.

Caries risk assessment

Caries risk assessment models involve a combination of factors:

- diet,
- fluoride exposure,
- susceptible host (socially or physically compromised, patients with fixed orthodontic appliances, xerostomy, change in DMF-T, etc.),
- microflora,
- behavioral factors.

The caries-risk assessment is also dependent on the age of the child. For easier assessment “Caries Risk Assessment Form” according to the age of the patient can be accessed from www.ada.org.

Basically we consider the patient to be at “high risk” the following cases:

- Three of more new lesions per year,
- Beginning of orthodontic treatment,
- Chronic illness (e.g. diabetes), or hospitalization,
- Medically compromised patients,
- Presence of social risk factors.
a. Indications of preventive pit and fissure sealing for deciduous and permanent teeth
   • Healthy pits and fissures of patients with high caries-risk.

b. Relative contraindications of preventive pit and fissure sealing:
   • when the concerned tooth is only partially erupted,
   • deciduous tooth close to exfoliation,
   • in case of restoration on other well-separated surfaces fissure sealing may be used on the intact fissures,
   • in the case of pit and fissure caries.

c. Absolute contraindications of preventive pit and fissure sealing:
   • teeth with cavitated carious lesion or dentin caries on the,
   • when there is caries on another surface of the tooth, where the filling would involve the fissures,
   • lack of patients cooperation,
   • teeth with large occlusal fillings,
   • tooth that has remained caries-free for 4 years or longer has no clinical indications for sealant placement, unless there is change in caries-risk.

d. Other factors to be considered:
   • The most preferable time for fissure sealing is as soon as possible (6 months) following the total eruption of the tooth (the enamel is undergoing post-eruptive maturation), however, sealant placement may be indicated at other times when a given child's/patient’s caries risk status is modified. The length of time elapsed since the eruption is not the main factor for deciding on fissure sealing.
   • The placement of sealants should be limited to previously unrestored pits and fissures.
   • In some cases the sealing of partially-erupted tooth may be indicated, where the gingival flap may interfere with application procedure, due to the difficulty of moisture control. In these cases the use of retraction cords may be necessary. Until the total eruption of the tooth less moisture sensitive materials (glassionomer-cements) may be used for sealing.
   • Marginal defects of occlusal amalgam fillings may be sealed, delaying the need for replacement of the old amalgam and protecting the tooth from secondary marginal caries.

49.4. Technique of pit and fissure sealing with resin based pit and fissure sealing materials
   • Surface preparation (cleaning),
   • Isolation,
   • Etching (conditioning),
   • Washing and drying with water-air syringe,
   • Application of fissure sealing material,
   • Polymerization,
   • Checking the polymerized sealant,
   • Polishing with fluoride containing paste.
a. Surface preparation:

A clean surface is necessary to achieve an optimal condition for pit and fissure sealing. For this reason attached plaque has to be fully removed. There are different methods for cleaning tooth surface:

- rotating brushes with fluoride- and oil free polishing pastes (fluoride may decrease the effectiveness of etching, oils may interfere with the resin based fissure sealing materials),
- rotating brushes without polishing paste,
- air polishing (sodium bicarbonate) / air abrasion (aluminum oxide).

The cleaning of tooth surface using rotating brushes with or without fluoride and oil-free polishing paste is a routine procedure before pit and fissure sealing (Gillerist et al 1998, Donnan & Ball 1988), although it has not been shown to be more beneficial than etching alone (Haris NO &Garcia-Godoy).

b. Isolation

To prevent contamination of tooth surface from the saliva isolating the tooth is necessary either with cotton wool rolls or with rubber dam. There is controversy whether relative isolation with cotton wool rolls is enough for the procedure or absolute isolation using rubber dam is needed. Choosing the isolation type is dependent on the cooperation of the patient, although it is well tolerated, by children if they are well prepared for it. Sealing of permanent molars happens in early stage of eruption when positioning of rubber dam clamp has to be done subgingivally which is painful without anesthesia (topical). (Eidelman et al. 1983). If assistance is not available rubber dam isolation is more preferable to assure isolation.

c. Etching, washing and drying

Etching is the essential for achieving a perfect retention between the sealing material and the tooth surface. The aim of conditioning is to make a micro retentive enamel surface which the hydrophobic sealing resin can perfectly attach to. To have the desired retentive surface 37% orthophosphoric acid containing gel has to be used for 15 seconds. Extending the etching time does not increase the clinical success of resin sealants. (Waggoner & Siegal 1996, Duggal et al. 1997). Acid etching removes approximately 10μm of enamel surface and creates a morphologically porous layer. The surface is enlarged to enable the sealing of tooth surface. Low-viscosity fluid resin contacts the surface and is attracted to the interior of these microporosities. Resin tags are formed in the microporosities of the enamel. Afterwards the acid gel is irrigated with air/water for at least at 30 seconds, and dried for 15 seconds. Waggoner & Siegal earlier stated that exact washing and drying times are not important, as long as all the etching material is being removed, and with drying a chalky white surface is obtained. The increased surface gained by etching is sufficient for the firm adhesion of the fissure sealing material, due to the low viscosity of the material, as it may flow into these microcavities, filling out this porous surface.

d. Application of sealing material, materials of fissure sealing

Requirements for fissure sealing material:

- Flowable,
- Wear resistant,
- Insoluble,
- Non-toxic,
- Adequate working time,
- Rapid cure,
- Good and prolonged adhesion,
- Sufficient strength and dimension stability,
- Same thermal conductivity as tooth,
• Chemically inert.

Sealing materials can be classified into generations according to their polymerization:

• I. generation: UV-light-polymerizing resin-based sealant,
• II. generation: auto-polymerizing resin-based sealants,
• III. generation: light-curing resin-based sealants,
• IV. generation: fluoride releasing light-curing resin-based sealants.

Glass-ionomer cement (GIC) based sealant materials or compomers (polyacid modified composite) may be used for fissure sealing. Flowable composite filling materials as a sealant have also been demonstrated by some authors. In this case the use of extra bonding (adhesive) material is indicated.

The best retention can be achieved by the use of resin-based sealant materials (light curing, auto-curing or fluoride releasing). The glass-ionomer cement and compomer based sealants have the lowest retention rates (Kühnisch et al; 2012).

The advantages of GIC based sealants have been described earlier in the case of partially-emerged teeth, where total isolation and moisture control is not achievable.

Sealants are available as clear, opaque or tinted. There is no difference between the retention, nevertheless opaque sealants have the advantage of more accurate evaluation by the dentist at recall. Some authors argue against the use of opaque sealants, as it precludes continual examination of the sealed fissure (Waggoner and Siegal, 1996, Simonsen, 2002).

The resin based materials may have filler particles in the resin matrix in order to increase physical properties. According to some studies unfilled sealants perform better than the filled ones, due to their better retention others demonstrated that the addition of filler particles to the sealant likewise appears to have little effect on clinical results (Waggoner and Siegal, 1996).

Fluoride-containing sealants have not shown superiority to regular sealant (Simonsen, 2002).

Application of the sealant material:

Resin based sealant materials are usually packed in a syringe delivery system, equipped with an applicator tip. The material should be applied on the cusp slopes, to let the material flow inside the fissures. The air bubbles and excess material have to be removed with either the tip of the probe or a small brush (some products may have a brush on the tip of the applicator). During the application of the material sealing the un-etched area should be avoided, to prevent marginal leakage. When using a light-curing material after the application 20 seconds waiting time is required before polymerization, to allow the material to flow inside the fissures.

e. Polymerization

Depending on the light intensity of the curing light, the time of polymerization is should be in accordance to the factory specifications of the material used.

f. Checking the polymerized sealant

Any excess material should be removed with a sharp scaler, or a tungsten-carbide (finishing diamond, Arkansas bur) finishing bur. The occlusion has to be examined with articulating paper for high spots. If it is necessary, occlusal adjustment is required.

g. Polishing with fluoride containing polishing paste

The significance of polishing after fissure sealing:

• Oxygen inhibits the polymerization on the surface of the resin based materials, leaving an thin layer of non-polymerized layer. This layer should be removed with the polishing.
• The etched surfaces that have not be sealed can be remineralized with fluoride.

h. Follow-up

A regular control of the retention after fissure sealing is necessary. The regularity of checkups depends on the caries risk of the patient (for children at least twice per year, for adults 1 once a year is recommended). Deficits and fractures need to be repaired. Controls and optimal oral hygiene prevents the development of caries (Chestnutt et al; 1994, Wendt et al; 2001a, 2001b, Lavonius et al; 2002).

The first checkup is recommended 6 months after treatment. All others should be planned like usual recalls, according to the caries risk of the patient.

49.5. Minimal invasive /extended pit and fissure sealing

Extended fissure sealing includes the treatment of small caries defects and is defined as a secondary preventive measure. It is the method or preventive resin restoration (PRR). Basically three types of PRR have been introduced.

The three types of PRRs which are performed are depending upon the carious lesion.

• Type A (Figure 5):
  • Caries of the pits and fissure is limited to the enamel.
  • The preparation size is very small.
  • Unfilled resin or sealant is used to restore the preparations of carious lesions and the rest of the fissure.

• Type B (Figure 6):
  • Small and confined lesion extending into the dentin.
  • Since the caries can be explored the preparation needs to be extended.
  • The preparation area is filled with composite resin material, with or without underlining and then the whole occlusal surface is sealed with filled resin sealant.

• Type C (Figure 7):
  • Deep caries into the dentin in a confined area of the fissures.
  • The caries is very definite and requires considerable preparation.
  • The preparation is filled with composite resin material, with underlining and then the whole occlusal surface is sealed with filled resin sealant.

In the case of type A and B hard metal (tungsten-carbide), conical fissurotomy burs are recommended with 6-8 cutting edges to preserve healthy tooth structures (Figure 8.). the length of the bur is 1.5-2.5mm, limiting the preparation to just below the DEJ (conservation). For the removal of the infected dentin slow stainless steel burs are used. Many types of fissurotomy burs are available It only cuts mostly enamel, patient discomfort is minimized and local anesthetics is unnecessary.

Figure 1.670. Figure 5. – Type A PRR
Figure 1.671. Figure 6. – Type B PRR

Figure 1.672. Figure 7. – Type C PRR

Figure 1.673. Figure 8. – Conventional cylindrical burs (left) compared with fissurotomy burs (right). Note the amount of extra healthy enamel removed by the conventional bur
49.6. Possible side effects

a. Local effects

Most side effects occur during etching/conditioning. For this reason neighboring soft tissues should be well isolated during application, and washing of the phosphoric acid. To prevent acid contact of mucosa and other tissues high volume evacuator is recommended for the washing procedure. Theoretically pulp irritation may occur due to etching with phosphoric acid.

b. Systemic effects

Literature mentions only few cases of systemic allergic reactions. (Hallström 1993, Ohlson et al. 2001)

- Contact allergy due to HEMA or TEGDMA (Kanerva et al. 1995, Ortengren 2000, Wrangsjö et al. 2001).
- Contact allergy to resin-modified or light-curing glass-ionomer cement (Laine et al. 1992, Kanerva & Lauerma 1998).

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50.1.50. Effect of feeding pattern to the oral diseases – Balazs Sandor

The science of dietetics slowly reaches the well deserved place among western medical sciences and gains more and more attention as many chronic diseases have been proven to be in association with inappropriate nutrition/diet.

Basically we can divide the oral effect of diet into two groups: the preresorptive phase (before absorption of food) and the postresorptive phase (the effect of absorbed nutrients). Both can seriously influence the oral and dental health conditions by affecting different attack points.

50.1. Basic terms

50.1.1. The transport of food
The time of food spent in the mouth is called the preresorptive (before absorption) state/phase. During this period local effects of food can influence the health state of the oral cavity.

Food in the mouth is well mixed with saliva during chewing. The main function of saliva is to keep the oral cavity humified, to moisten food thereby facilitating gustation and to form bolus being swallowed, to protect against infections, chemical substances and to neutralizes exogenous acids. During dehydration the amount of saliva decreases inducing thirst and fluid intake contributing to the maintenance of fluid homeostasis. Saliva is produced by main and minor salivary glands. The secretions by certain salivary glands is continuous but not of big amount (minor salivary glands, submandibular glands, parotid glands) while others (sublingual glands, submandibular glands) produce and release saliva only upon some kind of stimuli. Mechanical and aggressive chemical stimuli contribute to the production of watery saliva to dilute the critical substances. During food intake the saliva produced is rich in organic substances and enzymes involved in the chemical break-down of food. Saliva with digestive functions is secreted upon stimulation of gustatory receptors; however the entire system is controlled and synchronized by the central nervous system (medulla oblongata).

Human saliva mainly consists of water (99.27%), characterized by a pH ranging between 6.5-7.5 and its amount depends on various factors like hydration state, stress, medication, hormonal changes, chronic diseases. The average daily amount produced, differs between 700 to 1000 ml. The dry matter content is only 0.73%, and mainly consists of inorganic solutes/electrolytes and of trace elements (Cl, PO₄, Na, K, Ca, HCO₃, SCN, CO₂, O₂). Radicals of low half lives are partly produced during detoxifying processes while other part is a consequence of incorrect nutrition and food preparation (microwave oven). The presence of oxidative agents are at all events are harmful.

Most important organic components of saliva are mucin, α-amylase and maltase. Mucin is a glycoprotein that is able to neutralize both acids and alkali that means to maintain the normal H⁺-concentration. Enzymes play a crucial role in the initial phase of carbohydrate digestion as they break down starch to oligosaccharides. This means that digestion already starts with the saliva with the contribution of salivary enzymes producing an oral environment rich in low molecular weight, easily fermentable sugars. Normally the protective function of saliva counteracts on these harmful effects but human nutrition almost continuously acts against this delicate balance, thereby leading to oral diseases in the lack of adequate oral hygiene.

Some organic components of the saliva (lysozyme, lactoferrin, lactoperoxidase, cystatines, hystatins, secretory Ig-A, proline rich proteins) have antimicrobial role.

**50.1.2. BMI (body mass index)**

BMI is a measure for human body shape that is easily calculated and is a good index to rely on, in terms of physiological or medical nutrient questions.

Calculation: \( \text{BMI} = \frac{\text{body weight (kg)}}{\text{height}^2 \text{(m²)}} \), standards may vary according to age and gender.

The lowest statistical risk regarding chronic illnesses occurs if BMI is between 19 and 25 kg/m².

**50.1.3. Food guide pyramid, nutritional components**

Figure 1.674. Figure 1. – Food guide pyramid
This is well known figure, easy to understand and provides real information regarding the composition of a healthy diet. The figure refers to beneficial postresorptive effects, but local oral effects differ from the systemic ones, which are shown by the diagram.

Food intake provides substrates for bacteria found in the dental plaque, so it is not irrelevant what kind and how much food we consume, neither is the frequency and way of food intake. The physical characteristics of aliments (temperature, consistency, fiber content, stickiness) are also important, as for example foods that are more solid and increased mastication is needed, can clean the surface of teeth very well. In contrast sticky and tender foods attach easily to the surface of teeth and are more difficult to remove.

From all foods, easily fermentable carbohydrates (saccharides) are the most dangerous regarding the safety of teeth and enamel, as they are inevitable for the demineralization of dental hard tissue. The most harmful saccharides are the ones, from which acids are produced through bacterial fermentation, particularly monosaccharides (glucose, fructose, galactose, mannose) and disaccharides (sucrose, maltose, lactose).

50.2. Typical effects of food components

50.2.1. Proteins

Proteins are involved mainly in the period of tooth development, and play an important role in the structural development of dental and periodontal tissues; thereby they exert their effect mainly in the postresorptive phase and preeruptive stage. Malnutrition often causes dental developmental disorders, but the contrary is also true, as painful oral diseases can easily lead to eating disorders. This means that oral diseases may indirectly result in growth failure or abnormal weight loss.

The pH of proteins or the mixture of their structural elements, amino acids is approximately neutral (6-8), and it never falls to the critical point where demineralization of enamel is possible. Due to this phenomenon they do not expert harmful local effect regarding enamel.

Another issue is the consistency foods containing protein, as some of them are rich in fibers (meat, fish) while others are especially sticky (dairy products). According to these characteristics of the foods mentioned, they may facilitate plaque formation and can act as bacterial culture medium.

**Figure 1.675.** Figure 2. – Aliments with high protein content and rich in fibers
50.2.2. Carbohydrates

Carbohydrates are found in two forms in our food: one is starch, the other is sugar. Starch is a polysaccharide with low cariogenic effect. Due to its large molecular weight it cannot be metabolized bacteria. Saliva contains α-amylase, so the digestion of starch already starts in the oral cavity increasing the local sugar-concentration. Beside this the sticky consistency of starch is rather disadvantageous as it has a slower clearance in hidden areas of the oral cavity, leading to a prolonged sugar exposure in the mouth. Starch can be found in bread, rice, potato, flour and sweets. Nowadays bread is the aliment consumed to the greatest extent all over the world. In earlier times before sophisticated cooking techniques and among primitive tribes bread had a more erosive effect. However today bread is prepared from refined flour and consumed freshly hence it sticks more firmly to tooth surface. Bread without yeast could be preserved for more weeks and was eaten quite hard which caused the attrition of cusps and fissures on the occlusal surface, other than caries.

The general, postresorptive effect of sugars (monosaccharides, disaccharides) is the elevation of blood glucose level (to a higher extent and more rapidly than polysaccharides). According to some studies sugar-consumption alone does not increase the risk of diabetes mellitus, but may increase the risk of obesity, which is a great risk for diabetes. However other studies suggest that insulin resistance, β-cell dysfunction and inflammation caused by frequent and massive sugar ingestion lead to diabetes mellitus. Increased sugar intake also elevates the chance of developing cardiovascular diseases.

Of all sugars (mono- and disaccharides) sucrose exerts the highest cariogenic effect, but also fructose, maltose and glucose are significantly cariogenic, however galactose and lactose are of lower importance regarding this aspect.

Common characteristic of sugars is that they are the main nourishment for bacteria. Dental plaque microorganisms break down sugars into acids during fermentation thereby lowering the pH. All sugars containing molecules with six carbon atoms (lactose, sucrose, fructose) can be converted into various acids by plaque bacteria.

Factors influencing the cariogenic effect of acids:

1. the amount of consumed sugar,
2. consistency of food,
3. frequency of food intake,
4. time spent in the oral cavity: slowly dissolving lozenges and sweet beverages drunk sip by sip spend a long time in the mouth. Thereby teeth are exposed to the harmful effect of acids produced by fermenting bacteria for a much longer period.

With measuring the pH of the plaque we can draw conclusions about the listed factors. The Stephan’s curve shows the individual daily eating pattern based on pH measurements of the dental plaque. Crucial points of the
curve are where the pH drops below the critical level. If the duration of the pH level under this critical point increases there is a higher risk for caries development.

**Figure 1.676. Figure 3. – Stephan's curve.** The critical point is pH 5.5, where demineralization processes of the enamel begin. Chewing gum decreases the time under the critical pH level. Snacking increases the time under the critical pH level.

![Stephan's curve]

The presence of sugars in aliments: Nearly all aliments contain some amounts of sugar and starch. The cariogenicity of foods does not only depend on the sugar-content but also on the acidogenicity. This means that natural acid component of foods further deteriorate acid-base imbalance of the oral cavity. For example the fiber content of an apple, considered to be „tooth-friendly”, can clean the tooth surface very well however its fructose and fruit acid content is intensively cariogenic. Other fruits can be also cariogenic if they decrease the pH of the oral cavity. For instance banana is more cariogenic than chocolate or sucrose.

### 50.2.3. Fats and oils

As their pH is neutral they are not considered to be cariogenic. In the evolution of tooth decay fats and oils rather play a protective (prophylactic) role as they negatively influence the solubility of glucose, thereby preventing bacterial metabolism. Fat can coat the surface of carbohydrate containing food-granules so the cariogenic effect dominates less. According to some studies lipids are able to form a protective monolayer on the surface of acquired pellicle serving as a defensive structure against acidic attacks.

Further important role of fats and oils is that they facilitate the transport and absorption of lipid soluble vitamin (vitamin A, D, K, E) in the gastrointestinal system. Essential vitamins are indispensable for the healthy functioning of our organism that is why their intake should be well considered and ensured, even during fat free diet. All these effects of fats and oils belong to preresorptive processes.

Despite the sugar content some aliments with high fat and oil content are less cariogenic (e.g. cocoa powder, oily seeds).

**Figure 1.677. Figure 4. – Low cariogenic food, chocolate**
50.2.4. Vitamins, minerals and trace elements, water

Vitamins play an important role in the preeruptive phase, as they influence tooth development and mineralization of tooth hard tissues. After eruption of teeth (posteruptive phase) they do not have local effect. The lack of certain vitamins leads to severe deficiency diseases also manifesting in the oral cavity (e.g. vitamin C deficiency or scurvy). In the embryonic and fetal life, between the 4th and 16th gestational week, deficiency of calcium, vitamin D and vitamin A can easily result in oral malformations. Some data show that the deficiency of vitamin B6 and vitamin B12 can increase the risk of clefting (cheiloschisis or palatoschisis).

The fat soluble vitamin A effects the development of ectodermal tissues and also that of tooth buds. In case of vitamin A deficiency eruption is delayed and the differentiation and function of odontoblasts is impaired.

Water soluble vitamin B plays a role in cell-metabolism. If vitamin B1 deficiency is present the detrimental action of carbohydrates on teeth is even worse.

Vitamin C is water soluble and has a leading role in the formation of collagen and mucopolysaccharides, and the lack of it leads to inhibition of dentin-formation by ceasing the predentin formation.

Vitamin D is fat soluble and enhances the intestinal absorption of calcium and phosphate and ensures their optimal balance. The deficiency of the vitamin has negative influence on the mineralization of hard tissues, within the development of bones and enamel.

Minerals take part in the maintenance of the electrolyte-homeostasis and in metabolism of different tissues. Regarding dental hard tissue both calcium and phosphate play an extremely important role. Of course other minerals and trace elements are not to be missed for optimal structure and function, but in the aspect of mineralization calcium and phosphate are the most important ions. Their local role of the preresorptive phase is as essential as the systemic, postresorptive effect.

In the oral cavity ongoing de- and remineralization processes of the enamel are complicated chemical and physical reactions influenced by many various factors. It is important to emphasize the role of fluoride favorably influencing the hardness of dental substances and protecting against acidic exposures.

Víz
Water

Fluid intake and utilization is a key element of the organisms homeostasis. Water and crystalloid solutions play determinant roles in biochemical and biological processes. This also applies to the oral cavity where the amount of saliva is strongly influenced by our hydration status. In some stages of dental development (eruptive phase) hypersalivation is a normal reaction. Hyposalivation or sometimes xerostomy (dry mouth) is a much bigger problem leading to many chronic dental diseases like caries, sialolithiasis or it may be present as part of Sjögren syndrome.

According to these facts the calculated daily fluid requirement is very important information for patients and we have to highlight this during all kind of therapies. Further important note is that the requirement refers to still water and not to sparkling or sweet beverages. Osmotic and other effects of the latter seriously deteriorate absorption and the acidity of the oral cavity.

Table 1.4. Table 1. – Daily fluid requirement of children

<table>
<thead>
<tr>
<th>Weight Range</th>
<th>Requirement</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-10 kg</td>
<td>100 ml/bwkg/day</td>
</tr>
<tr>
<td>11-20 kg</td>
<td>1000 ml, plus 50 ml/bwkg/day</td>
</tr>
<tr>
<td>21 kg-tól</td>
<td>1500 ml, plus 20 ml/bwkg/day</td>
</tr>
</tbody>
</table>

50.2.5. Physical characteristics of foods

Temperature: excessively hot or cold foods and drinks, especially the rapid change between these can cause microscopical ruptures on the enamel and these anomalies further propagate erosion and the development of caries.

Sticky consistency: food debris may stay in the mouth for too long providing optimal conditions for bacterial growth leading to periodontal diseases and caries.

Soft food diet: sometimes it makes part of our therapy, but applying it for a longer period the self-cleansing processes of teeth by chewing are hindered and this again can lead to caries and periodontal problems.

Monotonous diet: it is characteristic of some diets, but these are contraindicated as they can cause deficiency of certain nutrients and absorption difficulties.

50.2.6. Utilization of sugar substitutes

Indication of usage: slimming diet, dental prevention, diabetes mellitus, relative cheapness

Sugar substitutes do not raise blood glucose level (except lycasin), and the overdose usually causes diarrhoea due to their massive osmotic effect. The sweetening effect of sugar substitutes is always compared to the the sweetening effect of sucrose which is considered to be 1.

Advantages in dental prevention:

• Acid-production during fermentation: formic is produced and very slowly.
• They do not have a role in the formation of bacterial extracellular matrix.
• Plaque adhesiveness changes: lower by xylitol consumption, higher by sorbitol because it is hygroscopic.
• Adaptation of microorganisms: there is no adaptation to xylitol, but there is to sorbitol.

Types:

1. Sugar substitutes (sugar alcohols, polyols): they possess a lower sweetening effect and have slightly lower energy content.

1. MANNITOL: E 421

• sweetening effect: 0.7
1. Oral biology

- It was first isolated from flowering ash (Fraxinus Ornus).
- Maximal daily dose 160mg/bwkg
- Symptoms of overdose: flatulence, diarrhoea

Use:
- In intensive therapy it is used to decrease intracranial pressure and it facilitates the distribution of medication in the brain
- Sweetening of diabetic foods
- Chewing gums
- Coating of candies (not hygroscopic)

2. Sorbitol: E 420

- Sweetening effect: 0.5
- Produced through fermentation of starch from corn, wheat or potato
- Delays rancidity
- Symptom of overdose: diarrhoea

Use:
- Used for preparation of diabetic foods (also for baking)
- Chewing gums
- Tooth pastes, mouthwashes
- Used as humectants in cosmetics

3. ISOMALT: E 953

- Sweetening effect: around 0.5
- Glucose and mannitol
- Symptoms of overdose: flatulence, diarrhea

Use: low-calorie and sugar free desserts, ice cream, sweets, chewing gums, sauces, mustard, dietary supplements

4. XYLITOL: E 967

- Sweetening effect: 1
- discovered by German and French chemists in the 19th century, the name xylitol comes from the Greek „xylo” = wood.
- Also available in crystalline form.
- As intermediate of carbohydrate metabolism it is a natural component of living organisms.
- Found in fruits: yellow greengage.
- Main use: chewing gums.
- Symptoms of overdose: flatulence, diarrhoea.
1. Oral biology

- Caries prevention: remineralizes the incipient caries, capable of binding to Ca\(^2+\).
- An „anti plaque” effect has been demonstrated.
- It is not suitable for preparing yeast containing foods.
- Daily use of 10,5 g of xylitol for 4 weeks decreases the germ-count of Streptococcus mutans by 17-20%.

2. Sweeteners

- They represent an intensive sweetening effect (30-300x compared to sucrose).
- Sweeteners energy free.
- There are two forms of sweeteners, natural or artificial derived sweeteners.
- Plaque bacteria can not ferment it.
- There is no effect on blood glucose level, or they rather lower it with other consequences.
- Synergistic effect: if used in combination the sweetening effect is bigger for example: aspartame + acesulfame 350x.
- Used combination: with each other and with sugar substitutes.
- Long term effect: is not yet known, till now no significant side effect has been described.

1. Acesulfame-K (C,H,KNO,S)

- Acceptable daily intake (ADI): 9 mg/bwkg.
- White crystal; first produced in 1967.
- One of the latest synthesized sweeteners.
- Has an intensive sweet taste, it is thermostable so it is appropriate for cooking and baking.
- Energy content is zero, it is not metabolized by the body It passes through unchanged.
- Synthesis: with a chemical reaction as a derivative of amidosulfonic acid.
- It has a synergistic effect with aspartame and other sweeteners.
- In sugar containing chewing gums it has a flavor enhancing effect.
- Safe to use, can be stored for a long period.
- Patients with diabetes mellitus can also consume it.
- It is used in tooth pastes, mouthwashes and foods.

2. Aspartam

- ADI 40mg/bwkg.
- sweetening effect: 200x, energy content is near zero.
- Phenylketonuria (PKU) is a metabolic disease where the use of aspartame is contraindicated, and those aliments with aspartame as sweetener have to be marked as phenylalanine sources.
- European Food Safety Authority (EFSA) considers it as a safe substance (EFSA-Q-2005-122).
- In Japan though it is not in use as sweetener.
3. Saccharin
- ADI: 2.5mg/bwkg.
- Artificial sweetener.
- Calorie free.
- Has a bitter, metallic aftertaste.
- Used to sweeten beverages, candies, cakes and tooth pastes.
- 10:1 mixture of cyclamate and saccharin is widely used in many countries.
- In certain studies (animal experiments with extremely high doses) it was proven to cause bladder cancer. Saccharin is excreted without any change within 24 hours with urine.

4. Cyclamate
- ADI: 7 mg/tkg;
- ADI: 7 mg/bwkg.
- Artificial sweetener.
- Sweetening effect: 30-50X.
- Compared to acesulfame it has a milder aftertaste.
- Usually distributed in combination with saccharin.
- Incidental side effects: animal studies proved bladder cancer (cyclohexamine), and fertility disturbing effect is evoking (data of animal experiments), has a salty aftertaste.
- Calorie free, good for slimming diets.
- Thermostable: appropriate for cooking and baking.
- Can be stored for a long period.
- Diabetic mellitus patients can consume it.

5. Sucralose
- ADI: 15 mg/bwkg.
- Diabetic mellitus patients can consume it.
- Calorie free, derived from sucrose.
- Not digested.
- No cariogenic effect.
- Good solubility, thermostable.
- Component of low calorie and or sugar free beverages, desserts, alcoholic drinks, jams, canned foods, dietary supplements.
- It is safe.

50.2.7. Snacking diet

Beside the composition of our foods we also have to control the frequency of our meals. A regular age specific diet is recommended including drinking habits. We cannot allow eating or drinking sugar containing sweets and
snacks between main meals. In case of snacking diet saliva cannot carry out its protecting buffering affect due to frequent acidic exposures.

50.2.8. Breastfeeding

Breastfeeding is undoubtedly ideal for the child until 18 months of age.

- Suggested at least until the age of 6 months (exclusively).
- Physiologic feeding type.
- Supplies the baby with maternal immunoglobulin.
- Hygienic.
- Ideal nutrient composition.
- Strong psychological relationship is formed between the mother and the baby.
- Muscle movements during breastfeeding are ideal for jaw and tooth development.

The micro flora of the oral cavity changes after the eruption of the first primary teeth. Pathogenic bacteria occur in the mouth, which may lead to the formation of caries. The feeding plan should be changed, breastfeeding before sleep, should be avoided, as milk contains sugar (lactose). Adequate cleaning of the oral cavity must be carried out according to the age of the child. Prolonged non-nutritional breastfeeding (over 1 year) may have severe cariogenic effects on the erupted teeth. In the case of socially underprivileged children this type of feeding may be the only source of proteins, so dental prevention is overshadowed.

50.2.9. Bad habits: smoking

In summary smoking, drinking alcohol and bad oral hygiene one by one lead to severe oral diseases, but together they exacerbate effect to each other. Thanks to the regulations of the european countries the rate of young smokers is decreasing. In Asia and the developing countries the number is still rising.

The primary place of smoke entry into the body is the oral cavity. The concentration of the smoke is the highest here. During smoking a high temperature, irritative smoke containing toxic substances, enters the oral cavity. It exsiccates and causes inflammation to the tissues that are in contact with the smoke. Due to the exsiccating effect the defensive function of the mucosa decreases, thereby facilitating the penetration of harmful substances. Smoking causes atherosclerosis with subsequent hypoxia, and this phenomenon also appears in the oral cavity. Consequences: gingivitis, periodontitis, and consequential early tooth loss. This disease appears in more severe forms and with impaired healing tendency in smoking patients.

It is a proven fact that in patients with periodontal diseases due to smoking the risk of myocardial infarct and of stroke increases. Studies and experiments have observed that the development of severe cardiovascular conditions is in association with small inflammations of the vascular wall evoked by oral bacteria invading the circulatory system. Also the risk of premature birth and of small for gestational age babies is higher.

Scaling in smokers is less effective and needs to be repeated more frequently. It might be a contraindication of oral implants because it reduces their success rate (to approximately 70%), side effects and complications are more often occurring. After oral surgical interventions such as tooth extractions healing processes are prolonged and complications are more frequent. In periodontal disease which is frequently caused by smoking, cleaning of teeth is more complicated and less effective thereby elevating the risk of caries.

Smoke discolors dental deposits (acquired pellicle, calculus) and its color may range from light brownish to black. Next to the esthetic disadvantage a characteristic breath odor (halitosis) is present. The discolored dental deposits are removed by professional polishing performed by a dentist or dental hygienist but they discolor rapidly again due to smoke. Passive smoking impairs defense of enamel and nicotine reduces the rate of fluoride incorporation into teeth.

It is extremely harmful to children whose parents are both smoking in their homes. Most severe effect of smoking is on the development of cancerous lesions in the oral cavity.
50.2.10. Erosion

Erosion due to eating occurs through the whole life and means the loss of dental hard substance. In late ages the total loss of enamel or even dentin loss may be present. The cause is complex as acidic effects temporarily weaken the enamel and as a result of following mechanical irritation enamel detaches from the tooth surface. In children consuming too much of sparkling beverages this phenomenon appears at a characteristic localization in early age. The prevalence of this condition can be reduced with preventive methods, however conscious eating habits and the knowledge and performance of appropriate oral hygiene is needed to prevent the development of this phenomenon.

50.2.11. Osteoporosis

Osteoporosis may lead to tooth loss. Teeth are attached to the alveolar bone, thus the lack of calcium, vitamin D and K may lead to tooth loss.

50.2.12. Diet for patients wearing dentures

Patient wearing dentures may have a varied diet. In the case of patients wearing partial dentures the cleaning of the remaining teeth is not negligible, to prevent caries and periodontal disease. In patients with total removable
dentures the main prevention of mucosal lesions is the main objective. Even with a precisely designed denture, the masticatory forces are reduced compared to natural teeth. Further problems may occur if the denture does not fit. Because of these difficulties these patient may prefer eating soft food, which do not require chewing. These foods are usually low nutrients, or even may be deleterious (excessive sugar content, sticky food and drinks). This may lead to deficiency diseases. The direct negative effects of denture wearing on general health have not been confirmed yet, presumably indirect effects apply.

50.2.13. Effects of physical and mental retardation, severe systemic diseases, patients requiring continuous care, effects of sugar containing medicines

These patients are considered to be in the high risk caries group. Due to the inadequate oral hygiene they are more prone to caries and periodontal disease. During the care of these patients we have to pay more attention to planning a caries protective diet (reduce plaque accumulation). Regular control and professional cleaning is necessary.

51. 1.51. Compex prevention concerning age, dental specialities and general diseases – Balazs Sandor

Most of the lesion in the oral cavity can be prevented. Regardless of the specialization, every dentist is responsible for educating and motivating their patients. Creating the right dentist-patient relationship, and the dentist’s personality is important. The main methods and goals of prevention can be determined, but the experience can only be acquired in practice.

51.1. Health education

- The complex of learning, teaching activities,
- maintaining health through health-care advice,
- development of positive behavioral forms and habits,
- is an organic part of prevention programs, but it cannot substitute preventive care service.

Selection of a suitable method and the appropriate application of the chosen method can be critical. Criteria of selection:

- target population – particularities of the age group, taking the level of awareness into consideration,
- educational objectives,
- given objective circumstances.

51.1.1. Methodological Principles of Health Education

Effective educational sessions should be precisely planned with the comprehensible and valuable knowledge by means of simple, clearly constructed sentences, avoiding the use of abstract expressions and foreign terminology

- "absorbable" amount of information,
- entertaining, playful demonstration,
- pursuit of active participation,
- evaluation of performance, response, highlighting success, correction of mistakes,
- conscious application of the principle of motivation – the most effective motivating factor is the presentation of results, for example oral hygiene, brushing teeth after the use of plaque disclosing agents,
- avoidance of arousing remorse, humiliation,
• avoidance of prohibition: seeking compromise may be of help: e.g. sweets are allowed, if we brush our teeth after consuming them,

• giving personal examples should be applied, where possible,

• intellectual and sensual influence,

• in the case of children motivation from the environment is also important (parent, kindergarten teacher, school teacher, etc.),

• practice, repetition, obtaining an adequate level of skills,

• two-way (interactive) communication, not merely the transfer of information (one-way communication).

51.1.2. Health Educational Methods

• Individual communication,

• group exchange of information and ideas, discussions,

• team organizations,

• spreading knowledge through mass communication channels.

51.1.3. Practical Opportunities for Health Education

• Active and passive,

• instruction applicable at the surgery (chair-side) and outside the surgery,

• the effect of "written word" and "spoken word",

• verbal and non-verbal (mimicry, look, eye-contact, gestures, vocal mimicry, etc.) methods.

51.1.4. Places of Health Education

• Children's dental surgery,

• waiting room,

• treatment premises,

• prevention room,

• health-care institutions,

• educational institutions.

51.2. Childhood

Lasts from 0-18 years of age and can be characterized by continuous change. This is the age when the quantitative and qualitative development of most organs takes place, which forms into a dynamic balance upon reaching adult age. Consequently, we cannot speak of uniform childhood guidelines, only prevention suitable for the actual age of the patient is conceivable. Based on the age related properties of dental diseases, various themes are relevant in each and every years age, as it would be in vain to speak about tumor prevention in the case of a new-born child, while caries prevention is superfluous in the case of patients with dentures.

All three levels of prevention (primary, secondary, tertiary) are included in treatment; in the present chapter we will mostly deal with primary prevention, in particular its most typical age related form. In case of child patients this includes the prevention of caries, periodontal disease, traumas and bad habit.
In Table 1, below, the 0-18 age group is divided into typical groups, listing the types of childhood preventive action. For didactic reasons dental prevention during pregnancy is also included here, as this specific period of life also has relevance to pediatric dentistry.

Table 1. Table 1. – Preventive action according to age groups

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Target Group</th>
<th>Instructor</th>
<th>What to Teach</th>
<th>Method</th>
<th>Location</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>preg.</td>
<td>mother, primary- primary prevention</td>
<td>dentist, dental hygienist, nurse, gynecologist, pediatrician</td>
<td>mother's teeth, dental problems during pregnancy, tooth development, fluoride dosage, breast feeding</td>
<td>for mother, personally or groups, pregnant club</td>
<td>regular controlling of pregnant (personally or groups)</td>
<td>obligatory, part of regular controlling</td>
</tr>
<tr>
<td>0-2 years</td>
<td>mother</td>
<td>dentist, dental hygienist, nurse, pediatrician</td>
<td>tooth development, fluoride dosage, baby oral hygiene, feeding, diet and drinks of this age</td>
<td>for mother, personally or groups, newborn club</td>
<td>baby advices</td>
<td>not obligatory</td>
</tr>
<tr>
<td>3-5 years</td>
<td>child, parents, teacher</td>
<td>dentist, dental hygienist, nurse, teacher, pediatrician</td>
<td>oral hygiene, eruption order, dietary advices, bad habits</td>
<td>in creative groups, play, toys, fairy tales</td>
<td>kindergarten, preschool, preventive rooms</td>
<td>obligatory, organized by the school</td>
</tr>
<tr>
<td>6-10 years</td>
<td>child, parents, teacher</td>
<td>dentist, dental hygienist, nurse, teacher, pediatrician</td>
<td>permanent dentition, oral hygiene, dietary advices, injuries, orthodontic problems</td>
<td>school, dental surgery, parents meeting</td>
<td>obligatory, organized by the school</td>
<td></td>
</tr>
<tr>
<td>11-14 years</td>
<td>child, teacher, parents, individually or groups</td>
<td>dentist, dental hygienist, nurse, teacher, pediatrician</td>
<td>oral hygiene, dietary advices, caries development, orthodontic problems, bad habits, smoking, drugs</td>
<td>school, dental surgery, parents meeting</td>
<td>obligatory, organized by the school</td>
<td></td>
</tr>
<tr>
<td>14-18 years</td>
<td>child, individually</td>
<td>dentist, dental hygienist, nurse, teacher, pediatrician</td>
<td>oral hygiene, dietary advices, caries development, bad habits, smoking, drugs, esthetic questions</td>
<td>school, dental surgery, parents meeting</td>
<td>obligatory, organized by the school</td>
<td></td>
</tr>
</tbody>
</table>

51.2.1. Dental Prevention Required During Pregnancy

Table 1. contains information necessary for the performance of preventive action. Ante-natal tasks are described in detail, including possible maternal dentition problems during pregnancy, and the question of dental development, nutrition, fluoride input and oral hygiene relevant to the unborn infant. Pregnancy is a highly responsive period in the lives of women, who call upon all available information with maximal commitment. Nowadays the opportunities for orientation provided by Internet allow patients'access to any information (perhaps even uncontrolled). This at times results in difficulties, as those not trained in the field of dentistry misinterpret, or are unable to interpret certain pieces of data.

51.2.1.1. Knowledge Relevant to Maternal Dentition

- Regular, appropriate dental care,
- proper nutrition,
- pregnancy gingivitis.
Proper nutrition during pregnancy principally means a balanced diet, which is moderate but rich in vitamins and nutrients. In fact nutritional extremities are what bring about potentially negative consequences, potentially undermining the mother's health condition, influencing the outcome of deliverance. In the case of excessive weight increase, there is a higher incidence of developing gestational diabetes. Inappropriate diet and the consequent diseases may increase the susceptibility of the teeth to caries.

A novel phenomena, referred to as pregorexia derived from the term anorexia adjusted to pregnancy, a nutritional disorder defined by the maintenance of a strict diet on behalf of the mother, and in addition to the desired sweets, she also refrains from the essentially necessary amounts of protein, carbohydrates and fat, in order to stay slim during and after her pregnancy.

**The Effects of Maternal Hormonal Changes in the Oral Cavity:**

Physiological hormonal changes, closely related to oral health. The increase in estrogen and progesterone levels also has an oral relevance, supporting the correlation of oral and systematic processes. Such hormonal changes cause a clearly outlined collective of symptoms: known as pregnancy gingivitis. Research has shown change in severity corresponding hormonal changes. Characteristic of pregnancy related gingivitis:

- prevalence of pregnancy related gingivitis ranges between 50 to 100%,
- chronic gingivitis present before pregnancy without complaints worsens,
- capillary proliferation,
- due to the effects of progesterone gums are more swollen, bleeding tendency increases,
- increase of probing depth,
- increased sulcular (crevicular) fluid secretion,
- gums are red, livid, its contour may change,
- enlargement of gums may develop,
- reversible.

The phenomena occurs in two phases: it may first aggravate at the end of the first trimester due to the high gonadotropin level, then at the end of the third trimester caused by high levels of estrogen/progesterone. Increased hormone levels subserve the proliferation of anaerobe bacteria: Prevotella intermedia, Prevotella nigrescens, Campylobacter rectus. Establishment and maintenance of oral care, tooth-brushing and an adequate oral hygiene can prevent pregnancy gingivitis, or can deter its exacerbation.

**Maternal or Pyogenic Granuloma:** (maternal tumor, granuloma gravidarum) the accumulation of the capillary network typical of maternal gingivitis is of such degree, that a characteristic local tissue proliferation develops, which may cause difficulties in the cleaning of teeth near-by. Imperfection or the entire lack of tooth-brushing can further deteriorate the patient's condition. Prevalence of the disease is 2-5%. The granuloma requires conservative treatment during pregnancy. If it does not regress after deliverance and nursing, or it reaches extreme dimensions in the course of pregnancy, surgical therapy is required. (Figure 1.)

**Figure 1.680. Figure 1. – Pyogenic granuloma**

There is a two-way relationships between systematic and oral health, the oral flora, the presence or absence of pathogens can influence the whole body. Studies have shown positive correlation between periodontal diseases of pregnant women and premature labor and low birth-weight. The treatment of periodontal diseases during
pregnancy is safe and effective, complications can be eliminated. The intensive therapy of premature new-borns and maternal oral pathogen infections in the fetal period represent a higher risk. Through immunology and serology examinations IgM immune-bodies produced against periodontal-pathogen bacteria can be shown from the umbilical cord blood. Maternal IgM does not pass through the placenta. Periodontal pathogens thus disseminate, enter the fetus, where they trigger the production of immune-bodies. In the course of the preventive program the attention of expectant women must also be directed to such correlations of oral hygiene and pregnancy, the outcome of deliverance, naturally by means of individually tailored guidance. Even mothers, who have no concern about their own oral health, oral hygiene, are willing to take action for the sake of the infant's health.

51.2.1.2. Knowledge Relevant to the Oral Health of the Infant

• Nourishment of new-born and infant, method of feeding.

• Normal primary tooth eruption sequence, possible pathological anomalies.

• Oral care.

• Fluoride therapy.

51.2.2. Dental Prevention in Early Childhood (1-3 years)

With reference to the table mentioned in the beginning of the chapter it can be observed that in the case of children with deciduous dentition the primary themes are the prevention of caries, gum- and periodontal diseases, and the avoidance of accidents.

Consultations between the pediatrician, health visitor, nursery governess, pediatric dentist and the parents can assist the flow of information. The age group between 0-3 is relatively difficult to access, while the possibilities of group prophylaxis are also limited. Leading information:

• cariogenic effect of sweetened pacifier, sweet liquids,

• importance of chewing,

• brushing of primary teeth,

• control of nourishment – at home, at the nursery,

• fluoridation with tablets (at nursery: group, at home: indicated by pediatrician, individually),

• pediatric dental check-up once a year, treatment when necessary,

• probable consequences of thumb-sucking.

51.2.3. In the case of kindergarten and elementary school children (3-6 years)

• Control of nourishment: sweets only at the time of main meals (always to be followed by brushing of teeth), access to sweets brought from home should not be allowed.

• Regular toothbrushing, toothbrushing exercises in groups twice a year.

• Guidance of parents, raising awareness of parental responsibility regarding the integrity and protection of teeth.

• Group dental check-up every half a year, treatment when necessary.

1. Teaching and practicing toothbrushing methods, is in accordance with age and developmental stage.

Table 1.6. Table 2. – The thematic of teaching toothbrushing methods and dietary advice to children with primary dentition and early mixed dentition

<table>
<thead>
<tr>
<th>Child group</th>
<th>Kindergarten in Autumn</th>
<th>Kindergarten in Spring</th>
</tr>
</thead>
</table>

Created by XMLmind XSL-FO Converter.
1. Oral biology

<table>
<thead>
<tr>
<th>Middle</th>
<th>Senior</th>
<th>School grade</th>
<th>Autumn</th>
<th>Spring</th>
</tr>
</thead>
<tbody>
<tr>
<td>familiarization, game, tools, glove</td>
<td>dry tooth-brushing practice</td>
<td>dry tooth-brushing practice</td>
<td>tooth-brushing with tooth-paste</td>
<td></td>
</tr>
<tr>
<td>Senior</td>
<td>dry tooth-brushing practice</td>
<td>tooth-brushing with tooth-paste</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. Irregular diet, sweetened drinks, eating, drinking after bed-time brushing

2. Trauma Prevention:

- Traffic accidents (use of 5-point security child-seats and seat-belts, awareness of the rules of traffic, measures to be taken in the case of an accident).
- Accidents while playing (presence of an adult is indispensable at all times).
- Sports related accidents (use of protective equipment: helmet with jaw protection, mouth guard).
- Child abuse (delivery of discreet information, comprehensible to a child).

51.2.4. Dental Prevention at School (age group between 7-14)

In the age group between 7-14, delivery of knowledge is still the most practicable in groups. Themes listed so far are similar in this childhood group as well, certainly by means of methods fitting the age group. These are as follows:

- preventive dental practice in groups twice a year,
- recommendation of tooth-pastes with fluoride content for home use,
- establishing the conditions for tooth-brushing at school,
- application of fluoride-gel once every week with teacher supervision,
- parental guidance, raising awareness of parental responsibilities,
- favorable influencing of dietary customs through the continuous modernization of child alimentation,
- regulation of school canteen supply, banishing sweets from schools,
- group dental examination, treatment where necessary,
- trauma prevention,
- crowding of teeth, orthodontic irregularities.

Additionally, self-destructive behavior (emergence of harmful habits) and bad habits must be mentioned, which endanger oral health and the condition of the whole of the body.

- smoking,
- drinking alcoholics,
- drug abuse,
- obesity or anorexia,
- bad oral habits (chewing, sucking),
- wearing oral piercings.
Table 1.7. Table 3. – Thematic of half-year dental prevention practices for children at school age (mixed dentition, young permanent teeth)

<table>
<thead>
<tr>
<th>School grade</th>
<th>Autumn</th>
<th>Spring</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>cleaning of distal teeth, autonomy, tooth-past amount</td>
<td>tooth related accidents, changing of teeth</td>
</tr>
<tr>
<td>4</td>
<td>fluoride, Elmex gel, varnish</td>
<td>fluoridation in group using Elmex gel</td>
</tr>
<tr>
<td>5</td>
<td>brushing, electric tooth-brush</td>
<td>tooth-brushing test</td>
</tr>
<tr>
<td>6</td>
<td>importance of orthodontics, gum-shield, accident prevention</td>
<td>fluoridation in group using Elmex gel with a deep vacuum splint</td>
</tr>
<tr>
<td>7</td>
<td>types of tooth-brushes, tongue cleaner</td>
<td>use of chewing gum</td>
</tr>
<tr>
<td>8</td>
<td>dental floss, cleaning of the interdental space</td>
<td>course of bed-time brushing, mouthrinses, rinsing</td>
</tr>
</tbody>
</table>

51.2.5. Dental Prevention of Secondary School Children

The age group between the age of 14-18 years fundamentally differs from the younger age groups; group-sessions are no longer effective. From approximately 16 years of age the posing and discussion of problems individually is the efficient tool. With approach of adulthood, esthetics and hygiene becomes more and more important to this group, and with this in the background, serious issues can be discussed with them (e.g. tumor prevention).

Table 1.8. Table 4. – Thematic of secondary school prevention

<table>
<thead>
<tr>
<th>School grade</th>
<th>Autumn</th>
<th>Spring</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>scale, smoking, erosion, bad breath</td>
<td>gingivitis and treatment</td>
</tr>
<tr>
<td>10</td>
<td>aesthetics (filling, cleaning, whitening, orthodontics)</td>
<td>polishing</td>
</tr>
<tr>
<td>11</td>
<td>individual, issues</td>
<td>individual, issues</td>
</tr>
<tr>
<td>12</td>
<td>individual, issues</td>
<td>individual, issues</td>
</tr>
</tbody>
</table>

Location: in general the dentist surgery, mandatory screening twice a year until 18 years of age, or until completion of secondary school studies.

51.3. Adulthood

In the case of healthy adult patients regular preventive advice is often suppressed with the discontinuation of organized, mandatory group screenings. Answerable patients continue to appear at dental screenings from time to time, however this is typical of 2-3% of the Hungarian population. Regular dental examination is integral to a conscious life-style, thus it is the task of dentists to deliver the necessary information to the people.

Primary themes with consideration to dental diseases characteristic of the age group:

• prevention of periodontal diseases, early loss of teeth,
• caries prevention,
• lesions of the oral mucosa and their prevention,
• benign lesions, pre-cancerous malformation, tumor prevention,
• opportunities for adult orthodontics.

51.4. Elderly Age groups
According to the WHO classification elderly age groups are the following:

• above 65 years of age – young old: relatively healthy and active,
• above 75 – middle old: activity depends on chronic diseases,
• above 85 – oldest old: decreased physical and intellectual activity.

51.4.1. Old-age Deformation

Unfold on physiological, pathological, psychological and socio-economical bases.

51.4.1.1. Sensation

• Hearing loss (30% prevalence in population above 60 years of age).
• Visual impairment (gradual from the age of 30).
• Loss of taste, scent.
• More frequent injuries due to a reduced touch sensation.
• Cold sensation: due to the reduction of sweat-gland function and the deterioration of circulation.
• Impairment of speech and communication skills, forgetfulness.
• Imbalance (increased risk for trauma).

51.4.1.2. Bones and Muscles, Connective Tissues

Weakening (loss of functionality), osteoporosis, confined joint movement, decrease of subcutaneous fat and elastic fiber quantity, leading to a higher susceptibility to injuries and more troublesome healing of wounds.

51.4.1.3. Oral Cavity Deformations

• From among hard tissues, typical of bones are: reduced vascularization, reduction of osteogenic cell quantities, reduced bone trabeculation, osteoporosis, inactivity: all these aggregately cause a ongoing resorption.
• Teeth: due to increased mineralization, caries activity is decreased, narrowing and obliteration of the pulp chamber.
• Soft tissues: thinning of the epithelium, reduction in the quantity of connective tissue (increased collagen density, decreased collagen turnover), loss of elasticity.
• Salivary glands: reduced salivation due to dehydration and structural alterations, at times due to the effects of medication.

51.4.1.4. Digestion

• Sensitive reaction to emotional changes (depression, nervousness, etc.), weakened bowel contractions cause constipation.
• Dehydration: typical as a result of decreased liquid intake.
• Malnutrition/cachexia may develop, thus the time of recovery declines.

51.4.1.5. Circulation

• Decrease of heart-rate, state of hypoxia, causing weakness, sensitivity to cold.
• Slowing circulation, lack of physical activity: stasis, leading to edema and finally ulcers and sores.

51.4.1.6. Immune System
A weakened immune system results in a higher incidence of neoplastic and age related autoimmune processes, frequency of opportunistic infections is typical.

51.4.2. Measurable Parameters of Daily Activity

- **ADL** – Activities of Daily Living (e.g.: clothing, lavation, feeding).
- **IADL** – Instrumental Activities of Daily Living (e.g.: making phone calls, brushing teeth).
- Advancement of age – augmentation of ADL/IADL issues.

51.4.3. Goals of the Maintenance of Oral Health

With the steady increase of life expectancy, the maintenance of the best possible quality of life (QoL). With respect to dental diseases it can stated that there positive correlation between the prevention of tooth loss and the quality of life, as well as general health.

- Maintaining 20 or more teeth, in the case of 20 or more natural teeth leads to "freedom of diet".
- the elimination of functional loss with adequately planned prosthetics.
- prevention of systematic diseases manifesting in the oral cavity.

51.4.3.1. Most typical deformations of the oral cavity, prevention of which should be sought

- Root caries,
- Periodontal disease with early teeth loss,
- Loss of tooth structure (physiological, cannot be prevented, but can be decelerated),
- Tumors.

**Causes of tooth structure loss:**

In general it is not a single, but rather a combined mechanical-chemical damage, which develops through long years. Its prevention or deceleration, i.e. the elimination of ethological factors, is a component of regular dental control. It's mayor types are:

- Erosion (acidic effect), which can be compensated by fluoridation, the elimination of acids.
- Attrition (occurs at the location of antagonist contacts, can be caused by tooth loss on the remaining teeth.
- Abrasion (mechanical factors, e.g. can be caused by metallic parts of dentures).

**Tumors:**

Certain types of tumors occur more frequently in the elderly compared to the younger population. This can be explained in a number of ways: reduced immune system, adverse noxas accumulating through the years and reduced activity of the elderly, each separately and jointly lead to the late diagnosis of malignant tumors. Another particularity is a relatively slow progression: at old age, previously rapidly metastasizing tumors will progress slower, while also tumors of different origin appear, which can be characterized slower progression. As a result of it all old-age tumor prevention is quite versatile. The main trends are supporting the immune system, regular control examinations and the elimination of adverse effects:

- Alcohol consumption,
- Smoking,
- Chronic irritation,
- UV-irradiation,
• HPV vaccine (currently under research),
• Viral infections,
• Insufficient oral hygiene,
• Early recognition and treatment of tumor preventive conditions.

51.5. Tesztkérdések az 1.46–51 fejezetekhez

1. Which are the soft deposits on the teeth?
   a. acquired pellicle, dental plaque, materia alba, debris
   b. calculus, dental plaque, debris
   c. caries, calculus, acquired pellicle
   d. materia alba, debris, caries, acquired pellicle

2. Goals of toothbrushing
   a. Removing plaque
   b. Removing debris and stains from the surface of the teeth
   c. Gingival stimulation
   d. Applying the toothpaste with special ingredients
   e. All of the above listed

3. Which answer is not correct for chisel scalers:
   a. hand instrument
   b. double ended
   c. activated with pulling movement
   d. used for scaling of interproximal surfaces

4. Which of the following caries types is not cavitated?
   a. superficial caries
   b. initial caries
   c. fissure caries
   d. root caries

5. The critical pH level of root caries formation:
   a. is the same as in the case of enamel caries
   b. 5,5
   c. 6,7
   d. there is no data about it

6. Safely Tolerated Dose of fluoride intake:
1. Oral biology

a. 1 mg/bwkg
b. 5 mg/bwkg
c. 32-64 mg/bwkg
d. fluoride is toxic in any dose

7. Effects of fluoride:
   a. endogenous effect in preeruptive stage
   b. exogenous effect in preeruptive stage
   c. endogenous and exogenous effect in preeruptive stage
   d. endogenous effect in posteruptive stage

8. Not correct for fissure sealing:
   a. Effective preventive method
   b. Cheap
   c. Painful
   d. Fast and easy to perform

9. Unfavorable fissure forms are:
   a. U, V, IK
   b. U, inverted Y, IK
   c. I, IK, inverted Y
   d. V, IK, I

10. Absolute contraindications of preventive pit and fissure sealing:
    a. incipient caries
    b. teeth with cavitated carious lesion or dentin caries in the fissures
    c. partially erupted teeth
    d. high risk of caries

11. Which of the following statements is correct:
    a. Vitamins have an important effect in the posteruptive stage of tooth development
    b. Vitamin D is essential in collagen formation
    c. Fats and oils are the most cariogenic part of our diet
    d. The relative sweetening effect of sugar substitutes is compared to sucralose

12. Primary-primary prevention is a prevention type for:
    a. children with primary dentition
    b. children before their tooth eruption
c. pregnant women
d. patients who never had dental prevention before

13. The properties of saliva:
   a. water content is 89.27%
   b. slightly acidic (pH 6.5-7.5)
   c. average daily amount 7000-10000 cm³
   d. dry matter content 10.73%

52. 1.52. Test questions–answers 1–45.

52.1. I.1. Development of tooth germ – Gabor VARGA

1. **Cell phenotype that is absolutely necessary for tooth formation:**
   c. both epithelial and mesenchymal

2. **The first mineralized structure during tooth development:**
   b. mantle-dentin

3. **The borderline of enamel organ facing preodontoblasts:**
   a. inner enamel epithelium

52.2. I.2. Fibers and extracellular matrix of hard tissues - Gabor VARGA

1. **Which protein gene mutations may lead to Osteogenesis imperfect?**
   a. collagen I

2. **Which one of the following structures do not contain collagen?**
   d. Mature enamel

3. **The absence of which vitamin leads to incomplete hydroxylation of key amino acids of collagen chain?**
   c. vitamin C

52.3. I.3. Osteogenesis – Gabor VARGA

1. **Key peptide group in bone morphogenesis:**
   b. BMP

2. **Cells of alveolar bone:**
   a. osteoblasts

3. **Cells that are not characteristic components of tooth formation**
   d. osteoclasts
52.4. I.4. Dentinogenesis and disturbances; formation of primary- , secondary- and tertiary dentin; dentin permeability – Gabor VARGA

1. Caries induced tooth pain is primarily induced by :
   b. opening of dentin tubules

2. Usual diameter of dentin tubules:
   a. 0,2-1 μm

3. Dentin hypersensitivity can be diminished by ….. of dentin tubules
   c. closure

52.5. I.5. Amelogenesis – Gabor VARGA

1. Cells forming enamel:
   d. ameloblasts

2. Percentual representation of amologenin among proteins in non-maturated enamel:
   e. 90%

3. Which protein gene mutations may lead to Amelogenesis imperfecta
   b. kallikrein-4


Which matrix proteins are typical in the immature enamel?
   e. amelogenin, enamelin

Which are the carioprotective trace elements?
   d. Sr, Sn, Mo

What does it mean that the calcium to phosphate ratio is typically 1.6 in apatite crystals?
   a. 10 Ca\(^{2+}\) to 6 PO\(_4\)\(^{3-}\)

52.7. I.7. Test - Calcium homeostasis – Dezso SZOMBATH

1. Complications of hypercalcaemia:
   b. obstipation, nephrolithiasis

2. May be a complication of primary hyperparathyroidism:
   a. mandibular cystic lesions
   b. epulis
d. lack of lamina dura

3. May be a complication of congenital hypophosphatasia:
   a. widened pulp chamber and root canal
   b. disturbed dentinogenesis
   c. retarded dentition
   d. insufficient cementogenesis
   e. loss of deciduous teeth before resorption is completed

52.8. I.8. Formation of hard tissues, mineralization, bone resorption and osteoclasts – Gábor VARGA

1. Average daily calcium excretion of the kidney in healthy adults:
   c. 175 mg

2. Directly stimulates the calcium uptake of intestinal epithelial cells:
   c. active vitamin D

3. Cells that are not characteristic components of tooth formation
   d. osteoclasts

52.9. I.9. Test - Cementogenesis – Balint MOLNAR

1. The coronal two-third of root surfaces is covered by this type of cementum:
   c. acellular fibrillar cementum

2. Multinuclear giant cells, responsible for cement resorption:
   e. cementoclasts

3. The main fiber component in the extracellular matrix of the fibrillar cementum is:
   a. type I collagen

4. Proteins synthesized by the epithelial cells of the Hertwig’s root sheath that induce cementogenesis:
   b. enamel matrix proteins

5. Localization of progeitor cells regulating the regeneration of cementum, PDL and alveolar bone:
   e. periodontal ligament

52.10. I.10. Test - Pathomechanism of bleeding and its relation to dentistry – Katalin VARNAI

1. Which defect(s) is (are) X-chromosome-linked?
   b. Factor VIII and Factor IX deficiencies

2. How long before a simple dental intervention should INR be tested in a stable patient?
   d. <24 hours
3. Dental treatment of a haemophilic patient can be carried out
   b. after factor supplementation and haematologic consultation at an expert institute

52.11. I.11 Test - Tooth eruption and tooth movement – Balint NEMES

1. What is the eruption mostly induced by?
   a. Local effects: The follicle binds different GFs, which results in bone resorption in the coronal region.

2. The eruption sequence of permanent teeth is:
   b. 6-1-2-4-5-3

3. Which statement is true?
   a. We need small forces to move the teeth as tooth movement is inversely proportional to the force acting on the tooth.

52.12. I.12. The morphology and function of salivary glands - Gabor VARGA

1. Fundamental importance for embryonic salivary development:
   d. all three

2. Location of secretory protein (export protein) storage in salivary glands
   b. zymogen granule

3. Which one of these transmitters stimulates both salivary secretion, gastric acid secretion and pancreatic enzyme secretion?
   b. acetylcholine

52.13. I.13. Salivary gland electrolyte, water and protein secretion - Gabor VARGA

1. Major channel(s) for salivary acinar electrolyte secretion
   d. Ca" dependent Cl- channels

2. Major channel(s) for salivary ductal electrolyte transport
eNaC

3. Primary ion in salivary acinar vectorial electrolyte transport
   b. Cl ion


1. Main source of epidermal growth factor (EGF) in humans
   d. parotid gland

2. The role of lysozyme in saliva:
1. Oral biology

3. Basic mechanism of the antibacterial action of lactoferrin

52.15. I.15. Dental stem cells for dental research - Gábor VARGA

1. The cells which form the pulp originate from the neuronal chrest, therefore we call them:

   b. ectomesenchymal

2. Fundamental characteristic(s) of stem cells:

   d. cells which have the capacity to renew themselves, and additionally able to produce differentiated

3. The stem cells of dental origin are able to regenerate the following structure(s):

   e. none of the above


1. Role of fat in caries formation

   a. cariostatic

2. Its deficiency leads to gingivitis and periodontitis

   c. vitamin C

3. The decrease of functional activity of chewing muscles may result in:

   d. malocclusion

52.17. I.17. Test – Chewing – Mate JASZ

1. While doing a mouth opening:

   b. The left and right condyles move symmetrically along a curved path forward and downwards, while a rotational movement also occurs

2. Under normal circumstances the left and right condyles perform different movements during:

   d. Chewing

3. Which one belongs to the temporomandibular joint?

   e. All of them

52.18. I.18. Test – Pathophysiology of inflammation – Beata Keremi

1. What is the consequence of increased capillary permeability?

   d. oedema formation

2. Which of the following proteins are required for granulocyte adhesion?

   d. integrin

3. Which is true for inflammatory cytokines?
b. have a role in the regulation of immune response

52.19. I.19. Structural and functional characteristics of dental pulp, blood supply to the oral tissues, pulpal pain and inflammation – Gábor VARGA

1. Main protein component of pulp matrix
   b. proteoglycan

2. Localization of nerve fibres of cold induced pain in tooth
   a. in pulp

3. Mediator liberated in the pulp during pulp inflammation:
   c. calcitonin gene-relate peptide (CGRP)

52.20. I.20. Test - Ionizing radiation and the oral effects therapeutic irradiation – Kristof KADAR

1. The most radiosensitive tissue of the followings is:
   a. blood/bone marrow

2. In typical salivary dysfunction related to radiotherapy the salivary flow after seven weeks is:
   c. about 20% of normal

3. Which of the following radiations is NOT considered ionizing?
   e. Infrared (IR) radiation


1. In the majority of cases, how many proteins are coded by an individual gene?
   a. one protein

2. Which disease can be cured by gene therapy of the mutation of the ADA gene?
   e. SCID

3. Secretory pathway of synthetized salivary proteins
   c. constitutive or regulated depending on the actual protein

52.22. I.22. Mechanisms of tumor formation and oral cancer – Gábor VARGA

1. Basic event(s) leading to tumor formation:
   a. successive mutations of somatic cells

2. Protooncogen, its mutation frequently play an importnt rola int he development of oral cancer
   e. k-ras

3. Etiologic factor for oral cancer
c. HPV virus


1. Which cranial nerve does not convey taste information?
   a. the third cranial nerve

2. What is not true for the mechanism of tasting?
   a. The sensing of each basic taste is localized to distinct areas of the tongue.

3. Which statement is false?
   d. The olfactory system is robust.

4. Which one of the drugs below does not cause gustatory dysfunction?
   d. Listerine

52.24. I.24. Test - Gingival sulcus and crevicular fluid – Zsolt LOHINAI

1. It is NOT the border of the gingival sulcus:
   b. alveolar process

2. Is is characteristic of the biological width:
   b. average width is 2 mm

3. Which statement is false:
   c. the sulcus epithelium ensures the real epithelial seal

52.25. I.25. Test - Oral aspects of salt and water household disturbances – József BLAZSEK

1. Which of the following conditions will NOT lead to a drastically increased fluid consumption?
   a. Increased vasopressin (ADH) production

2. The maximum survival time with water deprivation is:
   c. 8 to 10 days.

3. Expected after contusion, tissue injury:
   b. increased plasma potassium


1. Usual consequence of gastroesophageal reflux disease:
   b. dental erosion

2. The major cause of the gastric ulcer in the majority of cases:
b. bacterial infection

d. chronic alcoholism

52.27. I.27. Test - Oral aspects of acid-base regulation – József BLAZSEK

1. The following condition will lead to metabolic acidosis:

   d. in disorders of kidney carbonic anhydrase function

2. What is the amount of carbon dioxide produced daily by cellular respiration in a healthy adult human?

   c. 12000 to 13000 mmol

3. Which of the following acids can be eliminated by respiration?

   b. carbonate

4. Which of the following anions are not found in urine as an anion partner of hydrogen ion?

   d. bicarbonate


1. How does protein concentration change usually in the urine when the glomerular function is impaired?

   b. increased

2. In acute renal failure always happens:

   d. glomerular filtration is decreased

3. Approximate starting GFR value for hemodialysis:

   d. 8% of the normal GFR

52.29. I.29. Test - Pathophysiology of liver and oral aspects – Beata KEREMI

1. Blood clotting problems can occur when the synthesizing function of liver is damaged, because the elimination of endogenous compounds does not occur properly.

   c. true-false

2. Which statement is true for the formation of conjugated bilirubin?

   c. glucuronidation takes place

3. Which hepatitis virus can infect in the presence of another virus only?

   d. HDV

52.30. I.30. Test - Central and peripheral circulatory failure – Kádár Kristóf

1. When cardiac muscle inotropy is decreased:

   a. the stroke volume is decreased
2. Symptoms of left ventricular forward failure include:
   a. nycturia
   c. fatigue, weakness

3. In the initial phase of hypovolemic shock there is a generalized vasodilation (A), and the resulting centralization of the circulation leads to a generalized tissue hypoxia in most of the organs (B).
   d. false-true

52.31. I.31. Oral aspects of hypertension – Gábor VARGA

1. Characteristic for essential hypertension:
   a. incidence increases with age

2. Hyperthyreosis induces hypertension, since:
   c. hyperthyroid hormones sensitize the arterioles to adrenergic compounds

3. It does NOT play role in blood pressure regulation:
   c. alkaline phosphatase

52.32. I.32. Oral aspects of protein metabolism and energy balance – Gábor VARGA

1. Characteristic for the early, non-adapted phase of starvation:
   a. increased gluconeogenesis

2. Calculation of Body Mass Index (BMI)
   c. BMI= body mass (kg)/body height\(^2\) (m\(^2\))

3. Extreme low body weight is indicated by the following BMI value
   b. 16

52.33. I.33. Test - Diabetes mellitus and its oral aspects – Beata KEREMI

1. Can we measure high levels of insulin in II type DM patients?
   c. during the initial stage there may be high insulin levels due to insulin resistance

2. Which are the typical symptoms of metabolic X syndrome?
   c. diabetes mellitus
   e. hypertension

3. Patients with diabetes mellitus are more susceptible to infections because fewer neutrophils are formed and they cannot develop an effective protection.
   c. true-false

52.34. I.34. Test - Lipid metabolism – Beata Keremi

1. Which apoprotein is responsible for the activation of lipoprotein lipase?
c. ApoCII

2. Which lipoprotein/s transport/s triglycerides to the periphery to be used for energy?
   a. chylomicron and VLDL

3. What is the essence of reverse cholesterol transport?
   d. transport of excess cholesterol from the periphery to the liver

52.35. I.35. Test - Atherosclerosis – Beata Keremi

1. What is not characteristic of atherosclerosis?
   a. vessel wall thickening on small blood vessels

2. What plays a role in the development of atherosclerotic plaque?
   e. all of the above

3. Which are the risk factors for atherosclerosis?
   c. high oxLDL level
   d. deep, untreated periodontal pockets

52.36. I.36. Pain sensation – oral aspects – Gábor VARGA

1. Primary afferent fibers involved in pain development:
   b. A-delta and C fibers

2. Peptide transmitter releasing from nociceptive sensory nerves:
   b. CGRP

3. Meaning of „allodynia”:
   a. Decreased threshold of pain activation

52.37. I.37. Test - Oral aspects of endocrine disorders – Dezso SZOMBATH

1. What is the most likely diagnosis based on the following symptoms: apparent diastema, temporomandibular arthritis, distraction, macroglossia?
   c. GH-IGF-1 secretory dysfunction

2. Oral cavity alterations characteristic of Addison’s disease:
   a. patchy pigmentation on the tongue
   b. pigmentation all over the body including the gingiva

3. Retarded dentition with malocclusion is frequent in the following conditions:
   c. congenital hypothyroidism, cretenism

52.38. I.38. Test - Disorders of respiratory functions – Beata KEREMI
1. What is sensed by the brainstem chemoreceptors?
   b. carbon dioxide partial pressure in blood

2. Which is correct?
   d. vital capacity = inspiratory + expiratory reserve volumes + tidal volume

3. Which are possible reasons for breathlessness?
   d. all of the above


1. What is characteristic of the first phase of oral clearance?
   b. stimulated saliva secretion
   d. dilution and buffering are the primary characteristics

2. What is characteristic of stimulated saliva?
   a. Its bicarbonate concentration is higher than that of the unstimulated saliva and therefore it is better buffered.

3. What has a positive effect on oral clearance?
   d. high swallowing frequency and large swallowing volume

52.40. I.40. Test - Practice: Oral defense mechanisms – József Blazsek

1. Which is not a component of the oral defense barrier?
   e. secretory IgA

2. Which non-secretory immunoglobulin can stay functional for an extended time in the oral salivary milieu?
   c. IgM

3. Which is the most important in maintaining a normal oral microflora?
   d. secretory IgA mediated capture and elimination

4. Which is not directly related to the formation of secretory IgA and its transport to the oral cavity?
   c. C4 complement

52.41. I.41. Test - Practices: Analysis of dental plaque – Gabor KISS

1. What is the first step of dental plaque formation?
   d. acquired pellicle

2. What type of bacteria are the most frequent amongst the initial colonizers of dental plaque?
   c. Gram-positive cocci

3. What is the main ingredient of dental calculus?
   b. calcium phosphate
52.42. I.42. Practice: Salivary secretion – Gábor VARGA

1. Primary factor determining esophageal pH in humans:
   a. bicarbonate
2. Instrument for measurement small salivary gland secretion
   d. Periotron
3. Xerostomy by definition:
   c. decreased salivary secretion based on the subjective feeling of the patient

52.43. I.43. Test - Practice: Chewing – Mate JASZ

1. Which one does not fit on the sagittal Posselt diagram?
   b. 2 cm mouth opening
2. Which one is not true for the Bennett movement?
   c. It is usually more than 2 cm
3. During translational movement:
   e. All of the above answers are true

52.44. I.44. Test - Practices: PCR technique in dental research – Erzsebet BORI

1. You would like to separate longer and shorter PCR products (linear, double stranded DNA) by gel electrophoresis. During the separation ...
   b. shorter molecules migrate faster because they can move more easily through the pores of the gel.
2. Which statement is FALSE for the PCR reaction.
   d. DNA primase is used for the reaction.
3. We perform quantitative (Real Time) PCR reaction using Taqman chemistry. Which statement is TRUE?
   c. The Taqman probe increases the specificity of the reaction because not only the primers but also the Taqman probe must bind to the sequence to be detected.

52.45. I.45. Test - Practice: Investigation methods of mineralised tissues – Kristof KADAR

1. Which one is true for the quantitative light-induce fluorescence (QLF) method:
   a. fluorescence from an area with demineralised enamel is weaker
   c. the method is useful in caries diagnostics
2. Which one is true for static hardness testing methods:
   b. require a prepared, flat surface
3. Which of the following methods uses a ball (sphere)-shaped indenter?
   b. Rockwell hardness test
c. Brinell hardness test
Chapter 2. 2. Pediatric dentistry and orthodontics

1. 2.1. Diagnostic procedures in Pediatric Dentistry. Medication therapy. Developmental diseases – Balázs Sandor

1.1. Diagnostic procedures in pediatric dentistry

1.1.1. Patient history

The dental treatments are dependent on the detailed medical and dental history of the patient. History taking in children differs significantly from the one taken in adults. This is also a good opportunity to get to know the child and the parents. Table 1. shows a systematically built up questionnaire with mandatory questions to be asked about the medical condition of the child. Basically the questions can be grouped into four main categories: personal details, presenting complaints, medical history and dental history. The general medical history should also include questions regarding pregnancy and delivery, growth and development, current medical treatment. Family and social history would also be helpful in treatment planning, for some this may be too confidential. In the case of minors (under the age of 16) heteroanamnesis (information is not gathered from the patient, but from someone who knows the person) is required. It is important to note that parental access to their children may never be denied.

<table>
<thead>
<tr>
<th>Table 1. Table 1. – Patient history</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
</tr>
<tr>
<td>what happened, why visit</td>
</tr>
<tr>
<td>what is painful</td>
</tr>
<tr>
<td>other symptoms</td>
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<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>medication</td>
</tr>
<tr>
<td>general disease</td>
</tr>
<tr>
<td>family diseases</td>
</tr>
<tr>
<td>preventive anamnesis</td>
</tr>
<tr>
<td>vaccines</td>
</tr>
</tbody>
</table>

1.1.2. Extraoral examination

After the history taking visual, manual, and instrumental and imaging examinations are carried out. The first step is the extraoral examination. Table 2. shows the details of extraoral examination.

<table>
<thead>
<tr>
<th>Table 2.2. Table 2. – Extraoral examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>skull</td>
</tr>
<tr>
<td>symmetry of face</td>
</tr>
</tbody>
</table>
2. Pediatric dentistry and orthodontics

<table>
<thead>
<tr>
<th>eyes, periorbital tissues</th>
<th>size of pupilla</th>
<th>eye movements</th>
<th>periorbital bleeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>skin</td>
<td>wounds</td>
<td>scarf</td>
<td>spots (red-white)</td>
</tr>
<tr>
<td>lymnodes in all craniofacial region</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

1.1.3. Intraoral examination

Intraoral examination may only be carried out with the patient in the dental chair.

Important for examination:

- The position of the patient (sitting, reclined, supine position).
- Illumination (approximately 20000 lux).
- Equipment (dental forceps, dental probe, dental mirror, periodontal probe, water-air syringe).

Table 2.3. Table 3. – Intraoral examination

<table>
<thead>
<tr>
<th>lips</th>
<th>angulus</th>
<th>bucca</th>
<th>parotis</th>
</tr>
</thead>
<tbody>
<tr>
<td>vestibulum</td>
<td>mucous membrane</td>
<td>frenulums</td>
<td>curvatures</td>
</tr>
<tr>
<td>gingiva</td>
<td>vestibular side</td>
<td>oral side</td>
<td>papillas</td>
</tr>
<tr>
<td>palatum</td>
<td>hard</td>
<td>soft</td>
<td>palatal arches</td>
</tr>
<tr>
<td>tongue</td>
<td>surface</td>
<td>movement</td>
<td>size</td>
</tr>
<tr>
<td>floor of the mouth</td>
<td>sialoglands</td>
<td>retromolar, sublingual region</td>
<td></td>
</tr>
<tr>
<td>teeth</td>
<td>caries, periodontal and developmental diseases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>occlusion</td>
<td>simple classification</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 2.1. Figure 1. – Child examination with the help of the parent

Figure 2.2. Figure 2. – Child examination with the help of the parent (knee-to-knee position)
2. Pediatric dentistry and orthodontics

Figure 2.3. Figure 3. – Child examination with the help of the parent

Figure 2.4. Figure 4. – Child examination with the help of the parent
1.1.4. Imaging

Imaging used in dental routine:

- Photographs (important part of documentation)
- Intraoral radiographs
  - Periapical,
  - Bitewing,
  - Occlusal.
- Extraoral radiographs
  - Panoramic radiograph (orthopantomogram, OP),
  - Lateral cephalometric radiograph.

Other non-routine imaging may aid our diagnosis and treatment plan, such as cone-beam computer tomography (CBCT), magnetic resonance imaging (MRI), ultrasonography.

Generally radiographs are used in pediatric dentistry for diagnosis, for monitoring facial development and for monitoring the progress of our therapy. Because the radiation accumulates during life, every possible effort should be made to minimize the exposure. Radiographs should only be taken of the patient if the results affect our dental care.

Imaging is very important part of medical examination, but decision on treatments should never be based on it solely.

Sometimes the localization of a disorder or a lesion is needed (impacted tooth, supernumerary tooth, etc.). It is important before a treatment plan to know whether the disorder can be accessed from the buccal or the lingual side. CBCT may be one of our choices, however in some cases sufficient information may be gained from intraoral radiographs with special methods (Miller’s right angle method, Clark’s shift shot), and this way reducing the radiation exposure.
Miller’s right angle technique

After identifying the disorder on the periapical radiograph, an occlusal projection with the beam at a right angle (perpendicular) to the direction of the beam of the periapical exposure is taken.

Clark’s tube shift method (SLOB rule)

It is used to identify the buccal or lingual location of objects (impacted teeth, root canals, etc.) in relation to a reference object (usually a tooth). After making a radiograph of the disorder or lesion, the tube head is shifted in the mesial or distal direction. (When using a digital sensor, the sensor is left in the exact position). SLOB (Same Lingual, Opposite Buccal) rule:

- **Lingual localization**: on the radiograph, the examined object moves from the reference object in the same direction which the tube head has been moved (mesial or distal).

- **Buccal localization**: on the radiograph, the examined object moves from the reference object in the opposite direction which the tube head has been moved (mesial or distal). (Animation 1.)

1. video.

Adjunct detection tools:

**Fiberoptic transillumination (FOTI)**: ez a módszer a fogak áttetszőségén alapul. Az egészséges zománc más képet mutat, mint a caries. Az eljárás nem teljes értékű, kiegészítésként alkalmazható a szűrővizsgálatoknál, vagy a rtg kép készítése előtt. It is based on the transillumination of the tooth. The translucency of the carious enamel is different from the sound enamel. The diagnostic value is limited, however may be used to support indications of preventive treatment and bitewing radiography. (Huysmans MC, 2011).

**Laser/light fluorescence (Quantitative light fluorescence-QLF)**: There is loss of fluorescence in the carious enamel.

1.1.5. Other diagnostic possibilities

1. Pulp vitality tests

   - **Thermal tests** (cold, or hot),
   - **Electric pulp tests (EPT),**
   - **Laser Doppler flowmetry (LDF).**

   EPT and thermal testing only assess nerve supply of the pulp, whereas LDF is suitable for testing the vascular supply, without the possibilities of subjective responses. For thermal tests usually cold spray (-50°C) or hot gutta-percha may also be used. Gingival stimuli may produce fake reaction from the patient. The response in small children (especially in deciduous dentition) to the stimuli is not reliable, the results should be evaluated carefully. Teeth may react very differently, depending on the stage of root development, sometimes showing no reaction until root formation is complete, due to the incomplete innervation.

2. Diagnostic casts.

3. Caries activity tests.

4. Blood tests.

5. Microbiological investigations.

6. Histological examination.

ICDAS (International Caries Detection and Assessment System):

1. Sound even after 5 seconds of drying.
2. Pediatric dentistry and orthodontics

2. First Visual Change in Enamel (seen only after prolonged air drying or restricted to within the confines of a pit or fissure).

3. Distinct Visual Change in Enamel (tooth must be viewed wet).

4. Localized Enamel Breakdown after 5 seconds of drying (without clinical visual signs of dentinal involvement).

5. Underlying Dark Shadow from Dentin.

6. Distinct Cavity with Visible Dentin involving less than the half of the tooth surface.

7. Extensive Distinct Cavity with Visible Dentin involving at least the half of the tooth surface.

The established diagnosis determines the treatment plan, with the following sequence:

1. Emergency treatments (bleeding, inflammation, pain).


3. Surgical treatments.

4. Restorative treatments.


6. Accurate recall and follow up.

The method of examinations is the same in an unmanageable/uncoperative child, however we are forced to compromise. In extreme cases examination cannot be carried out, it has to be done in general anesthesia prior to treatment.

A personal relationship is formed between the patient and the dental staff during the examination process which is determining throughout the total treatment. This is especially important in children, where it has a decisive influence on the behavior of the child in the dental office later in life (fear, aggression, passivity, etc.).

In pediatric dentistry pay attention to the following:

1. Accept and do not try to change the child's temperament (calm-ADHD).

2. Show interest in things that are important for the child.

3. Be honest to the child (using words that are understandable on the level of the child).

4. The accompanying person/parent may be present during the treatment in accordance with legal regulations.

5. Provide horizontal eye contact, pay attention to the personal space of the child.

6. Always start with the simplest treatment.

7. Warm and comforting surroundings should be provided in the waiting room and in the office as well.

8. Be cheerful, liberated and talk constantly to child patient (positive feedback).

9. Control and recognize the non-verbal signs as it is the main information source for a child.

1.1.6. Special needs

There may be general medical conditions which may affect our timing of examinations and treatment. Although there are guidelines for the treatment of these patient, consultation with the physician is recommended.

1.1.6.1. Cardiovascular disorders (congenital or acquired)
Prevention of dental diseases is important in patients with cardiovascular disorders. For invasive treatment antibiotic prophylaxis is required according to the existing guidelines.

### 1.1.6.2. Disorders of the blood

#### 1.1.6.2.1. Bleeding disorders (Haemophilia, Von Willebrand’s disease Thrombocytopenia)

Communication with the haematologist is required, if replacement of blood clotting factors is necessary. Regional (nerve block) anesthesia should be avoided.

#### 1.1.6.2.2. Blood dyscrasias (anaemia, leukaemia)

Anemic patients have a greater chance of excessive bleeding. In patients with leukaemia prevention is essential. No elective treatment should be carried out until the remission of the patient.

### 1.1.6.3. Respiratory disorders (asthma, cystic fibrosis)

Dental care for patients with asthma is usually not a problem. The puff inhaler should be prepared during the treatment. General anesthesia should be avoided in patients with cystic fibrosis.

### 1.1.6.4. Convulsive disorder (epilepsy)

Some antiepileptic drugs (phenytoin) induce gingival hyperplasia. Removable dental appliances should be avoided.

### 1.1.6.5. Metabolic and endocrine disorders

#### 1.1.6.5.1. Diabetes mellitus

Prevention is very important in patients with diabetes mellitus. They are considered to be in the high caries risk group. When planning a treatment morning appointments are preferred after taking the medications and breakfast.

#### 1.1.6.5.2. Hyperthyreosis

Local anesthetics containing epinephrine should be avoided.

### 1.1.6.6. Neoplastic disorders

The oral symptoms associated with therapy of neoplastic disorders may be classified into:

- immediate symptoms (mucositis, exacerbation of oral inflammatory diseases)
- late symptoms may be (radiotherapy of the head and neck region may lead to the decreased salivary flow, mucosal ulcers).

### 1.1.6.7. Organ transplants

It is important to have a dental treatment prior to transplantation. All doubtful prognosis teeth should be extracted.

### 1.2. Pharmacological management of pain, fever and anxiety

#### 1.2.1. Systemic fever and pain relief

It is important to differentiate analgesia from anesthesia.

- Analgesia: reduction or relief of pain. There is no lack of sensation.
- Anesthesia: lack of sensation accompanied by analgesia.

Fever: a child is considered to have fever if the core temperature is above 37.9°C. Above 37.5°C orally or 37.2°C axillary measured temperature, during daytime physical and pharmacological management is indicated.
Physical reduction of the temperature may be achieved by undressing the child, cold bath (gradual reduction of the water temperature is necessary, using too cold water may make the situation worse inducing shivering, which raises the core body temperature) or with cold compress. There is a risk for hyperthermia in an overdressed child! These methods for fever relief are effective and should be used as a supplement to pharmacological management.

Pain: “Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (International Association for the Study of Pain, Pain Definitions”. Retrieved 12 October 2010.) Assessment is difficult, we have no objective measurement, yet non-verbal signs (grimacing, changes in vocalization, routine behavior, and mental status, etc.) and the parents may help us in recognizing and evaluating the quality and severity of pain.

Several anti-inflammatory, analgesic and antipyretic drugs are available in the market. They can be classified several ways, and may vary. Basically non-opioid analgesic drugs are used. Opioid drugs are only recommended in institutional settings, due to its severe adverse effect- respiratory depression. Some well known drugs are intentionally not mentioned here, and due to their severe non-dose-related (idiosyncratic) adverse effect there are excluded from our pediatric dental care. The use of acetylsalicylic acid or Aspirin (adverse effect: Reye syndrome –potentially fatal hepatonecephalopathy) or metamizole sodium (adverse effect: agranulocytosis) are not recommended.

1.2.1.1. Paracetamol (acetaminophen)

Antipyretic and mild analgesic, effect varies individually. It has no anti-inflammatory effect. Overdosing may cause severe liver damage (quite rare). Because of the low incidence of adverse effects it may be a good alternative to non-steroid anti-inflammatory drugs (NSAID). Usual dosage is 15 mg/kg, maximally 50 mg/kg/day divided into 4-6 parts, do not use continuously for more than 5 days, after this period change for another substance.

<table>
<thead>
<tr>
<th>Table 2.4.</th>
<th>Table 4. – Paracetamol dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Available forms</strong></td>
<td><strong>Packing</strong></td>
</tr>
<tr>
<td>tablets</td>
<td>500 mg</td>
</tr>
<tr>
<td>suspension</td>
<td>120 mg/5 ml</td>
</tr>
<tr>
<td>syrup</td>
<td>200 mg/5 ml</td>
</tr>
<tr>
<td>suppository</td>
<td>125 mg, 250 mg, 500 mg, 1000 mg</td>
</tr>
</tbody>
</table>

1.2.1.2. Ibuprofen

It is a widely used NSAID substance with anti-inflammatory effect, relieves pain very well. No prescription needed in Europe. Dosage according to Table 5. (Usually 30 mg/kg/day divided into 3 to 4 parts.)

<table>
<thead>
<tr>
<th>Table 2.5.</th>
<th>Table 5. – Dosage of ibuprophen</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Available forms</strong></td>
<td><strong>Packing</strong></td>
</tr>
<tr>
<td>suspension</td>
<td>100 mg/5 ml (strawberry and orange flavor)</td>
</tr>
<tr>
<td>oral soluble tablets</td>
<td>100 mg, 200 mg</td>
</tr>
<tr>
<td>suppository</td>
<td>60 and 125 mg</td>
</tr>
<tr>
<td>others</td>
<td>200-400 mg</td>
</tr>
</tbody>
</table>
1.2.1.3. Diclofenac (NSAID)

It is a very effective NSAID substance with anti-inflammatory action, able to reduce fever and relieve pain. According to our empirical observation duration of action may reach 12 hours, but may change individually. Recommended dosage is 0.5-2 mg/kg/day divided into 2-3 parts.

Table 2.6. Table 6. – Dosage of diclofenac

<table>
<thead>
<tr>
<th>Available forms</th>
<th>Packing</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>drops (suspension)</td>
<td>15 mg/ml 0.5 mg/drop</td>
<td>0.5-2 mg/kg/day divided into 2 to 3 parts</td>
</tr>
<tr>
<td>tablets</td>
<td>25 mg</td>
<td>0.5-2 mg/kg/day divided into 2 to 3 parts</td>
</tr>
<tr>
<td>tablets</td>
<td>25-150 mg</td>
<td>for children above 14 years 75-100 (150 mg) mg/day divided into 2 to 3 parts</td>
</tr>
</tbody>
</table>

1.2.1.4. Combination of analgesic and antipyretic drugs

In particular cases of infectious and systemic diseases the analgesic and antipyretic effect of the drugs is insufficient. The distressing symptoms may recur after 1-2 hours. Supplemental physical methods of reducing the temperature or combined use of analgesic drugs (to mineralize risk of the adverse effect), changed every 4-5 hours, may be indicated for 24-36 hours in the acute phase of the disease.

1.2.1.5. Supportive management in case of fever and pain

- Increased fluid intake,
- Maintenance of normal blood glucose level,
- Electrolyte supplementation,
- Cold compress.

1.2.2. Local anesthetics

1.2.2.1. Infiltration anaesthetics

Local anesthetics are distributed with or without epinephrine. Epinephrine causes local vasoconstriction which prolongs the duration of action.

1.2.2.1.1. Lidocaine hydrochloride injection

Lidocaine hydrochloride injection is an amide type local anesthetic and antiarrhythmic drug. Maximum dose of lidocaine without epinephrine is 4.5 mg/bwkg, with epinephrine 7 mg/bwkg. Several concentrations are available in the market (1%, 2% and 10%). The maximum dose of epinephrine is 1-2 ug/bwkg, with the maximum concentration of 10 mg/l. The generally used 2 ml solution containing 40 mg of lidocaine, if mixed with epinephrine it contains 0.02 mg of epinephrine. In small children diluted solutions may be used (diluted to 0.5%-1%), to reduce the toxicity.

1.2.2.1.2. Articaine hydrochloride injection

Articaine hydrochloride injection is an amide type anesthetic solution distributed in a 4% concentration always with, either 1:80000, 1:100000 or 1:200000 epinephrine.. Maximum dose is 7 mg articaine hydrochloride/bwkg (0.175 ml/bwkg). The use of articaine is not recommended under the age of 4. Usually for a child the weighing 20-30 kg, 0.25-1 ml, weighing 30-45 kg 0.5-2 ml solution is sufficient.

Ultracain DSForte: contains 0.024 mg epinephrine-hydrochloride and 80,0 mg articaine-hidrochloride in 2 ml ampoules. Maximum dose is 7 mg/bwkg.
2. Pediatric dentistry and orthodontics

Ubistesin: 1 ml solution contains 40 mg articaine hydrochloride and 0.005 mg (1:200000) epinephrine hydrochloride. The 1.7 ml ampoules contain 68 mg articaine hydrochloride and 0.0085 mg epinephrine hydrochloride).

Ubistesin forte: 1 ml solution contains 40 mg articaine hydrochloride and 0.01 mg (1:200000) epinephrine hydrochloride. Recommended for longer treatments, or for surgical treatments.

Septanest: contains 68 mg articaine hydrochloride and 0.017 mg epinephrine in 1.8 ml ampoules.

1.2.2.1.3. Bupivacaine-hydrochloride injection

Bupivacaine-hydrochloride injection amide type long-acting local anesthetic solution marketed in 2.5 mg/ml (50 mg bupivacaine hydrochloride/bottle) and 5 mg/ml (100 mg bupivacaine-hydrochloride/bottle) concentration in 20 ml bottles. “Bupivacaine is generally not recommended for children aged less than 12 years since there is insufficient information on the effects of its use in this age group” (WHO Model Prescribing Information: Drugs Used in Anaesthesia).

1.2.2.1.4. Tools of local anesthesia

Tools of local infiltration anesthesia

- Disposable syringe and needle.

- Pen style (Paroject®-Figure 5.) device cylinder ampoules, permitting a slow non-traumatic injection. Drawback is not possible. Although only a part of the solution is injected from the ampoule, it must not be reused with other patients (Gräf W, 1981).

- CCLADS (Computer Controlled Local Anaesthetic Delivery System- Figure 6.) minimizes pressure and pain during infiltration. Studies revealed no difference in pain scores.

Figure 2.5. Figure 5. – Pen style (Paroject®) syringe

Figure 2.6. Figure 6. – CCLADS Computer Controlled Local Anaesthetic Delivery System
1.2.2.2. Topical anesthesia

May be used for the extraction of remnants of deciduous teeth, and prior to infiltration anesthesia. They are effective 1-2 mm deep, the area should be dried, and sufficient times is required for the on-set of its effect. The area of use is limited to 1-2 cm².

1.2.2.2.1. Lidocaine spray

3,80 g lidocaine in 38 g alcoholic solution (10% concentration). One puff contains 4,8 mg lidocaine. It penetrate 1-2 mm deep in the oral mucosa, limiting its indications. Another disadvantage in children is spraying in the oral cavity may cause reflex apnea. Application is recommended with cotton wool ball. Because of the high lidocaine concentration only professional use is allowed.

1.2.2.2.2. Lidocain containing gel (Dentinox® gel)

1g gel contain 150 mg chamomile tincture, 3,4 mg lidocaine hydrochloride-monohydrate and 3,2 mg macrogol-lauryl-ether. Because of the low lidocaine concentration it is indicated for home use especially in teething pain.

1.2.2.2.3. Lidocain ointment

5 % concentration, may be used in home care and in professional care as well.

1.2.2.2.4. Benzocaine containing anesthetic suspension

Contains bezocaine in 3% concentration, basically sweet, but may be prescribed in special flavors. The flavored suspension contain sucrose, which should be taken into account during long time usage. Prescribed for home use.

1.2.2.2.5. Benzocaine gel for professional use: 20% concentration

Preferred by children due to the flavoring (bubble gum, butter rum, menthol, pina colada, walterberry). Penetrates 2-3 mm deep in the oral mucosa. According to the instruction manual the onset time is 10-30s, our empirical observations suggest longer onset time (2-4 minutes).

Table 2.7. Table 7. – Dosage of local anesthetics

<table>
<thead>
<tr>
<th>Active substance</th>
<th>Product name</th>
<th>Maximum dose</th>
<th>10 kg child</th>
<th>20 kg child</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lidocaine</td>
<td>Lidocain 2%</td>
<td>4,5 mg/ttkg</td>
<td>45 mg → 2,25 ml</td>
<td>90 mg → 4,5 ml</td>
</tr>
<tr>
<td></td>
<td>Lidocain 2% epinephrine 1:100</td>
<td>7 mg/ttkg</td>
<td>70 mg → 3,5 ml</td>
<td>140 mg → 7 ml</td>
</tr>
<tr>
<td>Articaine</td>
<td>Ubistesin (4% articaine, epinephrine 1:200 000)</td>
<td>7 mg/ttkg</td>
<td>Only over the age of 4.</td>
<td>20-30 kg 1,5 ml/treatment, 2,5 ml/24 hours 30-45 kg 2 ml/24 hours 5 ml/24 hours</td>
</tr>
<tr>
<td></td>
<td>Ubistesin forte (4% articaine, epinephrine 1:100 000)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ultracain DS Forte</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>Marcain/ Bucain 0,25; 0,5%</td>
<td>2 mg/ttkg</td>
<td>not recommended</td>
<td>not recommended</td>
</tr>
</tbody>
</table>

1.2.2.3. Procedure of local anesthesia

- Written consent of the parents and accurate history is required
- Communication with the child, describing the numbness to the child
- Previous topical anesthesia is required before injection
• Short and fine needles are recommended disposable needles are used (25G, 27G, 30G)
• Assistance is required (fixing the head of the child)
• Infiltration has to be done slowly with warm anesthetic solution
• The patient should avoid eating anything until the numbness lasts (mucosal injury, Figure 7.)

Figure 2.7. Figure 7. – Morsication of the lips and buccal mucosa after lower anesthesia

Pain-free anesthesia:

The appropriate technique of pain-free anesthesia is shown in Figure 8. The administration of the solution has to be slow, and the needle should always be inserted only in the previously topically anaesthetised area, proceeding with continuous deposition of the anesthetic solution. In cases of serious surgical treatment (extraction) of deciduous the palatal anesthesia is also required. After topical anesthesia and buccal injection the anesthesia is carried out via the interdental papillae, with stepwise advancement of the needle. With slow retraction the solution is being administered. Finally the injection is done directly on the palatal mucosa.

Figure 2.8. Figure 8. – The steps of pain-free anesthesia
Lower block anesthesia:

Until the eruption of the lower first permanent molars the mandibular foramen on the ramus of the mandible is located in the level of the occlusal plane of the primary molars. According to this the injection should be infiltrated in the height of the occlusal plane of the lower primary molars. After eruption it is located 1 cm above the occlusal plane. The method is similar that of adults.

Intraligamentary anesthesia:

It may be used in anxious, very young or mentally retarded children for the removal of primary tooth root remnants. Usually 0.2 ml solution is enough for each root. The use contraindicated in the risk of endocarditis.

1.2.3. Behavior management (sedation)
In some cases, due to the anxiety of the child, effective communication and intervention is impossible. With local pain relief may be achieved, but may be supported with sedation if needed. In some cases general anesthesia is the only solution (mental retardation, small children, big surgical intervention, etc.). The discussion of general anesthesia exceeds this topic. In outpatient (ambulatory) treatment conscious sedation is an accepted method of behavior management. The regulations of conscious sedation may differ in countries. The use of nitrous oxide, commonly known as laughing gas is well established method of sedation (according to the existing regulation of Hungary conscious sedation can only be performed in the presence of a doctor familiar with airway management.)

In our practice we use pharmacological behavior management with benzodiazepine and its’ derivatives.

Midazolam: short acting benzodiazepine derivative, mostly used for sedation. Available in many forms: intravenous or intramuscular injection, clysma, suspension and tablet. Usually the tablet (7.5 mg and 15mg) and the suspension (1mg/ml) form is used in practice. The dose ranges between 0.3-0.5 mg/bwkg. The onset is about 20-30 minutes after the administration. We recommend the use of the lower limit of the dosage. The adverse effect may be respiratory depression (contraindicated in patients with respiratory disease). Paradoxical effect occasionally occurs. It causes congrade and anterograde amnesia. The child should never be left unattended until the end of the duration of action. “As soon as the patient reaches the state of consciousness in which he arrived, he may be discharged from the surgery” This should take about 3-4 hours.

1.2.4. Antibiotic treatment

Conservative use of antibiotics is required to reduce the chance of bacterial resistance. The prescription has to be followed by the patient. Inadequate antibiotic therapy (underdose, short time administration) may also develop resistance. Oral wounds may be classified as clean, potentially contaminated, or contaminated. In the case of contaminated wounds antibiotic treatment is needed with the minimum of 5 days. (Usually 5-7 depending on the choice of drug) Antibiotic treatment is not indicated in dental infections limited to the pulp or periapical inflammations (pulpitis, apical periodontitis, sinus tract, or localized intraoral swelling) Acute facial swelling of dental origin usually requires antibiotic treatment. Sometimes referral to medical management and intravenous therapy is needed. Chronic periodontal disease may require long term antibiotic therapy especially if there is immunodeficiency in the background. Viral infections should never be treated with antibiotics. In some cases antibiotic prophylaxis may be required.

1.3. Developmental diseases and their treatment

Developmental disorders may be classified as dental developmental disorders and craniofacial anomalies (cleidocranial dysplasia, etc.). Craniofacial anomalies and usually lie on the basis of genetic disorders. For the treatment of these anomalies extensive teamwork is necessary.

**Table 2.8. Table 8. – Developmental disorders according to developmental stage**

<table>
<thead>
<tr>
<th>Developmental stage</th>
<th>Possible disorders</th>
</tr>
</thead>
</table>
| Formation of the dental lamina Induction and proliferation | changes in the number of teeth:  
oligodontia (Figure 9.)  
germinated or fused teeth, concrescence (Figure 10.)  
primordial cysts, and odontogenic tumors |
| Histodifferentiation | regional odontodysplasia |
| Morphodifferentiation | changes in size  
dens invaginatus (Figure 12.)  
dens evaginatus  
Carabelli cusp, talon cusp  
Hutchinson’s incisor and mulberry molars (congenital) |
## Developmental stage

<table>
<thead>
<tr>
<th>Possible disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>syphilis)</td>
</tr>
<tr>
<td>supernumerary roots</td>
</tr>
<tr>
<td>taurodontism</td>
</tr>
<tr>
<td>enamel pearls</td>
</tr>
<tr>
<td>dilacerations</td>
</tr>
<tr>
<td>peg shaped incisor (Figure 11.)</td>
</tr>
</tbody>
</table>

### Dentin developmental disorder

- dentinal dysplasia
- dentinal cysts
- Dentinogenesis imperfecta

### Enamel developmental disorder

- enamel hypoplasia
- fluorosis
- amelogenesis imperfecta (Figure 13.)
- Turner-tooth (Figure 14.)

### Eruption

- stage premature or delayed eruption
- natal and neonatal teeth
- ectopic eruption
- eruption cysts
- transposition (Figure 15.)
- impaction

---

**Figure 2.9. Figure 9. – Isolated oligodontia**
Figure 2.10. Figure 10. – Fusion of the teeth. Compared to geminated there is a change in the number of teeth (less)

Figure 2.11. Figure 11. – Peg shaped lateral incisor

Figure 2.12. Figure 12. – Dens invaginatus. Preoperative radiograph, with periapical lesion (left), after treatment (right)
2. Pediatric dentistry and orthodontics

1.4. Suggestions for therapy in particular dental developmental disorders

1. Changes in the number of teeth:

- Oligodontia hypodontia: orthodontic and prosthodontic treatment. The prosthodontic appliances should be in accordance the principles of pediatric prosthodontic care.

- Supernumerary tooth (e.g.: mesiodens): usually extraction or surgical removal is indicated.

2. Geminated, fused teeth, changes in size, supernumerary roots, Turner-tooth: depending on the shape of the tooth and the needs of the patient preventive (fissure sealing) or restorative treatment is needed, with special attention to further caries prevention.

3. Concrescence: the joining of to teeth (may be supernumerary) trough the cementum: preventive treatment, in some cases extraction may be needed.
4. Primordial cysts, odontogenic tumors: after the accurate diagnosis surgical and oncologic treatment is required.

5. Dens invaginatus, evaginatus, Carabelli cusp, talon cusp: after recognition immediate preventive treatment is needed (fissure sealing, fluoridation). They are more prone to caries due to the change in the anatomical shape, root canal treatment in the case of dens invaginatus is difficult and sometimes unsuccessful, therefore these teeth may require extraction.

6. Hutchinson’s incisor: Part of Hutchinson’s triad (keratitis parenchymatosa, cochlear nerve degeneration (deafness), dental disorders). Very rare these days. Basically infectological and dermatological treatment is necessary. The teeth may be restored.

7. Regional dentinal dysplasia, dentinogenesis imperfecta, amelogenesis imperfecta and enamel hypoplasia are histological disorders which are sometimes hard to diagnose. It is desirable to clarify the etiology; treatment plan is based upon the expected therapeutic advantages and risks. (restorations, preventive treatment, extraction, orthodontic and prosthodontic therapy).

8. Taurodontism: Root developmental disorder. Root canal treatment is very difficult in these cases. Preventive treatment and regular follow up is recommended.

9. Dilaceration: due to trauma during development, causing an abnormal angulation of the root. May lead to the impaction of the tooth. If erupted endodontic or orthodontic treatment is difficult. Sometimes surgical removal is indicated.

10. Enamel pearl: by the time discovered, it may cause periodontal problems. Extraction may be indicated.

11. Ectopic eruption, transposition, impaction: surgical or orthodontic treatment is necessary.

12. Fluorosis: depending on the severity micro abrasive or restorative treatment is needed.

13. Premature eruption: basically no intervention is needed, preventive care is necessary. Delayed eruption: may need orthodontic treatment.

14. Connatal or neonatal teeth: the premature eruption of the primary incisors. The root is not fully developed yet, which may lead to excessive mobility. In the case feeding problems the grinding of the edges or in the case of excessive mobility extraction is indicated.

15. Eruption cysts: harmless bluish swelling of the mucosa around the erupting teeth. Usually no treatment is needed, they disappears on their own. If there is bleeding, infected or causes an esthetic problem, excision of the top of the cyst is necessary.

References


AMERICAN ACADEMY ON PEDIATRIC DENTISTRY COUNCIL ON CLINICAL AFFAIRS: Guideline on appropriate use of antibiotic therapy for pediatric dental patients. Pediatr Dent. 2008 2009;30(7 Suppl):212-4


2.2. Cariologic lesions and consequent diseases in primary dentition – Balazs Sandor

2.1. Characteristics of primary dentition

Primary tooth caries and consequent diseases histologically represent that of the permanent dentition. The clinical differences are due to the anatomical (morphological) differences between the permanent and primary dentition.

Characteristics of deciduous teeth compared to permanent teeth:

- Smaller crown,
- Narrow occlusal surfaces,
- The dentin and enamel layer is thinner,
- Relatively wide pulp chamber,
- The pulp horns are closer to the surface,
- Relatively longer roots (compared to the crown-root ratio of the permanent teeth),
- The roots flare outwards from the cervical area in a greater extent (to accommodate the permanent successors),
- Root canals irregular, numerous accessory canals on the floor of the pulp chamber,
- The crown is wide and flat,
- Interdental papillae are short,
- The contact points are closer to the gingiva,
• There are generally lower mineral levels in deciduous teeth,
• Pulp chamber is relatively wide.

**Figure 2.15. Figure 1. – Deciduous dentition**

![Deciduous teeth](image)

The typical localization of caries in primary dentition may be:

• On the front teeth: smooth and interproximal surfaces.
• On molar teeth: occlusal and interproximal surfaces.

• Early childhood caries (ECC) which is: "the presence of one or more decayed (non-cavitated or cavitated lesions), missing (due to caries) or filled tooth surfaces in any primary tooth in a preschool-age child between birth and 71 months of age." (American Dental Association) Most frequently the maxillary incisors are affected. In severe cases the first molars are also affected. Other terms may describe the etiology, localization or progression of the disease (rampant caries, nursing caries, baby bottle caries, circular caries). In less severe cases it occurs before the age of six years, in severe cases before the age of three years.

**Figure 2.16. Figure 2. – Clinical appearance of circular caries (ECC)**

![Circular caries](image)
2.2. The pathological progression of caries

**Incipient caries** (white spot lesion) demineralization, the translucency of the enamel decreases causing white opaque white discoloration, non-cavitated. With preventive intervention it can be held in balance, the progression may be arrested.

![Figure 2.17. Clinical appearance of incipient caries](image)

**Superficial caries**: loss of enamel matrix, with dental probe a superficial roughness is observed, may exhibit yellowish discoloration, due to the collapse of the enamel prisms (cavitation) restorative treatment is needed.

![Figure 2.18. Clinical appearance of superficial caries (permanent tooth)](image)

**Moderate caries** (caries media): the dentin is involved maximally until the half of its width. In the area of the dentino-enamel junction (DEJ) the caries spreads laterally (this area of the tooth is the least resistant against caries) and towards the pulp, the enamel surface is collapsed, the bottom of the cavity is discolored (brownish), may cause transient clinical complaints.

Five histologic zones can be distinguished in dentin caries:

- Normal dentin: deepest part, no bacteria are present, normal tubular histology.
- Sub transparent dentin: demineralized intertubular dentin, with crystal formation in the tubules, no bacteria are present, the remineralization of this layer is possible.
- Transparent dentin: soft demineralized intertubular dentin, with no bacteria present.
- Turbid dentin: bacterial invasion, widened tubules filled with bacteria, must be removed during operative treatment.
- Infected dentin: the outer most region of the dentin caries, no minerals are present, only bacteria, collagen. No dentin structure can be identified.
2. Pediatric dentistry and orthodontics

**Figure 2.19. Figure 5. – Clinical appearance of moderate caries in primary tooth**

Deep caries (caries profunda): the lesion spreads deeper in the dentin towards the pulp with a thin intact dentin bridge. Usually causes clinical complaints.

**Figure 2.20. Figure 6. – Clinical appearance deep caries**

Complicated caries (penetrant caries): the lesion reaches the pulp, symptoms are dependent on the elapsed time, and the depth of penetration.

**Time course of the disease:**

Caries is a chronic disease, but its progression is dependent on many factors. Usually for an incipient caries to reach the cavitated irreversible stage 12-24 months are needed. The progression in primary dentition is faster, due to the wide dentin tubules the caries is wet dentinal caries is present with fast progression (caries humida).

**2.3. Treatment of primary tooth caries**

Our main goal is the prevention of childhood dental diseases. It is important to note that a well planned individual preventive program may totally (100%) prevent the occurrence of dental caries in childhood. Every preventive method should be taken in account, and regular control is necessary even on the healthy patients. In childhood a checkup and control examination is recommended every six months.

The lesions diagnosed during regular checkups are being treated, and additional preventive measures are essential. According to this approach, after effective restorative treatment, prevention of further caries is
necessary. After the teeth have been restored, depending on the caries activity of the patient 3-6 months regular control is recommended.

Before the treatment of the diagnosed lesions, treatment planning is necessary with the following factors, which should be considered:

- development and the status of the dentition and teeth,
- caries risk,
- oral hygiene,
- compliance (mainly dependant on the parents),
- cooperation.

2.3.1. Incipient caries

Macroscopic enamel lesion cannot be detected, preventive oral hygienic and dietary advice have to be given to the patient (accompanying parent/caregiver). The lesion can be remineralized with local fluoridation. In the case of proximal and smooth surface non-cavitated enamel caries of primary teeth, resin infiltration can stop the progression.

2.3.2. Superficial and moderate caries

Depending on the localization, size of the lesion, the age the complaints and the cooperation of the child restorative therapy is needed. Stainless steel crown is indicated in the case of bigger lesions (3 or more surfaces involved), in patient with high caries risk or when the child is treated in general anesthesia. Superficial interproximal lesions of the primary incisors in uncooperative children may be treated with interproximal stripping (opening a gap between the teeth) and impregnation. Basically this is a minimal intervention, to allow the saliva to remineralize the surface. It is not recommended in the molar region (space maintenance). It is a basic rule to use minimally invasive treatments, only to remove the infected tissue, and to choose fast setting reliable filling material. Local anesthesia and pain relieving is usually needed.

2.3.2.1. Recommended materials

2.3.2.1.1. Glassionomer cements (GIC)

During their setting reaction they are less sensitive to moisture compared to resins, they release fluoride continuously, chemically binds to the tooth, their thermal expansion is similar that of the tooth, can be etched and polished. Wide range is available. conventional glass ionomers have decreased mechanical resistance against wear, while resin modified glass ionomers (RM-GIC) with improved wear resistance is ideal for the fillings of primary tooth. They are less esthetic than composite filling materials.

2.3.2.1.2. Composites

Esthetic, with good mechanical characteristics, can be well polished, more technique sensitive (moisture sensitive), and to prevent the possible pulp irritation caused by the unreacted monomers, underlining of the deep lesion with GIC is indicated. The use of composite is not recommended for uncooperative children, where the isolation is compromised.

2.3.2.1.3. Compomers

Basically the features glass ionomers and composites are combined, as it is a mixture of the two. It can be well used in pediatric dentistry.

2.3.3. Profound caries regarding its localization

Our treatment should always be minimally invasive, but the total infected dentin has to be removed. Dentin preparation must be carried out with water cooling when using rotary instruments. For the removal of carious dentin low speed handpiece is recommended with stainless steel bur. Hand instruments (sharp excavators) may be used as well. If the proximal marginal ridges are already involved by the interproximal caries, or on the
radiograph the proximal caries has already reached more than half of the dentin, pulpotomy is the choice of treatment, direct pulp capping is not recommended. Regarding the localization of the caries the following treatments can be carried out:

1. **Filling with plastic material (composite, GIC, RM-GIC, compomer).** See the picture below.

**Figure 2.21. Figure 7. – Clinical picture of glass ionomer filling (permanent molar)**

2. **ART:** atraumatic restorative technique: is an interim restoration until definitive treatment cannot be achieved due to the lack of cooperation. It is indicated in teeth without the history of pain. Some of the carious dentin is removed without local anesthesia, and the cavity is restored with GIC until the definitive treatment.

3. **IPC:** indirect pulp capping: the use in primary teeth is doubtful, can only be used in teeth without symptoms or with reversible pulpitis, in children with low caries risk, and if the child has no pulpal disease in the dental history. Used in the case of deep cavities. In the case of IPC a material with good coronal seal is necessary with glassionomer lining.

4. **Prefabricated stainless steel crowns (SSC)** are indicated in primary molars. Esthetic versions are available in the market for the front region (prefabricated polycarbonate, porcelain fused to metal, zirconium ceramic and acrylic crowns).

Preparation:

- 1-1,5 mm occlusal reduction.

- Minimal preparation on the interproximal walls, without undercuts, the contact surfaces should be removed.

- The crown should follow the gingival margin.

Hall technique is being used recently. Hall technique is a restorative technique, where the tooth is restored with a SSC, without any or minimal preparation after the separation of the teeth. If the caries proceeding into the dentin should be removed.

**Figure 2.22. Figure 8. – Prefabricated zirconium ceramic crowns**
5. Strip crowns are transparent templates for composite restorations in the front region. The strip crown is filled with composite material and positioned on the prepared tooth. The tooth is prepared as normally it would be for a composite filling and a chamfer margin crown. After positioning the crown it is polymerized. The strip crown is removed. Occlusal adjustment and polishing are the final steps.

2.3.4. Complicated caries

In the case of deep/complicated caries with clinical and/or radiographic signs, endodontic therapy is indicated in accordance with the pulpal conditions (see later). Direct pulp capping is contraindicated in milk teeth, due to its low success rate.

2.4. ECC (Early Childhood Caries)

This caries type requires special attention, due to the followings:

• in severe cases it affects young (under the age of 3 years) patients,
• treatment is difficult,
• it is caused by inappropriate diet, and bad oral hygiene, thus can be prevented,
• dietary instructions must be given to the parent immediately,
• characterized by fast progression,
• urgent treatment should be carried out.

2.4.1. First steps of treatment

The parents should always be informed, and oral hygiene habits have to be changed:

• Information has to be given about the characteristics of this caries type, and the possible prevention.
• Information has to be given about the bad habits responsible for the formation of this disease.
• The parent should be informed about their duties.
• Consultation may be needed with the child’s family doctor.
• Age specific healthy diet in needed.
• Snacking should be avoided.
• Between meals water should be consumed.
• Appropriate oral hygiene methods should be used.

2.4.2. Mild lesions effecting small surfaces

In addition to the foregoing professional fluoridation (solutions, varnishes, gels) and regular control is indicated every 1-3 months.

2.4.3. Advanced ECC

The lesion spreads on the surfaces. Conventional cavity preparation (box form) cannot be carried out, due to the small size of the tooth and the lesion. Because of the irregular spreading of the caries the rate of surface/marginal sealing is disadvantageous, therefore according to the localization of the teeth the use of prefabricated esthetic crowns (incisors) and prefabricated SCC (molars) is indicated.

2.5. Consequent diseases of primary tooth caries

2.5.1. Consequent diseases of the pulp: inflammation of the pulp (pulpitis)
1. Acute reversible pulpitis (earlier termed as pulp hyperemia): usually caused by profound caries, no spontaneous pain is present, but sensitive to cold and hot is specific. There are no radiographic signs. Treatment depends on the extent of the carious lesion. If the pulp is exposed during preparation preventive pulpotomy should be carried out, since direct pulp capping has a bad prognosis in primary dentition (high undifferentiated mesenchymal cell content → osteoclast differentiation → internal resorption or pulp becomes necrotic). IPC may only be used if the the pulp is not exposed after caries removal.

2. Acute irreversible pulpitis: may be defined as total or partial. Sooner or later (in primary dentition sooner) untreated reversible pulpitis becomes irreversible. It causes spontaneous severe pain, which may intensify in lying position due to congestion. There are no radiographic signs. There is intensive prolonged pain to cold and hot, requiring pain relief. Depending on the condition of the pulp pulpectomy is indicated in the case of total irreversible pulpitis, and pulpotomy (therapeutic, not preventive) is indicated in partial irreversible pulpitis.

3. Chronic hyperplastic pulpitis (pulp polyp): very specific in primary teeth but sometimes it may also appear in young permanent teeth. This is the proliferation of the pulp tissues into the carious cavity. The top of the pulp is covered by oral epithelium. Usually causes no complain. There are no radiographic signs. Treatment choices may be: vital pulpotomy, or if the pulp is exposed to the oral cavity extraction and space maintenance is necessary (Figure 9.).

4. Pulp necrosis: usually causes no pain. There are no radiographic signs. In primary dentition partial necrosis of the pulp occurs very frequently, even in the case of developed periradicular lesions we may find inflamed but vital pulp sections in the pulp chamber. It is essential to treat the teeth only after local anesthesia, even if our clinical diagnosis assumes the necrosis of the pulp. The treatment is usually pulpectomy.

**2.5.2. Consequent diseases of the periradicular region (complicated gangrena)**

As earlier described in multi rooted teeth primary teeth the inflammation usually spreads interradicularly, due to the high number of accessory canals orientated from the pulp chamber. The periradicular inflammations of the primary teeth may affect the developing permanent successor, causing enamel hypoplasia (Turner tooth), to prevent this situation extraction and space maintaining must be considered.

1. Acute periradicular periodontitis can occur after pulp necrosis, or unsucssfull endodontic treatment. It is important to note that irreversible inflammation of the pulp may also spread to the periradicular tissues without necrosis. There is intensive pain to percussion and to biting. Radiographic signs only develop later (preiradicular radiolucency, widened periodontal space). Treatment: pulpectomy.


3. Acute and chronic periradicular submucous abscess is a collection of pus in a well defined area which only occurs after the necrosis of the pulp (or partial necrosis!). Sign are usually very conspicuous:
• Teeth may become mobile (not obligatory).

• Spontaneous pain which intensifies pain to percussion and biting.

• Abscess is visible next to the tooth on the gingiva.

• The drainage of the pus through a sinus tract (fistula) leads to the chronic form of the abscess.

• Usually symptoms resolve as long as the sinus tract is open.

• It is not accompanied by general symptoms, although abnormal regional lymph nodes may be present.

Treatment: the pus may be drained through the tooth, by opening the pulp chambers, with the disappearance of the swelling.

4. Periostitis is the inflammation of the periosteum: the spreading of the periradicular inflammation in the cancellous bone (osteomyelitis) will reach and penetrates through the cortical layer of the bone causing periostitis. If abscess is formed it is termed as subperiostal abscess. It is characterized by the swelling of the face according to the localization of the tooth, accompanied by general symptoms.

Treatment: usually extraction of the tooth (danger of Turner tooth!), but initially the elimination or reduction of the inflammation is necessary by the drainage of the pus (if present), warm-moist compress, antibiotic and analgesic medication. In the case of risk for phlegmone, hospitalization is indicated with intravenous antibiotic treatment.

2.6. Endodontic treatment in primary dentition

Careful consideration is needed:

• Accurate diagnosis is hard

• Extraction or endodontic treatment

• Prolonged treatment (small child).

2.6.1. When is endodontic treatment needed

• Pulpexposure during dental treatment

• trauma (fracture)

• due to inflammation.

2.6.2. Extraction or endodontic treatment

• In young age when the time of eruption of the permanent successor is far, mainatnance of arch length is important (space maintanace), endodontic treatment may be the choice

• If extraction is the choice, space maintenance is necessary, but balanced extraction may be considered, which is the extraction of the contralateral tooth to maintain the symmetry.

• Endodontic therapy is needed where extraction should be avoided:

• Bleeding disorders

• Already crowded arch

• Lack of permanent successor.

2.6.3. Indications and contraindications
Indications and contraindications can be seen in Table 1.

### Table 2.9. Table 1. – Indications and contraindications of primary tooth endodontic treatment

<table>
<thead>
<tr>
<th>Indications</th>
<th>Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulp necrosis</td>
<td>Systematic disease</td>
</tr>
<tr>
<td>Symptoms of pulpitis</td>
<td>If the patient wants extraction</td>
</tr>
<tr>
<td>Clinical or radiographic assessment suggest pulp treatment</td>
<td>Uncooperative child</td>
</tr>
<tr>
<td></td>
<td>Unrestorable tooth</td>
</tr>
<tr>
<td></td>
<td>Bad oral hygiene</td>
</tr>
<tr>
<td></td>
<td>Floor of pulp chamber perforated</td>
</tr>
<tr>
<td></td>
<td>Advanced root resorption (more than 1/3)</td>
</tr>
<tr>
<td>Close to exfoliation</td>
<td></td>
</tr>
</tbody>
</table>

#### 2.6.4. Treatment options

- Indirect pulp capping,
- Direct pulp capping,
- Pulpotomy,
- Pulpectomy.

1. **Indirect pulp capping (IPC):** described earlier.

2. **Direct pulp capping:** bad prognosis, described earlier.

3. **Pulpotomy:**

   3.1. **Vital pulpotomy:** the aim is to keep the radicular pulp vital with the removal of the inflamed coronal pulp tissue. It is indicated in the case of reversible pulpitis (preventive pulpotomy), partial irreversible pulpitis (therapeutic pulpotomy) and carious exposure of the pulp and in cases described earlier.

   **Contraindicated:**
   - fistula or swelling, total irreversible pulpitis,
   - the tooth crown is non-restorable,
   - absent hemorrhage; profuse hemorrhage (more than 5 minutes),
   - marked tenderness to percussion,
   - mobility,
   - radiolucency exists in the furcal or periradicular areas,
   - spontaneous pain, especially at night,
   - necrotic pulp,
   - dystrophic calcification (pulp stones),
   - perforation of the pulp chamber.
Method of vital pulpotomy (Figure 10-17)

- Local anesthesia,
- Removing caries,
- Opening, and removing the total top of the pulp chamber,
- Coronal pulp tissue is removed using a large sterile, sharp excavator or sterile steel round bur,
- Haemostasis with sterile cotton wool moistened with saline, it is important to make sure, there is no blood clot in the pulp chamber as it will induce necrosis,
- Treatment of the remaining pulp (dressing material or tissue training, Table 2.),
- Zinc-oxide eugenol cement (may be mixed with formaldehyde),
- GIC covering and SSC is preferred.

**Figure 2.24.** Figure 10. – Clinical appearance of profound caries on lower right second primary molar, prior to vital pulpotomy, in rubber dam isolation

**Figure 2.25.** Figure 11. – The tooth after caries removal, and the opened pulp chamber

**Figure 2.26.** Figure 12. – The removal of the coronal pulp tissue with a sterile bur
Figure 2.27. Figure 13. – The coronal pulp tissue removed, haemostasis achieved with sterile cotton wool moistened with saline

Figure 2.28. Figure 14. – Application of zinc-oxide eugenol (ZOE)
Figure 2.29. Figure 15. – ZOE cement covered with glassionomer cement

Figure 2.30. Figure 16. – Metal matrix strip and wooden wedge positioned for the final clinical restoration
2. Pediatric dentistry and orthodontics

Figure 2.31. Figure 17. – The final coronal restoration with composite resin

Table 2.10. Table 2. – Materials used for vital pulpotomy

<table>
<thead>
<tr>
<th>Material</th>
<th>Success rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Formocresol</td>
<td>90–98%</td>
</tr>
<tr>
<td>MTA (mineral trioxide aggregate)</td>
<td>~100%</td>
</tr>
<tr>
<td>Calcium-hydroxide</td>
<td>60% (internal resorption)</td>
</tr>
<tr>
<td>ZOE paste with formaldehyde (N2)</td>
<td>Unknown</td>
</tr>
<tr>
<td>ZOE paste without formaldehyde</td>
<td>Unknown</td>
</tr>
<tr>
<td>Laser therapy/electro surgery</td>
<td>Unknown (less than ferric sulfate)</td>
</tr>
<tr>
<td>Ferric sulfate</td>
<td>92.7%</td>
</tr>
<tr>
<td>Glutaraldehyde</td>
<td>Unknown</td>
</tr>
</tbody>
</table>

3.2. Devitalization pulpotomy: two stage procedure, when anesthesia cannot be obtained to permit the extirpation of the pulp. In the first step paraformaldehyde is applied on a thin dentin layer covering the vital pulp to devitalize the pulp in the pulp chamber and leave the radicular pulp vital. After 1-2 weeks coronal pulp can be removed. The success rate is low, because the pulp in the root canal may also become devitalized. This technique is not recommended these days.

3.3. Non-vital pulpotomy is used where pulpectomy is indicated due to irreversible change in the radicular pulp, but is impractical because of the root morphology or lack of cooperation. In the first appointment the coronal pulp is removed and calcium-hydroxide or Ledermix paste is applied on root pulp, and the crown is closed. 1-2 weeks later if symptoms have resolved the tooth may be restored. If the symptoms do not resolve the previous procedure may be repeated, or extraction may be indicated.

4. Pulpectomy

Indications:

• necrotic pulp,
• total irreversible pulpitis,
• periradicular inflammation.

The removal of pulp tissue is followed by filling the root canals with a resorbable cement. The objectives of our treatment are:
• Maintain the tooth free of infection,

• Promote physiologic root resorption,

• Hold the space for the erupting permanent tooth,

• Obtrurate the root canals.

Method of treatment:

• Biomechanically cleansing and disinfection of the root canals.

Pulpectomy is contraindicated:

• Teeth with non-restorable crowns.

• Periradicular involvement extending to the permanent tooth bud.

• Pathologic resorption of at least one-third of the root with a fistulous sinus tract.

• Excessive internal resorption.

• Extensive pulp floor opening into the bifurcation.

• Systemic illness such as congenital or rheumatic heart disease, hepatitis, leukemia, and children on long-term corticosteroid therapy, or those who are immunocompromised.

• Primary teeth with underlying follicular cysts.

Pulpotomy techniques:

• One stage technique,

• Two stage technique.

4.1. One stage technique

• Anesthesia,

• Rubber dam isolation (preferred),

• Finding the root canals and measuring the length,

• Working length estimated from preoperative radiograph,

• For the assessment of the working length, EAL may be used, as shown in studies. Accuracy is not affected by root resorption (accuracy: 96.7%),

• Disinfection of the canals (sodium-hypochlorite),

• Drying the canals with paper point,

• Then root canals can be filled resorbable root canal filling material with the use of rotary paste filer (zinc-oxide eugenol paste, non-setting calcium hydroxide paste, calcium hydroxide polymeric root canal sealant-Sybronendo: Sealapex).

One stage technique is recommended in symptomless necrosis and there is no radiologic sign of inflammation. In all other cases two stage technique is recommended.

4.2. Two stage technique

The task in the first appointment is to disinfect the canals, and to reduce inflammation by applying calcium hydroxide or Ledermix paste in the root. If the symptoms resolve 1-2 weeks later, root canal filling can be carried out the same way as described in the one-stage technique.
References

AMERICAN ACADEMY OF PEDIATRIC DENTISTRY CLINICAL AFFAIRS COMMITTEE--PULP THERAPY SUBCOMMITTEE; AMERICAN ACADEMY OF PEDIATRIC DENTISTRY COUNCIL ON CLINICAL AFFAIRS: Guideline on pulp therapy for primary and young permanent teeth; Pediatr Dent. 2005-2006;27(7 Suppl):130-4.

AMERICAN DENTAL ASSOCIATION: Statement on Early Childhood Caries (www.ada.org)


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3. 2.3. Traumatic lesions in primary dentition – Balazs Sandor

3.1. Epidemiology and etiology of dental traumas

During the first 10 years of life oral trauma in children is very frequent. The incidence is decreasing gradually with age. Five percent of all injuries are localized to the oral region. In preschool children 17% of all injuries are oral injuries. Later in life the hands and feet are injured. Thirty percent of the children suffer injuries to their primary dentition, and 22% to their permanent dentition. The boys are twice as much affected than the girls are. Most commonly the upper central incisors are injured.

Diagnosis and treatment for traumatic dental injuries of primary and permanent teeth are challenging due to the diversity of the trauma (six types of luxation and nine types of fracture). The injury types may be combined leading more than a hundred variations. Fractures dominate in the permanent dentition and luxations are the most common injury types in primary dentition. This difference is due to the physiologic resorption or the root of the primary tooth from the age of three years, and the structure of the bones (thinner and more elastic). Every trauma case may have a different treatment sequence and prognosis. A web-based knowledge center, the www.dentaltraumaguide.org is helpful in establishing an evidence-based diagnosis and a treatment plan.

Injuries to the deciduous dentition are the most frequent between the age of 1 and 3 years. From the first year of age the child learns to walk (toddler), later to run, climb and play, with a limited control of their movement. In permanent dentition there is a peak in the incidence between the age of 7 and 11 years which is due to falls, bicycles, collisions and sports. Prevalence of the injuries is the number of cases in a given population at any time. Incidence of the injury is the increase of cases over a given period of time (Figure 1;2).

Figure 2.32. Figure 1. – The incidence and prevalence of dental injuries in deciduous dentition (Origin: Andreasen JO: Traumatic injuries to the teeth (2nd edition). Redrawn form: Roberts G, Longhurst P: Oral and Dental Trauma in Children and Adolescents
The trauma affecting the tooth may be classified as direct or indirect:

- **Direct trauma:** the tooth itself is hit by an object. The front region is affected.

- **Indirect trauma** is usually due to forceful contact with the opposing arch. Mainly the premolars and molars are affected. It may be accompanied by the fracture of the lower jaw.

Factors influencing the type of trauma:

- energy impact,

- resilience of impacting object,

- shape of impacting object,

- direction of force.
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Blunt object may cause greater damage to the soft tissues, and the supporting structures, high-velocity, sharp object usually cause fractures and luxations of the teeth.

Predisposing factors to dental injuries may be:

- Increased overjet,
- protrusion of upper incisors,
- insufficient lip closure are.

During examination and treatment the possibility of child abuse should never be underestimated. What constitutes abuse is dependent on the culture of the child, the country, or maybe even a subculture of a city. The diagnosis is very challenging intellectual and emotional exercise. The failure to diagnose a child abuse, may have severe influence on the child’s future life. Child abuse may be categorized into four types:

- physical abuse,
- sexual abuse,
- emotional abuse,
- neglect.

Since the orofacial region is the most frequently traumatized area of the body during child abuse, the dentist, pediatrician may be the first one to meet the child. The signs of physical abuse may be:

- injuries, not matching the history,
- bruising and injuries of the soft tissues in hidden areas of the body, not overlying bony prominences (armpit, inner surface of thigh),
- multiple injuries of different stage,
- injuries with the shape of a recognizable object,
- bite marks.

3.2. The classification of dental injuries (Table 1.)

World health Organization (WHO) classification: comprehensive system. Jaw fractures are not involved in the classification of oral injuries, they are classified separately as fracture of face bones

- Andreasen’s classification: separated into 19 groups including injuries to the teeth, the supporting structures, gingiva and oral mucosa. It is a modification of the WHO classification.
- Garcia-Godoy’s classification: also a modification of the WHO system. It separates tooth fractures upon the involvement of the cementum.
- Ellis classification: also the modification of the WHO system used widely in studies.

Table 2.11. Table 1. – Most frequently used classifications (Modified after Bastone et al. 2000)

<table>
<thead>
<tr>
<th>Andreasen</th>
<th>World Health Organization</th>
<th>Health Organization</th>
<th>Garcia-Godoy</th>
<th>Ellis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crown infraction</td>
<td>Fracture of enamel tooth</td>
<td>Enamel crack</td>
<td>Class I: enamel fracture</td>
<td></td>
</tr>
<tr>
<td>Uncomplicated crown fracture</td>
<td>Fracture of crown without pulp involvement</td>
<td>Enamel fracture</td>
<td>Class II: enamel-dentin fracture without pulp involvement</td>
<td></td>
</tr>
</tbody>
</table>
2. Pediatric dentistry and orthodontics

<table>
<thead>
<tr>
<th>Andreasen</th>
<th>World Organization</th>
<th>Health Organization</th>
<th>Garcia-Godoy</th>
<th>Ellis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complicated crown fracture</td>
<td>Fracture of crown with pulpal involvement</td>
<td>Enamel-dentin fracture with pulp exposure</td>
<td>Class III: enamel-dentin fracture with pulp involvement</td>
<td></td>
</tr>
<tr>
<td>Uncomplicated crown-root fracture</td>
<td>Fracture of root of tooth</td>
<td>Enamel-dentin-cementum fracture with pulp exposure</td>
<td>Class IV: traumatized tooth that becomes non-vital, with or without loss of crown structure</td>
<td></td>
</tr>
<tr>
<td>Complicated crown-root fracture</td>
<td>Fracture of crown and root of tooth</td>
<td>Enamel-dentin-cementum fracture with pulp exposure</td>
<td>Class V: Luxation</td>
<td></td>
</tr>
<tr>
<td>Root fracture</td>
<td>Luxation of tooth</td>
<td>Enamel-dentin-cementum fracture with pulp exposure</td>
<td>Class VI: Avulsion</td>
<td></td>
</tr>
<tr>
<td>Concussion</td>
<td>Intrusion or extrusion of tooth</td>
<td>Root fracture</td>
<td>Class VII: Displacement of tooth, without fracture of crown or root</td>
<td></td>
</tr>
<tr>
<td>Subluxation</td>
<td>Avulsion of tooth</td>
<td>Concussion</td>
<td>Class VIII: Fracture of the crown en masse and its replacement</td>
<td></td>
</tr>
<tr>
<td>Intrusive luxation</td>
<td>Other injuries including laceration of oral soft tissues</td>
<td>Luxation</td>
<td>Class IX: Fracture of deciduous teeth</td>
<td></td>
</tr>
<tr>
<td>Extrusive luxation</td>
<td></td>
<td>Lateral displacement</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lateral luxation</td>
<td></td>
<td>Intrusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avulsion/Complete luxation</td>
<td></td>
<td>Extrusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Comminution of the alveolar socket</td>
<td></td>
<td>Avulsion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fracture of the facial or lingual alveolar socket wall</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fracture of the alveolar process with and without the involvement of the socket</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fractures of the mandible or maxilla with and without the involvement of the socket</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Laceration of the gingival or oral mucosa</td>
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<tr>
<td>Contusion of the gingival or oral mucosa</td>
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<td></td>
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</tr>
<tr>
<td>Abrasion of the gingival or oral mucosa</td>
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<td></td>
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</tbody>
</table>

3.3. Injuries to the deciduous teeth

Trauma of the deciduous incisors may conclude in developmental anomalies of the permanent dentition, depending on the age, when the injury occurred and the type of trauma that occurred. In the case of primary tooth avulsion and intrusion the chances for subsequent permanent tooth anomaly is higher compared to the
other types of luxation injuries. The rate of developmental disorders in permanent dentition is higher when the trauma to the deciduous tooth happens under the age of four.

### 3.4. Patient examination

Dental trauma is one of the emergency cases in pediatric dentistry demanding urgent treatment, to relieve pain, and to improve the prognosis of our treatment.

Steps of examination:

- History,
- Clinical examination:
  - Extraoral examination,
  - Intraoral examination,
- Radiographic examination.

To follow steps of examination is essential for a well established treatment plan. It gives us important information about the status of the dentition at presentation, prognosis of the injuries, other injuries sustained, medical complications or possible litigation. If it is possible photographs should be included in the documentation. The sequence of the steps of examination may be altered in cases where there is an excessive bleeding, sign of life threatening injuries, or the postponed treatment of the trauma may jeopardize the prognosis of the tooth (avulsion-repositioning).

a., History

To take a thorough history of the patient and the trauma pre-printed charts may be useful. The answers given by the patient, parent or caregiver should be documented. If there is great discrepancy between the history of the trauma gathered from the patient and the parents/caregivers the possibility of abuse should be evaluated.

The patient history should include the general medical history of the patient (medications, allergy, immunization, systemic diseases) and the trauma itself. Immunization is important regarding tetanus vaccination.

Questions to ask about the history of the trauma:

- When did the trauma occur?
- Where did the trauma occur?
- How did the injury take place?
- Has there been any initial treatment done elsewhere?
- Have there been any other dental injuries in the past?
- Have all the tooth fragments or avulsed teeth been accounted for?
- Questions regarding the signs of closed head injury.

When did the trauma occur?

The time elapsed between the trauma and the initial treatment is important in some special cases, especially in permanent dentition, where repositioning of the avulsed or intruded tooth needs to be done.

Where did the trauma occur?

Gives us important information about the site where the injury has happened. In some cases tetanus prophylaxis may be needed.
How did the injury take place?

It is important to know who was there during the accident. This way we may get information about the way the injury happened (accident, epileptic seizure, assault).

Has there been any initial treatment done elsewhere?

Previous treatment may influence our diagnosis. Analgesic medication may alter the responses to some of the physical examinations (percussion, vitality check), or if restoration has already been done, the exact extent of the injury may not be perfectly determined (pulp involvement).

Have there been any other dental injuries in the past?

Previous trauma to the teeth may explain radiographic or clinical findings which are not consistent with the present trauma.

Have all the tooth fragments or avulsed teeth been accounted for?

If the fragments have not been found at the site of the injury, the possibility of aspiration must be taken into account, or radiographs should be taken of lip or cheek lacerations to search for the tooth fragments.

Questions regarding the signs of closed head injury

Closed-head injuries are the leading cause of death in children under 4 years old. Up to 50% of all accidents involve the head. If symptoms of a head injury are present, immediate medical care is necessary. Without treatment, it may lead to further brain damage, disability, or death. The symptoms may be overlooked by parents/caregivers after a dental trauma, especially when there is excessive bleeding after a soft tissue injury, which leads the patient to the dental office first. The signs of closed head injury are listed in Table 2.

Table 2.12. Table 2. – Symptoms of traumatic brain injury

<table>
<thead>
<tr>
<th>Mild traumatic brain injury symptoms</th>
<th>Moderate and severe brain injury symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immediate symptoms</td>
<td>Delayed or secondary symptoms</td>
</tr>
<tr>
<td>Confusion</td>
<td>Mood swings</td>
</tr>
<tr>
<td>Temporary memory loss</td>
<td>Headaches or migraines</td>
</tr>
<tr>
<td>Tinnitus</td>
<td>Insomnia</td>
</tr>
<tr>
<td>Nausea</td>
<td>Memory problems</td>
</tr>
<tr>
<td>Slurred speech</td>
<td>Change in sense of smell or taste</td>
</tr>
<tr>
<td>Headache</td>
<td>Inability to concentrate</td>
</tr>
<tr>
<td>Loss of consciousness for less than 30 minutes</td>
<td>Fatigue</td>
</tr>
<tr>
<td>Drowsiness</td>
<td>Sensitivity to light and sounds</td>
</tr>
<tr>
<td>Dilated pupils or anisocoria</td>
<td>Trouble with balance</td>
</tr>
</tbody>
</table>

b.; Clinical examination

Extraoral examination
As described earlier the dentist may be the first doctor to see the child after an accident, which imposes a great responsibility to the personnel. Before examining the oral region, other serious injuries of the body should be excluded. This also includes the search for signs of closed head injury.

In the oral region bruises, abrasions, cuts should be noted. The borders of the maxilla and the mandible, the temporo-mandibular joint should be palpated, to exclude fracture. Swellings on the face may make the palpation difficult and painful for the patient.

Intraoral examination

Before we carry out the intraoral examination, the face should be washed with a detergent, and mouth should be rinsed out. First the oral soft tissues, the occlusion of the patient and the color of the traumatized tooth are being examined. Any changes due to the trauma should be noted. The specific examination of the traumatized teeth is described separately at the types of the injury.

Vitality testing: after trauma the teeth may fail to respond to vitality testing, and because the child may be distressed after the accident reliable interpretation to pulp vitality testing cannot be obtained. In every day practice usually we use cold test as thermal stimuli.

Percussion: test should be carried out with vertical and horizontal percussion. Before testing the traumatized tooth, percussion should be carried out on an intact adjacent tooth, to make the patient realize the difference between the two teeth.

Mobility testing: is carried out horizontally and vertically with one of our fingers gently supporting the traumatized tooth on the labial side, and the handle of our mirror or dental probe on the lingual side.

In primary dentition reliable answers may not always be obtained, due to the age of the child. When we are testing the traumatized tooth of a small child, we should never forget that these procedures may cause severe pain, therefore, losing the ability of the child to cooperate.

c.: Radiographic examination

Radiographic examination is essential to determine extent of the injury to the supporting tissues, the stage of root development, and the relation to the permanent successors. Always consider minimizing the risk of radiation to the child. After the clinical examination the area of the injury is already determined. If clinical examination cannot fully exclude the chance of bone fracture, panoramic radiograph should be made. The most frequently traumatized teeth are the upper incisors, which cannot be safely examined on panoramic radiographs, so usually intraoral periapical or occlusal radiographs are needed. If there is penetrating lip lesion, soft tissue radiographs should be taken by placing the dental film/digital dental sensor between the lips and the dental arch, in order to locate foreign object. Foreign object are usually impossible to palpate, due to the physiologic reactions of the perioral muscles.

3.5. Treatment of traumatic lesions in primary dentition according to the diagnosis

The diagnosis and treatment of the child with primary tooth injury is always challenging, due to the lack of cooperation and fear. Some cases may only be solved in general anesthesia or in conscious sedation.

Several factors may affect our treatment plan:

- Age and maturation of the child, dental developmental stage.
- Type and extent of injury.
- Cooperation of the child.
- Social background of the child.
- The relation between the traumatized primary tooth and the underlying permanent successor germ.

Possible consequences of primary tooth trauma (mainly avulsion and intrusion) or alveolar fractures are to the permanent teeth (Figure 3.).
• Tooth malformation,
• impacted teeth,
• eruption disturbances,
• White or yellow-brown discoloration of crown,
• hypoplasia of permanent incisors.

**Figure 2.34. Figure 3. – The late consequences of primary tooth trauma. Impaction of the upper permanent incisor**

Because of the potential sequel, our treatment plan should aim to minimize further damage to the permanent dentition.
Splinting of the teeth in primary dentition is only indicated in the case of alveolar fracture or intra-alveolar root fracture (further described in II/5: Traumatic cases in young permanent dentition.).

So far there is no evidence for the use of antibiotics after luxation injuries. It is the choice of the clinician. At the pediatric dentistry division in the department of Dentistry, Oral and Maxillofacial Surgery, University of Pécs, we recommend the use of antibiotics in the case of intrusive luxations and where there is extensive soft tissue injury or significant surgical intervention.

After luxation injuries the dark (grey) discoloration of the traumatized tooth is a frequent late complication. Usually it is asymptomatic clinically. The parents complain about the esthetic disadvantage. The late discoloration of the tooth is usually associated with the necrosis of the pulp. No treatment is needed, unless there are signs of infection.

**Figure 2.35. Figure 4.** „Trauma pathfinder” obtained and redrawn after http://dentaltraumaguide.org/Trauma_Pathfinder.aspx

a. **Enamel infraction of primary teeth**
   
   Definition: incomplete fracture of the enamel, with a visible fracture line on the tooth surface
   
   Percussion: not tender. If the tooth is tender to percussion luxation injury or root fracture may be in the background.
   
   Vitality test: not reliable
   
   Mobility test: normal
   
   Radiographic examination is usually unnecessary, unless there is tenderness to percussion
   
   Treatment: no treatment is needed. If the child is cooperative enough, etching and sealing with an adhesive bond resin may prevent the discoloration of the crack.

b. **Enamel fracture** (uncomplicated crown fracture limited to the enamel)
   
   Definition: uncomplicated fracture of the crown that only involves the enamel
   
   Percussion: not tender. If the tooth is tender to percussion luxation injury or root fracture may be in the background.
   
   Vitality test: not reliable
   
   Mobility test: normal
   
   Radiographic examination is usually unnecessary, unless there is tenderness to percussion. If the patient has penetrating lip lesion soft tissue radiograph should be done to exclude the penetration of the fragment.
   
   Treatment: smooth the sharp edges with a fine diamond bur or with and abrasive metal strip, fluoridating the fracture surface. 3-4 weeks later clinical and radiographic control is recommended.
c. **Enamel-dentin fracture** (uncomplicated crown fracture with enamel and dentin involvement)

Definition: fracture of the crown that only involves the enamel and the dentin, but not the pulp

Percussion: not tender. If the tooth is tender to percussion luxation injury or root fracture may be in the background.

Vitality test: not reliable

Mobility test: normal

Radiographic examination: The distance between the fracture surface and the pulp may be evaluated on periapical radiographs. If the patient has penetrating lip lesion soft tissue radiograph should be done to exclude the penetration of the fragment.

Treatment: Lining with glass ionomer cement and resin (composite or compomer) restoration. 3–4 weeks later clinical and radiographic control is recommended.

d. **Complicated crown fracture**

Definition: fracture of the crown that only involves enamel and the dentin exposing the pulp

Percussion: not tender. If the tooth is tender to percussion luxation injury or root fracture may be in the background.

Vitality test: not reliable

Mobility test: normal

Radiographic examination: If the patient has penetrating lip lesion soft tissue radiograph should be done to exclude the penetration of the fragment.

Treatment: to keep the vitality of the tooth pulotomy or partial (Čvek) pulpotomy is indicated with, zinc-oxide eugenol, or mineral trioxide aggregate (MTA) or calcium-hydroxide (less) dressing. Depending on the cooperation of the child extraction may also be considered Clinical and radiographic control is necessary after 1 week, 6-8 weeks and 1 year. If there are signs of periodontal inflammation the extraction or root canal treatment is favorable.

e. **Uncomplicated crown-root fracture**

Definition: fracture involving enamel, dentin and cementum but not involving the pulp. The fracture line is extending below the gingival margin. The crown is split into two or more parts

Percussion: tender to percussion due to the subgingival fracture

Vitality test: not reliable

Mobility test: coronal fragment is mobile

Radiographic examination: the fracture line in not visible. Occlusal radiograph is recommended.

Treatment: If the involvement of the root is small, after the removal of the coronal fragment the tooth may be restored. Otherwise extraction is indicated. Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0,1 % chlorhexidine application is needed twice a day for one week. The parents should be informed about the possible complications (swelling, fistula, or increased mobility). In the case of coronal fragment removal clinical and radiographic control is indicated after 1 week, 3-4 week and after 1 year. If the tooth has been extracted radiographic control is needed at 1 year and every year until the eruption of the permanent tooth.

f. **Complicated crown–root fracture**

Definition: fracture involving enamel, dentin and cementum, and also involving the pulp. The fracture line is extending below the gingival margin. The crown is split into two or more parts
Percussion: tender to percussion due to the subgingival fracture

Vitality test: not reliable

Mobility test: coronal fragment is mobile

Radiographic examination: the fracture line is not visible. Occlusal radiograph is recommended.

Treatment: If the involvement of the root is small, after the removal of the coronal fragment the tooth may be restored with pulpotomy. Otherwise extraction is indicated. Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0.1% chlorhexidine application is needed twice a day for one week. The parents should be informed about the possible complications (swelling, fistula, or increased mobility). In the case of coronal fragment removal clinical and radiographic control is indicated after 1 week, 3-4 week and after 1 year. If the tooth has been extracted radiographic control is needed at 1 year and every year until the eruption of the permanent tooth.

g. Root fracture

Definition: fracture confined to the root of the tooth, involving the cementum, the dentin and the pulp.

Percussion: tender to percussion

Vitality test: not reliable

Mobility test: coronal fragment is usually mobile

Radiographic examination: occlusal and multi-angle periapical radiograph is indicated. The fracture line is usually in the middle or the apical third of the root.

Treatment: if the coronal fragment is not displaced and not mobile no treatment is needed. If there is minor displacement the repositioning and splinting of the tooth may be indicated. Otherwise extraction is needed. The apical fragment should be left inside for physiologic resorption. Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0.1% chlorhexidine application is needed twice a day for one week. The parents should be informed about the possible complications (swelling, fistula, or increased mobility). In the case of coronal fragment removal clinical and radiographic control is indicated after 1 week, 3-4 week and after 1 year. If the tooth has been extracted radiographic control is needed at 1 year and every year until the eruption of the permanent tooth.

h. Alveolar fracture

Definition: A fracture of the alveolar process which may or may not involve the alveolar bone socket. Several teeth may be mobile as one unit. Occlusal interference is usually present.

Percussion: tender to percussion

Vitality test: not reliable

Mobility test: a segment is mobile

Radiographic examination: occlusal, multi-angle periapical and panoramic radiograph is indicated. Vertical fracture may run along the PDL, horizontal fracture line may be located apically. It may be combined with root fractures. The horizontal fracture line may run in the level of the permanent successor.

Treatment: due to the great extent of the trauma, usually general anesthesia is indicated. After repositioning the segment flexible splinting is required for 4 weeks. Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0.1% chlorhexidine application is needed twice a day for one week. The parents should be informed about the possible complications (swelling, fistula, or increased mobility). Splint removal and radiographic control is needed after 4 weeks, further control 6-8 weeks later and every year until the exfoliation of the affected teeth.

i. Concussion
2. Pediatric dentistry and orthodontics

Definition: injury to the periodontal structures without increased mobility or displacement of the tooth, without gingival bleeding. The periodontal ligaments are not torn.

Percussion: tender to percussion due to the contusion of the periodontal ligaments.

Vitality test: not reliable

Mobility test: normal

Radiographic examination: no radiographic signs

Treatment: Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0.1 % chlorhexidine application is needed twice a day for one week. The parents should be informed about the possible complications (swelling, fistula, increased mobility or discoloration of the tooth). Clinical and radiographic control is indicated after 1 week and 6-8 weeks.

j. **Subluxation**

Definition: injury to the periodontal structures. Some of the periodontal ligaments are torn resulting in increased mobility of the tooth and bleeding from the sulcus. The tooth is not displaced.

Percussion: tender to percussion

Vitality test: not reliable

Mobility test: increased mobility

Radiographic examination: no radiographic signs, normal periodontal space

Treatment: Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0.1 % chlorhexidine application is needed twice a day for one week. The parents should be informed about the possible complications (swelling, fistula, increased mobility or discoloration of the tooth). Clinical and radiographic control is indicated after 1 week and 6-8 weeks.

k. **Extrusion** (Figure 5.)

Definition: Partial displacement of the tooth out of its socket, partial or total separation of the periodontal ligaments resulting in loosening and displacement of the tooth. Compared with lateral luxation the alveolar bone is intact. Axial displacement is combined with the retraction or protrusion of the tooth.

Percussion: tender to percussion

Vitality test: not reliable

Mobility test: excessively mobile

Radiographic examination: increased periodontal space

Treatment: in minor extrusion (<3mm) with immature root repositioning, or left for spontaneous alignment. In the case of severe extrusion or tooth with fully developed roots extraction is needed.

Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0.1 % chlorhexidine application is needed twice a day for one week. The parents should be informed about the possible complications (swelling, fistula, increased mobility or discoloration of the tooth). Clinical and radiographic control is indicated after 1 week, 6-8 weeks and 1 year.

**Figure 2.36. Figure 5. – Extrusion of tooth 6.1 and 6.2.**
1. **Lateral luxation**

Definition: Displacement of the tooth other than axially, usually accompanied by the fracture of the alveolar bone either on the lingual or labial side. According to the terminology if both the labial and lingual alveolar bone is fractured it is classified as alveolar fracture. Occlusal interference can be seen very often, due to the palatal or lingual displacement, causing premature contact with the opposing tooth. Similarly to extrusion it is characterized by partial or total separation of the periodontal ligament. The apex of the tooth is usually wedged into the bone due to the displacement, the tooth is non-mobile.

Percussion: increased tenderness, high metallic (ankylosis) sound to percussion,

Vitality test: not reliable

Mobility test: non-mobile

Radiographic examination: increased periodontal space, best seen on occlusal radiograph

Treatment: in retrusion (lingual displacement of the crown) with no occlusal interference spontaneous repositioning is the choice of treatment. In the case of occlusal interference, when the crown of the tooth is displaced lingually it may be repositioned. If crown is dislocated labially extraction is indicated, because of the collision between the root of the primary tooth and the permanent successor germ.

If the displacement is minor and there is slight interference in the occlusion, careful grinding of the tooth is the treatment.

Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0,1% chlorhexidine application is needed twice a day for one week. The parents should be informed about the possible complications (swelling, fistula, increased mobility or discoloration of the tooth). Clinical and radiographic control is indicated after 1 week, 2-3 weeks, 6-8 weeks and 1 year.

m. **Intrusion**

Definition: Displacement of the tooth into the alveolar bone, usually accompanied by the fracture of the alveolar bone either on the lingual or labial side. According to the terminology if both the labial and lingual alveolar bone is fractured it is classified as alveolar fracture. The injury can be confused with avulsion when the tooth may disappear totally in the tissues (Figure 6., 7.). The apex of the tooth is usually wedged into the bone due to the displacement, the tooth is non-mobile. The tooth may penetrate into the nasal cavity, which can be diagnosed upon bleeding from the nose.

Percussion: increased tenderness, high metallic (ankylosis) sound to percussion,
Vitality test: not reliable

Mobility test: non-mobile

Radiographic examination: no periodontal space is visible. Periapical radiograph is very important to determine the relationship between the root of the primary tooth and the permanent tooth germ. If there is retrusion the root will be displaced away from the tooth germ. The root of the traumatized primary tooth looks shortened compared to the contralateral incisor. In protrusion the root is dislocated towards the permanent tooth. On radiograph the root is elongated compared to the contralateral incisor.

Treatment: in the case of retrusion the tooth is left for spontaneous re-eruption. When the primary tooth is protruded extraction should be done to minimize the damage caused to the permanent tooth germ.

Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0.1 % chlorhexidine application is needed twice a day for one week. The parents should be informed about the possible complications (swelling, fistula, increased mobility or discoloration of the tooth). Clinical and radiographic control is indicated after 1 week, 2-3 weeks, 6-8 weeks and once every year until the eruption of the permanent successor.

**Figure 2.37. Figure 6. – The intrusion of tooth 6.1 may be clinically mistaken as avulsion**

![Figure 2.37. Figure 6. – The intrusion of tooth 6.1 may be clinically mistaken as avulsion](image)

**Figure 2.38. Figure 7. – Periapical radiograph reveals the intrusion of the tooth**

![Figure 2.38. Figure 7. – Periapical radiograph reveals the intrusion of the tooth](image)
n. **Avulsion**

Definition: Complete displacement of the tooth out of the alveolar socket. May be confused with total intrusion.

Percussion: not available

Vitality test: not available

Mobility test: not available

Radiographic examination: necessary to differentiate from intrusion. On radiograph empty alveolar socket can be seen.
Treatment: avulsed primary tooth should never be repositioned.

Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. Clinical and radiographic control is indicated after 1 week, 6 months and once every year until the eruption of the permanent successor.

3.6. Pathologic sequel of trauma to the teeth

Complications may appear shortly after an injury.

6.1. Pulpitis: pulpitis accompanies almost every trauma. In minor injuries it is reversible. In greater traumas the irreversible pulpitis and later necrosis may form.

6.2. Pulp necrosis can occur when the blood supply is damaged after luxation injuries.

6.3. Tooth discoloration is very frequent in the case of primary incisors. Immediately after the tooth may be discolored due to the rupture of the pulp blood vessels. This causes a pink, brown discoloration to the tooth. If late dark discoloration occurs, that is usually the sign of pulp necrosis.

6.4. Pulp canal obliteration is common after primary incisor luxation injury, and in immature teeth.

6.5. Inflammatory resorption can be either internal or external. It is usually associated with the infected pulp and the inflamed periodontal ligament

6.6. Replacement resorption: if there is no periodontal ligament between the root surface and the bone, the tooth becomes anklyotic. With the physiologic growth of the child, the root will be replaced by bone.

3.7. Prevention of traumas

The objectives of dental trauma prevention are to identify the risk sports, to describe the preventive measures, and their effects. Mouth guards can reduce the risk of severe injury in contact sports, such as ice hockey, football, soccer and basketball, boxing karate etc. The forces can be reduced and distributed to a greater area due to the elasticity of the mouth guard. Commonly over the counter mouth guards are found in sporting goods stores. These are typically molded to an individual’s dentition using a “boil and bite” type material. Due to the improper extension of these mouth guards the preventive effect is limited.

According to the recommendation of the American Dental Association custom made mouth guards are preferable, it is constructed of a more comfortable material that is also more durable, and it does not interfere with breathing as the store bought variety. „Participants in sporting events are encouraged to contact their dentist for fabrication of a custom mouth guard. In addition, organizers and coaches of children’s sports are encouraged to recommend and/or require the use of mouth guards for all of their participants."

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4.2.4. Cariologic lesions and consequent diseases in young permanent dentition – Balazs Sandor

4.1. Sequence of eruption

The sequence of permanent tooth eruption has significantly changed in the past years, due the acceleration in development, and early maturation. The first permanent molars still erupt around the age of 6, but it is not uncommon that they appear by the age of 4. The eruption of the first permanent molars may go unnoticed because they erupt behind the primary teeth without changing them, which leads to a high incidence of caries in these teeth. The changing of the incisor teeth also appears in a very young age (6-8 years). Although this change is visible, in the case of overcrowding of the teeth due to the insufficient length of the dental arch, the contact areas will increase, thus these teeth may also be more prone to caries (Figure 1;2).

Immediately after eruption the enamel is immature, maturation, secondary mineralization is incomplete. This secondary mineralization increases the hardness and resistance of the enamel. These teeth are more susceptible to caries due to their lower mineral content and because these teeth remain in infra-occlusion for a long time, which leads to a difficulty in obtaining a good oral hygiene, children are unable to perform their teeth brushing properly.

The other very important characteristic of freshly erupted young permanent teeth is that their root development is still in progress, they have short roots, with open apices, the dentine walls of the tooth are thin and more prone to fracture. Total root development may take 2-3 years after eruption.

The treatment of freshly erupted teeth is not easy, everything must be done to prevent caries. Restorative treatment should not be limited to the treatment of the carious lesion, by removing the infected tissues and restoring it with a filling material, but the identification of risk factors and preventive measures also have to be taken in account.

Everything must be done to maintain the vitality of the pulp as it is the prerequisite for root development.

Figure 2.39. Figure 1. – The time sequence of permanent tooth eruption (with an average age)
Figure 2.40. Figure 2. – On this picture the difference between girls and boys can be observed

4.2. Principles of treatment of young permanent teeth dentition

The results of our caries risk assessment are important in treatment planning:

- When to treat?
- When to monitor?
• What materials should be used?

• What kind of preventive treatments should be proposed?

1. In children caries appears mainly on the occlusal surface rather than approximally. In patient with an increased risk for caries, fissure sealing should always be carried out.

2. To maintain the vitality of the pulp it is essential to reduce the chance of pulp irritation during cavity preparation (adequate water cooling, sharp excavator close to the pulp), and also during the restoration.

3. As dentin tubules are wider in these teeth, lining the cavity with glass-ionomer cement before composite resin restoration, may reduce the chance of postoperative sensitivity, and the chance of irritation by etching or by the unpolimerized monomers of the filling material. 4. The use of self-etching adhesives has been shown to significantly decrease the postoperative sensitivity.

5. In deep cavities, spreading deeper than the half of the dentin layer indirect pulp capping should be carried out in order to maintain pulp vitality, to promote tertiary dentin formation. (It is limited to cases with normal pulp with no symptoms of pulpitis or in teeth diagnose with reversible pulpitis)

4.2.1. Stepwise or total caries removal

There is a controversy between stepwise and total caries removal.

They both have advantages and disadvantages.

1. In the case of step-wise caries treatment the final treatment is done in a second appointment. The first step is removing the caries until the dentin-enamel junction, and removing only the outermost infected dentin. The aim is to prevent unintentional pulp exposure. Calcium-hydroxide is placed over the carious dentin to slow or arrest the caries development, induce remineralization and promote tertiary dentin formation. Temporary restoration is placed (glass-ionomer cement) over the calcium-hydroxide.

In the second step the rest of the caries is removed, and the final restoration is placed with indirect pulp capping (calcium-hydroxide+glass-ionomer cement+composite resin) The time interval between the two steps is 3-6 months, to allow sufficient time for the formation of tertiary dentin and a definitive pulpal diagnosis. There is a chance for subsequent pulp inflammation and necrosis between the two steps of caries treatment which leads to arrest in root development. We do not recommend this type of treatment. In the case of pulp exposure during preparation according to the condition and infection of the pulp endodontic therapy is indicated.

2. In the case of total caries removal there is a higher chance for the exposure of the pulp during excavation. In these cases direct pulp capping partial or total preventive pulpotomy may be our choice of treatment. Partial and total pulpotomy has shown to be successful in maintaining the vitality of the pulp.

4.3. Treatment of consequent diseases

Consequent diseases of immature teeth are the same that of mature teeth, presenting the same symptoms. Various guidelines are available for treatment planning.

Two main types of treatment are the apexogenesis and the apexification (Table 1).

Table 2.13. Table 1. – Apexogenesis vs. apexification

<table>
<thead>
<tr>
<th>Apexogenesis</th>
<th>Apexification (root end closure)</th>
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</table>
| Continued physiologic development and formation of the root. Apexogenesis can be accomplished by treatments that aim to maintain the vitality of the pulp:  
  • Indirect pulp capping | A method of inducing root end closure of a non-vital immature permanent tooth, by the removal of the necrotic pulp tissue, and the placement of a bioactive material (calcium-hydroxide, MTA) in the root canal to promote the formation of an artificial calcified barrier. The mineralized tissue can be composed of osteocementum, osteodentin, |
### 4.3.1. Apexogenesis

Originally the term apexogenesis defines the physiologic process of root development. Since the aim of some treatments (indirect pulp capping, direct pulp capping, pulpotomy) is to assure this developmental process, these treatments are termed the same way.

#### 4.3.1.1. Indirect, direct pulp capping

The differences between the two procedures are seen on figure 3.

**Figure 2.41. Figure 3. – Schematic drawing of direct and indirect pulp capping**

#### 4.3.1.2. Pulpotomy in immature tooth (Figure 4-6)

Steps of pulpotomy are the same that of primary dentition (II./2.,II./5.). Immature teeth undergoing endodontic therapy, aiming to preserve the vitality should be monitored until the total development of the root. Pulpotomy is not recommended in teeth with closed apex. Where possible complete root canal treatment should be done (success rate is higher).

**Figure 2.42. Figure 4. – The picture shows the pulp chamber of a lower first molar after the removal of the pulp tissue from the pulp chamber. Complete isolation with rubber-dam is indispensable**
Figure 2.43. Figure 5. – An MTA derivative (Biodentine™) placed over the remaining pulp tissue

Figure 2.44. Figure 6. – Postoperative radiograph of the tooth after coronal restoration
Indirect, direct pulp capping and pulpotomy is only recommended in the case of symptomless teeth or in the case of reversible pulpitis and partial irreversible pulpitis.

In immature teeth with total irreversible pulpitis, or pulp necrosis apexification should be carried out.

### 4.3.2. Apexification

The definition of apexification can be seen in Table 1. There are two main types of materials used for apexification. (Table 2.)

**Table 2.14. Table 2.**

<table>
<thead>
<tr>
<th>Calcium hydroxide</th>
<th>MTA and derivatives</th>
</tr>
</thead>
<tbody>
<tr>
<td>• creates an apical hard tissue barrier against which the root canal filling can be placed</td>
<td>• Immediate root canal filling can be made after making an apical stop with MTA</td>
</tr>
<tr>
<td>• highly alkaline material (pH 12-13) which may activate alkaline phosphatase activity</td>
<td>• High pH (12.5)</td>
</tr>
<tr>
<td>• increases the activity of calcium dependent pyrophosphatase inducing calcium phosphate precipitation (mineralization)</td>
<td>• Appetite like interfacial deposits form during the maturation which may improve the frictional resistance</td>
</tr>
<tr>
<td>• Direct effect on the apical and periapical soft-tissue</td>
<td>• 5mm artificial barrier made by MTA is significantly stronger and shows less leakage than the 2mm barrier induced by calcium-hydroxide</td>
</tr>
<tr>
<td>• Antibacterial activity</td>
<td>• One appointment (or two if previously disinfected with calcium-hydroxide) apexification can be carried out, which reduces the chance of infection and fracture</td>
</tr>
<tr>
<td>• Several visits are needed until barrier is formed</td>
<td></td>
</tr>
<tr>
<td>• Decreases the flexural and fracture</td>
<td></td>
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</tbody>
</table>
2. Pediatric dentistry and orthodontics

<table>
<thead>
<tr>
<th>Calcium hydroxide</th>
<th>MTA and derivatives</th>
</tr>
</thead>
<tbody>
<tr>
<td>strength of the dentin</td>
<td></td>
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</table>

4.3.2.1. Method of apexification with calcium-hydroxide

- Anaesthesia
- Access cavity
- Necrotic pulp exstriation (removal)
- Mild preparation 1-1.5 mm short of radiographic apex (excessive mechanical preparation should be avoided due to the inherently thin dentin walls)
- Irrigation (1% NaOCl)
- Ca(OH)2 placement in the root canal for two weeks (disinfection)
- After two weeks Ca(OH)2 should be changed
- Access cavity is closed with GIC or IRM
- Review: 3-6 months
- Check for barrier gently with paper points (files may break the thin barriers) (Figure 7.)
- If barrier is formed root canal filling may be carried out by warm vertical condensation of gutta-percha.

**Figure 2.45. Figure 7. – Picture of calcified barrier formed after calcium-hydroxide treatment made with operating microscope. (Case and picture of Dr. Krajczár Károly)**

4.3.2.2. Method of apexification with MTA and derivatives

- Basic procedure is the same
- The placement of calcium-hydroxide into the root canal for disinfection is not required, unless pain is present, as it has been shown in numerous studies the total apical healing was achieved after single-appointment treatments with MTA.
Due to the higher possibility for fracture in teeth after apexification (due to the thin dentin walls), instead of filling the root canal with gutta-percha, the placement of prefabricated fiber post has been shown to be more advantageous. (Figure 8-10.)

**Figure 2.46. Figure 8. – Radiographic measurement of working length**

**Figure 2.47. Figure 9. – Placement of MTA derivative (Biodentine™) in the root canal**
Figure 2.48. Figure 10. – The radiograph shows the fiber post in the root canal. The radiograph was taken 3 months after the placement of the MTA and the post. Note the apical barrier formed.

For further reading on the treatment of freshly erupted teeth read II./5: Traumatic cases in young permanent dentition.

References


5.2.5. Traumatic cases in young permanent dentition – Balazs Sandor

Traumatic epidemiology etiology and the classifications of the traumatic cases have earlier been described in II./3: Traumatic lesions in primary dentition. The specific examination and the treatment of the traumatized tooth are significantly different from the primary dentition. The treatment of a young permanent tooth, or in other terms called freshly erupted, or immature permanent tooth, is a great challenge for the practitioners.

5.1. Young permanent teeth

The treatment of young permanent teeth is different in some aspects from the treatment of mature, fully developed teeth. Root development starts after the crown formation has reached the cemento-enamel junction. The part or the enamel organ where the inner and the outer enamel epithelium meets is called the cervical loop. The growth of the cervical loop will form the Hertwig’s Epithelial Root Sheath (HERS), which is responsible for the root development of the tooth. The pulp is necessary to continue apexogenesis. The loss of vitality ceases root development; therefore, it is our main treatment objective to maintain the vitality of the pulp of a tooth affected by caries, traumatic injury, or other causes. An open apex is found in the developing roots of immature teeth until apical closure occurs approximately 3 years after eruption. The stages of root development have been described by several authors, such as Andreasen, Čvek or Moorrees (Figure 1.). Generally we consider the apex to be open if the apical constriction is wider than 1 mm.

Figure 2.49. Figure 1. – The Moorrees classification of root development. Prognosis of treatment of immature teeth can be determined upon the stage of root development

5.2. Endodontic treatment in young permanent teeth

The exact steps of the procedures are described in II./4 Cariologic leasions and consequent diseases in young permanent dentition.

Protective liner, indirect pulp capping: a thin layer applied on the pulpal surface of the cavity or fracture site, to close down dentin tubules, to act as a protective layer between the restoration and the pulp.

Apexogenesis: is a vital pulp therapy procedure performed to encourage physiological development and formation of the root end. Three basic procedures are used: direct pulp capping, partial pulpotomy (Čvek), conventional vital pulpotomy).

• Direct pulp capping: direct pulp capping is not indicated in teeth with immature roots, due to the possible contamination and inflammation of the pulp tissue near the fracture site.

• Pulpotomy:

  • Čvek pulpotomy: Čvek et al demonstrated that in teeth with complicated crown fractures only the most superficial 2 mm of the pulp is inflamed and requires removal.

  • Conventional pulpotomy: the pulp is removed, from the whole pulp chamber.
2. Pediatric dentistry and orthodontics

The goals of pulpotomy was earlier described by Webber et al:

- Sustaining a viable Hertwig’s sheath, thus allowing continued development of root length for a more favorable crown-to-root ratio.
- Maintaining pulpal vitality, thus allowing the remaining odontoblasts to lay down dentine, producing a thicker root and decreasing the chance of root fracture.
- Promoting root end closure, thus creating a natural apical constriction for root canal filling.
- Generating a dentinal bridge at the site of the pulpotomy. While the bridging is not essential for the success of the procedure, it does suggest that the pulp has maintained its vitality.

Apexification: inducing a calcified barrier in a root with an open apex or the continued apical development of an incompletely formed root in teeth with necrotic pulp.

Regenerative endodontics: Even if apexification is successful, the crown-root ratio is unfavorable and the dentin walls are thin, leaving the teeth with a great risk of fracture. Revascularization of the teeth with necrotic pulp and periapical lesions is a procedure allowing continued maturation of the teeth. The treatment is carried out by the induction of intracanal bleeding after the chemical disinfection of the root canal. Mineral trioxide aggregate (MTA) is placed over the blood clot, and the crown is restored.

5.3. Treatment and diagnosis of injuries to the young permanent dentitions

Several factors may affect our treatment plan:

- Age and maturation of the child, dental developmental stage,
- Type and extent of injury,
- Cooperation of the child,
- Social background of the child.

Splinting:

„Current evidence supports short-term, non-rigid splints for splinting of luxated, avulsed and root-fractured teeth. While neither the specific type of splint nor the duration of splinting are significantly related to healing outcomes (except for avulsion where the time may be of importance), it is considered best practice in order to maintain the repositioned tooth in correct position, provide patient comfort and improved function”

Various types of materials can be used for splinting, such as nickel-titanium orthodontic wires, multi-strand coaxial stainless steel wires, or 50 lb monofilament fishing lines luted to the traumatized and the adjacent teeth with resin. The duration of splinting is dependent on the type of the trauma.

So far there is no evidence for the use of systemic antibiotics after luxation injuries. It is the choice of the clinician. The beneficial effect of the use of antibiotics on the surface of the immature root before replantation, increasing the chance of revascularization, has been described by several authors. At the pediatric dentistry division in the department of Dentistry, Oral and Maxillofacial Surgery, University of Pécs, we recommend the use of antibiotics in the case of intrusive luxations and where there is extensive soft tissue injury or significant surgical intervention.

Please note that the treatment of luxation injuries and some fractures should be carried out in anesthesia. (Depending on the child’s age, the type of trauma, and the cooperation it may include local anesthesia, conscious sedation combined with local anesthesia, or general anesthesia.)

According to the clinical findings “trauma pathfinder”, accessible on the website: www.dentaltrumaguide.org, may be useful for establishing the diagnosis.

a. Infraction
Definition: incomplete fracture of the enamel, with a visible fracture line on the tooth surface

Percussion: not tender. If the tooth is tender to percussion luxation injury or root fracture may be in the background.

Vitality test: usually positive. Transient loss of vitality increases the risk of subsequent pulp necrosis.

Mobility test: normal

Radiographic examination: no abnormalities can be observed

Treatment: no treatment is needed. Etching and sealing with an adhesive bond resin may prevent the discoloration of the crack.

No follow up is needed unless the infraction is associated with luxation injury.

b. Enamel fracture

Definition: uncomplicated fracture of the crown that only involves the enamel

Percussion: not tender. If the tooth is tender to percussion luxation injury or root fracture may be in the background.

Vitality test: positive. Transient loss of vitality increases the risk of subsequent pulp necrosis.

Mobility test: normal

Radiographic examination: periapical and occlusal exposures are recommended to exclude root fractures. If the patient has penetrating lip lesion soft tissue radiograph should be done to exclude the penetration of the fragment.

Treatment:

• If the fractured fragment is available, it can be bonded to the teeth.

• Smooth the sharp edges with a fine diamond bur or with an abrasive metal strip, fluoridating the fracture surface.

• Restoration with composite resin.

6-8 weeks and 1 year later clinical and radiographic control is recommended.

c. Enamel-dentin fracture (uncomplicated crown fracture with enamel and dentin involvement)

Definition: fracture of the crown that only involves the enamel and the dentin, but not the pulp

Percussion: not tender. If the tooth is tender to percussion luxation injury or root fracture may be in the background.

Vitality test: positive. Transient loss of vitality increases the risk of subsequent pulp necrosis.

Mobility test: normal

Radiographic examination: periapical and occlusal exposures are recommended to exclude root fractures. If the patient has penetrating lip lesion soft tissue radiograph should be done to exclude the penetration of the fragment. The distance between the fracture surface and the pulp may be evaluated on periapical radiographs.

Treatment:

• If the fractured fragment is available, it can be bonded to the teeth.

• Restoration with composite resin, after the lining of the dentin surface, to reduce chemical irritation of the pulp.
6-8 weeks and 1 year later clinical and radiographic control is recommended.

d. Complicated crown fracture

Definition: fracture of the crown that only involves enamel and the dentin exposing the pulp

Percussion: not tender. If the tooth is tender to percussion luxation injury or root fracture may be in the background.

Vitality test: positive. Transient loss of vitality increases the risk of subsequent pulp necrosis.

Mobility test: normal

Radiographic examination: If the patient has penetrating lip lesion soft tissue radiograph should be done to exclude the penetration of the fragment.

Treatment: to keep the vitality of the tooth pulotomy or partial (Čvek) pulpotomy is indicated with calcium-hydroxide, or MTA dressing. In the case of teeth with closed apex, associated with luxation injury root canal treatment is indicated. Clinical and radiographic control is necessary after 6-8 weeks and 1 year. (Figure 2-6.)

**Figure 2.50.** Figure 2. – Complicated fracture of immature tooth after isolation, desinfection and debridement of the fracture surface

**Figure 2.51.** Figure 3. – 2 mm deep partial removal of the pulp tissue with sterile bur and sterile saline administration
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Figure 2.52. Figure 4. – Covering the pulp with Biodentine™ (Modified MTA)

Figure 2.53. Figure 5. – The whole fracture surface overed with composite, to prevent indirect infection of the pulp
e. Uncomplicated crown-root fracture

Definition: fracture involving enamel, dentin and cementum but not involving the pulp. The fracture line is extending below the gingival margin. The crown is split into two or more parts

Percussion: tender to percussion due to the subgingival fracture
Vitality test: apical fragment usually vital. Transient loss of vitality increases the risk of subsequent pulp necrosis.

Mobility test: coronal fragment is mobile

Radiographic examination: the fracture line in not visible. Occlusal radiograph is recommended.

Treatment: treatment options can be found in Table 1. Surgical and orthodontic extrusion may be used in teeth with closed apices. In the case of immature teeth to maintain pulp vitality the fracture site should be covered with composite material. In these cases definitive treatment is postponed until the total development of the root.

Follow up: clinical and radiographic control is indicated after 6 weeks and 1 year.

Table 2.15. Table 1. – Treatment options for uncomplicated crown-root fractures

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Indication</th>
<th>Advantage</th>
<th>Disadvantage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Removal of coronal fragment and subsequent</td>
<td>Superficial fractures, can be used in teeth with open apices.</td>
<td>Fast treatment.</td>
<td>None.</td>
</tr>
<tr>
<td>restoration</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Removal of coronal fragment, gingivectomy,</td>
<td>Recommended in areas where gingivectomy does not compromise esthetics,</td>
<td>Relatively fast treatment.</td>
<td>The tooth may migrate labially, due to the palatal</td>
</tr>
<tr>
<td>subsequent restoration</td>
<td>can be used in teeth with open apices.</td>
<td></td>
<td>pocket.</td>
</tr>
<tr>
<td>Orthodontic extrusion of apical fragment</td>
<td>All types of fractures, assuming that reasonable root length can be</td>
<td>Stable position of the restored tooth.</td>
<td>Time consuming procedure with late completion of final</td>
</tr>
<tr>
<td></td>
<td>achieved after extrusion. May only be used in mature teeth.</td>
<td>Optimal gingival health.</td>
<td>restoration.</td>
</tr>
<tr>
<td>Surgical extrusion of apical fragment</td>
<td>All types of fractures, except crown-root fractures in young teeth with</td>
<td>Rapid procedure. Stable position of the tooth.</td>
<td>Limited risk for root resorption and marginal</td>
</tr>
<tr>
<td></td>
<td>open apices where vitality should be preserved, assuming that reasonable</td>
<td>The method allows inspection of the root for</td>
<td>breakdown of the periodontium.</td>
</tr>
<tr>
<td></td>
<td>root length can be achieved. The tooth may be rotated in 90°-180° to</td>
<td>additional fractures.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>expose the subgingival fracture site, allowing better periodontal healing.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decoronation</td>
<td>Can be used in cases where the root cannot support a post-retained crown</td>
<td>Preserves the alveolar process.</td>
<td>Postpones definitive restoration.</td>
</tr>
<tr>
<td></td>
<td>restoration.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extraction</td>
<td>Extraction in cases of extensive deep crown-root fractures.</td>
<td>None.</td>
<td>Tooth loss.</td>
</tr>
</tbody>
</table>

f. Complicated crown–root fracture (see figure 7.)

Definition: fracture involving enamel, dentin and cementum, and also involving the pulp. The fracture line is extending below the gingival margin. The crown is split into two or more parts

Percussion: tender to percussion due to the subgingival fracture
Vitality test: apical fragment usually vital. Transient loss of vitality increases the risk of subsequent pulp necrosis.

Mobility test: coronal fragment is mobile

Radiographic examination: the fracture line in not visible. Occlusal radiograph is recommended.

Treatment: in immature teeth pulpotomy should be carries out. The final treatment is postponed until the total development of the root. In mature teeth root canal treatment may be the option. In other aspects the treatment options are the same as in the uncomplicated crown-root fracture except for the fragment removal and subsequent definitive restoration, which is impractical due to the extent of the fracture.

Follow up: clinical and radiographic control is indicated after 6 weeks and 1 year.

**Figure 2.55. Figure 7. – Complicated crown-root fracture of tooth 1.1**

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g. Root fracture

Definition: fracture confined to the root of the tooth, involving the cementum, the dentin and the pulp. Can be further classified upon the luxation of the coronal fragment

Percussion: tender to percussion

Vitality test: due to the pulp damage may give negative results

Mobility test: coronal fragment may be mobile, depending upon luxation injury

Radiographic examination: occlusal and multi-angle periapical radiograph is indicated. The fracture line is usually in the middle or the apical third of the root.

Treatment: Repositioning of the coronal fragment, and reducing the fracture by firm digital pressure, and splinting in this position for 4 weeks with flexible splint. Before stabilizing the tooth the position of the tooth has to be checked with radiograph. If the fracture is in the cervical third of the root, the period of splinting may range up to 4 months. The vitality of the tooth should be checked at least for one year. Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0,1% chlorhexidine application is needed twice a day for one week.

The aim of our treatment is to encourage hard tissue union between the fragments, to maintain vitality to enable maturation of young permanent tooth. Immature teeth have a much better healing ability, than mature.

Healing of root fracture:

- Type I: hard tissue union (30%),
- Type II: connective tissue or connective tissue and bone interposition between the fragments.
Failure of healing is due to the necrosis of the pulp with the interposition of granulation tissue. If there is pulp necrosis, but the tooth is fixed, endodontic treatment is indicated (root canal treatment a filling). If the tooth is still mobile the treatment is done according to the length of the coronal fragment. In teeth with longer coronal fragment, an artificial barrier is induced by calcium-hydroxide or MTA.

Radiographic control and splint removal is indicated after 4 weeks, except for the cervical fracture described earlier. Further control after 6 months and yearly for 5 years.

h. Alveolar fracture

Definition: A fracture of the alveolar process which may or may not involve the alveolar bone socket. Several teeth may be mobile as one unit. Occlusal interference is usually present.

Percussion: tender to percussion

Vitality test: usually negative

Mobility test: a segment is mobile

Radiographic examination: occlusal, multi-angle periapical and panoramic radiograph is indicated. Vertical fracture may run along the PDL, horizontal fracture line may be located apically. It may be combined with root fractures.

Treatment: due to the great extent of the trauma, usually general anesthesia is indicated. After repositioning the segment flexible splinting is required for 4 weeks. Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0,1% chlorhexidine application is needed twice a day for one week. Splint removal and radiographic control is needed after 4 weeks, further control 6-8 weeks, 4 months, 6 months later and yearly for 5 years.

i. Concussion

Definition: injury to the periodontal structures without increased mobility or displacement of the tooth, without gingival bleeding. The periodontal ligaments are not torn.

Percussion: tender to percussion due to the contusion of the periodontal ligaments.

Vitality test: positive. Transient loss of vitality increases the risk of subsequent pulp necrosis. The vitality may be false negative for 3 months. The root development should be controlled with radiographs.

Mobility: normal

Radiographic examination: no radiographic signs

Treatment: Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0,1% chlorhexidine application is needed twice a day for one week.

Clinical and radiographic control is indicated after 4 weeks, 6-8 weeks and 1 year.

j. Subluxation

Definition: injury to the periodontal structures. Some of the periodontal ligaments are torn resulting in increased mobility of the tooth and bleeding from the sulcular gap. The tooth is not displaced.

Percussion: tender to percussion

Vitality test: positive. Transient loss of vitality increases the risk of subsequent pulp necrosis. The vitality may be false negative for 3 months. The root development should be controlled with radiographs.

Mobility test: increased mobility

Radiographic examination: no radiographic signs, normal periodontal space
Treatment: No treatment is needed. For patient comfort flexible splinting may be required for 2 weeks. Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0,1% chlorhexidine application is needed twice a day for one week.

Clinical and radiographic control is indicated after 2 weeks, 4 weeks, 6-8 weeks, 6 months and 1 year. In mature teeth with lack of response to vitality tests for 3 months, in immature teeth with ceased root development or signs of inflammation, external root resorption in both mature and immature teeth, endodontic treatment is indicated according to the stage of root development.

k. Extrusion

Definition: Partial displacement of the tooth out of its socket partial or total separation of the periodontal ligament resulting in loosening and displacement of the tooth. Compared with lateral luxation the alveolar bone is intact. Axial displacement is combined with the retraction or protrusion of the tooth.

Percussion: tender to percussion

Vitality test: negative results

Mobility test: excessively mobile

Radiographic examination: increased periodontal space

Treatment: repositioning the tooth and flexible splinting for two weeks. Revascularization of the tooth with open apex can be confirmed with x-ray showing continued root development and pulp canal obliteration (PCO). Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0,1% chlorhexidine application is needed twice a day for one week. Splint removal and control is needed after 2 weeks. Further control at 4 weeks, 6-8 weeks, 6 months, and 1 year Clinical and radiographic control is indicated after 1 week, 6-8 weeks and 1 year. In mature teeth with lack of response to vitality tests for 3 months, in immature teeth with ceased root development or signs of inflammation, external root resorption in both mature and immature teeth, endodontic treatment is indicated according to the stage of root development.

l. Lateral luxation

Definition: Displacement of the tooth other than axially, usually accompanied by the fracture of the alveolar bone either on the lingual or labial side. According to the terminology if both the labial and lingual alveolar bone is fractured it is classified as alveolar fracture. Occlusal interference can be seen very often, due to the palatal or lingual displacement, causing premature contact with the opposing tooth. Similarly to extrusion it is characterized by partial or total separation of the periodontal ligament. The apex of the tooth is usually wedged into the bone due to the displacement, the tooth is non-mobile.

Percussion: increased tenderness, high metallic (ankylosic) sound to percussion,

Vitality test: negative results, unless there is minor displacement

Mobility test: non-mobile

Radiographic examination: increased periodontal space, best seen on occlusal radiograph

Treatment: repositioning the tooth and flexible splinting for four weeks (because of the alveolar bone involvement). Revascularization of the tooth with open apex can be confirmed with x-ray showing continued root development and pulp canal obliteration (PCO). Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0,1% chlorhexidine application is needed twice a day for one week. Control is indicated after 2 weeks, 4 weeks, 6-8 weeks, 6 months, and yearly for 5 years. Splint removal is carried out on the 4th week. In immature teeth with ceased root development or signs of inflammation, external root resorption in both mature and immature teeth, endodontic treatment is indicated according to the stage of root development.

m. Intrusion (Figure 8.)

Definition: Displacement of the tooth into the alveolar bone, usually accompanied by the fracture of the alveolar bone either on the lingual or labial side. According to the terminology if both the labial and lingual...
alveolar bone is fractured it is classified as alveolar fracture. The apex of the tooth is usually wedged into the bone due to the displacement, the tooth is non-mobile.

Percussion: increased tenderness, high metallic (ankylosic) sound to percussion,

Vitality test: negative

Mobility test: non-mobile

Radiographic examination: no periodontal space is visible.

Treatment: The treatment choice depends on the stage of root development and the degree of the intrusion (Table 2.). The chosen treatment is observation if spontaneous re-eruption can be expected. If there is no change after 2-4 weeks, orthodontic repositioning is required to prevent ankylosis. Surgical repositioning of the tooth is recommended in the acute phase of the injury. Otherwise orthodontic extrusion is indicated. After surgical and orthodontic repositioning 4-8 weeks of flexible splinting is required. To prevent inflammatory external resorption, endodontic treatment is indicated in teeth with closed apex 3-4 weeks after the initial treatment. Revascularization of the tooth with open apex can be confirmed with x-ray showing continued root development and pulp canal obliteration (PCO).

Soft food diet and toothbrushing with soft brush is recommended for 1-2 weeks. 0.1% chlorhexidine application is needed twice a day for one week. Control is indicated after 2 weeks, 4 weeks, 6-8 weeks, 6 months, and yearly for 5 years. Splint removal is carried out on the 4th week. In immature teeth with ceased root development or signs of inflammation, external root resorption in both mature and immature teeth, endodontic treatment is indicated according to the stage of root development.

**Table 2.16. Table 2. – Treatment options in intrusion according to the stage of root development and the degree of intrusion**

<table>
<thead>
<tr>
<th>Degree of intursion</th>
<th>Spontaneous</th>
<th>Orthodontic</th>
<th>Surgical</th>
<th>Repositioning</th>
</tr>
</thead>
<tbody>
<tr>
<td>Open apex</td>
<td>Up to 7 mm</td>
<td>X</td>
<td>7 mm</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>X</td>
<td>7 mm</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Closed apex</td>
<td>Up to 3 mm</td>
<td>X</td>
<td>7 mm</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>3–7 mm</td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>&gt; 7 mm</td>
<td></td>
<td></td>
<td>X</td>
</tr>
</tbody>
</table>

**Figure 2.56. Figure 8. – Intrusion of tooth 1.1**
n. Avulsion

Definition: Complete displacement of the tooth out of the alveolar socket. One of the most serious injuries to permanent teeth. This type of trauma can be seen in 0.5-3% of all dental injuries.

Percussion: not available

Vitality test: not available

Mobility test: not available

Radiographic examination: On radiograph empty alveolar socket can be seen.

Treatment:

First aid: Immediate treatment is of great significance, but this cannot always be carried out, due to the lack of dentist in the close area. Information may be given in the case of trauma on telephone to older children or adults around the child about what to do until they find a dentist. If the avulsed tooth is found, it may only be held by the crown. The prognosis is the best if the replantation is done immediately at the site of the injury after washing it for 10 seconds under cold running water, without mechanical cleaning. Replantation is contraindicated in the case of severe caries, non cooperating patient or severe medical conditions, such as immunosuppression, and severe cardiac disease. The contraindications cannot be cleared through telephone, thus we recommend the patient to be taken to a dentist as soon as possible. Several storage media may be recommended to keep the periodontal ligaments viable (saliva, saline, milk, tap water, special tooth saving box). After 60 minutes of dry storage the cells are not viable. The treatment choice is dependent on the stage of root development the condition of the periodontal ligament (PDL) cells.

Classification of the teeth into groups upon the condition of the PDL cells:

- PDL cells are most likely viable (immediate replantation),
- PDL cells may be viable but compromised (tooth held in storage media such as saline, saliva, milk, tissue culture medium-tooth rescue box, and the total dry time is less than 60 minutes),
- PDL cells are non-viable due to dry-time more than 60 minutes.

The treatment options can be seen in Table 3.

Table 2.17. Table 3. – Treatment options in the case of avulsion, according to the guidelines of the International Association of Dental Traumatology

<table>
<thead>
<tr>
<th>Apex</th>
<th>Closed</th>
</tr>
</thead>
<tbody>
<tr>
<td>PDL</td>
<td>Viable</td>
</tr>
<tr>
<td>Condition of tooth</td>
<td>Immediately replanted</td>
</tr>
<tr>
<td>Treatment</td>
<td>i. Leave tooth in place</td>
</tr>
<tr>
<td></td>
<td>ii. Wash with saline and chlorhexidine</td>
</tr>
<tr>
<td></td>
<td>iii. Suture gingival lacerations if present</td>
</tr>
</tbody>
</table>
v. Apply flexible splint for 2 weeks
vi. Administer antibiotics
vii. Check tetanus protection
Root canal treatment after 7-10 days, before splint removal.

iv. Examine alveolar bone. In the case of fracture reposition
v. Replant the tooth with slight pressure
vi. Suture gingival lacerations if present
Verify position of tooth clinically and with radiograph
vii. Apply flexible splint for 2 weeks
viii. Administer antibiotics
ix. Check tetanus protection
Root canal treatment after 7-10 days, before splint removal.

ix. Check tetanus protection
Revascularization and the continuation of root development is the aim.
ii. Administer local anesthesia
iii. Administer antibiotics
viii. Suture gingival lacerations if present
Verify position of tooth clinically and with radiograph
ix. Apply flexible splint for 2 weeks
x. Administer antibiotics
xi. Check tetanus protection
The risk of infection related resorption should be weighed up against the chances of revascularization. In the case of no revascularization apexification is needed.

2% sodium-fluoride solution root surface treatment for 20 minutes may slow down resorption.

Instructions
Soft diet for 2 weeks, toothbrushing with soft toothbrush, 0.1% chlorhexidine mouth rinse twice a day, no contact sports.

Follow up
Ankylosis is frequent outcome due to infraocclusion. If infraocclusion is >1mm decoronation is needed to maintain the contour of the alveolar bone until definitive treatment. Follow up: 4 weeks, 3 months, 6 months, 1 year and yearly thereafter.

The following picture show the method of replantation (Figure 9-11).

**Figure 2.57. Figure 9. – Avulsed tooth in saline storage media**
Figure 2.58. Figure 10. – Site of avulsion

Figure 2.59. Figure 11. – After replantation the splinting of the tooth with coaxial wire and composit material
Endodontic considerations: In the case of closed apex endodontic treatment should be initiated 7-10 after the injury to reduce the chances of pulp inflammation related external resorption. After instrumentation calcium hydroxide is applied in the canal for 2-4 weeks. before final root canal filling. The endodontic treatment of the tooth extraorally before replantation is not recommended. There is a chance for revascularization in immature teeth. If there is no sign of revascularization after 3 month, namely the root development is ceased, apexification is indicated.

Favorable outcome of treatment: asymptomatic, normal mobility, normal percussion sound, no radiologic abnormality, continuing root canal development, and root canal obliteration (immature tooth)

Unfavorable outcome: ankylosis, no mobility, resorption, ceased root formation. Ankylosis in a growing patient may lead to disturbance of alveolar and facial growth.

5.4. Prevention of traumas

Described in II.3: Traumatic lesions in primary dentition.

References


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ROBERTS G., LONGHURST P.: Oral and Dental Trauma in Children and Adolescents; Oxford Medical Publications, 1996
2. Pediatric dentistry and orthodontics


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5.5. Test

1. **Order of steps of diagnostic procedures in pediatric dentistry**
   a. Patient history, imaging, extraoral examination, intraoral examination
   b. Patient history, extraoral examination, intraoral examination, imaging
   c. Imaging, patient history, extraoral examination, intraoral examination
   d. Patient history, intraoral examination, extraoral examination, imaging

   Right answer: B

1. **Which of the following statements is incorrect?**
   a. Opioid analgesics are only recommended in an institutional setting
   b. Acetaminophen has a strong anti-inflammatory effect
   c. Combined use of analgesic and antipyretic drugs may be recommended in the acute phase of the disease
   d. All of the above are correct

   Right answer: B

1. **Which of the following developmental disorder is formed in the stage of morphodifferentation?**
   a. Taurodontism
   b. Eruption cyst
   c. Fluorosis
   d. Turner-tooth

   Right answer: A

1. **Which of the following is not used for primary molar restorations?**
   a. Stainless steel crowns
   b. Compomer filling material
   c. Amalgam filling material
   d. Composite filling material

   Right answer: C
1. Possible endodontic treatment methods for primary teeth diagnosed with reversible pulpitis
   a. Indirect pulp capping, preventive pulpotomy
   b. Preventive pulpotomy, pulpectomy
   c. Direct pulp capping, pulpectomy
   d. Extraction and maintaining the space

Right answer: A

1. Treatment choice in the case of primary tooth avulsion
   a. Repositioning and splinting the tooth
   b. Etching and sealing with adhesive resin to prevent discoloration
   c. Pulpotomy is indicated
   d. No intervention is indicated, only the advise should be given

Right answer: D

1. In the case of intrusion of the primary incisors:
   a. the permanent tooth buds may be damaged
   b. orthodontic treatment may be used to reposition the tooth
   c. there is no chance for spontaneous re-eruption
   d. Extraction is always indicated

Right answer: A

1. Stepwise caries removal:
   a. more successful than total caries removal
   b. is carried out in 3 appointments
   c. aims to prevent unintentional pulp exposure
   d. All the above are correct

Right answer: C

1. Apexification:
   a. a method of inducing root end closure of a non-vital immature permanent tooth
   b. is a treatment method to assure the physiologic development and formation of the root
   c. is a treatment method to assure the physiologic resorption of the root
   d. neither one is true

Right answer: A

1. Which of the following statement is correct?
   a. Right after the eruption of the teeth, the enamel is already totally matured.
b. MTA creates a hard tissue barrier in the crown of the teeth during apexification

c. The layers of direct pulp capping, starting with the deepset layer are: glass ionomer, composite, calcium hydroxide

d. The use of fiber post in teeth after apexification may reduce the chance of root fracture

Right answer: D

1. **Subluxation is characterized by:**

   a. Tenderness to percussion

   b. Increased mobility

   c. Pulp necrosis

   d. a.) and c.) answers are correct

   e. a.) and b.) answers are correct

Right answer: E

1. **Primary treatment option in the case of intrusion less than 7 mm in the case of open apex:**

   a. Orthodontic extrusion

   b. Surgical repositioning

   c. Splinting

   d. No treatment is needed, because there is a chance for re-eruption

Right answer: D

1. **Condition if PDL cells in the case of avulsion after a dry-time of more than 60 minutes**

   a. Most likely viable

   b. Viable but compromised

   c. Non-viable

   d. Neither one of the above

Right answer: C

6. 2.6. Most common dental and jaw disorders – Zsuzsanna Gurdan

Orthodontic disorders involve disadvantageous dental and facial structures due to either developmental or acquired malformations, which can be associated with unfavourable consequences. Nevertheless, these are only severe in rare cases, in the majority of cases they cause functional and aesthetic disorders.

**Eugnathic** dentition is an anatomically, functionally and aesthetically flawless dentition. A healthy dentition can develop only in a regular, well structured skull. Any deviation of dentition from the normal is considered to be dysgnathic.

6.1. **Etiology of orthodontic anomalies**

Detecting the etiological factors of jaw and dental anomalies is a major field of orthodontics, as determining the origin of deformities can be decisive regarding the treatment plan and the chosen treatment modality. Anomalies
occur more frequently in the upper jaw and in permanent dentition. Causes can be inherited or acquired, but often the combination of the two can be seen.

**Congenital disorders:** dental disorders due to inherited (genetic) abnormalities or teratogenic and environmental deleterious factors manifesting during the period between conception and birth. The size and shape of teeth, facial bones and skull base are always determined by genetic developmental programmes, therefore the treatment of genetic disorders is always more difficult and more complex than that of the acquired disorders. When taking the medical history and making the diagnosis these should be considered, especially when planning the retention phase.

1.1. Congenital disorders of the jaw and face (cheiloschisis and palatoschisis)

2. Congenital disorders of the dentition

2.1. Congenital disorders of the teeth

2.1.1. Hutchinson tooth

2.1.2. Amelogenesis imperfecta

2.1.3. Dentinogenesis imperfecta

2.1.4. Odontogenesis imperfecta

2.2. Numerary disorders

2.2.1. Hypodontia: hereditary hypodontia can be explained with Bolk’s theory of terminal reduction, according to which the last element of every tooth group disappears from the dentition. Most frequently the third molars, upper lateral incisors, upper and lower second incisors, second premolars and lower incisors are absent. Lack of one tooth is defined as aplasia, lack of several teeth is called oligodontia, an extended lack of teeth is anodontia partialis, complete lack of teeth is termed as anodontia totalis, which can affect both primary and permanent teeth. Multiple hypodontia can be associated with different syndromes (Down syndrome, ectodermal dysplasia triad).

**Figure 2.60. Figure 1. – Upper lateral incisor hypodontia**
2.2.2. Hyperdontia: causes involve development of more tooth buds in the dental lamina or one of the regularly formed tooth buds is split. They can be of regular shape and size, or smaller, conical, peg-shaped. Most frequently hyperdontia occurs in the area of front and molar teeth. Typical supernumerary teeth (dens supranumerarius) are teeth with regular tooth shape. Most frequently it affects premolars and upper lateral incisors. Atypical supernumerary teeth (dens supplementarius) usually occur in the upper jaw due to overproduction of the dental lamina. It cannot be compared to regular teeth, an example for it is the mesiodens. A radiographic examination is always required to make the diagnosis, on physical examination a bulge can be palpated on the palate or in the vestibular region. An inclination of the adjacent teeth may indicate the presence of supernumerary teeth. In most cases their removal is indicated.

2.2.3. Dens retromolaris: 4th molar

2.3. Locational disorders: can be due to supernumerary teeth, difficulties of eruption and lack of space.

2.3.1. Rotation, torsion (rotating around an axis).

**Figure 2.61. Figure 2. – Upper lateral incisor in rotated position**

2.3.2. Proclination/retroclination (axis orientation of the tooth is angled anteriorly or posteriorly, root apices stay in situ).

**Figure 2.62. Figure 3. – Proclined front teeth**
2.3.3. Transposition (two teeth change places)

2.3.4. Buccal, palatal, lingual occlusion/ version

2.3.5. Mesio-, distoocclusion/ version (following extraction the tooth moves to mesial or distal direction)

2.3.6. Infra-, supraocclusion/version (the tooth extends above the occlusal plane/ tooth is beneath the occlusal plane)

2.3.7. Impaction/ retention: can be due to supernumerary teeth, trauma, abnormal position of the tooth bud, lack of space. Most frequently it affects the third molars and upper canines. In impaction eruption of the tooth may be impeded due to some anatomical obstruction, the retained tooth does not show the signs of eruption.

**Figure 2.63. Figure 4. – Impacted canines following surgical exposure**
2.3.8. Ectopic tooth: the position of the tooth is located labially/buccally or palatally/lingually to the dental arch. Most frequently it occurs in the case of the upper canines. It is due to lack of space between the lateral incisor and the first premolar, as the canines erupt/change subsequently. In the lower arch due to similar causes the second premolars can be in ectopic position.

**Figure 2.64. Figure 5. – Canine ectopia**

2.4. Morphologic disorders
2.4.1. Micro-, macrodontia (abnormally smaller or bigger teeth)

2.4.2. Geminatio dentinum (due to splitting of the tooth crown of the tooth bud, common root, separated crown)

2.4.3. Fusion (due to the fusion of two tooth buds leading to the development of a tooth double in width).

**Figure 2.65. Figure 6. – Fusion of teeth**

2.4.5. Tooth concretion (fusion of the teeth in the cemented area)

2.4.6. Tooth dilacertion (due to some kind of trauma to the primary tooth, the root is formed in an angle to the crown)

2.4.7. Invaginated tooth (dens invaginatus: tooth-like formations inside the tooth)

2.4.8. Dens evaginatus (dens evaginatus: additional cusp on the chewing surface of the premolars)

2.4.9. Taurodens (elongation of the crown of the molars at the expense of the roots, over-sized pulp chamber)

3. Inherited disorders of bones

3.1. Structure and size of dental arches

The regular upper dental arch is elliptical, the lower dental arch is parabolic. Abnormalities in form may vary.

3.2. Disorders of the jaw

3.2.1. Prognathism/ retrognathism (maxilla is in an abnormal anterior/ posterior position)

3.2.2. Progenism/ retrogenism (mandible is in an abnormal anterior/ posterior position)

3.2.3. Micrognathism/ microgenism (upper jaw/ lower jaw is underdeveloped)

3.2.4. Macrognathism/ macrogenism (upper jaw/ lower jaw is bigger in size)
3.2.5. Asymmetry: there is a visible discrepancy between the two sides of the face correlating to the median-sagittal plane.

Disorders are usually combined, rather than being present independently.

II. Acquired disorders: any factors that interfere with the normal course of development lead to anomalies or aggravate the already existing disorder.

1. Caries and consequent diseases

2. Feeding of the new-born: due to active muscle contractions during breastfeeding, it is more advantageous than artificial feeding.

3. Nutrition: intake of vitamins, trace elements, minerals and proteins of proper amount and quality. Solid food, which require chewing are recommended, instead of soft food.

4. Position of the baby in bed: if the head is placed higher, there is an increased risk for mesial bite, whereas a lower position of the head is associated with distal bite.

5. Mouth breathing: before the onset of orthodontic treatment the cause of breathing through the mouth should be revealed. An otorhinolaryngologic examination and a consultation with a speech therapist are often needed prior to the treatment. Without cessation of the etiologic factor no result can be achieved by orthodontic treatment alone. The sooner the obstructed nasal breathing is noticed and treated, the less facial and dental anomalies can be expected.

6. Childhood oral habits: to a great extent, these are responsible for the development of different anomalies. These should be considered when planning the treatment and choosing the appliance. Managing to break oral habits during orthodontic treatment will facilitate the correction of orthodontic anomalies.

6.1. Using a pacifier: the developed anomalies are the same as that of digit sucking

6.2. Digit sucking: duration, intensity and frequency all influence the extent of anomalies. Prolonged digit sucking is always associated with open bite, speech disturbances, protrusion of upper incisors, retroclination of lower incisors and mandible, narrowing of the upper dental arch, gothic palate.

6.3. Nail biting: there is a peak in the incidence is between the age of 3-6 years and in teenagers. Presumably it is associated with stress. In childhood it may be a source of joy.

6.4. Bruxism: mainly psychological causes can be found in the background, however it can be due to systemic and local causes. Wearing off the tooth surfaces and joint pain can present as accompanying symptoms.

6.5. Difficulties swallowing. squeezing of lip during swallowing may cause increased overjet, while tongue thrust swallowing usually causes anterior open bite.

6.6. Premature extraction of primary teeth: when eruption of the permanent teeth cannot be expected six months following the extraction of the primary tooth. It contributes to the development of ectopia in the frontal region and to deep bite. Early removal of primary molars accounts for the mesialization of permanent molars.

6.7. Trauma: an injury to primary teeth may damage the tooth buds of permanent teeth. As a result eruption disorder, ankylosis, inflammation or tooth loss may occur. Most frequently the middle incisors are affected leading to severe aesthetic disadvantages.

6.8. Effects of general diseases on the development of malocclusion: consultations with pediatricians and endocrinologists are needed. Acute infectious childhood diseases and hormonal disorders may play a role in the development of different anomalies. Endogenous secretory disorders, such as diabetes mellitus, disorders of calcium metabolism, embryonic harms (teratogenic drugs, viral infections, irradiation) may provoke late eruption of the teeth and underdevelopment of the jaws.

6.2. Classification of dentition disorders

The classification described by E. H. Angle is used to make a dentoalveolar diagnosis. Its advantage is that it is simple, dentition disorders can be assessed even without X-ray examinations by visual examination during
physical examination, analysis of study samples on sagittal anomalies. Disadvantages of the classification should be regarded as well. Vertical and transverse examinations are also required to make the diagnosis. Functional anomalies are not involved in the classification which should always be considered when making the diagnosis. The position of the upper sixth teeth is not considered to be always ideal or constant as Angle had stated.

Angle’s classification:

1. **Class I**: the mesiobuccal cusp of the upper first molar should be aligned with the groove of the mesio- and centrobuccal cusp of the lower first molar, whereas the cusp of the upper canine is aligned in between the lower canine and first premolar.

   • I/1: this is Class I according to Angle’s classification method, however there is a narrow dental arch in the front region
   
   • I/2: upper incisors are inclined labially, combined with diastema

   • I/3: cross bite in the area of incisors.

   **Figure 2.66. Figure 7. – Cross bite of the front teeth**

   • I/4: cross bite in the lateral region.

   Cross bite: in anterior cross bite the one or more incisors misalign behind the lower antagonist teeth. In buccal lateral cross bite the upper molars are situated buccally to normal, in lingual cross bite they are situated more linguually to normal. The disorder can uni- or bilateral.

   • I/5: narrow dental arch in the lateral region

   • I/6: perfect occlusion, normocclusion.

The degree of **overjet** (sagittal overbite) can be determined with sagittal examination. In a normal overjet the incisal edge of the upper incisors are aligned 1-2 mm in front of to the lower incisors. The extent of an **overbite** (vertical overbite) can be determined with vertical examination. According to an appropriate overbite the upper incisors cover the crown of lower incisors in an extent of 1-2 mm.
Class I anomalies:

1. Deep bite: when overbite exceeds 2 mm. It can be of dental (shape of the teeth, axis of the teeth) or skeletal origin. Its most severe form is cover-bite, where the upper incisors completely cover the lower ones.

**Figure 2.67. Figure 8. – Cover-bite**

2. Open bite: in occlusion there is no contact between some of the lower and upper teeth or groups of teeth. It can be due to infantile swallowing, mouth breathing, digit sucking, but can be also of skeletal origin.

**Figure 2.68. Figure 9. – Open bite**
3. **Crowding:** can be primary (inherited), secondary (acquired), tertiary (multifactoral). It is important to clarify the dimensions (sagittal, transverse or both) of crowding since it largely influences the treatment plan. The most controversial issue of orthodontic diagnosis is whether extractions are needed or not in case of crowding. To make the right decision the degree of dental arch narrowing and the relationship between apical-coronal bases should be considered.

4. **Protrusion:** can be due to tongue thrust, a pronounced frenulum labii superioris, Bolton deviation.

5. **Median diastema:** mesiodens, thickened frenulum labii, proclined incisors can be found as underlying causes. A similar clinical appearance is present in the case of the disproportion between the size of teeth and the dental arch.

**Figure 2.69. Figure 10. – Median diastema**
6. Cross bite: if it is found in the front region, differential diagnosis from Class III is needed.

2. Class II: the mesiobuccal cusp of the upper first molar is positioned in front of the mesiobuccal cusp of the lower first molar, whereas the cusp of the upper canine onto or in front of the cusp of the lower canine.

Video 1.

Video 2.

Video 3.

Video 4.

Video 5.

- II/1. Proclined upper incisors with a major overjet. Underlying causes often involve enlarged tonsils, childhood oral habits (digit sucking). Typical symptoms of this disorder include mouth breathing, gothic palate, pronounced Spee curve, increased ANB in cephalometric measurements.

- II/2. Front teeth can be situated as follows:
  - upper central incisors retroclined,
  - all upper incisors retroclined,
  - ectopic upper canines.

Due to the location of upper incisors the mandible is often in distal position, in forced bite.

Class II/subtype: if Class II malocclusion is seen on one of the sides and Class I malocclusion is present on the other side. Joint examination is required due to asymmetry.

Deep and open bite can also be seen in Angle’s Class II. Along with sagittal deviations a narrow upper dental arch can also be observed, as a transverse problem.

3. Class III: the mesiobuccal cusp of the upper first molar is positioned behind the distobuccal cusp of the lower first molar, the cusp of the upper canine is positioned behind the cusp of the lower first premolar. When making
the diagnosis it should be differentiated from I/3 subdivision, in which only one or two incisors are in anterior crossbite.

• Pseudo-progenism: with some pressure applied onto the mentum, edge-to-edge relationship of the mandible can be corrected. In this case the axis of the incisors is atypical and during the cephalometric examination ANB angle is either negative or zero.

• Real progenism: compensational positioning of the incisor (upper incisors are proclined, lower incisors are reclined) and an extended mentum can be seen. ANB angle is negative, monkey space is sustained. Vertical deviations (deep and open bite) can also be found here.

Figure 2.70. Figure 11a. – Real progenism, frontal view

Figure 2.71. Figure 11b. – Real progenism, lateral view
Treatment of orthodontic disorders is more effective and treatment time is shorter when they are detected on time. It is worth starting the therapy before the disorder is fully developed. Treatment of inherited disorders is always more complicated, requiring more time and showing an increased tendency of relapse. Some parts respond to effects exerted on them in different ways during the developmental process, therefore they vary in their adaptability to impacts. This explains why orthodontic work is difficult: the orthodontist needs to find the appropriate therapeutic appliance and method -in a system made up of elements responding differently to impacts- which result in a harmonious, aesthetic and functionally stable dentition.

Questions:

- Define lack of tooth buds, what types are there?
- Define overjet and overbite!

7.2.7. The diagnostic methods and treatment plan of orthodontic anomalies – Zsuzsanna Gurdan

Irregular dentition provides an unfavourable impression regarding facial aesthetics and beauty. Besides aesthetics there has always been a major emphasis on functional aspects. Functional disorders may be associated with speech disturbances, impaired mastication, resulting in nutritional difficulties and later digestive problems. The particularity of orthodontic treatments starting in childhood is the influence on growth direction and size of dentofacial structures during the growth period. Directing growth can result in more harmonious features and provides conditions for proper muscle functioning.

Orthodontic diagnosis aims at detecting skeletal and dental discrepancies of the orofacial region and at preparing the treatment plan based on these.

The necessary results required to make the comprehensive diagnosis are provided by medical history, clinical examination, radiographic-cephalometric analysis, photo analysis and model analysis.

7.1. Medical history

During the first meeting with the patient we get to know the main complaints and etiological factors. It consists of two parts: general and orthodontic history taking.
1. Taking the general medical history starts when the patient presents to the doctor. We observe his/her movements, stature, and in children we can have an impression on prospective cooperation. It is of utmost importance to gather information on general health status, immunizations, allergies, sensitivity to drugs, current medications, major health problems and oral habits (digit sucking, lip biting, tongue thrusting). To earn the trust of the patient conversation is the primary consideration. While we are learning about his/her habits (especially wind instruments and sports) different speech disturbances may be revealed.

2. Orthodontic history taking aims at collecting information on facial profile, temporomandibular joint, previous orthodontic treatments, injuries to the teeth, eruption sequence, time and course of shedding teeth.

**Figure 2.72. Figure 1. – Past medical history**
2. Pediatric dentistry and orthodontics

**MEDICAL HISTORY**
(Child/Adolescent)

- **Patient name:**
- **Birth date:**
- **Name of your child's physician:**
- **Address of your child's physician:**
- **Office phone:**
- **Date of last examination:**

1. **Is your child in good health?**
   - Yes
   - No
   - Don't know

2. **Does your child have a health problem?**
   - Yes
   - No
   - Don't know

3. **Has your child ever been hospitalized or had general anesthesia or emergency room visit?**
   - Yes
   - No
   - Don’t know

4. **Are your child's immunizations up to date?**
   - Yes
   - No
   - Don’t know

5. **Does your child have allergies to medications (drugs), medical products (latex), or the environment (dust, mites, pollen, mold)?**
   - Yes
   - No
   - Don’t know

6. **List past medications taken by child:**

7. **List daily medications child is now taking:**

8. **Has your child ever had or been treated by a physician for any of the following:**

   **Check one for each condition**

<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
<th>?</th>
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<tbody>
<tr>
<td>a. Problems at birth</td>
<td>p. Cancer</td>
<td></td>
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<tr>
<td>b. Heart murmur</td>
<td>q. Cerebral palsy</td>
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<tr>
<td>c. Heart disease</td>
<td>r. Seizures</td>
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<tr>
<td>d. Rheumatic fever</td>
<td>s. Asthma</td>
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<tr>
<td>e. Anemia</td>
<td>t. Cleft lip/palate</td>
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<td>f. Sickle cell anemia</td>
<td>u. Speech or hearing problems</td>
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<tr>
<td>g. Bleeding/hemophilia</td>
<td>v. Eye problems/contact lenses</td>
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<td>h. Blood transfusion</td>
<td>w. Skin problems</td>
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<td>i. Hepatitis</td>
<td>x. Tonsil/adenoid/sinus problems</td>
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<td>j. AIDS or HIV+</td>
<td>y. Sleep problems</td>
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<td>k. Tuberculosis</td>
<td>z. Emotional/behavior problems</td>
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<td>l. Liver disease</td>
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<td>m. Kidney disease</td>
<td>bb. Growth problems</td>
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<td>n. Diabetes</td>
<td>cc. Attention deficit disorders</td>
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<tr>
<td>o. Arthritis</td>
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</table>

9. **Has your child had any recent rapid growth?**
   - If so, how much?

10. **Parents:**
    - (Father) Ht: ______ Wt: ______
    - (Mother) Ht: ______ Wt: ______

11. **Older brothers and sisters:**
    - (1) Ht: ______ Wt: ______
    - (2) Ht: ______ Wt: ______
    - (3) Ht: ______ Wt: ______

12. **Females: Has menstruation begun?**
    - If yes, when? ______
    - Pregnant? ______

13. **Using birth control pills?**

14. **Child's grade in school:**
    - Child's school

15. **Do you consider your child to be (check one):**
    - [ ] Advanced in learning
    - [ ] Progressing normally
    - [ ] Slow learner

**DENTAL HISTORY**

16. **What is your main concern about your child's dental condition?**

17. **Has your child been to a dentist before?**
    - No ____ Yes ____ If yes, date of last visit:

18. **Regular dentist's name:**

19. **Check one for each condition:**

<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
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<tbody>
<tr>
<td>a. Has your child ever had dental x-rays? Date of last x-rays?</td>
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<tr>
<td>b. Will your child be uncooperative? If yes, explain</td>
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<td>c. Has your child experienced any complications following dental treatment? If yes, explain</td>
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<td>d. Has your child had cavities and/or toothaches?</td>
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<td>e. Are your child's teeth sensitive to temperature or food?</td>
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<tr>
<td>f. Did you or your child ever get instructions in brushing?</td>
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<tr>
<td>g. Do your child's gums bleed when brushed?</td>
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<tr>
<td>h. Does your child use fluoride products: rinses, drops, tablets?</td>
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<tr>
<td>i. Does or has your child had any clicking or pain in the jaw joint?</td>
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<tr>
<td>j. Does or has your child had any problems opening or closing the mouth?</td>
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<tr>
<td>k. Has your child inherited any family facial or dental characteristics? If yes, explain</td>
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7.2. Clinical examination

Clinical examinations involve three fields: general, extraoral and intraoral parameters.

1. General properties (individual parameters)
   - mental development of the patient,
   - stature,
   - body weight,
   - body height,
   - parents-child relationship,
   - posture and head: examination of these is important since the posture of the patient depends on the balance of muscle tone and the way one holds his/her head influences mandibular position at rest.

2. Extraoral examination (characteristic features of the face and soft tissue properties)
   - examination of the profile: examined when head and trunk are straight, the Frankfort plane should always be horizontal.
     - frontal view: brachycephaly, mezocephaly, dolichocephaly,
     - aesthetic parameters of the face: Steiner’s sign, Ricketts’ aesthetic line, E. H. Angle’s ‘Line of Harmony’, nasolabial angle,
     - facial prognathism: orthognath-retrognath-prognath,
     - anterior facial height index.
   - examination of the lips: length and width of the lips, lip closure, examination of lips profile (definition of Korkhaus), relation of upper incisors to the lower lip, gummy smile.
   - nose: size, nature of glabella, width of nostrils.
   - chin prominence.

3. Intraoral examination is made up of three main areas: dental, dentoalveolar, soft tissues.

3.1. Dental examinations:
   - status, oral hygiene (orthodontic treatment can only begin following the required periodontal, cariologic and prosthetic care),
   - number of teeth (supernumerary teeth, hypodontia),
   - abrasion, attrition,
   - dental, skeletal, chronological age,
   - occlusion,
   - midline,
   - overbite, overjet,
   - Angle’s molar-canine relationship,
• dental discrepancies regarding shape and numbers.

3.2. Dentoalveolar examinations: the relation of apical basis (an imaginary line connecting the apices of labial segment and apices of buccal segments) and coronal basis (a curve interconnecting the incisor edges of front teeth and the buccal cusps of occlusal surfaces of lateral teeth) is a key issue in making the proper decision regarding tooth extractions.

3.3. Examination of soft tissues: lips, palate, gingiva, bucca, tongue and the treatment of pathological disorders.

7.3. Functional analysis

The examination of the temporomandibular joint (TMJ) is an important step in diagnostics. Orthodontic treatments do not aggravate the already existing anomalies, however it is necessary to diagnose joint problems prior to the treatment. Our treatment plan may be modified and prior to the intervention we refer the patient to a TMJ specialist.

1. Prior to the physical examination enquiries should be made regarding:
   • any difficulties, pain when opening the mouth, chewing or speaking,
   • frequency of headache, neck pain, toothache,
   • joint clicking, popping, pain recently,
   • any previous treatment due to painful joints.

2. Physical examination

2.1. Auscultation: auscultation with a stethoscope during lateral and anteroposterior movement.

2.2. Palpation: examination of m. pterygoideus lateralis, m. temporalis, m. masseter, if they are tender on palpation.

2.3. Functional examination of TMJ: during lateral and opening-closing movements. Palpating the joint is important both when opening the mouth and when closing it. Any deviations of the mentum, asymmetry when opening the mouth should be regarded. Measuring the maximal distance between the incisors is also important, and it should not exceed 40-45 mm.

2.4. Radiographic examinations should only be performed when indicated.

7.4. Examination of functional anomalies

1. Examination of deglutition: infantile, transitional, mature swallowing.

2. Examination of tongue dysfunction: tongue thrusting, high/low positioning of the tongue, macroglossia, squeezing the lips during swallowing.

3. Examination of lip dysfunction: incomplete closure of lips, excessive activity of m. mentalis.

4. Examination of buccal dysfunction: sucking the bucca, chewing on it.

5. Examination of breathing: mouth breathing (“adenoid facies”).

7.5. Photo analysis

1. Extraoral photos: frontal view (rest, smile) - lateral view.

2. Intraoral photos: frontal view, lateral view from both sides, occlusal view.

7.6. Radiographic examinations
An indispensable element of orthodontics is a radiographic examination to make the diagnosis, to provide prognosis and to evaluate changes.

1. **Periapical image**: examination of the teeth, tooth buds regarding any disorders in position, shape, numbers

2. **Orthopantomographic image**: maxilla and mandible can be seen together. Position of joint, teeth, symmetries and other anatomical structures can be visualized.

**Figure 2.73. Figure 2. – Orthopantomograph**

3. **Posteroanterior cephalometry**: facial bones and bones of the skull can be seen in the posteroanterior image. When adjusting the head position, Frankfort plane is perpendicular to the film. Midline anomalies (skeletal and dental), asymmetries, intermolar and intercaninal distances can be examined.

4. **Radiographic image of TMJ**: structure and form of condylar head of the mandible can be analysed.

5. **Carpal index** (growth stages can be determined based on the radiograph of distal epiphysis of radius and ulna, carpals and metacarpals) is not used anymore. This method is replaced by T. Baccetti’s (1966-2011) method of examining vertebral maturity, which helps assess the skeletal maturation of the patient without performing extra radiographic examination.

**Figure 2.74. Figure 3. – 6 stages of cervical vertebra maturation**

6. **Cephalometry**: with its help the sagittal and vertical dimensions of the skull, diagnostics of craniofacial deformities and development of the skull can be observed. During radiographic imaging the following aspects should be considered, otherwise the image cannot be evaluated:

- teeth should be in central occlusion,
- Frankfort plane should be horizontal,
• lips should be in natural position,
• all metallic objects should be removed.

**Figure 2.75. Figure 4. – Cephalometric radiograph**

![Cephalometric radiograph](image)

After the overview of the cephalometric radiograph the following steps are the tracing of the image and marking the cephalometric landmarks.


Angular measurements determine the axis of front teeth, mandibular growth, maxilla – mandible relationship and their vertical, sagittal relationship to the skull.

Angular measurements:
• SNA, SNB, ANB, SNPg.
• NSBa, Gn-tgo-Ar angle, Norderval (N) angle,
• NL-NSL, ML-NSL, ML-NL angles,
• Interincisal angle, I-Na, T-NB angles,
• Holdaway (H) angle.

Linear measurements: I-Na, T-NB, T-NPg, Pg-NB, N-Sp’(anterior, superior facial height), Sp’-Gn (anterior facial height), N-Sp’/Sp’-Gn · 100 = 100 = facial height index.

**Figure 2.76. Figure 5. – Cephalometric analyses**
### Figure 2.77. Figure 6. – Hasund’s harmony box

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2. Pediatric dentistry and orthodontics
Most frequently used cephalometric analyses are those from Ricketts, Steiner and Hasund. Hasund’s analysis is popular and applied also in the everyday work of our clinic.

The point in Hasund’s cephalometry is that it implements harmony within the given facial type considering the harmonic relations between the structures in every patient. Its unique, advantageous feature is the concept of floating norms. The value of all five variables changes linearly in the Hasund chart and the ratios of a harmonious face are termed by Hasund as floating norms. Every horizontal line contains the basal indicators of a harmonious facial type. To define facial harmony there is an edited harmony schema available that is placed on a chart containing the patient’s indicators. A subject whose values lie inside the schema displays a harmonious facial pattern. The horizontal axis of the schema is the “harmony line” indicating the patient’s facial type (orthognathic, retrognathic, prognathic).

Results are correlated to normal values, which according to Gauss curve, display scattering. Serial examinations allow the analysing of the consequences of growth and treatment (changes in soft tissue, dentoalveolar changes and mandibular rotation).

At our clinic there is an orthodontic diagnostic analysing software available which assists the assessment of the images (Smile for Windows3.0, Markella Zsolt).

During treatment, the superpositioning method of the cephalometric radiographs taken of the patient, previous data can be compared.

Factors to be examined during cephalometric analysis:

- Facial type: maxillary and mandibular prognathism can be determined sagittally (SNA, SNB angles. Vertical (ML-NSL, NI-NSL angles).


- Growth analysis: it is important regarding spontaneous growth, since the direction of growth can be influenced with therapy. (form of condyle, arch of canalis mandibulae, inferior outline of mandible, symphysis inclination, anteroinferior facial height, degree of gonion angle, rotational types of the jaw).

- Cephalometric norms of incisors: should be aligned with the basal facial structure detecting the presence of pathological muscle function.

- Soft tissues: are not in concordance with the bony anomalies beneath. Holdaway’s soft tissue angle, nose, lip profile.

- Dentoalveolar norms: model analysis, Steiner box.

**Figure 2.78. Figure 7. – Steiner box**

### 7.7. Computed tomography

Computed tomography: computerized cross-sectional imaging. It is suitable to detect cancerous, inflammatory diseases of the skull, to diagnose genetic malformations and different processes of the bones.
7.8. Model analysis

**Model analysis**: based on the study cast made by taking an impression with orthodontic impression trays and alginate impression material. The followings are examined:

- Edward H. Angle’s diagnostic system examining the mesio-distal relationships.
- Shape of dental arches, that can be arrow-headed, U shaped, elliptical or parabolic.
- Position of the teeth within the dental arch should also be regarded: crowding, normal, spacing, lack of teeth, symmetries and asymmetries.
- By using Pont’s index we can determine the degree of narrowness of the dental arches. The total of the greatest width of the four upper incisors is multiplied by 100 and divided by 80, we get the degree of ideal distance between the upper first premolars. This value is compared to that, measured by us. We get the transversal difference of the dental arch in millimeters. This measurement can be performed also in the molar region with changing the measurement points. Instead of 80, there are 64 in the denominator of the formula. The degree of narrowness of the dental arch is minor until 5 mm, average above 5 mm and major above 8 mm.
- Length of the anterior dental arch can be defined with the help of Korkhaus Index. The total width of four upper incisors is multiplied by 100, divided by 160, providing us an ideal value, which can be compared with the values measured by us.
- Transversal examination of symmetric relationships is done by determining the midline. Tuberal plane is authoritative in the anteroposterior direction.

**Figure 2.79. Figure 8. – Midline shift**

1. Assessment of local relationships:

- *Moyers’ analysis* on mixed dentition: from the mesio-distal diameter of the four lower incisors we can conclude to the required space of the lower and upper permanent supporting zone. The chart indicates the space demand for canines and premolars with a possibility of 75%.

**Figure 2.80. Figure 9. – Moyers table**
In Gross’ analysis on mixed dentition we can conclude to the space demand of permanent teeth using a multiplex regressive equation.

\[ Y_{33-36} = 0,63 \cdot X_{32} + 0,84 \cdot X_{32} + 0,67 \cdot X_{36bl} + 4,47 \]

**Figure 2.81. Figure 10. – Gross equation**

A disadvantage of this analysis is that it provides results regarding only the lower dental arch and only one side at a time.

**Bolton analysis** provides the ideal ratio of upper-lower teeth from the total of their width in eugnath dentition. According to Bolton, proper alignment of teeth (without spacing or crowding) in the dental arches is provided by the ideal width of the teeth. We can calculate the full ratio in permanent dentition, in mixed dentition the front ratio is applied.

**Figure 2.82. Figure 11. – Bolton table**

Its practical significance is the following: if there is a 2 mm difference between the measured and ideal values, discrepancies can be solved by interproximal stripping (reduction of the mesial and distal approximal surfaces of teeth) of lower incisors. Extraction is needed if the difference is 4 mm or >.

- Dental arch discrepancy is measured in permanent dentitions determining the degree of crowding or lack of space. The total of mesio-distal diameter of all teeth in a jaw and the ratio of the length of the complete dental arch.

- Determining of intercaninal distance cannot be altered during the treatment, otherwise relapse can occur.

- **Overjet**: measured parrelly with the occlusal plane, it is determined by the distance between the vestibular surface of the lower central incisor and the edge of the upper middle incisor.

- **Overbite**: measured parrelly with the occlusal plane, the difference in mm between the vestibular surface of the lower incisor edge and the palatinal surface of the upper incisor.

Based on all the information collected when making the diagnosis, a list of problems and individualized treatment plans are set up with the help of a thorough analysis. Also general dentists play a major role in the early detection of anomalies. Prevention of anomalies is difficult, since a major part of them are inherited. If anomalies are revealed in mixed dentition periods, prevention of a fully developed anomaly can promote development favourably (interceptive orthodontics). When treating fully developed anomalies, limits and difficulties of the treatment should be considered (curative orthodontics). Treatment aims at reaching a harmony
between aesthetics and function. This harmony refers to the foursome of facial profile, function, occlusion and articulation. Prior to the initiation of the treatment, conversation between a child patient together with his/her relative and the doctor is of utmost importance. Patients should be informed of treatment modalities, possible side-effects, changes of eating habits and speech, financial issues, time of wearing, frequency of check-ups. Orthodontic treatment can start after a written consent and all documentations will be retained even after the end of the treatment.

*Questions:*

- What are the aspects of an extraoral examination?
- What methods are suitable to assess space relationships?

### 8. 2.8. The methods of orthodontic treatments – Zsuzsanna Gurdan

A diagnosis should be established prior to every medical intervention. Orthodontic diagnosis is a complex list of abnormalities involving facial structures, location of jaws, relationship of dental arches, location of some tooth groups and abnormal location of teeth. For an accurate list of these errors, all diagnostic steps should be completed to avoid mistakes. The treating physician should be familiar with the facial character of the patient, skeletal relationships and dento-alveolar discrepancies in all the three dimensions.

During the therapy phase the aim of the treatment should be defined ranging from the realignment of certain teeth to the complete rehabilitation of the complex masticatory apparatus. It is recommended to divide the treatment plan into shorter phases and discuss them with the patient or relatives to help them to understand the processes better and to achieve better cooperation.

Diagnostics of dental deviations have constituted a standardized system since Angle’s classification method. Its significance comes from the discovery of Angle of relationships between anatomic discrepancies and functional disorders and from the fact that patients seek medical advice due to orthodontic anomalies resulting from sagittal deviations. Patients do not recognize bone deviations, they never see their own profile, they always see their look facing the mirror therefore they formulate their wishes based on these looks. Similarly, judging the smile happens through this facing aspect, however the orthodontist knows well that the treatment result is to interfere with all three dimensions.

The available space of tooth movement is being determined upon the making of the diagnosis. The dentoalveolar arch of both the lower and upper jaw is horse-shoe shaped, is covered and lined by cortical bone layer and the teeth are located in the articulation within the trabecular bone, the gomphosis. The natural positioning of teeth has not changed much throughout the evolution, the alignment of cusps and interdental spaces is the same in all species, since they are only able to function this way. Therefore, following the rules of our orthodontic forefathers is of utmost importance, as they proceeded from observations of nature.

**Figure 2.83. Figure 1. – Dentition of dogs and piranhas**

Characteristic features of human dentition:
1. In complete dentition three teeth make up an occlusal unit, except for the lower central incisor and the upper third molar.

2. Ideal interincisal angle is cca. 130, regardless of the form of the head and face.

3. Positioning of the teeth is defined by the prosthetic space, its size and shape change as children grow but remains practically stable in adults.

4. Teeth are positioned in their place spontaneously (tilt, occlusal plane, Curve of Monson and Spee, etc.) during eruption according to anatomical and functional circumstances. In an optimal occlusion co-functioning of three joints (left and right temporomandibular joint and dental articulation) can be observed. (see gnathological skills).

5. Expectations following a completed orthodontic treatment were defined by Andrews, termed as the “six keys to occlusion”, which has been further developed since then, mainly according to the observations of Roth and Williams. It should be emphasized that during an orthodontic treatment an “artificial” condition is created that still changes significantly after the active period of moving the teeth, especially in children, whose development has not come to an end yet.

Treatment plans should be carried out regarding all the above mentioned aspects, including the objective and subjective limits of the treatment.

**Treatment of Class I discrepancies**

Characteristic features of this type of anomalies are that there is neither sagittal deviation nor does the skeletal configuration shows significant aberration. Problems are mainly due to dento-alveolar deviations. One of the most frequent ones is:

1. **Crowding**: lack of space.

   There are two ways of treating this problem:

   1. Using the maximum space, available, that is formed by nature and in an inherited way.

   2. Creating more space artificially by expanding into the appropriate direction of space or extraction of optimally chosen tooth/teeth.

   Most frequently this problem is caused by the discrepancy between the size of the jaw and the size of the teeth (primary crowding - inherited).

   In other cases, the mesio-distal size of erupted teeth decreases due to decay or tooth loss narrowing the originally sufficient space (secondary crowding - acquired).

   During the development of children, the evolving dentition preserves its adaptability towards changing circumstances, therefore any later impact results in a change. It is well known that the positioning of incisors is not final even after the eruption of permanent teeth, the interincisal angle is altered by an increased muscle tone originating from the force exerted from the muscles of lips, leading to an increased interincisal angle, that in some cases may result in crowding (tertiary crowding - late).

   A variety of appliances are available ranging from removable to fixed appliances. The mesiodistal dimension of the primary molars is larger, than that of the permanent successors (premolar), providing us with space (leeway space), that may be used. In a crowding of lesser extent this space can be adequate to solve the problem but the gap should be closed backwards and forward (retention appliances).

   Rhiss space is often used without appliances, by interproximal reduction of primary molars, thereby providing enough space for the erupted permanent teeth. It is recommended to perform expansions and extractions of permanent teeth based on a consultation with an orthodontic specialist.

2. **Excess of space**, forms include proclined teeth or spacing between the incisors.

   In differential diagnosis it should be differentiated from Class II /1 anomalies, which is often not easy. If there is an inherited small size of teeth present, treatment of the anomaly becomes impossible, since there would be a greater lateral gap after closing a midline diastema and vice versa: the gap will be larger in the middle. At last
but not least, there is a chance for relapse in the achieved results. Prior to the treatment, a model analysis is indispensable to be able to inform the patient of the expected results. It is important to note that a good supporting zone is not to be ruined in favour of gap closure.

3. **Cross bite.** It can found in any regions of dentition, commonly alongside with some other anomalies.

   In front teeth it can be due to early loss of primary teeth or abnormal positioning of the tooth bud. To direct erupting teeth into a good direction, **wooden spatulas** can be used, held in a 45° angle and chewed by the child for several minutes many times a day.

   Another possibility is to use an inclined plane, which needs special handling and care in every case. It may be only used with cautions, because it may overload of the temporomandibular joints and open the bite. There are different removable appliances with expansion screws that are able to move teeth into the proper direction within a few weeks’ time. The use of fixed appliances for a partial or complete dental arch may also be indicated.

   In lateral cross-bite the upper teeth can be in a vestibular position compared to the lower teeth or on the contrary, can be located orally so as the buccal cusps bites on the lower central groove, or more orally within the line. The three cases should be treated only following the exact diagnosis, since the discrepancy can result from forced bite or facial asymmetry.

4. **Deep bite** is where the upper teeth cover the lower ones by more than 2 mm. Because of didactical reasons deep bite should be differentiated from increased deep bite of Class II that is due to distal bite and from lowered bite that can be caused by continuous abrasion or by the loss of teeth in the supporting zone. Deep bite can evolve from morphological variability of teeth and also from steep axis positioning.

   Therefore, treatment of front teeth can be confined to axis correction and intrusion of incisors. In some cases extrusion of the supporting zone can settle the problem by levelling the curve of Spee.

5. **Open bite** is the situation where the upper and lower teeth are not in contact. In the case of dental anomaly treatment includes the cessation of bad habits (digit sucking, pacifier, tongue thrusting), since in many cases the elimination of the etiologic factor solves the problem. Age of the child, mental development and psychic conditions all should be regarded in these interventions.

   Vestibular plates can be used with great efficacy along with some other removable appliances.

   Intrusion of the supporting zone can lead to adequate results in skeletal discrepancies, however extrusion of front teeth should be avoided.

**Treatment of Class II discrepancies**

   Characteristic features of this anomaly involve sagittal discrepancies of the upper and lower teeth resulting from teeth or jaws position. Also Angle had distinguished the two subtypes according to the axis of the incisors. Subgroup II/1 is often characterized by mouth breathing, progenism of the upper incisors, narrow, gothic palate. Mouth breathing is not present in subgroup II/2, dental arches are wide and crowding of the front teeth can be observed.

**Figure 2.84. Figure 2.** – Typical clinical appearance of Class II bite
Based on European traditions, especially in growing children different bimaxillary functional appliances are used, with appropriate accessories they can even be used in orthognatic treatments of the jaw. It is important to note that due to acceleration, unlike dental age, the development of the body finishes at younger ages. Consequently, growth tendencies needed for the treatment are available only at young ages.

Fixed appliances can be successful if bite modifying appliances are used in between the upper and lower teeth. These can involve a simple elastic rubber bands, or special bite modifying appliances fixed extra- or intraorally.

When growth is finished, treatment can be continued with extraction of premolars or some type of orthognathic surgeries.

III. Treatment of Class III discrepancies

Anomalies are primarily due to inherited factors, which are most likely passed on by dominant genes. Mesial growth of the mandible significantly exceeds that of the maxilla, however it can also be due to underdevelopment of the maxilla. Aim of the treatment is cessation of the real cause, thus it is very important to identify jaw responsible for the anomaly. While growth direction of the maxilla and to a certain limit the size of the maxilla can be influenced, mandibular growth cannot be interfered with. Due to dental compensations, upper incisors are inclined forward, lower incisors are inclined inward. Therefore the distance between incisal edges is reduced.

Functional appliances can be well used in the treatment of mesial bites. Almost every author has an appliance to treat Class III discrepancies (Progenic activator, Fränkel III appliance, Bionator, Hansa).

If the maxilla is smaller and located skeletally more backward, a maxillary orthodontic treatment can be performed in the growth period wearing a so called reverse headgear (Delaire mask). In many cases, when the above mentioned treatment modalities do not produce satisfactory results, wearing these appliances is still worth it, until the end of growth period. Their wear may prevent the formation of negative sagittal overbite- that may not even be corrected with orthognatic surgery- by keeping the jaws together.

**Figure 2.85. Figure 3. – Wear of a Delaire mask**
As conservative treatment of inherited disorders is limited, after growth only surgical interventions can be considered.

A special type of mesial bite develops not in an inherited way, but is due to bad oral habits or forced bite, during which the mandible slips forward in the last phase of closing the teeth (pseudo-progenism). The skeletal ANB value is not negative, upper incisors are inclined inward, lower incisors outward, with a pressure to the mentum incisors can be positioned into an edge bite. The anomaly can be treated well by the aligning of the incisors.

Methods of orthodontic treatments vary according to geographical location of countries and the preferred treatment modalities of the experts. Standardized templates cannot be set up, since every case differs from the other and requires in individualized therapy. During the history of orthodontics the schools playing a decisive role in the development of this discipline are well known and this diversity enables the practicing orthodontist to choose the optimal treatment modality for the patient.

9. 2.9. Complex orthodontic cases – Judit Rostasi-Szabo

“Complex orthodontic treatment” is referred to cases where comprehensive treatment is carried out in cooperation with other fields of dentistry, to restore oral health.

1. The first and most important relationship is the cooperation between orthodontics and pediatric dentistry.

This connection is related to the pedodontist, who follows the child from preschool age until adulthood. The task of the pediatric dentist in addition to cariologic care, also involves the examination of the development of all structures of the oral cavity, continuous control of anomalies, observation of dentition sequence, natural formation of the dental arches, occlusal changes and functions. Speech disorders may also be discovered.

In the case of anomalies in tooth number and position the pediatric dentist consults the orthodontist, but simple cases may be treated by themselves. (See below.)

2. Preprosthetic orthodontic treatment is more and more common nowadays, as a cooperation with a prosthodontist. It cannot be overemphasized that -tooth loss is not an orthodontic problem. So basically replacing the missing teeth is the task of the prosthodontist. However orthodontic anomalies may occur as a sequelae of tooth loss (elongation, drift, inclinations). Better prostodontic results may be achieved in these cases if the teeth are aligned in a more favorable position in cooperation with an orthodontist in patients with no previous orthodontic anomalies. The case is different if previously abnormal occlusion was already present in the patient. In these cases the two professions must work together during the dental rehabilitation of the patient. The abnormal occlusion should be corrected prior to prostodontic treatment.

3. There is also a great need for cooperation between orthodontics and periodontology. Abnormal position of the teeth itself may cause periodontal disease, or may aggravate periodontitis. It is an important fact that by moving a tooth, periodontium rebuilds, the statically weakened teeth have chance to regain the ability of chewing. Cooperation of the two professions have to begin before the treatment, and continue on during
orthodontic treatment, because fixed orthodontic appliances lead to a difficulty in maintaining oral hygiene, toothbrushing is harder, more plaque is present. Any periodontal inflammation during orthodontic treatment may lead to failure.

4. Cooperation between dentoalveolar surgery and orthodontics plays an important role in daily routine care, such as moving teeth to their right position with surgical-orthodontic treatment, removing supernumerary, impacted, retained teeth, minor soft-tissue surgery or orthognathic surgery of the jaws. For successful treatment cooperation is necessary during the making of the diagnosis and the treatment plan.

9.1. Cooperation with pediatric dentistry

In accordance with the previously described, the aims of the cooperation are to reach a healthy deciduous and permanent dentition, to control and facilitate a harmonic alignment of the dental arches during development regarding the esthetic and harmonic functions of the face, to achieve a healthy periodontium, and a well-functioning temporomandibular joint. This is a part of prevention, but also involves interceptive orthodontic interventions as well. The pediatric dentist meets the child patients most frequently during regular checkups.

**Figure 2.86. Figure 1. – The aim of orthodontic treatment**

1. Healthy deciduous teeth are necessary for further dental formation. In the period of permanent tooth eruption a gaps appear between the deciduous teeth as a consequence of jaw growth, thereby providing space for the permanent, wider incisors.

To maintain the normal sequence of tooth eruption and to extinguish initial (natural) crowding of the teeth is the task of pediatric dentistry. Interproximal reduction (stripping) may provide enough space, thereby eliminating the need for orthodontic appliances.

In the case of severe crowding the orthodontist must take part in the work.

2. Due to bad cariologic conditions of the Hungarian children deciduous teeth are often removed, when crown of the tooth cannot be restored. Prefabricated crowns are suitable for restoring the ability of chewing and the size of the teeth.

Deciduous teeth play a role in maintaining the space for the permanent dentition. If the early extraction of the deciduous teeth is necessary, space maintainers should be used. The early extraction of the deciduous teeth – more than half a year before the normal exfoliation - causes secondary constriction of the dental arch. The space maintainer can be a local partial tool (fixed), a lingual-palatal arch, expanding the whole denture, or a simply removable retention plate. In the case of smaller crowding, “lip bumper” can still ensure the sufficient place.

**Figure 2.87. Figure 2. – Deciduous molars restored with NiCr crown**
Figure 2.88. Figure 3. – Lingual arch appliance

Figure 2.89. Figure 4. – Lip Bumper appliance
3. If the dentition is not corresponding to the age of the child, it should be recognized during checkups. It may be caused by aplasia, supernumerary tooth, ectopic eruption, impaction, retention, ectopic resorption deciduous roots, or abnormal size (smaller, bigger), morphology of the teeth, or premature or delayed eruption. In these cases radiographic examination is necessary. Depending on the diagnosis, with the cooperation of the other dental professions, the problems can be solved. It must be emphasized that early recognition is essential in the success of the treatment. Some authors suggest a radiographic control examination at the age of 6 and 10 years, to assure early diagnosis.

4. Graduate and postgraduate dental education involve orthodontic studies, thereby anomalies regarding a tooth, a group of teeth, or the whole dentition can be recognized easily, and the pediatric dentist may refer the patient to an orthodontist in time, after the teeth are restored (if necessary).

9.2. Cooperation with prosthetic dentistry

1. The main difficulties of treatment of anodontia in childhood are the continuous growth, development and morphological changes, which may be ceased by dental interventions, causing iatrogenic disorders.

Fixed prosthodontic appliances are postponed because of the wide pulp chambers and incomplete root formation. Temporary solutions are not able to prevent the atrophy of the alveolar bone, thereby surgical-prosthetic treatment may be needed.

A previously mentioned, the replacement of missing teeth is not an orthodontic problem, but in some cases closing the gap from the right direction can be a treatment - if the intercuspitation permits that. Regarding the children the time from which they wear a foreign material in their mouth should be considered. These procedures should considered as a compromise. Undesirable functions may appear primarily in the temporomandibular joint, leading to its dysfunction. Fortunately children have the ability to adapt easily, thus taking gnathological aspects into consideration, and with sufficient experience we can achieve good results. When the replaced tooth shape cannot be physiologic, it may be changed, in accordance with esthetic and functional aspects. Asymmetry should be avoided due to the, because of the asymmetric movements of TM joint, as described earlier.

**Figure 2.90. Figure 5. – Bilateral aplasia (lateral incisor)**
Expansion of the space according to the size of the contralateral tooth, before prosthodontic treatment is mainly needed in the case of asymmetry. In most of the cases this may be solved with partial appliances, but sometimes the movement of the total dental arch may be necessary. Our aim should be the treatment in accordance with the aspects mentioned above.

If this cannot be achieved without tampering with the intercuspidation, removing the contralateral tooth and closing the gaps may be the choice of our treatment.

Based on the current approaches of implantology, implantation is childhood is not considered to be a permanent solution. The vertical growth of the alveolar process present in a child is ceased in the area of the implant. After a period of time the replaced tooth will be relatively shorter, compared to the natural teeth. This cannot be corrected always esthetically by the changing of the abutment. An implant may be used as an orthodontic anchorage reinforcement (temporary anchorage devices- TAD), because they are not mobile.

Implants in the front region may cause further esthetic problems, as it has a greyish transparency through the gingiva. Autotransplantation is a better choice. Autotransplanted teeth may show signs of vitality, perfect esthetic and functional results. Later they can be moved in their optimal position with orthodontic treatment. The risk of autotransplantation includes ankylosis, root resorption, which may lead to tooth loss.

2. The difficulties of childhood are not present in adulthood prosthodontics. The role of the orthodontist is only secondary. In these cases the aim of this team work is to restore the occlusion, fulfilling the functional and esthetic requirements. The task of the orthodontist regarding the position and the movement of the tooth is dependent on the treatment plan of the prosthodontist, by taking the dental and gingival anchorage and support into account.
In most of the cases this means the correction of the tooth axis, the intrusion of elongated teeth, occasionally raising or lowering the bite. To achieve the planned result in adults, fixed orthodontic appliances are needed. In some cases orthodontic temporary anchorage devices (mini-implants) are required as anchorage reinforcement.

A similar problem can occur when the late eruption of wisdom teeth-usually in an incorrect axial direction-is used as a primary abutment for fixed prosthodontic appliances.

**Figure 2.92. Figure 7. – Separation wire**

![Separation wire](image)

**Figure 2.93. Figure 8. – Partial appliance for the aligning of the wisdom teeth**

![Partial appliance](image)

Sometimes it is enough to separate the space between the 2nd and the 3rd molar, and the tooth can erupt in a correct axis. In other cases a partial fixed appliance may align the teeth in a few month.

3. In the case of orthodontic anomaly, the treatment of the anomaly has priority. If conservative treatment is insufficient, surgical treatment is used, with prosthodontic rehabilitation afterwards. Wrong diagnosis, poorly designed restoration, or switching the order of treatment endangers the success of our rehabilitation. Most common anomalies, where making well-functioning prosthodontic appliances is impossible are:

1. deep bite,

2. elongation (because the missing antagonist),

3. mesially or distally tilted tooth (due to the loss of the adjacent tooth),

4. increased Spee curve (mainly in distalocclusion),

5. lateral cross-bite,

6. frontal cross-bite,

7. retained or impacted teeth,
8. Ectopic teeth.

**Figure 2.94. Figure 9. – The impacted canine erupted under the bridge**

Prior to starting the prosthodontic treatment, we may have to wait for years (depending on the malformation) until the orthodontic treatment is finished.

In the cases of missing teeth the orthodontic treatment is difficult, due to the loss of anchorage needed for the movement of the teeth, so either extraoral anchorage reinforcement or mini-screw implants are required.

This technique allows us to only move the affected tooth without the unnecessary loading of the other teeth.

**Figure 2.95. Figure 10. – Mini implants used for elongated teeth**

9.3. Cooperation with periodontology

One of the aims of this cooperation may be restoring the original state after pathologic tooth migration due to periodontal disease. Treatment planning, choosing and controlling the dimensions of forces, dissolving inflammation is a complicated task. Tooth movement should be done cautiously, as it may even worsen the situation. In these cases the periodontologist first has to eliminate the inflammation, and the patients have to adopt to the newly formed situation in their everyday oral hygiene. During the orthodontic treatment strict periodontal control is needed. By the moving the tooth a new periodontium is formed, creating the potential for a healthy oral cavity. Some authors achieve the stabilization of the tooth by vertical movement, but this often requires the devitalization of the tooth.

**Figure 2.96. Figure 11. – Tooth movement towards the pocket, and away from the pocket in accordance to the periodontal state**
The movement of the tooth towards, or away from the periodontal pocket is dependent upon the decision of the specialists, if tooth replacement is also required prosthodontist is also needed in the team.

The other important aspect of the cooperation is when the oral hygiene of the child wearing orthodontic appliance, is inadequate, therefore contraindicating the treatment. According to the statistics only 20 per cent of children between the age of 6-12 years brush their teeth well. It is another sad fact, that parents cannot motivate their child to brush normally, because their oral hygiene habits is not better. There is no motivating “role model” for the child. Sometimes the treatment has to be postponed until the periodontologist can improve the condition of the periodontium. In some cases the periodontal conditions may indicate the discontinuation of the treatment. Orthodontic treatment -especially with fixed appliances- is contraindicated in patients with bad oral hygiene.

During orthodontic treatment children are regularly controlled, the orthodontist plays an important role in teaching the good tooth brushing technique. It may be favorable to have a clinical dental hygienist in the staff, who is well prepared about the oral hygiene of patient wearing orthodontic appliances. It is important to note that the movement of teeth is based on the remodeling of the periodontium, thus if there is any inflammation in the periodontal tissues, forces applied on the teeth will lead to the breakdown of the periodontium, impairing the stability of the tooth and eventually causing to tooth loss. The prognosis of these cases is unfavorable.

9.4. Cooperation with dentoalveolar and maxillofacial surgery

The relationship between orthodontics and oral surgery is very complex, beginning with a simple tooth extraction, through the soft and hard tissue surgical treatments, up to the total rehabilitation of facial deformities and severe developmental disorders by orthognatic surgery. Listing all these would exceed the limits of this chapter, but some important aspects are listed below.

**Tooth extraction**: routine surgical treatment, relieving crowding, when there is a discrepancy between the size of the teeth and the jaw. In other cases it may be the indication of shortening of maxillary arch in class II. malocclusion, where the cephalometric analysis shows the anteroposition of the maxilla. This way space can be made behind the incisors, the overjet can reduced by their distalization. A special case of orthodontic-surgical cooperation is the movement of impacted teeth. After the surgical exposure of the tooth an anchorage device (bracket, hook, etc.) is bonded to the tooth, this way making it a part of the force-system.
Surgical cooperation is needed for the removal of supernumerary teeth or odontomas, which may alter the normal eruption of the teeth. If this is diagnosed in time – where the pediatric dentist plays an important role – after the removal of the malformation we can expect normal, spontaneous eruption and no further treatment is needed. If the tooth does not erupt or it is in a wrong position, orthodontic treatment is necessary.

**Soft tissue operations:** most frequent type of soft tissue surgery is the frenectomy, in our country unfortunately often without correct indication. In the case of tongue it may be crucial to examine in an open mouth if the child is able to touch the frontal part of the palate with the tip of the tongue. It is usually the speech therapists who indicates the surgery, without real functional background, subjecting the child to unnecessary negative psychological effects.

Other area is the upper labial frenulum, according to the literature this can be one of the causes of midline diastema. It may be necessary to cut the excessive tissue, the question is the time of treatment. By the vertical growth of alveolar process, this frenulum moves toward the vestibular fold, thus considering later development and tooth eruption, unnecessary surgical interventions can be avoided.

**Figure 2.97. Figure 12. – Enlarged frenulum without diastema**

An orthodontist may need the help of the oral surgeon in many cases in accomplishing the orthodontic plan. In the case of transverse constriction of the maxillary arch, rapid palatal expansion is very important treatment method. The aim of the treatment in this case is not to move the teeth, but to move the two maxillae away from each other separated along the mid-palatal suture. Loading of the bone is transferred through the teeth. To reduce the risks of this procedure (resorption) corticotomy may be carried out, thereby reducing the resistance.

Due to a developmental acceleration apparent in 20th and 21st century, this treatment should be performed at an earlier age of 15 or 16 years. Meanwhile direct rapid expansion is associated with severe pain depending on the pain threshold of the child, the surgical pretreatment, reduces the pain and chance of relapse, nevertheless increasing the efficiency.

**Figure 2.98. Figure 13. – Corticotomy for rapid palatal expansion**
In the field of maxillofacial surgery, the orthognatic surgery plays a significant role. These orthognatic surgical treatments can offer solution for the sagittal, transverse and vertical discrepancies. These treatments may be performed on either one jaw or on both of them. Jaw distraction surgery is gaining ground next to traditional surgical interventions.

As we can see the information above, neither disciplines of dentistry nor the whole medicine can work separately. Medical work is teamwork. In order to treat the patient successfully, we need to think as a team, and the treatments should be synchronized.

10.2.10. Cleft patients – Judit Rostasi-Szabo

Around the 21st day of fetal development the embryonic disk becomes infolded into the amnionic cavity. Craniocaudal and lateral folding will form the primitive stomadeum. This process is followed by the growth of the adjacent tissues, forming processes, the boundaries of the primitive mouth. The upper border is the frontonasal process, and the maxillary (lateral border) and mandibular (lower border) processes are formed from the first branchial arch. The fusion between the continuously growing processes will form the primary walls of the primitive stomadeum. (Flerkó B. Az ember fejlődése – POTE egyetemi jegyzet 1. 11 és 1. 24.)

The anomalies of this fusion is responsible for clefting. By the 14th week, the crucial elements of the head are fully developed. The clinical appearance and extent of the clefts may reflect the time when (6th week [36-42. day]) and how strong was the noxa affecting the process involved in the closure.

10.1. Etiology

Hereditary: a correlation between the family history and the developmental anomalies has been shown.

Acquired: several co-existing factors may produce a more severe case. Responsible factors:

- Virus (rubella, herpes, cytomegalo, human papilloma),
- Chemicals (drugs, organic solvents),
• Ionizing adiation (x-ray, radiation isotopes),

• Hormonal problems,

• It could be associated with congenital defects or any other syndromes.

10.2. Incidence

Hungary has a quite favourable position according to the survey of the WHO. Less than 100 children are affected, that are born annually. The cleft lip and the total clefting (cheilo-gnatho-palatoschisis) are more frequent males, meanwhile there is a higher incidence for soft- and hard palate clefting in females. In the case of unilateral clefting usually the left side is affected.

Figure 2.99. Figure 1. – The Statistics of People Born with Cleft Lip and Palate

10.3. The Classifications of Clefts

There are many classifications known from the 20th century. One of the most relevant today is the Tassier classification (1979), which also indicates the incidence of the type of cleft with numbers (Figure 2.).

Figure 2.100. Figure 2. – Tessier classification
Apart from the previous systems, the LAHSAL system permits a more effective digital data transfer as well as computer processing.

L – Lip
A – Alveolus
H – Hard palate
S – Soft palate

The position of each letter indicates the side of the face, the small letters stand for the partial and the capital ones for the total cleft.

10.4. The therapy of cleft patients

1. Nutrition problems: In case of clefts, the oral cavity is communicates with the maxillary sinus, or the clefting of the labial muscles may interfere with the feeding of the baby. The vacuum necessary during feeding cannot be formed, but it also hinders the grip of the nipples.

The Haberman feeder was specially developed for the purpose of feeding infants with clefts. This instrument has a valve on it that is not only to control the slow and smooth flow of milk, but also ensures that the closure of the cleft during feeding.

Figure 2.101. Figure 3. – Haberman feeder
The **SNS appliance** stimulates breast feeding, and thus stimulating the production of milk as well. Apart from infants with cleft, it could be effective in the case of premature and slowly developing babies, it is also useful when the feeding is to be re-started.

**Figure 2.102. Figure 4. – SNS appliance**

The **SoftCup** feeder is a spoon-shaped feeder that allows the stimulation of the lips and does and prevents the milk from spilling. It enhances both the subsequent inclination to breast feeding and his or her willingness to accept fluids from glass.

**Figure 2.103. Figure 5. – Soft Cup Feeder**
2. **Otorhinolaryngological disorders** – one of the most frequent problems accompanied by cleft lip and cleft palate is the **altered Eustachian tube function**. Because of the Eustachian tube dysfunction, the pressure is unable to level off along both sides of the eardrum, which causes the retraction of the eardrum, leading to serous otitis media that of bacterial infection could turn a suppurative process. This recurrent inflammation may lead to hearing impairment as well as frequent upper respiratory tract infections.

The medical screening and hearing test (evaluation of auditory functions), which is to be carried out during his or her sleep by the audiologist, is done first months.

Good hearing is essential for the complete development of the infant, for his or her proper social aptitude as well as advanced oral skills.

The implementation of a „gromet” (respiratory pipe) into the eardrum could provide an effective solution to the dysfunction, since it permits both the pressure being properly levelled off and controls the direction of the infectious subjects being produced in the middle ear.

3. **Speech acquisition** – it is proven that the speech acquisition of infants with cleft lip and cleft palate is delayed with half a year or so in general compared to that of the healthy infants. The anomalies of the morphology cause different speech dysfunctions, such as nasal sounding speech and the potential mispronunciation of certain sounds due to the air leak.

4. **Dental problems; chewing- and biting dysfunctions/disorders.** Depending on the social background, an increased caries risk should be taken into account.

Scarring after the surgical closure of cleft has a rather negative effect on the development of the middle face, and therefore on the development of the dentition as well. The three-dimensional developmental deficit causes a characteristic facial appearance, deformity of the nose due to the shortage of tissues, concave facial profile. The frontal and lateral cross-bites as well as the open-bites are frequent, and the face displays similarities to the characteristics of the class III. profile.

There is a higher chance for congenitally missing teeth in the area of the cleft, but sometimes odontomas may be present as well. The interrupted continuity of the alveolar process mainly in the area of the upper canines may interfere with the normal formation of the dental arch and the periodontium.

5. **Psychic problems** – the psychic problems of patients born with cleft lip and cleft palate mostly derive from their physical appearance, as they are often ridiculed in the course of social interaction. The frequent surgical interventions, the too much time spent in a clinical environment as well as the receptiveness to various forms of
disease could often result in the emergence of a persistent awareness of illness, and thus leading to low self-esteem and the constant feeling of inferiority.

10.5. The complex therapy prescribed for children with cleft lip and cleft palate

Due to the earlier described complexity of the disorder, the therapy is to be carried out by a special team whose members are the following:

1. Pediatric surgeon,
2. Dental and maxillofacial surgeon,
3. Audiologist,
4. Otorhinolaryngologist,
5. Speech therapist,
6. Pediatric dentist,
7. Orthodontist,
8. Psychologist,
9. Sociologist,
10. Social worker.

Apart from the permanent members, the therapy could be enhanced by the contribution of obstetricians, neonatologists, geneticists, prosthodontist, plastic surgeons, anesthesiologists and pediatricians.

I. The Steps of the Therapy

- Cleft lip closure around the age 3rd month,
- Nutrition,
- Maturity,
- Vaccinations delayed.

Figure 2.104. Figure 6. – The course and result of the therapy

Figure 2.105. Figure 7. – Schematic drawing of the Milliard-surgery
II. The Steps of the Therapy

- Soft palate closure until the age of two years, before the starting of speech,
- Audiology,
- Otologic surgeries,
- Speech therapy.

III. The Steps of the Therapy

- Hard palate closure until the age of 6-10 years; the palate closure is usually carried out at one time nowadays, depending on the severity of the disorder.
- Subsequent speech therapy.
- Speech corrective surgery.

Figure 2.106. Figure 8. – Isolated cleft palate
IV. The Steps of the Therapy

- Dental prevention- and treatment,
- Orthodontic control and if necessary treatment after the changing of dentition.

Figure 2.107. Figure 9. – Bone defect at the border of the premaxilla in the case of cleft lip and palate

V. The Steps of the Therapy
• Bone augmentation around the alveolar process before the eruption of the canines.

VI. The Steps of the Therapy

• Corrective surgeries,

• Prosthodontic treatment.

Primary surgery. The advantage of the Millard technique is that it is able to unite the layers of the skin, the muscles and of the mucosa at the same time, with providing stable function. This way the need for secondary surgery can be prevented, because it usually provides adequate functional and esthetic results later in life.

Secondary operations. Lip correction is needed in cases where:

1. the philtrum shortens,

2. the vermilion zone is partially constricted, or there is triangular deviation from the normal vermilion border, it is asymmetric.

Speech correction with surgery. Prior to surgery the patient should be examined by a speech therapist, a phoniater, an audiologist and an otorhinolaryngologist. The problem is due to the shortness of the soft palate. The soft palate cannot reach the posterior wall of the oropharynx, thereby it is unable to separate it from the nasopharynx. In the case of VPI (velopharyngeal inadequacy) certain sounds may be lost; but operation itself could engender quality of the already existing speech.

Nasal Surgical Correction. The most frequent problem is the shortness of the columella that can be aggravated by the lack of the inferior bones of the anterior nasal aperture, the underdeveloped premaxilla, the posterior position of the maxilla and the bone defect of the alveolar process as well. The soft tissues become hollow without a massive hard tissue base, therefore there is an acute angle between the two nasolabial sulci, giving the face a characteristic appearance.

In the course of the operation, the fact that the cartilages are still in a growing phase shall be a primary concern, as these units can be still highly vulnerable for being disturbed in their further development, which also has a negative effect on the aesthetic harmony.

Accordingly, these type of surgeries should be planned for a later time, after puberty.

Figure 2.108. Figure 11. – Results before and after the surgical correction of the nose
10.6. The Orthodontic Therapy of Patients with Cleft Lip and Palate

The main aim of the early therapy is to support the development of the maxilla until the lip closure surgery, and to separate the nasal and oral cavities by means of an artificial plate. The treatment should be carried out during the first two weeks, as the majority of children do not accept the appliance in their mouth after this time interval.

The plate (McNeil) is made out of thin acrylic fibers (<1mm) based on the impression taken from the mouth, the plate may also be vacuum formed. The plate is to be changed monthly in accordance with the development pace.
The appliance is especially suitable in the case of a bilateral cleft, for retraction the protrusive premaxilla, and it may help in preparing the surgery by converging the parts of the lips.

This therapy, however, has become less popular recently due to the necessity of the frequent check-ups, the expenses of the appliance and the latest views of its effectiveness.

The elastic tapes, which is placed into the mouth of the infant during the non-feeding intervals, is able to reduce the extension of the cleft. This procedure can be used to prevent tension around the surgical area in the course of the post-operative period, thus contributing to the undisturbed healing process and the decreased potentials for scarring.

2. The Orthodontic Therapy during Primary, Mixed Dentition Stage

In primary dentition, the frequent medical consultations may provide a psychological preparation for children to accept further therapeutic interventions.

Children and their parents should be kindly taught to devote more attention to the proper dental hygiene and in cooperation with the pediatric dentists to sustain a dentition free of caries, and thus enhancing the development of a healthy permanent dentition.

Right after the surgical closure of the cleft a concrete, goal-oriented orthodontic therapy has to be started, if the child is mentally prepared for the cooperation.

Those devices which support the co-development of the jaws, in order to harmonize the dental arches should be taken into consideration. These functional appliances have an effect primarily on the perioral muscles, and thus on the development of the jaws; they are to support the adequate movement of the lip and the tongue and are beneficial not only for the work of the speech therapist, but also for the advancement of the oral skills.

Nowadays the frequently used trainers, with individual adaptation may be suitable to carry out the procedures mentioned above.

One of the greatest advantages of these trainers is the fact that they are made out of an elastic material and could therefore be adapted in the dental office; eliminating the necessity of taking impression each and every time and, nonetheless, the children get use to them easily. According to the parents, well-marked results can be seen after a few months of use.

During mixed dentition the number and position of the tooth buds should be examined. Due to the lack of calcium salts in earlier stage, the diagnostic value of the radiographic examination is doubtful. It is essential to evaluate the extension of the bone defect, which may be effectively carried out by advanced radiological examinations nowadays. Because of the bone defect of the alveolar process, the development of the complete dental arch is hindered, and consequently, right after the evaluation of both the bone maturity and the condition of the tooth germs, the bone augmentation becomes necessary. This should be carried out soon before the expected eruption time of the upper canines. Prior to surgery, if the transversal dimension of the teeth and the alveolar process is not sufficient, expansion is indicated. During expansion the scar of the cleft closure may be lacerated (fistula), and it may result in the failure of the bone augmentation.

The abnormal position of the incisors may mean esthetic problems in the majority of the children, but can be easily corrected with special partial fixed orthodontic appliances. In those cases where despite the previous therapeutic interventions the development of the maxilla is delayed compared to the mandible, there is an alternative orthopedic treatment for the maxilla up until the end of the growing phase.

Figure 2.109. Figure 12. – Orthopedic treatment
3. The Orthodontic Therapy during Permanent Dentition

The ultimate aim of the therapy is to create an overall harmonious, functionally adequate dentition, and to create the esthetic parameters of the face; briefly speaking, it is an objective that has never been successfully carried out. After the complete development of the permanent dentition, the movement of the teeth is only possible by means of fixed appliances. During this period the prosthodontic treatment should be planned in cooperation with a prosthodontist to replace the missing teeth the adjustment of the abutment teeth is carried out in this phase according to this.

If the correction of the facial deformities is inevitable, the orthodontic therapies preceding the eventual orthognatic surgery should be carried out during this time interval as well. The surgical treatment is planned by the orthodontist and the maxillofacial surgeon according to the cephalometric analysis (three dimensional software is also available). Apart from the Le Fort osteosynthesis of the maxilla and the Dal Pont-Obwegeser surgery of the mandible, the osteo-distraction may provide an adequate surgical solution for the severest anomalies.

Figure 2.110. Figure 13. – The schematic drawing of the Dal Pont-Obwegeser Surgery

Complete Rehabilitation:

- Care up until age 18.
- Recovering the lost functions.
- Creating harmonious, and esthetic facial formation, and to help the patient in socialization and to facilitate them to be a useful, equal part of the society.

It shall be clearly seen from the above explained facts that the therapy of children born with cleft lip and palate is a great challenge including multidisciplinary and constant co-operation up until age 18. Unfortunately, one could come across even nowadays such incidences that have been far too ignored, interrupted or delayed in a proper treatment. Accordingly, the extensive information distribution job of private physicians and health visitors is on every account invaluable; however, the early diagnosis of any developmental anomalies, the long-term family consultations to support the complete social integration of invalid and underprivileged child patients until they reach adulthood are to be primarily concerned as well.
Prevention:

1. Family planning – it is essential to give repeatedly high emphasis on awareness; having children entails enormous responsibility for each and every family member, therefore they have to be prepared.

2. Mapping the genetic history of the family – without specific genetic knowledge, it is widely known that a marriage knot within the family or close kinship leads to increased incidence of developmental anomalies.

3. Eliminating noxae – awareness. During the conscious parenting, the mother is supposed to reduce any possibilities for obtaining acquired etiological factors. It is widely known that certain substances (such as the folic acid and the vitamin C) are beneficial for the embryo and therefore advised to be taken in an adequate dose before and during the period of the pregnancy (according to the seasonal presence of natural vitamin sources like vegetables and fruits).

4. Genetic consultation – necessary in the case of a positive anamnestic family response, before the parenting and for making a responsible long-term decision.

In view of all these facts (see the introduction and the part about embryo development), it is crucial to accentuate that while the pregnant woman is still not aware of her pregnancy, the development of the embryo had already commenced a phase of essential advancements, such as the closure of the neural tube or the facial formation. Consequently, the importance and role of prevention is the elimination of developmental disorders should be emphasized repeatedly.

11. 2.11. Screening, controlling – Zsuzsanna Gurdan

Our orthodontic treatment aims at achieving a healthy and stable denture harmonious both in appearance and function. As Albin Oppenheim had put it: “the biggest and most difficult task of orthodontics is to maintain the attained results.” (Oppenheim, 1934)

Moving of the teeth during an orthodontics treatment is achieved by metabolic processes in the alveolar bone. The aligned function of osteoclasts and osteoblasts warrants the rebuilding of alveolar bone and the anchoring of moved teeth in the required site. However, this alveolar process and the rebuilding of periodontal tissues do not occur promptly, they require a minimum of 3-4 months. (Proffit et al. 2013) This is the most frequent cause of relapse and allows the teeth to move back to its original position.

11.1. Definition of retention

Retention was defined by Riedel in 1969: fixation of the teeth in an ideally aesthetic and functional position. The basis of the treatment involves reorganization of periodontal and gingival fibres and minimizing changes in jaw growth and also ensuring neuromuscular adaptation so that the teeth remain in the most appropriate position.

11.2. Etiology

Factors responsible for instability are not exactly known. The presence of several factors is thought to be in the background of relapse. There are four different schools of thought regarding relapse and stability:

1. Occlusion school of thought: according to Kingsley correct occlusion is the most important, with it stability can be achieved.

2. Apical base school of thought: Lundstrom suggests a major apical basis is necessary to move the teeth and achieve stability. McCaully states that the distance between canines and molars should not be altered in order to avoid relapse.

3. School of thought regarding the lower front teeth: Grives and Tweed emphasized the exact adjustment of lower canines to achieve stability

4. Musculature school of thought: Rojer emphasized the the balance of functional muscles to avoid relapse.

11.3. Factors influencing the prevalence of relapse
2. Pediatric dentistry and orthodontics

1. Reorganization of the gingival fibrous system.

2. Tendency and direction of jaw growth, growth line (skeletal open bite, progenia).

3. Functional adaptation of adjacent soft tissues (lips, bucca, tongue), lacking adaptation of the neuromuscular system.

4. Presence or reoccurring bad habits (digit sucking, mouth breathing, lip squeezing during swallowing, tongue thrusting during swallowing).

5. Misdiagnosing and setting up the wrong treatment plan (excessive force, constantly altered direction).

6. Improper planning of retention phase (short retentional retention time, improper retention appliance).

7. Effect of the wisdom tooth (questionable).

8. The dental arch shortens with time.

9. Tertiary malocclusion (of multiple causes) that can be due to the constant movement of the teeth, due to lacking contact, growth and muscular pressure.

11.4. Planning of the retention phase

Following an active treatment it is of utmost importance to plan the maintaining treatment, in other words the retention phase should be planned at the beginning of the treatment. Skipping this phase leads to relapse. In more severe disorders and improper planning of the retention phase there is always a higher risk for relapse.

Main aspects of retention planning:

1. Severity of malocclusion: the more extended the disorder that is to be corrected, the longer the retention phase should take.

2. Type of malocclusion (basal, dento-basal, dento-alveolar, sagittal, vertical, transverse).

   Skeletal alterations may relapse in all three directions (vertical, sagittal, transverse planes). Until the final stage of growth relapse can be expected. Relapse in transverse corrections can be seen less frequently, as growth in this direction ends at the earliest age.

3. Age of the patient: the younger the patient, the shorter the retention phase, due to better regeneration ability and faster tissue remodelling.

4. Growth stages, growth prognosis: unless growth stops, the risk for relapse is higher.

5. Effect of soft tissues, muscle balance: by the end of the treatment the aim is the balance between outer and inner muscles, which contributes to the stability of the results.

6. Causes in the background: only the complete cessation of the bad habits can lead to stable results.

7. The extent of moving the teeth: if the treatment requires the moving of the teeth of a greater extent, the retention phase is longer.

8. Speed of moving the teeth: a faster moving of the teeth is associated with a longer retention phase.

9. Inherited factors: stable results are easier to achieve in acquired disorders; inherited malformations require a longer retention phase by all means.

10. Periodontal condition: periodontal condition is a very significant factor both in the active and in the retention phase. Stable results can be achieved only with healthy periodontium.

11. Systemic disorders, eating habits: general diseases influence the ability of regeneration. In certain systemic disorders both the active and the retention phase are longer.

12. Approximal contact points.
13. Height of the cusps and condition of adjacent tissues.

14. Patient cooperation: in lacking or failing cooperation fixed retention is recommended.

One of the most difficult tasks of orthodontic work is the planning of retention phase and its implementation. It should be therefore regarded, since it is easier to make up the optimal treatment plan than to treat any relapse.

11.5. Duration of retention time

There is no scientific consensus regarding the currently used retention appliances in orthodontic treatments.

Duration of retention is varied and its time should be individually determined in every patient. In complicated treatments (such as those of adults) the retention time is usually longer than in treatments performed in simple, adolescent patients.

There are several different protocols determining the duration of retention treatment. Retention time should be two times longer than the duration of active treatment.

In average the retention phase lasts from the end of 10-years of age until the beginning of 20-years of age and is independent from the type of retention (total, partial or only night retention).

Every patient with a fixed appliance is to receive a retention treatment. A constant 3-4 month and a partial 12-month retention time are required to rebuild periodontal tissues, fibres. These are the minimal time periods required for the above mentioned processes.

General rule: the patient should always quit the use of the retention appliance gradually, and it should be regularly monitored even in this phase.

A life-long treatment is the only guarantee to prevent relapse.

11.6. Types of retention appliances

The usage of a removable appliance is crucial in order to maintain perfect results. Alongside the removable appliance a fixed, bonded retainer can be applied on the lingual surfaces of the upper and lower front teeth.

Most common types of retention appliances:

1. Lingual permanent retainer

This is a 0.032-0.036” rounded steel arch, bonded onto the lingual tooth surface. It is recommended on the surface of teeth susceptible to relapse.

Figure 2.111. Figure 1. – Permanent: retainer on the upper dental arch

Figure 2.112. Figure 2. – Permanent: retainer on the lower dental arch
2. Retention plate (Hawley-retainer)

Parts: basal plate, anchored to the palatal mucosa and the labial wire that is anchored to the front teeth.

Figure 2.113. Figure 3. – Upper and lower retention plates

Figure 2.114. Figure 4. – Upper and lower retention plates

3. Positioner: irreversible elastomeric splint surrounding every upper and lower tooth.

Figure 2.115. Figure 5. – Positioner

**Figure 2.116. Figure 6. – Vacuum-formed retainer**

5. Intermaxillary functional appliance
   - Hansa, Aktivátor, Twin-block, Bionátor, Doppel platte.

6. Multiband appliance – wearing it for a prolonged time

7. Active retention appliance
   - see later.

8. (Myofunctional treatment).

**11.7. Malocclusions requiring the usage of a removable retention appliance**

- Class I, non-extraction cases with protruding front teeth and diasthema in the past medical history.
- Class I and II, in cases of patients with malocclusion also requiring extractions.
- Class II, after non-extraction treatments.
• Deep bite, open bite, following the correction of ectopic teeth.
• After treated lateral cross-bite.

11.8. Malocclusions requiring fixed retention appliances
• Crowding of lower front teeth from canine to canine.
• Severely rotated tooth.
• Treated median diasthema or lack of tooth.
• In periodontal involvement.
• In pre-prosthetic orthodontic treatments (if space is to be kept free for an implant or bridgework).

11.9. Malocclusions that requires no retention appliances
• Frontal cross bite, when teeth are stabilized with a correct overjet or overbite.
• Following a successful diasthema closing treatment in permanent dentition, as the central incisors are stabilized by the lateral incisors.
• Following the realignment of impacted teeth.
• Following a successful serial extraction (Hotz).

11.10. Active retention appliances
Intermaxillary functional appliances can be used as active retention appliances in patients treated with Class II, Class III deviations, where no skeletal deviation was detected. These different appliances can be used in occlusal discrepancies exceeding 3 mm. In this case the main indication is the crowding of lower front teeth, since the stability of the lower incisors is the most problematic job of orthodontics. If necessary, it is possible to gain some extra space in the area of the lower incisors by enamel reduction, stripping and therefore creating favourable conditions.

Possible retention appliances:
a. clips from canine to canine,
b. multiband appliance from the first premolar to the contralateral premolar,
c. intermaxillary functional appliance.

Retention appliances are merely passive devices, however they can be used as active appliances in the case of relapse.

11.11. Retention possibilities, systems for different occlusion problems
1. Abnormalities of tooth position
a. Rotation

The transseptal gingival fibrous system is the cause of relapse

Retention possibilities applied:
• Lingually bonded retainer.
• Contralateral overcorrection of a rotated tooth and a prolonged wear of multiband appliance.
2. Pediatric dentistry and orthodontics

- Surgical incision of the transseptal fibre (not recommended in lower teeth because of the narrow gingiva and the alveolar bone between the teeth).
- Medication (altering metalloproteinase activity by chemically modified tetracycline).

b. Crowding

The transseptal gingival fibrous system is the cause of relapse

Retention possibilities applied:
- Lingually bonded retainer.
- Retention splint.
- Retention plate.
- Medication (altering metalloproteinase activity by chemically modified tetracycline).

c. Gap, diastema

The transseptal gingival fibrous system is the cause of relapse

Retention possibilities applied:
- Lingually bonded retainer.

Figure 2.117. Figure 7. – Lingually bonded retainer after diastema is closed

- Retention splint.
- Retention plate.
- Medication (altering metalloproteinase activity by chemically modified tetracycline).
- Restorative dentistry, prosthetic dentistry.

2. Occlusal deviations in the sagittal direction:

a. Class II malocclusion

Causes of relapse include an altered jaw growth, transseptal gingival system and lip position.

Retention possibilities applied:
- Intermaxillary functional appliance.
2. Pediatric dentistry and orthodontics

- Lingually bonded retainer, where the front teeth are protruded and the incisors are straightened by the force exerted by the lip muscles.

- Myofunctional treatment: increasing the tone of the orbicularis oris muscle with exercises following an overjet treatment.

- Prolonged wear of a multiband appliance without activation.

b. Class III malocclusion

The usual cause of relapse is the growth of the mandible.

Retention possibilities applied:

- Functional appliance.
- Positioner.
- Surgical correction, where relapse has developed.

3. Occlusal deviations in vertical direction:

a. Deep bite

Relapse is caused by the jaw growth in vertical direction.

Retention possibilities applied:

- Retention splint with or without bite-elevation in order to control an overbite.
- Retention plate with or without bite-elevation in order to control an overbite.
- Retention plate combined with a finger spring, to prevent the elongation of the front teeth.

b. Open bite

Causes of relapse include jaw growth in a vertical direction, elongation of the molars, intrusion of front teeth, bad habits (if bad habits are still present after the treatment, the risk for relapse is 100%).

Retention possibilities applied:

- Positioner.
- Intermaxillary functional appliances (Bionator).
- Retention appliance with bite elevation in the molar region to block the molars therefore preventing their elongation.
- Combination of a retention plate and a high-pull headgear.

4. Occlusal deviations of the maxilla in a transverse direction

Relapse is caused by the functional alignment of soft tissues.

Retention possibilities applied:

- Multiband appliance is worn 3-4 months longer following the active treatment period.
- Later transpalatal arch.
- Retention plate.

The finishing of the active treatment is associated with the careful planning of the retention phase focusing of the following criteria:
6 keys to a harmonious occlusion (Stephen W. Andrews):

- at the end of the treatment molar relation is Class I or Class II,
- at the end of the treatment canines are always in Class I occlusion,
- correct incisor relation,
- free from gaps,
- free from rotation,
- flat Spee curve.

Expansion of the lower dental arch is not possible, or only minimally possible, therefore the treatment of the upper dental arch always depends on that of the lower dental arch.

Intercaninal distance cannot be expanded, transversal molar distance can be expanded with the minimum of 3 mm according to William R. Proffit, this distance is 1-2 mm according to Asbjørn Hasund.

5. starting the treatment at the earliest possible time, most optimal in the growing phase

- Form of the dental arch should not be altered.
- In certain cases the most appropriate retention is chewing force.
- Torsion and overcorrection of rotation in the opposite direction.
- Retention depends on the structural and functional changes of bone tissue, which are influenced by different endocrine disorders.
- Lower front teeth are to be maintained in proper axis position compared to the mandibular basis.
- Retention can be complicated by different tooth size discrepancies.

11.12. Postretentional phase

It cannot be stated unequivocally that the aimed result can be maintained even after the active retention treatment. Therefore a postretentional phase should follow. It is recommended to call the patients back for check-ups in the time period of 1-2 years. In this phase there is a good chance to notice and detect some possible pathological growth tendencies (fully developed progenia, relapse of a skeletal open bite). Treatment failures and misdiagnoses all contribute to gather experiences and therefore to improve our own work.

Questions:

- When is fixed retention required?
- What are the six keys to a harmonious occlusion?
Chapter 3. 3. Reconstructive dentistry

1. 3.1. The Modern Concept of Endodontics, Examination Methodes, Treatment Plan – Edina Lempel

1.1. The Modern Concept of Endodontics

Endodontics is a dental science, that deals with the morphology, histology and pathology of the human dental pulp and periapical tissues. Endodontics describes the activity of healthy pulp, the factors, that play an important role in damage of pulp and periapical tissue and help to establish a correct diagnosis and effective treatment plan.

The aim of the endodontic treatment is to keep the vitality of the pulp. In case of irreversible pulpal damage or pulp necrosis, root canal treatment is necessary to prevent the consequences of pulpal inflammation or necrosis.

Requirements for endodontic treatment:

• The dentist should possess the knowledge of endodontic diseases and their pathology.
• The dentist should know and apply the diagnostic methodes and should establish a proper clinical diagnosis.
• The dentist should adjudge the prognosis of the tooth and should establish a treatment plan with the patient acceptance.
• A requirement of a successful root canal treatment is the knowledge of the anatomy of the pulp.
• The dentist should know the nature of the endodontic infections and their local and systemic treatment.
• The dentist should apply different chemical agents in order to disinfect and chemically prepare the root canal.
• The dentist should know and put into practice the special endodontic armamentarium and materials used for preparation, disinfection, measurement, root canal filling and for post-reconstruction.
• The endodontic treatment is not finished with the root canal filling, there is a need of a proper coronal sealing with a restoration in order to prevent failures.
• There is a need of regular recall to make sure of the successfullness of the treatment. In case of endodontic failure there is possibility for endodontic revision or endodontic surgical method or the combination of them.


1.1.1. Diseases of the pulp

• Inflammatory reaction of the pulp tissue.
• Etiological factors: microbes (toxins, enzymes, metabolic byproducts) or other irritative agents.
• It could lead to irreversible pulpitis (Langeland 1981).
• Pulpitis is caused by the cariogenic bacteria in ~95% of the cases (Schäfer 2001).
• If the caries reaches the pulp it leads to the necrosis of the coronal pulp tissue (Lin 1981).
• The cause of pulpitis and pulpal necrosis is usually bacterial invasion and infection (Schroeder 1991).
In development and maintenance of **periapical diseases** there is an important role of microbes (especially bacteria and fungi, rarely viruses).

- However microbes can stay hidden in the root canal from the defensive reactions of the immune system (http://www.sld.cu/galerias/pdf/endodoncia_parte_1.pdf) hollow tube theory Rickert és Dixon 1931).
- These are not specific microbes; it is a mixed infection with 15-30 species.
- The treatment should have a wide spectrum.
  - 95% of the microbes could be removed by mechanical preparation.
  - The root canal is disinfected by chemical preparation.
  - With the combination of chemical agents the disinfective effect can reach a wider spectrum.

**Figure 3.1.** Figure 1. – MB1 and MB2 root canals in tooth 16 after preparation (The left figure is in lower, the right one is higher magnification.) The anastomoses between root canals creates a complicated tube network that is hard to prepare mechanically and fill completely. They can stay hollow after the root canal filling

**Figure 3.2.** Figure 2. – Mesiocentral third root canal is visible between the mesiobuccal and mesiolingual root canal orifices in tooth 36. (The orifice of the mesiocentral root canal is next to the orifice of the mesiolingual root canal.) The undiscovered root canal would be a significant volume of dead space

1.1.2. The endodontic treatment
The aim of the **endodontic treatment**:

- **For the patient:**
  - Maintain the function and aesthetics of the teeth.
  - Provide painless and asymptomatic state.

- **For the dentist:**
  - Come up to the patient expectations (aesthetics, function, painless and asymptomatic state).
  - Eliminate the pathogens.
  - Prevent reinfection.

The aim of the root canal treatment is to keep the diseased tooth. The treatment executed with competence is safe and effective.

**The following steps describe the technical aspects:**

- Anamnesis, clinical and radiological examination.
- Diagnosis establishment.
- Treatment plan establishment.
- Local anesthesia and isolation.
- Opening of the pulp-space, preparation of trepanation cavity and extirpation of the pulp tissue from the pulp chamber.
- Localization of the root canal orifices, extirpation of the pulp tissue from the root canals.
- Provision of the penetrable root canals.
- Working length determination, chemo-mechanical preparation.
- Provisional root canal filling and restoration.
- Definitive root canal filling, control radiograph.
- Accomplishment of the coronal restoration.
- Recall of patient for control examinations.

**Root canal treatment in one or two appointments:**

- In case of vital pulp tissue the treatment should be accomplished in one appointment.
- In case of necrotic pulp tissue (infected tooth) stronger disinfectant agents are used for longer action-time with ultrasonic cleaning if the treatment is accomplished in one appointment.
- There is no statistically significant difference in success if one or two-sitting treatment is compared.
- Postoperative sensitivity or pain is a frequent occurrence after one appointment treatment.
- One appointment treatment is not a higher risk factor in case of acute exacerbations (acute periapical abscess).
- In certain cases (apexification, resorption, non-dryable root canal, complications) two or more appointments are necessary.
1.2. Endodontic examination methods, treatment plan
(http://www.sld.cu/galerias/pdf/endodoncia_parte_2.pdf)

The aim of the endodontic examination:

• To find the diseased tooth
• To establish the diagnosis.
• To discover the anatomic features during the treatment.
• To map the possible difficulties of the treatment.
• To calculate the chance of success.
• To control the quality of the treatment.
• To control the success of the treatment during the recall.
• To indicate further treatments if it is necessary (endodontic/parodontal/surgical treatment).

Establish a treatment plan:
(http://www.aae.org/uploadedFiles/Dental_Professionals/Endodontic_Case_Assessment/2006CaseDifficultyAssessmentFormB_Edited2010.pdf)

• Is there any need for premedication?
• Is the coronal reconstruction of the diseased tooth possible?
• Is the treatment aggravated by abnormal anatomic features or pathologic processes?
• Is the pulp vital or is there any possibility to keep the vitality of the pulp?
• Is the root canal system infected?
• Is there any sign of periapical pathosis?
• What kind of instruments and materials are available for the treatment?
• Is the treatment accomplished in one or more appointments?
• How much time is available to implement the treatment? (Lack of time caused by unplanned treatment or unexpected difficulties can change our decision near more appointments.)
• Does the patient accede to the treatment being alive to the expected success of the process?
• Does the patient need antibiotics therapeutically (diagnosis+anamnesis), or what kind of painkiller is recommended?
• Is it necessary to supplement the primary endodontic treatment with parodontal or surgical treatment?
• After how much time and how often is it necessary to check the results of the treatment?
• What should be applied in case of unsuccessful treatment: conservative or surgical solution?

Figure 3.3. Figure 3. – Left: The root canal treatment is difficult to accomplish due to a rare anatomical variation (two distal roots) in tooth 46. Middle: Working length determination with X-ray. The location of the root canals is distinctly visible on the radiograph. Right: Root canal filling control X-ray
Figure 3.4. Figure 4. – The endodontic success depends on the elimination of infection and prevention of reinfection. A: Middle-sized, diffuse periapical radiolucency and fractured instruments in the MB, DB root canals of tooth 26. B: Passing by the fractured fragments is successful, disinfection of the apical part is available. C: Root canal filling control. The fractured fragments are left behind in the root canals. D: 2-years control, intact periapical area. E: 9 years later the coronal restoration (composite filling) is damaged. The periapical area is intact. To prevent the reinfection the prompt repair of the coronal restoration is necessary.

2.3.2. Pulp Diseases and Their Diagnosis – Edina Lempel

Clinical diagnosis establishment has to focus on the therapy and clinical classification of pulp diseases (and their consequences) should be based on this relation. The diagnosis will clearly determine the prognosis and the treatment of the tooth.

2.1. The healthy pulp

- Asymptomatic.
- There is a mild, casual sensitivity or mildly painful response at vitality/cold test.
- When the stimulus is folded the pain disappears immediately.
- Pain could not be triggered by palpation or percussion.
• Lamina dura is intact on the radiograph and there is not sign of root resorption or calcification.

2.2. Reversible pulpitis (hyperaemia pulpae)

• Inflamed pulp tissue.

• The response for thermal (cold) tests is quick, sharp and hypersensitive.

• After triggering the pain disappears immediately.

• From other aspects the pulp remains asymptomatic.

• This is a symptom and not a disease (If the pulp irritation is terminated the pulp will return to its normal, healthy state).

• Continuous irritation can maintain the symptom or irreversible pulpitis can develop.

• Differentiation from irreversible pulpitis:

  • The response for cold test is instantaneous, painful, however disappears immediately after triggering. (In case of symptomatic irreversible pulpitis the pain is maintained for longer time.)

  • There is not spontaneous pain. (In case of symptomatic irreversible pulpitis spontaneous pain is frequent.)

2.3. Cracked tooth syndrome

When a crack progresses into dentin, mastication could be painful because of the deformation of the tooth.

Symptoms:

• Anamnesis: trauma, bruxism, bad habits (knacking nuts with tooth), or a former cracked tooth.

• Pain while biting (there is more frequent presence of pain upon release of biting pressure than it is elicited during the pressure phase).

• Normal response for vitality/cold test (vit. - positive).

• Usually the response is negative for percussion, ther is not radiological sign.

• It is often difficult to achieve an objective definitive diagnosis, therefore it could have long history.

• The crack not always progresses into the root system to involve the pulp.

• The pulpal pathosis caused by a crack can extend to the periodontal ligament space, creating a periradicular periodontitis.

• Diagnosis establishment:

  • A variety of devices have been used for bite tests, including cotton applicators, toothpicks, orangewood sticks, and rubber polishing wheels.

  • Dye penetration test could be informative (caries indicator, or methylene blue).

  • Operating microscope, or lupe.

  • Transillumination.

  • Periodontal sounding.

Figure 3.5. Figure 1. – Caries detector dye (Caries Marker, VOCO) makes visible the mesio-distal fracture in tooth 15
Visible changes are rarely seen on X-ray images, only if the pulp or periodontal ligaments are involved.

Treatment:

• Occlusal protection, splinting of the tooth structure (metal-ceramic or full ceramic crown).
• Root canal treatment in case of involved pulp tissue, splinting of the tooth structure.

2.4. **Irreversible pulpitis**

Classification:

• acute, subacute, chronic.
• partial, total.
• sterile, or infected.

Chronic pulpitis is usually asymptomatic. Clinically, the maximum extent of the inflammation can not be determined until the periodontal ligaments are not affected (sensitivity for percussion).

2.4.1. **Asymptomatic irreversible pulpitis (Pulpitis chronica)**

Common causes are caries, trauma, harmful effects of dental procedures.

It may be asymptomatic or presents itself with a few symptoms.

Clinical manifestations:

• Pulpitis chronica clausa abscedens:
  • Often asymptomatic.

• Figure 3.6. Figure 2 – Pulpitis chronica clausa abscedens in tooth 37. Coronal destruction is marked on the preliminary X-ray. The patient complained of mild spontaneous pain intensifying and then disappearing at intervals. The tooth was sensitive for warm
• Pulpitis chronica clausa granulomatosa (internal granuloma, Palazzi-granuloma):
  
  • Internal resorption is formed.
  
  • It could be displayed as an incidental finding on X-ray images.
  
  • Coronally it may have a „pink spot” appearence.

Figure 3.7. Figure 3. – Internal resorption in the pulp chamber in tooth 31. The tooth is vital, periapically intact

Figure 3.8. Figure 4. – Internal resorption at the border of the middle and apical third of the root canal. Root canal filling was performed with thermo-compaction technique
3. Reconstructive dentistry

- Pulpitis chronica aperta ulcerosa:
  - Caries penetrans.
  - Asymptomatic, mild pain after biting.

- Pulpitis chronica aperta granulomatosa:
  - Hyperplastic pulpitis, pulp-polyp, pulpitis chronica aperta proliferativa
  - Exuberant inflammatory tissue proliferates through the exposed pulp chamber and forms a cauliflower-like „pulp polyp”.
  - Low-grade chronic irritation and good vascularization (In primary and immature permanent teeth).
  - Chronic pulpitis can lead to calcification and obstruction of the root canal.

**Figure 3.9. Figure 5. – Calcification of the middle and apical third of the root canal in tooth 12. The complaints were similar to pulpitis. The tooth is periapically intact**
2.4.2. Symptomatic irreversible pulpitis (Pulpitis acuta)

The most frequent cause is dental caries.

Symptoms:

- Spontaneous, intermittent, paroxysmal or persistent pain.
  - Intensity: mild or aggravating.
  - Quality of pain: sharp or dull.
  - Localized or referred.
- Rapid exposure to dramatic temperature changes (especially to cold stimuli) will elicit heightened and prolonged episodes of pain even after the source of the pain is removed.
- Hot or cold compress can reduce the symptoms.
- Changing position can trigger pain (decumbence, getting up from lying position).
• Painkillers can not ease the pain, therefore it can interfere with sleep.

Diagnosis establishment:

• Anamnesis focused on pain (questionary).
• Oral examination (cariologic, parodontal).
• Carefully constructed and evaluated thermal test.
• Crown and periapical X-ray images
  • Based on indirect signs (caries, extent of filling, etc.), the suspected tooth will be selected.
  • In advanced cases the periapical area is involved, which leads to periapical widening (edema).
• In case of referred pain, selective anesthesia test should be performed.

Figure 3.11. Figure 7. – Pulpitis acuta in tooth 47. Prolonged, strong, spontaneous pain, may provoked by cold test. The periapical bone structure and the periodontal gap is intact on the X-ray. The extensive caries refers to the pulp involvement

Figure 3.12. Figure 8. – Pulpitis acuta in tooth 25. The periapical bone structure and the periodontal gap is intact on the X-ray. The extensive caries refers to the pulp involvement
2.5. Pulpal necrosis (gangraena pulpae)

The cause of necrosis could be untreated irreversible pulpitis, trauma, dental treatment (surgical or orthodontic treatment), or other effects that could block the pulpal blood supply which is succumbed.

The pulp tissue remnants liquidify or coagulate. Tooth discoloration could be a sign of pulpal necrosis.

Pulpal necrosis could be partial or total:

- Partial necrosis:
  - Characterized by the symptoms of irreversible pulpitis.
  - More frequent in multi-rooted teeth.

- Total necrosis: vitality is negative
  - Gangraena simplex (pulp chamber and root canals are not infected), asymptomatic.

Figure 3.13. Figure 9. – Gangraena simplex in tooth 47. The tooth is asymptomatic, vitality negative. The tooth is periapically intact (left). Test cavity preparation during trepanation was painless. Right: Root canal filling control X-ray
• Gangraena complicata

• Asymptomatic (Maintains an asymptomatic periapical periodontitis).

• Symptomatic (Maintains a symptomatic periapical periodontitis).

2.6. Acute periapical periodontitis (periodontitis periapicalis acuta, symptomatic periapical periodontitis)

**Painful** periradicular inflammation. Predominantly occurs in *gangrenous teeth*. Less often chronic occlusal trauma may lead to acute periapical periodontitis in vital teeth. The patient’s complaint is **sensitivity** or mild to intensive **pain while mastication**, percussion or touch with tongue.

**Vitality test** is negative (or could be positive in case of partial necrosis), **percussion test** could be positive – these results are decisive in diagnosis establishment.

**Periodontal gap** could be **intact** or **expanded** on X-ray images. After longer time (~7-10 days) lamina dura is interrupted, **radiolucency** becomes visible.

Figure 3.14. Figure 10. – Periodontitis periapicalis acuta in tooth 35. The tooth is periapically intact
Figure 3.15. Figure 11. – Periodontitis periapicalis acuta in tooth 15. Widening of the periapical ligament space is visible, however the continuity of lamina durea is not disrupted.

Figure 3.16. Figure 12. – Periodontitis periapicalis acuta in tooth 15. Left: Periapical ligament space is widened. Right: Calcium-hydroxide paste medication was applied for 2 months and afterwards the root canal was permanently filled. The periapical area is healed.
Untreated acute periapical periodontitis can lead to acute periapical abscess formation.

### 2.7. Acute periapical abscess (abscessus periapicalis acuta, symptomatic periapical periodontitis)

Painful periapical inflammation, purulent exudate around the apex.
General symptoms could be present (subfebrility, febrile, malaise).

Intra- and later extraoral swelling. Extent and location is determined by:

- Position of the affected tooth’s apex.
- Thickness of the cortex.
- Position of the skeletal traction.

The swelling is typically located vestibularly, however is frequent palatally (eg. palatal roots of upper premolars and molars).

The pain could be mild or severe.

**Figure 3.18. Figure 14. – Acute periapical abscess. Painful vestibular swelling near tooth 23**

The tooth is **mobile** and painful for percussion. **Vitality is negative.**

On radiograph:

- X-ray image is the same as seen in acute periapical periodontitis. **Periodontal ligament space** may be **intact** or mildly **widened.** **Radiolucency** is visible. Due to the rapid progress of the process, the cortical and trabecular bone is not demineralized yet.

- Acute exacerbation of the chronic periapical periodontitis is the so-called **phoenix abscess,** characterized by **localized** or **diffuse periapical radiolucency.**

**Figure 3.19. Figure 15. – Phoenix abscess. On the x-ray small, diffuse radiolucency is visible, the coronal restoration (glass ionomer cement filling) is extended. Purulent exudate was evacuated during trepanation**
3. Reconstructive dentistry

Differential diagnosis: in case of lateral parodontal abscess the tooth is **vital** and there is the presence of a **parodontal pocket**.

### 2.8. Chronic periapical periodontitis (periodontitis periapicalis chronica, asymptomatic periapical periodontitis)

**Asymptomatic.**

- The symptoms are similar as seen in gangraena pulpa (**tooth discoloration, vitality negative**).
- Pressure performed on the apical area can cause different sensation than on vital tooth.
- The tooth could be **sensitive** for **percussion, palpation**.
- **Fistula** could be present (abscessus periapicalis chronica):
  - Suppurrent process, **pus** will be excreted for pressure.
  - Fistula could be positioned away from the troubled apex, therefore X-ray image is necessary with a gutta percha cone positioned **in the sinus tract**.

**Röntgenfelvételen:**

- Radiographic signs:
  - Radiolucency is visible at the foramen apicale. The size can be small or big, diffuse or localized:
    - Granuloma periapicale,
    - Cysta periodontalis apicalis,
    - Abscessus periapicalis chronica.

**Figure 3.20. Figure 16.** – Left: Small, diffuse radiolucency is visible at the mesial and distal periapical area of tooth 46 (periodontitis periapicalis chronica). Right: Root canal filling control X-ray
Figure 3.21. Figure 17. – Small, diffuse radiolucency is visible at the mesial and distal periapical area of tooth 46 (periodontitis periapicalis chronica). On the apex of the distal root external resorption is present.

Figure 3.22. Figure 18. – Middle-sized periapical radiolucency is visible on tooth 47 (periodontitis periapicalis chronica). A: Incomplete root canal filling. B: Root canal filling control X-ray after revision. C: Control X-ray after 2 years. The bone is periapically intact.
Figure 3.23. Figure 19. – Middle-sized periapical radiolucency is visible around the apex of tooth 22 (perodontitis periapicalis chronica). Left: Diagnostic, preliminary X-ray. Middle: Control X-ray from the apical gutta-percha closure (warm vertical compaction), sealer extrusion. Right: Root canal filling control X-ray

Figure 3.24. Figure 20. – A: Small, diffuse radiolucency around the apex of tooth 12. B: Working length determination with X-ray. C: Root canal filling control X-ray. D: X-ray 18 months later
3. Reconstructive dentistry

Figure 3.25. Figure 21. – Left: Middle-sized radiolucency with well-defined outlines next to the root of tooth 34 distally. Middle: Working length determination with X-ray. Right: Root canal filling control X-ray, lateral canals are filled with sealer.

Figure 3.26. Figure 22. – Diffuse periapical radiolucency is visible on the CBCT image. It may be associated with tooth 22. (22 gangraena pulpa, perforating internal resorption. 23 tooth is vital.) Right: 3D image. Horizontal cross-section, the arrow shows the apex of tooth 22.

Figure 3.27. Figure 23. – Extended radiolucency with well-defined outlines. It is associated with the apex of tooth 21, however more teeth are involved. A: orthopantomogram detail. B: Preliminary X-ray. C: Working length determination with X-ray, correction is necessary in tooth 11. D: Root canal filling control X-ray.

- The termination of the sinus tract is determined by following the path taken by the gutta percha cone. This will direct the clinician to which tooth is involved.
3. Reconstructive dentistry

- Abscessus periapicalis chronica.

Figure 3.28. Figure 24. – Abscessus periapicalis chronica. Guttapercha cone shows to the apex of tooth 11. (Incomplete root canal filling, internal resorption, radiolucency are visible)

Figure 3.29. Figure 25. – Abscessus periapicalis chronica. Palatal orifice of sinus tract in relation with the palatal root of tooth 26. Adjacent teeth are vital

- Excessive bone mineralization could be present around the apex (as a response to chronic irritation):
3. Reconstructive dentistry

- Osteosclerosis periapicalis.

**Figure 3.30.** Figure 26. – Osteosclerosis periapicalis. Incomplete root canal filling in tooth 12 with excessive periapical bone mineralization. (Fractured instrument in tooth 13 with short but well-condensed root canal filling, periapically intact)

Periapical inflammation origined from endodontic infections can lead to acute or chronic inflamations of the jaws (periostitis, abscess, phlegmone). The discussion of these diseases is included in dento-alveolar surgery scope.

## 2.9. Periodonto-Endodontic Interrelationships

The interrelationships between pulpal and periodontal disease primarily occur due to the intimate anatomic and vascular connection between the pulp and the periodontium.

### 2.9.1. Primary endodontic lesions

**Diagnosis:**
- Pulpitis, necrotic pulp.
- Izolated parodontal damage (pocket or swelling).
- Furcation could be affected in premolars and molars.
- Alveolar bone margin is held mesially and distally.
- Small amount of plaque/calculus in the sulcus/pocket.

**Treatment:**
- Root canal treatment, root canal filling.
- Control of bone and periodontal regeneration after 4-6 months.

**Good prognosis.**

**Figure 3.31.** Figure 27. – 47 caries penetrans, primer endodontic lesion, periodontium is affected (periodontitis periapicalis chronica, furcation involvement). Good prognosis (85 persistant primary molar, 45 agenesis)
2.9.2. Primary endodontic lesions with secondary periodontal involvement

When a lesion of endodontic origin is not treated or failed, usually pathosis will continue, leading to destruction of the periapical alveolar bone and progressing into the interradicular area, causing breakdown of surrounding hard and soft tissues.

Diagnosis:
- Necrotic pulp, incomplett root canal filling or root fracture.
- Isolated, deep pocket.
- Breakdown of periodontal hard and soft tissues, significant amount of plaque/calculus in the pocket.

Treatment:
- Root canal treatment, endodontic revision.
- Periodontal treatment.
- Vertical fracture = tooth extraction.

Usually good prognosis.
Figure 3.33. Figure 29. – Primer endodontic lesion with severe periodontal involvement. A: Extended periapical radiolucency around the distal root of tooth 46, lateral periodontium is affected. The distal margin of the alveolar bone has disappeared. Furcation involvement is visible. B: Root canal filling control X-ray. C: Signs of healing are visible on the X-ray after 3 months. D: Intact bone structure and periodontal ligament space after 7 months

Figure 3.34. Figure 30. – 35 vertical fracture. Dislocation and periapical radiolucency is visible. The tooth is endodontically treated and restored with intrapulpal post (fiber reinforced resin post). Preci-Vertix abutment is over-loaded. Extraction is indicated

Figure 3.35. Figure 31. – A: Small, diffuse radiolucency, incomplete root canal filling and fractured instrument in tooth 44. B: Root canal filling control X-ray. (Before permanent filling calcium-hydroxide paste medication was used for 3 months.) C: control X-ray after 6 years. D: Pain and swelling near the tooth after 7 years. Pus excretion from the 9 mm deep pocket. E: Deep vertical bone resorption. Signs of vertical fracture
2.9.3. Primary periodontal lesions

Diagnosis:
- Periodontitis.
- The tooth is vital (however degenerative processes could be present).
- Deep periodontal pocket, periodontal pain.
- Increased tooth mobility.
- More teeth are affected (usually).

Treatment:
- Motivation for good oral hygiene.
- Scaling, smoothing of root surface.
- Elimination of local causative factors (overhang of a filling or crown, opened contacts).
- Periodontal surgical treatment may be required.

Prognosis is poor in advanced cases.

2.9.4. Primary periodontal lesions with secondary endodontic involvement

Diagnosis:
- Periodontal disease has an effect on the pulp through dentinal tubules, lateral canals or apical foramen.
- There is a defluxion for pressure from the deep pocket.
- Increased tooth mobility.
- Pulpal necrosis.

Treatment:
- Endodontic treatment, conservative or surgical periodontal treatment.

Prognosis is poor in advanced cases.

Figure 3.36. Figure 32. – 32 primer endodontic lesion with secondary periodontal involvement (periodontitis periapicalis chronica)
3. Reconstructive dentistry

Figure 3.37. Figure 33. – Primer endodontic lesion with secondary periodontal involvement. 42 extended, well-defined periapical radiolucency. 42, 43 external root resorption in the middle third. (Trauma or over-loading is suspected)

Figure 3.38. Figure 34. – External root resorption originated from the marginal periodontium. Pulpitis developed via the involvement of the root canal
3. 3.3. Endodontic Examination Methods, Differential Diagnosis of the Oro-Facial Pains – Edina Lempel

The pain is an unpleasant sensory, emotional perception, which is associated with tissue degradation. It is affected by the physiological and psychological state of the patient.

3.1. Endodontic examination methods

The purpose of a diagnosis is to determine what problem the patient is having, and why the patient is having that problem.

The diagnostic methods are simple, commonly used techniques in endodontic diagnosis establishment.

3.1.1. Anamnesis

3.1.1.1. Medical history

Endodontic treatment is not contraindicated by any general diseases, however in certain diseases or conditions special care or premedication may be necessary.

- Recent myocardial infarction (within 6 months). Uncontrolled diabetes, uncontrolled hyperthyreosis.
- Drug sensitivity.
- Antibiotic profilaxis is indicated if bacteremia may occur during the procedure and because of the patient’s underlying disease (http://www.aae.org/uploadedFiles/Publications_and_Research/Guidelines_and_Position_Statements/antibioticprophylaxisquickrefguide.pdf) (eg. prosthetic valve, rheumatic endocarditis, etc.).
- Pregnancy (X-ray of the first trimester can only be performed if it is absolutely necessary to confirm the diagnosis and perform the treatment).
- In case of bleeding disorders or anticoagulated state the patient should be premedicated before the surgical treatment.
- Known infectious diseases.

3.1.1.2. Endodontic anamnesis

Pain-centric history should be recorded:

- Pain (localization, commencement, intensity, provocation and relief of pain, duration),
- Trauma,
- Past symptoms,
- Esthetic problems.

Easier anamnesis recording is based on a structured questionnaire.

Figure 3.39. Figure 1. – Pain-centric questionnaire
The pain is information:

- From accurately recorded history sufficient information is obtained:
  - It could be specified whether the inflammation is limited only to the pulp.
  - When the periodontal ligament is not affected, the patient is unable to locate the painful tooth.
  - If the inflammation extends to the periodontal ligaments, it is easier to identify the tooth due to the proprioceptive receptors.
  - Pain is present when lying down, or standing up:
    - Pressure changes occur at the head-neck region and in the pulp as well.
    - Suggests pulpitis.
  - Diagnostic difficulties of referred pain:
    - To direction of neck, temporal region, ear (differential diagnosis)
    - To antagonist quadrant.
    - It is rare that the pain eradiate through the midline.

### 3.1.2. Extraoral examination

Visual examination: facial asymmetry, swelling, skin (color-change, sinus tract, color changes, fistula orifices, sign of previous damage or operation).

Palpation: examination of lymph nodes, swellings.

### 3.1.3. Intraoral examination
• Visual examination (magnification), palpation, percussion.
• Cariologic examination.
• Vitality test.
• Test-cavity.
• Mobility.
• Selective aneshtesia.
• Bite test.
• Simple periodontal examination.
• Fiber optic test.

3.1.3.1. Vitality test of the pulp

Provocation with cold and hot:

• Cold is the primary pulp testing method. The most popular method to performing cold testing is with a refrigerant spray (difluor-dichlorine methane). The sprayed cotton pellet should be applied to the gingival area of the tooth or crown for minimum 5 seconds or until the patient has a painful sensation.

Figure 3.40. Figure 2. – Cold test with cotton ball

• Ice-rod

• A method for heat testing is to apply heated guttapercha or compound stick to the surface of the tooth for 5 seconds or until the patient has a painful sensation.

Reactions:

• Absence of pain (does not define unambiguously gangraena).
• Mild or moderate pain is felt but disappears immediately (within 1-2 seconds) upon removal of the thermal stimulus.
• Immediate, excruciating painful sensation as soon as the stimulus is placed upon the tooth. It is released after 1-2 seconds upon removal of the thermal stimulus.
• Mild or intensive lingering pain or intensification of a painful sensation after the stimulus is removed.

The electric pulp tester has limitations in providing information about the vitality of the pulp. A positive response only denotes that some viable nerve fibers are present in the pulp and capable of responding. These tester primarily may be added advantage in case of crowned tooth.
3.1.3.2. Test-cavity

This method is used for assessing pulp vitality only when all other test methods are deemed impossible or the results of the other tests are inconclusive. A small Class I cavity preparation is made through the occlusal surface without anesthesia. If the patient feels pain once the bur contacts sound dentin, or cold test could be performed in the prepared cavity on dentin surface. This sensation signifies only that there is some viable nerve tissue remaining in the pulp, not that the pulp is totally healthy. If the result is negative trepanation will be continued.

3.1.3.3. Palpation of periradicular mucosa and gingiva

Palpation should be performed by the tip of fingers or with a silicate-condensing (ball) instrument.

Figure 3.41. Figure 3. – Palpation with a blunt-ended instrument

In the course of the soft tissue examination, the alveolar hard tissues should also be palpated. Emphasis should be placed on detecting any soft tissue swelling or boney expansion, especially noting how it compares with and relates to the adjacent and contralateral tissues. The clinician should question the patient on any areas that feel unusually sensitive during this palpation.

3.1.3.4. Selective anesthesia

Sometimes the patient may not even be able to specify whether the symptoms are emanating from the maxillary or mandibular arch (referred pain). In these instances, when pulp testing is inconclusive, selective anesthesia may be helpful. The clinician should first selectively anesthetize the maxillary arch. This should be accomplished by using a periodontal ligament injection (intraligamentary). It is placed more forward, one tooth at a time, until the pain is eliminated. If, after a period of time, the pain is not eliminated, this technic should be repeated on the mandibular teeth below.

Technique:

- Intraligamentary anesthesia: with 0,2ml Lidocain-Adrenalin or Articain-Adrenalin solution.

- The injection is administered to the most posterior tooth in the quadrant that may be suspected, starting from the distal sulcus.

Figure 3.42. Figure 4. – Selective anaesthesia
3.1.3.5. Bite test

Bite tests are indicated when a patient presents with pain while biting and may help to localize the tooth involved. The tooth may be sensitive to biting when the pulpal pathosis has extended into the periodontal ligament space, however there is no radiographic sign, or the sensitivity may be present secondary to a crack in the tooth. A variety of devices have been used for bite tests, including cotton applicators, toothpicks, orangewood sticks, and rubber polishing wheels. Adjacent teeth should be used as controls.

Signs of incomplete fracture during the bite test:

- Pain.
- The crack will be visible (magnification, dye penetration test may help with caries indicator).
- The pain is released upon the removal of the bite tester.

**Figure 3.43. Figure 5. – Testing of incomplete fracture with bond applicator**

3.1.3.6. Simple periodontal examination
Endodontic pathosis can affect the periodontal tissues and periodontal diseases can affect the pulp as well.

The measurement of periodontal pocket depth is an indication of the depth of the gingival sulcus, which corresponds to the distance between the height of the free gingival margin and the height of the attachment apparatus below. Deep pocket depths indicate pathologic horizontal or vertical bone loss. Using a calibrated periodontal probe, the clinician should record the periodontal pocket depths on the mesial, middle and distal aspects of both the buccal and lingual of the tooth, noting the depths in millimeters. Furcation bone loss can be secondary to periodontal or pulpal disease. The amount of furcation bone loss, as observed both clinically and radiographically, should be documented.

Based on the endodontic and periodontal examination the clinician should define the prognosis of the tooth.

**Figure 3.44. Figure 6. – Periodontal examination**

3.1.3.7. **Fiber-optic examination**

The light can only penetrate through an intact tooth surface, cracks or fractures block it.

3.1.3.8. **Magnification**

Operating microscope or lupe.

It is useful primarily in the perception of cracks and fractures.

**Figure 3.45. Figure 7. – Endodontic examination with operating microscope (Leica M320)**
3. Reconstructive dentistry

Figure 3.46. Figure 8. – Fracture next to the amalgam filling on the disto-lingual cusp of tooth 46. Cusp protection is necessary

Figure 3.47. Figure 9. – Tooth 16 is endodontically treated and restored with crown. After removal of the crown and partial removal of glass-ionomer core the mesio-distal fracture is clearly visible under magnification

3.1.4. Imaging tests

The image should be used only as one sign, providing important clues in the diagnostic investigation. When not coupled with a proper history and clinical examination and testing, the radiograph alone can lead to a misinterpretation of normality and pathosis. When the diagnosis is difficult, multiple exposures may be necessary in order to determine the presence of multiple roots, multiple canals, resorptive defects, caries, restoration defects, root fractures, and the extent of root maturation and apical development. The radiograph is able to show the mineralized tissues. The radiographic appearance of endodontic pathosis can sometimes be very subjective. This emphasizes the necessity for other objective diagnostic tests, as well as the importance of obtaining and comparing older radiographs. When endodontic pathosis appears radiographically, it appears as bone loss in the area of the periapex.

Types of X-rays used for endodontic purpose:

• **Intraoral, periapical radiograph**
  
  • It is **essential** in root canal treatment.
  
  • It is made from one or more directions (with 10-15 degree of horizontal deflection).
3. Reconstructive dentistry

- If a sinus tract is present, to trace it a gutta-percha cone is threaded into the opening of the sinus tract and should be inserted until resistance is felt. After a periapical radiograph is exposed as a contrast uptake.

- **Crown** or **bite-wing images** are useful to detect crown-lesions and to assess the situation of the pulp chamber.

- **Orthopan radiograph** gives an overview, extensive lesions, anatomical relationships are shown well.

- **Occlusal radiograph** is advisable for the assessment of the oro-vestibular dimension of the oral structures and lesions. Primarily it is useful in endodontic surgery.

- Three-dimensional [CBCT images](http://www.aae.org/uploadedFiles/Publications_and_Research/Guidelines_and_Position_Statements/conebeamstatement.pdf): primarily it is useful in diagnostic difficulties (vertical fracture is suspected) or in the treatment of a tooth with unusual anatomy.

**Figure 3.48. Figure 10. – CBCT image in three dimension. Extreme anatomical variation of tooth 23 (dens invaginatus)**

![CBCT image](image-url)  

Main outcomes of the evaluation of an endodontic radiograph:

- Is the **lamina dura** intact or is missing section shown in the periapical area?

- Does the **bone structure** show normal architecture or are any signs of demineralization visible?

- What is the location and width of the **pulp chamber**?

- Is the **root canal system** within normal limits or does it show signs of resorption or calcification?

- Is the **root canal system** easily followable?

**Figure 3.49. Figure 11. – 35 pulpitis acuta. Left: Ramification of root canal. Right: Root canal filling control X-ray**

![X-ray image](image-url)
3. Reconstructive dentistry

- What kind of anatomical structures are seen on the radiograph?

- In case of previous endodontic procedures, how do we judge the quality of the root canal filling or is there any preparation error, which makes it hard to retreatment?

- Does the analysed radiograph give a satisfactory information or additional X-rays are needed?

Further questions:

- One or more root canals are present in the root?
  
  - In case of one root canal, it is followable in the entire length of the root.
  
  - In case of ramified root canal, the clearly visible root canal will be blurred suddenly.

**Figure 3.50. Figure 12. – The dentist could not reach the optimal length in tooth 34.**

A: Preliminary X-ray, calcium-hydroxide releasing guttapercha cone was applied in the root canal. The run of the root canal is blurred from the middle of the root. B: The ramification of the root canal is clearly visible with operating microscope. Both root canals were enlarged. C, D: Control X-rays after root canal filling

- Duplicated apex could be present.
• In case of radiographic working length determination, if the file is not positioned in the centre of the root canal, the presence of another channel arises.

• Is the pulp necrotic?
  • There is no radiological sign of the pulp necrosis.
  • Degradation products of the pulpal degeneration, bacterial metabolites and toxins may cause demineralization of the periapical bone.
  • These can penetrate from the pulpal space not only through the apical foramen but also through the lateral root canals and the furcation.
  • Demineralization starts in the trabecular bone at first and the cortical plates are affected later.

• Are other pulpal lesions present?
  • Pulp stones, denticules, other calcifications (it is not pathologic in itself).
  • Internal resorption (endodontic treatment is necessary as soon as possible).
  • Perforated external resorption (endodontic treatment is necessary).

• Maturity of the apex.

Figure 3.51. Figure 13. – Immatured teeth (11, 12) are injured in trauma. 11 permanently root canal filled and calcium-hydroxide paste medication in tooth 12

• Presence of root fracture:
  • After traumatic injury and in case of intrapulpal posts.
  • Only the horizontal fracture is visible in early stage.
  • In case of vertical and transversal root fracture, bone demineralization may be present.
  • Mock-moon-like lesion.
  • If the fracture occurs during a root canal, filling outflow may appear.

Figure 3.52. Figure 14. – A: Horizontal fracture in the middle root-third of tooth 11 and the apical root-third of tooth 21 due to trauma. 11 tooth is vital, 21 tooth has symptomatic pulpitis and mobility. B: The fractured fragments are dislocated in tooth
21, therefore the root canal preparation was performed only in the coronal fragment. C: Root canal filling and flexible splint. D: Control X-ray after 2 months

3.1.5. Laboratory diagnostic methods

In endodontic practice laboratory testing is rarely performed. For high risk patients or who have persistent or severe infection may be indicated. The purulent secretion is sampled at the beginning of treatment for the determination of the infectious microorganism and antibiotic sensitivity testing. Pus or the contents of the root canal are absorbed in a sterile syringe without bubbles (do not use disinfectant solution in this step). The sample injected into an anaerobic transport medium shall be sent to the lab within 2 hours.

3.2. Differential diagnosis of oro-facial and dental pain

The patient may complain of a „dental pain”, however the origin of the pain is not dental:

- Temporo- mandibular disorders = TMD (gnatology deals with this subject).
- Pain in the head-neck region – there is not correlation with teeth
  - Often pain with non-odontogenic origin can mimic odontogenic pain.
  - The pain could be experienced as an odontogenic pain and the patient insists of this fact.
  - Some common features:
    - The pain follows the nerves and may arise from the innervated structures.
    - It can occur on a large area.
    - The pain is frequently diffuse and unilateral (similar to irreversible pulpitis).
  - Acute and chronic orofacial pain should be differentiated.

3.2.1. Acute disorders with facial pain

3.2.1.1. Trigeminus neuralgia

The most common neuralgia. It is typical in elderly females (age: 50-60). It is extremely inconvenient for the patient. Trigeminus neuralgia is a characteristic (http://www.fpa-support.org/2011/01/about-neuropathic-facial-pain-and-tn-trigeminal-neuralgia/) syndrome.

- Extreme pain attack in trigeminus branch (usually unilateral).
- Intensive dental pain sensation.
- The quality is electric-shock-like, sharp, stabbing.
3. Reconstructive dentistry

- Sometimes there is a prodromal symptom too (formication).
- Localization: the patient points out the area supplied by the trigeminus.
- **Trigger points:** the pain is induced by pressure of certain points (intra-extraoral).
- The pain attack (ictus) is short, lasts for a few seconds, **shorter than 1 minute.**
- Secondary pain is burning. The patient usually tries to ease the pain with a massage.
- The patient learns to avoid the triggering of these zones.

The pulpal origin must be excluded.


Other type of neuralgia may cause „dental pain“-like sensation as well, however these are very rare (cluster headache - ggl. sphenopalatinum neuralgia).

### 3.2.1.2. Acute sinusitis maxillaris

Inflammation of sinus mucosa can cause dental discomfort, especially biting sensitivity in the premolar-molar region. This disease is characterized by biting sensitivity. The face could be painful for pressure at the area of the sinus. Can eradiate to the mandible on the same side.

Differential diagnosis:

- The response for thermal test is positive.
- Sensitivity for palpation and percussion.
- Pain is intensified by leaning forward.
- Forehead could be painful.
- Coryza.
- Have to differentiate from odontogen sinusitis.

### 3.2.1.3. Acute otitis media

- Pain in otitis media can eradiate to the upper-lower molars on the same side.
- Other symptoms are present.
- Pain: acute, throbbing.
- Pain is intensified by leaning forward.

### 3.2.1.4. Myocardial pain

- General anamnesis is very important.
- In case of angina there is eradiation of pain in 10% into the mandibule.
- If the patient relaxes it could be released.
- Mostly the pain is present on the left side, but could be bilateral.
- Pain of shoulders, arms, neck, back.
- The patient could be in serious state of health.
3.2.1.5. **Herpes zoster**

Mostly the 2., 3. branches of Trigeminus are affected.

In rare cases it can cause symptomatic pulpitis in the prodromal phase (lasts for weeks).

- The pain is unilateral, one or more teeth are affected, sharp, throbbing intermittent. Easy to point out the painful tooth.
- It is hard to diagnose the zoster in the prodromal phase (lasts for weeks).
- Early endodontic treatment can release the pulpal pain.
- After the infection the health of the pulp and the periapical area must be controlled.

3.2.1.6. **Sialolithiasis**

Swelling of the salivary gland is similar to a lymphadenitis.

Pain and swelling:

- The pain starts at salivation and intensifies.
- The character of the pain is tension-type.
- Teeth’s response is normal for thermal and mechanical tests.
- Diagnosis: RTG, ultrasound examination

3.2.2. **Chronic disorders with facial pain**

3.2.2.1. **Temporo-mandibular disfunction (Temporomandibular disorder, craniomandibular disorder)**

- Causing factor: stress (bruxism).
- Neurological, joint and dental components.
- Mostly young females are affected.
- Pain is dull and diffuse.
- Exacerbation at mastication, at mouth-opening.
- Myalgia: pain is triggered by pressure of the muscles.

3.2.2.2. **Atypic facial pain**

It is not a characteristic dental pain. Its origin is unclear.

The pain:

- Constant, throbbing, burning.
- Difficult to localize.
- Can migrate.
- There are not trigger points.
- Pain killers can not ease the pain.
- Physical/psychical stress can increase the intensity.
3. Reconstructive dentistry

Differential diagnosis: painful teeth’s response is normal for examination tests and the periodontium is healthy as well.

3.2.2.3. Phantom dental pain

- This is a persistant pain of oral structures after pulp extirpation, apicectomy or extraction.
- The incidence is 3% after pulpectomy.
- The pain is constant, dull, deep, spontaneous.
- In case of chronic pain it is difficult to localize.
- Treatment (abusively) of adjacent teeth can release the pain.
- Clinical and radiological tests are negative.
- Differential diagnosis:
  - Must be differenciated from dental pain, neuralgia, atypic facial pain (dental history).

3.2.2.4. Cancer

Myeloma multiplex (plasmocytoma): bone destruction and pain (in other bones as well).
- Pain is caused by compression.
- Associated with disorders of motoric functions.
- Associated with visual, auditve, gustation, tactile disorders.
- Pain is flaming, associated with paresthesia.
- Not typical pulpal pain.

4. 3.4. Pathology of pulp and periapical tissues – Edina Lempel

4.1. The healthy pulp

From many perspectives, the pulp is a unique soft tissue with ecto-mesenchymal origin. Pulp consists of specialized cells, the odontoblasts, arranged peripherally in direct contact with dentin matrix. This close relationship referred to as the dentin-pulp complex, is one of the reason that dentin and pulp should be considered as a functional entity made up of histologically distinct elements. Pulp is enclosed in the rigid mineralized dentin which results in special peculiarities. Thus it is situated within a low-compliance environment. As a consequence, inflammatory reactions result in an increase in tissue pressure instead of volume.

The mature pulp bears a resemblance to embryonic connective tissue and is therefore a relatively rich source of stem cells. The pulp houses a number of tissue elements, including nerves, vascular tissue, connective tissue fibers, ground substance, interstitial fluid, odontoblasts, fibroblasts, immunocompetent cells, and other cellular components.

Figure 3.53. Figure 1. – Layers of pulp tissue
Function of the pulp:

- Cells, blood vessels, nerves protect the vitality of the pulp.
- Protective function,
- Nutritive function,
- Reparative function.

Etiology of pulp diseases:

- Bacterial infection (caries),
- Dentintubules open to the pulp,
- Bacteria and their products get into the periapical area from the pulp.

Figure 3.54. Figure 2. – Progression of pulp diseases and development of periradicular pathosis
Pulp diseases could be acute or chronic, depends on the virulence of bacteria and the immunologic response of the host.

**Figure 3.55. Figure 3. – Pulp responses to caries progression**

4.1.1. **Anatomic features affecting inflammation of the dental pulp**

- The dental pulp is surrounded by unyielding calcified walls that limits its ability to increase in volume.
- Capillary dilation and the transudation of fluids increases the volume of tissue.
- Swelling causes increased pressure stimulating pulpal nerves to register pain.
- Pulpal blood vessels are supplied by small feeder vessels entering through, usually, a single narrow channel at the apical foramen.
- Secondary blood supply is missing.
- The pulp less capable of carrying out an inflammatory response.
- As the pulp swells, the constricted source is cut off which leads to necrosis
- Because the tooth is embedded in the jaws, pulp infection will invariably extend into the surrounding bone.

4.1.2. **Histopathologic classification of pulpal diseases**

- Regressive changes,
- Inflammatory changes,
  - Pulpal hyperaemia,
  - Chronic pulpitis,
  - Acute pulpitis,
  - Necrosis of the pulp,
4.2. Regressive changes of the pulp

4.2.1. Vascular degeneration of odonotoblasts

Odontoblasts react very quickly for many stimulies. This reaction leads to often vacuolization of odontoblasts. This change can be result of pathologic damage of the pulp. It is the result of intracellular or extracellular breakdown of cells metabolism.

4.2.2. Hyaline degeneration of the pulp

In this regressive change, albumenic substances are found in the pulp. This type of degeneratin is typical in elder teeth with chronic inflammation.

4.2.3. Lipidic degeneration of the pulp

Chronic inflammation results in this form of change. Lipidic degeneration is characterized by the breakdown of metabolism, which leads to lipid accumulation in the pulpal cells.

4.2.4. Reticular atrophy

Lost odontoblasts and pulpal cells are compensated by interstitial tissue formation in matured teeth.

4.2.5. Pathological calcification

Pathological calcification results from normal aging, caries, trauma, periodontal disease, operative procedures and, systemic factors. Histologically, it is characterized by the reduction in the quantity and size of pulp cells (including odontoblasts, fibroblasts, undifferentiated mesenchymal cells), increase in pulp stones, dystrophic mineralization of fibers, vascular and neural elements, increased collagen deposition, increased peritubular and reparative dentin formation in the coronal and radicular parts of the tooth, and decreased blood and nerve supply.

A coagulation type of necrosis may be found. The pulp cells are shrunken, and the nuclei are pyknotic or karyorrhectic. Collagen fibers persist, but they may be heavily mineralized. Nerves and blood vessels are also sites for dystrophic mineralizations. Pulp stones are abundant, and the dentinal walls, both coronally and radicularly, are covered with large amounts of reparative, amorphous dentin, tending toward obliteration of pulp and space.

4.3. Inflammatory diseases of the pulp

Acute inflammation is accompanied by the classic signs and symptoms of heat, redness, pain, swelling and loss of function; these are muted or absent in chronic inflammation. Heat, redness, and swelling are not clinical features of pulpal inflammation because the dental pulp is hidden from view. Pain and the loss of function is the only feature of inflammation that accompanies pulpal inflammation.

4.3.1. Reversible pulpitis (hyperaemia pulpa)

Reversible pulpitis represents the first stage of pulp inflammation. This form is mild to medium pulp inflammation caused by stimulation and the defense system of the pulp tissue is still able to recover. In this condition the pulp is actively responding to an irritant which may be bacterial toxin or product reached the pulp. The immune response is mostly characterized by cell-mediated immunity in reversible pulpitis. The immune responses could be innate and/or adaptive.

Figure 3.56. Figure 4. – Immune responses to caries progression
The components of the innate response of the dentin/pulp complex to caries include at least the following six:

- outward flow of dentinal fluid,
- odontoblasts,
- neuropeptides and neurogenic inflammation,
- innate immune cells, including immature dendritic cells (DCs), natural killer (NK) cells, and T cells,
- cytokines,
- chemokines.

Although the first two items are not classic components of innate immunity, they are uniquely involved in the initial inflammatory response to caries.

Odontoblasts have cellular processes that extend into dentinal tubules and are the first to encounter the caries bacterial antigens. They express low levels of interleukin 8 (IL-8) and genes related to chemokines and chemokine receptors. The odontoblasts have been shown to attract immature dendritic cells.

Dendritic cells (DCs) are a heterogeneous leukocyte (white blood cell) population. DCs in healthy peripheral tissues (steady state) are in an immature state. The cells are capable of sensing microbes as well as antigen capture and processing capabilities. A rapid accumulation of pulpal DCs has been observed beneath cavity preparations, and an increased number of DCs accumulated under caries. Immature DCs are therefore considered to be part of the innate phase of pulpal immune response.

**Figure 3.57. Figure 5. – Function of dendritic cell (Antigen presentation)**
Macrophages are professional phagocytes in innate immune responses. Activated macrophages are effective killers that eliminate pathogens in both innate and adaptive immune responses, and are also important in tissue homeostasis, through the clearance of senescent cells, and in remodeling and repair of tissue after inflammation. The number of macrophages increases with the progression of caries and is always higher than that of DCs at all stages of the caries invasion.

Pulp hyperaemia is characterised by the destruction of odontoblasts and the invasion of the subodontoblastic region by lymphocytes and plasma cells, as well as dilation of arteries and arterioles with a clearly increased flow velocity of the blood.

The affected tooth does not hurt all the time; pain is elicited with thermal stimulation, particularly application of cold. The symptoms seem to be associated with dilated blood vessels and transudation of fluids.

4.3.2. Cracked tooth syndrome

If a crack penetrates into dentin, mastication will be painful. Pain produced by A fibers in response to the hydrodynamic mechanism has a sharp or bright quality. The crack may progress into the root system to involve the pulp, or it may even split the entire tooth into two separate pieces. Biting sensitivity may be caused by pulpal pathosishas extended into the periodontal ligament space, creating a periradicular periodontitis.

4.3.3. Irreversible pulpitis

This is the condition where the pulp is irreversibly damaged. The pulp can not recover from the insult and damage. For example, decay that has reached the pulp of the tooth introduces bacteria into the pulp. The pulp is still alive, but the introduction of bacteria into the pulp will not allow the pulp to heal and it will ultimately result in necrosis, or death, of the pulp tissue. The acute pulp inflammation is characterized by inflammatory changes in the odontoblastic layer, directly under the involved dentinal tubules. The inflammation generally does not extend into the deep pulp tissue. Edema causes disruption of the odontoblastic layer and a reduction in the number of odontoblasts. Dilated capillaries, filled with erythrocytes, are evident. Polymorphonuclear leukocytes (neutrophils and eosinophils) are seen both in the capillaries and extravascularly. The immune response is mostly characterized by humoral immunity in irreversible pulpitis.

4.3.3.1. Asymptomatic irreversible pulpitis (Pulpitis chronica)

Chronic pulpitis, resulting from mild to moderate irritating operative manipulations, is usually seen in the subodontoblastic layer under the involved dentinal tubules. The cell-rich and cell-free zones are invaded by macrophages, lymphocytes, and, occasionally, plasma cells. These cells are also found, after more severe chemical injuries, in the deeper pulp tissues. Abundant dilated capillaries, new fibroblasts, and reticular and collagen fibers are also present. In the older lesions, abundant quantities of reparative dentin are elaborated, presumably by undifferentiated reserve cells. The odontoblastic layer lining this reparative dentin usually consists of a single layer of flattened, fibroblastic appearing cells. The radicular pulp tissue usually is intact but contains widely dilated blood vessels.

- Pulpitis chronica clausa ascenden:

"Pulpitis chronica clausa ascenden:"

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3. Reconstructive dentistry

Under dental caries, chronically inflamed pulps are usually densely infiltrated with lymphocytes and plasma cells. Under deep carious dentinal lesions, pulp abscesses develop under the heavily infected or necrotic dentinal tubules. Polymorphonuclear leukocytes are abundantly present around the liquefaction necrosis.

- **Pulpitis chronica clausa granulomatosa (internal granuloma, Palazzi-granuloma):**

Granulation tissue shows collagen, numerous blood vessels, edema, and a loose immune system. Granulation tissue is develop when fibroblasts synthesize big amount of collagen and proteoglycans and neovascularization occurs. Migration and proliferation of fibroblasts is induced by growth factors. Granulation tissue may involve the remainder of the coronal pulp tissue and an internal resorption is develop. Inflammatory stimuli that could elicit or enhance internal resorption include three possibilities: cytokines, prostaglandins and possibly increased internal tissue pressure.

The internal granuloma is a very rare occurrence (1.6%). Internal resorption exhibits a high number of vessels. Visible are neutrophilic granulocytes and lymphocytes as well as multinucleated giant cells (dentinoclasts).

The radicular tissue may remain resistant. However, periapical changes, such as dilated blood vessels, edema, alveolar bone resorption, and scattered chronic inflammatory cells, may be noted. Huge quantities of amorphous reparative dentin, lined with fibroblast-like cells, are found under the involved dentinal tubules.

- **Pulpitis chronica aperta ulcerosa:**

This pathosis consists of three zones: ulcerative zone externally, contamination zone in the middle and the proliferative zone pulpally. Especially primary and immature permanent teeth are affected. The disease caused by low-grade virulence microorganisms. The defenses of the host is good and because the pulp chamber is opened by the penetrans caries there is a possibility to release the inflammatory exudate. It is characterized by local vascular dilatation, infiltration of mononuclear leukocytes, partial tissue necrosis and ulcus formation.

- **Pulpitis chronica aperta granulomatosa:**

Pulpal exposure with long-continued irritation in primary or immature permanent teeth can lead to a proliferative response, or hyperplastic pulpitis. Exuberant inflammatory tissue proliferates through the exposure and forms a „pulp polyp“. It is presumed that a rich blood supply coupled with ample lymphatic and oral drainage allows this proliferative response. This pathosis is characterized by lymphocytes, macrophages, plasmocytes and histiocytes. In deeper regions there is an accumulation of fibroblast with collagen production. The surface is covered with fibrin and epithelial cells originated from the oral mucosa.

**4.3.3.2. Symptomatic irreversible pulpitis (Pulpitis acuta)**

Persistent infection leads to the activation of adaptive immunity. A transition to an adaptive immune response will take place in the dental pulp as the caries and bacteria approach the pulp. Antigens are recognized individually and lines of lymphocytes are developed to produce specific antibodies which attack to the recognized cells and initiate their destruction. Phagocytes remove the remains. B cells and T cells are the major lymphocytes involved.

Accumulation of neutrophils that discharge lysozymes to digest phagocytozed bacteria can lead to micro-abscess formation.

A variety of cytokines have been observed in the pulp. Patients with symptomatic and asymptomatic irreversible pulpitis have been shown to have an almost 23-fold increase in the cytokine IL-8 in the pulp. Cytokines in the pulp interact with each other. The ultimate effect on pulpal inflammation and healing is dependent upon the integrated actions of these inflammatory mediators.

In addition to the lymphocytes, macrophages also provide defense against certain intracellular pathogens. Activated macrophages can function as class II antigen-presenting cells, similar to pulpal dendritic and B cells. In addition, activated macrophages secrete many inflammatory mediators.

The dull aches are associated with C-fibers and slow Aδ-fibers. As inflammation intensifies, the A-fibers are increasingly activated. C-fiber innervation and Aδ-fibers are polymodal receptors that are sensitive to inflammatory mediators. The pain mechanisms associated with pulpitis are similar to those of the rest of the body (i.e. receptors, intracellular signaling, transmitters, etc.). The inflammatory mediators act on specific...
3. Reconstructive dentistry

receptors relating to nociceptive neurons, leading to the production of second messengers and activation of phospholipases and protein kinases. The second messengers regulate receptors ion channels that deal with sensitization. The ion channels open based on pain stimuli propagating action potentials in sensory neurons.

4.3.4. Pulpal necrosis (gangraena pulpa)

In severe pulpitis of operative origin and under deep-seated caries, portions of the coronal pulp undergo liquefaction necrosis (abcess). Such a reaction usually leads eventually to total pulp necrosis. Histologically, partial pulp necrosis exhibits a liquefied zone, surrounded by live, dead, and dying polymorphonuclear leukocytes. Portions of, or the remainder of, the coronal pulp tissue are converted to granulation tissue rich in macrophages, lymphocytes, and plasma cells. It is tempting to consider the presence of lymphocytes and plasma cells as a sign of localized antigen-antibody reaction. In time, the radicular pulp tissue succumbs, and granulation tissue is found in the apical portion of the root canal and also in the periodontal ligament.

4.4. Inflammatory diseases of the periapical tissues

Periapical tissues consist of the periodontal ligament and the alveolar bone. Periapical lesions resulting from necrotic dental pulp are among the most frequently occurring pathologies found in alveolar bone. Exposure of the dental pulp to bacteria and their by-products, acting as antigens, may elicit nonspecific inflammatory responses as well as specific immunological reactions in the periradicular tissues, and cause the periapical lesion.

The principal cause of apical periodontitis is the persistence of microorganisms in the root canal system. The microorganisms found in these cases are predominantly Gram-positive microorganisms (cocos, bacillus and filaments, such as Actinomyces, Enterococcus and Propionibacterium) and fungi mainly Candida albicans.

Figure 3.58. Figure 6. – The host response in apical periodontitis (Source: Nair PNR: Periodontol 2000 13:121,1997)

4.4.1. Acute Inflammatory Periapical Diseases

The structural components of a periapical lesion depend on the balance between the microbiological factors and the host defenses. Thus, when the pulpal infection spreads to the periapex, a symptomatic inflammatory response of the periapical connective tissue is produced in the form of an abscess or acute lesion.

4.4.1.1. Acute periapical periodontitis (periodontitis periapicalis acuta, symptomatic periapical periodontitis)
When the infection reaches the periapex a predominantly anaerobic mixed flora is established; in response, the host releases defense mechanisms, in the form of various cell types, intercellular messengers and antibodies. The microbiological factors and the host defense mechanism interact, destroying are large amount of periapical tissue, giving rise to the different types of periapical lesion. The primary response at vascular level is rapid vasoconstriction, followed almost immediately by vasodilation, giving rise to an accumulation of red blood cells in the center of the vessel, and a migration of leukocytes to the peripheral areas, adhering to the vascular walls. This provokes the formation of small fissures in the endothelium of the vessel, inducing an extravasation of plasma towards the connective tissue; edema is produced which increases local pressure and compressing the nerve endings, causing pain.

4.4.1.2. Acute periapical abscess (abscessus periapicalis acuta, symptomatic periapical periodontitis)

The final consequence of the inflammatory process is an infiltrate containing lymphocytes, macrophages and plasmatic cells. In the acute phase of the inflammation, an exudate is produced as a response to the aggression of the pulp and periapical tissue, with predominance of polymorphonuclear neutrophils. The degranulation and release of lysosomal (myeloperoxidase, beta-glucuronidase, lysozyme) and cytoplasmic (lactate dehydrogenase) enzymes from polymorphonuclear neutrophil granulocytes during phagocytosis of bacteria and degraded tissue particles leads to pus formation.

4.4.1.3. Chronic periapical periodontitis (periodontitis periapicalis chronica, asymptomatic periapical periodontitis)

Once the inflammation has reached the chronic stage, the host responds with a proliferation of new cells, vessels and fibers, in an attempt to repair the lesion, resulting in the formation of new tissue.

- Granuloma periapicale

Periapical granuloma is a localized mass of chronic inflammatory tissue, with acute inflammatory infiltrate containing macrophages and polymorphonuclear cells; and chronic inflammatory infiltrate containing B and T lymphocytes.

In chronic periapical periodontitis it is common to find nests of epithelium, formed from epithelial cell rests of Malassez, having a latent capacity to proliferate. The epithelial cells are normally arranged in layers forming small islands, strands and / or trabecula of varying thickness. Thus, the periapical granuloma histopathologically consist of a granulomatous tissue with infiltrate cells, fibroblasts, and a well-developed fibrous capsule. The epithelial cells generate an „epithelial attachment“ to the root surface or canal wall.

Extraradicular periapical actinomycosis is a chronic granulomatous infection, caused by species of types Actinomyces and Propionibacterium; with Actinomyces israelii being the most frequently isolated species. These microorganisms are able to build cohesive colonies and so escape the body’s defense mechanisms, thus establishing themselves in the periapical tissue.

Figure 3.59. Figure 7. – Periapical granuloma on the apex of extracted tooth
• Cysta periodontalis apicalis

The radicular cyst is a chronic inflammatory lesion with a closed pathologic cavity.

Figure 3.60. Figure 8. – X-ray image of a periapical cyst on tooth 12
Figure 3.61. Figure 9. – A periapical cyst in the alveolar bone
Figure 3.62. Figure 10. – Exstirpated periapical cyst with fibrous capsule
The cyst is lined either partially or completely by non-keratinized stratified squamous epithelium. The underlying fibrous connective tissue wall is inflamed with varying degrees of cell infiltration, which consists mainly of macrophages and small blood vessels. In radicular cysts, cholesterol crystals move in the direction of the epithelium-lined cyst cavity, since the outer collagenous capsule of the lesion is too tough for the crystals to move through.

The cholesterol crystals are surrounded by macrophages and giant multinucleate cells that are unable to degrade the crystals and act as mediators, increasing inflammation and bone resorption.

The etiopathogeny of cysts is particularly controversial, there formation has been explained by diverse theories, such as epithelial colonization, epithelial cavitation, or the formation of microabscesses.

**Figure 3.63. Figure 11. – Periapical cyst with purulent product**

**Figure 3.64. Figure 12. – Resorbed alveolar bone after cyst exstirpation**

**Classifications of cysts:**

**Figure 3.65. Figure 13. – Types of periapical cysts**
- **Periapical true cyst**: When the cystic cavity is surrounded completely by epithelium and is not directly connected with the radicular canal. Histopathologically has four major elements: the cyst cavity, the epithelial cyst wall, the extraepithelial tissue, and the collagenous capsule. The cavity completely enclosed in an epithelial lining, generally reveals necrotic tissue and sometimes cholesterol clefts. The tissue between the epithelial lining and the fibrous capsule consists of numerous blood vessels and infiltrating cells (T, B lymphocytes, plasma cells. The epithelial lining contains neutrophils. The process of cyst formation has three stages: 1. the dormant cell rests of Malassez proliferate, 2. epithelium-lined cavity forms, 3. growing of the cyst (osmotic pressure theory).

- **Periapical pocket cyst**: When the epithelium-lined cystic cavity is directly connected with the radicular canal. Pocket cyst is initiated by the accumulation of neutrophils around the apical foramen in response to the bacteria. The microluminal space becomes enclosed in a stratified, squamous epithelium, which grows and form an epithelial collar around the apex tip with an epithelial attachment. As the necrotic tissues and microbial products accumulate, the sacklike lumen enlarges, forming a voluminous diverticulum of the root canal space into the periapical area.

- **Abscessus periapicalis chronica**

  Sometimes a small pocket of acute inflammation will arise within CAP but not become an acute abscess. Because this pocket of acute inflammation produces a purulent exudate it is known as "suppurative apical periodontitis."

**Figure 3.66. Figure 14. – Chronic periapical abscess with purulent exudate**
The pathosis is characterized by polymorphonuclear neutrophil granulocytes, thus pus formation and epithelial cells. The purulent exudate is transported/drainaged through a channel lined by fibrous connective tissue and chronic inflammation cells. This channel is called "fistula" or "sinus track."

**Figure 3.67. Figure 15. – Extraoral sinus tract**
**Osteosclerosis periapicalis**

This pathosis (Focal sclerosing osteomyelitis) is a reaction of bone resulting in the formation of dense bone induced by inflammation. The infection of the pulp progresses to the apical tissues to produce a small periapical radiolucency called rarefying osteitis. The small rarefying osteitis may be either a periapical granuloma, a radicular cyst or an abscess. The bone surrounding this rarefying osteitis becomes dense in order to prevent further spread of the lesion.

5.3.5. **NiTi rotary systems and usage of supplementary electronic devices, and operating microscope in endodontic treatment – Dora Ottoffy-Kende**

5.1. **Rotary NiTi preparation instruments**

The root canal treatment is a great technical challenge for a dentist. Working with traditional techniques and devices are exhausting and time consuming. Several rotary instruments were developed for making the dentists work easier and faster. The greatest change was the appearance and spreading of the NiTi rotary instruments 20 years ago. The success caused by the super elasticity of the NiTi alloy, permitting of continuous rotary movements in the curved canals without clinically significant preparation mishaps. Developing NiTi rotary instruments is permanent. Preparation movements, properties and fracture resistance of the instruments are developed continuously by the manufacturers. The shape, the preparation movements (rotary or non rotary movements), the rotary speed and the sequence of the instruments could be different in each NiTi systems. The basic characteristics of the NiTi instruments cannot be defined, only the alloy which is the same in all systems. The instruments can be otherwise classified in different subgroups with same characteristic features.

![NiTi rotary file and stainless steel hand instrument](image)

However the different instruments can create a separate systems with a proper indication based on the manufacturer’s recommendation the specialists usually combine the different systems acting upon the difficulties of the preparation.

5.1.1. **Features of the NiTi rotary instruments**
5.1.1.1. The shape of the files

All of the NiTi instruments have non-cutting tip. The conicity of the instruments is various usually bigger than the conicity of the stainless steel hand instruments with 0.02 conicity. The length of the cutting zone can be differ from the ordinary 16mm. The cross section of the instruments are various can be symmetric and asymmetric. Instruments with active and passive cutting edges and instruments with another edge design can be distinguished.

- **Passive blade design:**
  - No cutting edges.
  - Slower preparation, but the preparation keeps better the original shape of the root canale.
  - Preparation mishaps (1) rarely occur.
  - (ProFile, System GT, FlexMaster, K3 – more effective because of the positive Rake-angle).

**Figure 3.69.** Figure 2. – Passive cutting edge with triple U cross section

- **Active blade design:**
  - Usually three (triangle cross-section), or two (S shape cross section), sharp cutting edge.
  - The cutting edges work the whole length of the cutting zone of the instrument.
  - Effective preparation, preparation mishaps occur more frequently.
  - (ProTaper Universal, Mtwo, RaCe, Reciproc, WaveOne, HERO Shaper, Endo Sequence, Twisted Files).

**Figure 3.70.** Figure 3. – Active cutting edge, triangular shaped cross section

**Figure 3.71.** Figure 4. – Active cutting edge, S shaped cross section

- **Active cutting edges, but asymmetric design:**
• The edges not work the whole length of the cutting zone.

• The load affect on the instruments and nipping probability is smaller.

• (Revo S, ProTaper Next – the axis of the instrument is not the symmetry axis of the cross section; G-files – asymmetric cross section; One Shape – various shaped cross section along to the axis of the instrument).

**Figure 3.72. Figure 5. – Excentric, rectangular shaped cross section with asymmetric preparation design**

**Figure 3.73. Figure 6. – Excentric, irregular shaped cross section with asymmetric preparation design**

• Others:

  • Different edge design which cannot be classified to the previous two categories.

  • (LightSpeed, Light Speed LSX – passive and active instruments with short cutting zone; Liberator – straight not helical cutting edges parallel to the axis of the instrument; SafeSiders - intermittent, plane surface without cutting edges on the surface of the instrument etc.)

5.1.1.2. Preparation movement

• Continuous rotary movement, at accurately defined speed

  • **Torque control** suggested (ProFile, System GT, ProTaper Universal, ProTaper Next, Mtwo, FlexMaster, K3, Twisted Files).

  • **Torque control not certainly suggested** (HERO Shaper, Revo S, G-files, One Shape, RaCe, Endo Sequence, Liberator, LightSpeed, Light Speed LSX).

• Alternating movement

  • Alternating (90 °) rotary movements, established by the contra-angle hand piece from the rotary movements of the micro motor (SafeSider).

• **Reciproc** movements

  • The active direction of the preparation movement is bigger than the opposite direction. So that the file pass out rotary movement, but not continuously leading to significantly decreasing loading effect. ([http://www.youtube.com/watch?v=ar8t7jxFLwM](http://www.youtube.com/watch?v=ar8t7jxFLwM)) Reciproc, WaveOne).

The examples mentioned above illustrate the variegation of the NiTi rotary instruments, smaller or radical differentiation between them and the hard decision making regarding to select a system for using.
5.1.2. The preparation rules

There are big differences between the systems according to the strategy of the preparation. But the basic rules under mentioned are the same:


- Ensure the proper access.

- Prepare the root canal with hand instrument till the prescribed rate in a whole working length.

- Prepare with light movements at low speed.

- Observe the prescribed preparation time.

- Observe the prescribed sequence of the instruments.

- **Figure 3.74. Figure 7.** – sterilized endodontic storage unit which help observing the prescribed sequence of the instruments (Mtwo System Box, VDW)

- The NiTi rotary files more and more considered disposable instruments. If more than one usage is permitted the used instruments must be changed without any visible injury after the certain number of usages recommended by the manufacturers.

**Figure 3.75. Figure 8.** – Used, different types of rotary NiTi files
Figure 3.76. Figure 9. — Usage of proper preparation technique fracture of the instrument is rarely occurred. Control RVG of the root canal filling of the tooth 48 treated through the metal ceramic crown. Severe curved roots, two roots distally. Fractured tip of 20/.06-os Mtwo file (VDW) in the distolingual canal.
5.1.2.1. How we should use the instruments during the preparation?

- The different instruments require for different technique for the usage. The „pecking” type of movement (eg. System GT, Reciproc, Wave One), and the painting type movement, filling the walls of the canals (eg. ProTaper Universal, Mtwo) are common.

- The pressure toward the apical part of the canal should be very mild. The file should pass through the canal in self-moving. If the movement of the instrument in the canal is hampered, rinsing and, recapitulation (1) with hand instrument are necessary, the conicity of the already prepared coronal part of the canal should be increased. After these we can try again to go deeper in the canal.

- Usage of EDTA solution or gel is recommended by several manufacturers, (eg. RCPrep, Glyde, FileCare) for making the preparation easier, but the canal should always be filled with solution, and the debris should be removed by regular irrigation.

- Working of the whole cutting zone of the file should be avoided because of nipping and fracture possibility of it.

- The ledge type preparation mishap should be treated with hand instrument (K-file).

- The control of the working length is important, refine the working length with apexlocator can be necessary near to the apex (possibility of working length foreshortening).

5.1.2.2. Preparation strategies

- Most of the rotary NiTi instruments should be used with crown-down preparation technique. Usage of files with bigger diameter followed by the smaller sizes during the preparation pass toward apical. The permeability of the canal should be ensured canal pre-treatment with small size hand instruments and with recapitulation of the apical part of the canal. The disadvantages of the crown-down technique is the difficult file sequence (eg. ProFile, (http://www.youtube.com/watch?v=ZZN5OjONmXU) ProTaper Universal, (http://www.youtube.com/watch?v=9ZwakhVec8A) HERO Shaper, Light Speed CRX/MRX/LSX).

- In the case of some instruments each files should be used at the whole working length from the smaller to the bigger sizes. The main advantages of the technique are the simple file sequence and the lucidity of the technique. The working length determination should be done prior to the preparation (eg.Mtwo).

- In the case of apico-coronal technique, the conical shape of the canal is prepared like with step back technique. (Lightspeed, (http://www.youtube.com/watch?v=nLk_8cV6AkU) Lightspeed LSX)
• In the case of instruments preparing with reciproc movements one file could be enough for the final preparation. The exploration of the whole length of the canal with a small file is also necessary. Widening the canal with bigger and bigger files gradually is not necessary. One bigger size instrument do the preparation and pass through the canal from coronal to apical. Refine the working length before the last step of the preparation is also needed. (eg. [http://www.youtube.com/watch?v=w6ur8yHZoMg] Reciproc, WaveOne).

1. video – „S” shaped curvature of the mesial canals of the tooth 37. Preparation with Reciproc file (VDW).

5.2. Electronic endodontic devices

5.2.1. Endodontic motors

Figure 3.77. Figure 10. – Endodontic motors (Reciproc Silver, VDW)

Driving of the NiTi rotary instruments can be solved safety with endodontic motors. The speed of the instrument is regulated precisely. It has torque control function which helps to prevent the fracture of the instrument in the case of file nipping by stopping the preparation. It can be able to do special eg. reciproc movements. Normal and wireless motor types are exist. The vertical position of the file can be continuously monitored by built-in apexlocator. The signal of the apexlocator can stop the preparation avoiding the over instrumentation.

5.2.2. Apexlocators

Figure 3.78. Figure 11. – Apexlocator (Raypex 5, VDW)
5.2.3. Laser instruments


5.2.4. Ultrasonic preparation devices

Figure 3.79. Figure 12. – Ultrasonic instrument used for endodontic tasks (UDS-E, Woodpecker)
Ultrasonic device can be used for several tasks in the endodontics. Sufficient tip for the task, and adjustable capacity of the device are needed for usage of ultrasonic device for solving different problems. The ultrasonic instruments are used for non-surgical endodontics:

• Refinement of the trepanation cavity, preparation of the floor of the pulp chamber, explore the orifices of the root canals (operating microscope or magnification, usage of the instrument in medium speed with water cooling or in wet pulp chamber are recommended).

**Figure 3.80. Figure 13. – Ultrasonic preparation tip for refinement of the trepanation cavity**

![Ultrasonic preparation tip](image)

**Figure 3.81. Figure 14. – Usage of ultrasonic device to open the orifice of the MB2 canal of tooth 26**

![Usage of ultrasonic device](image)

2. video – Shaping of the pulp chamber with ultrasonic device.
• Solving the obstructions and preparation mishaps of the root canals (operating microscope, medium speed, dry root canal).

**Figure 3.82. Figure 15.** – Ultrasonic file latch with stainless steel K-file curved according to the shape of the canal

![Image](https://via.placeholder.com/150)

• Cleaning the already prepared walls of the root canals, activating the desinficient irrigating liquid (low speed, root canal filled with irrigation liquid, passive file, 3X20s).

• Removing fractured files (operating microscope, low/medium speed, dry root canal, active file).

**Figure 3.83. Figure 16.** – Removing of broken file from the MB canal of tooth 26 with ultrasonic preparation device. A: approaching the coronal part of the fragment with Gates-Glidden instrument. B: preparation around the fragment with ultrasonic K-file-al. C: the released fragment. D: The removed fragment of the instrument

![Image](https://via.placeholder.com/150)

3. video – Usage of ultrasonic K-file to removing the broken fragment of hand instrument.

• Removing intrapulpal posts (high speed, strong water cooling, vibrating tip).

• Making of root canal fillings (medium speed, without water cooling, spreader or plugger tips).

**Figure 3.84. Figure 17.** – Exploration of the connection between the MB1 and MB2 canals with ultrasonic preparation (retreatment of tooth 26). Left: infected debris can be occurred at the connective area also in the case of already prepared canals. Middle: Two MB canals are connected with ultrasonic K-file. Right: As the paper point shows the lumens of the two root canals were joined through the total length of the root canal

![Image](https://via.placeholder.com/150)
5.2.5. Electronic devices for heat guttapercha techniques

- The final result is surer and the working procedure is easier if electronic devices are used for heating of the guttapercha in the cases of using thermoplastic root canal filling techniques. **Electronic heat carrier plugger** can be used for vertical condensation techniques instead of classic heat carrier plugger. (eg. System B Pack Unit, BeeFill Pack, E&Q Master Pen).

**Figure 3.85. Figure 18. – Elektronic heat carrier plugger (E&Q Master Pen, META)**

![Image](image1.png)

- Heating and injecting the injectable guttapercha can be done by different **guttapercha pistols** (eg. System B Fill Unit, BeeFill, E&Q Master Gun).

**Figure 3.86. Figure 19. – Guttapercha pistol (E&Q Master Gun, META)**

![Image](image2.png)

- The standard form of the canals prepared with rotary NiTi files are permit of the usage of **thermafil obturators** for filling the root canals. Heating for the tips different **ovens** can be used.(eg. Thermaprep Plus, GuttaMaster, HEROfillOven).

**Figure 3.87. Figure 20. – Oven used for heating thermafil obturators(Thermaprep Plus, Dentsply-Maillefer)**

![Image](image3.png)
5.3. Operating microscopes

(Magnification (~4-24 x) and the quality of the pictures are better than which are available with other magnification glasses.

Coaxial, strong irradiation (ordinary halogen lamp, in the latest developed microscopes have LED or xenon lights).

Possibility for introspection and projection for the assistance also.

Making of excellent documentation (photo, video).

Ergonomic tretment position is possible.

Quality of the endodontic treatment is increased by the microscope.

Figure 3.88. Figure 21. – Usage of rhodium coated mirror lead to clear shadow free picture
Figure 3.89. Figure 22. – Removing of pulp stone from the pulp chamber of the tooth 16. The operating microscope ensure the good visual control.

Figure 3.90. Figure 23. – A: In the case of tooth 37 the orifice of the root canals were not found by the dentist. Extreme tooth structure removing leading to perforation of the furcation. B: At the beginning of the treatment the closure of the perforation is incomplete, the canals are not opened: Usage of the operating microscope permit of the precise closure of the perforation (Biodentine, Septodont), and the localisation and exploration of the canals. D: Root canal filling and control RVG.
There are two categories of microscopes suggested for endodontic reasons:

  - Magnification changing gradually by hand.
  - Focus pulling by hand.
  - Adjustable frictioned joints on the stage of the microscope.
  - Different level of facility.
  - Well useable.

**Figure 3.91. Figure 24. – Dental basic microscopes**

  - Motor-driven zoom without scales.
  - Motor-driven focus pulling.
  - Foot switch possible to order.
  - Magnetic stoppers on the microscope stand.
  - Several expansion facilities.
  - Very comfortable usage.

**6. 3.6. Working length determination of the root canal – Dora Ottoffy-Kende**
Creating optimal root canal form during the root canal preparation, which is fit for the requirements of the chemo-mechanical root canal preparation, ensure proper pre treatment for the obturation without over instrumentation of the canal which can lead to the weaken of the tooth is important.

The one most important aim of the root canal treatment is to explore the whole length of the root canals. One of the most important data used during the root canal treatment is the working length.

The working length is the biggest distance in which the preparation instruments are used for the preparation in the root canal which is measured from a coronal reference point determined by the dentist.

**Figure 3.92. Figure 1. – Desired preparation length marked with silicone stops on the files. The stops positioned with the working length measuring device (VDW)**

Ideal apical end point of the root canal preparation is considered the physiological apical constriction of the root canal (foramen physiologicum or apical constriction) written in the traditional anatomic patterns. (Kuttler). During the working length determination we looking for that length of the file which just reach the apical constriction during the preparation.

The vertical extension of the root canal treatment and filling are mainly determined by the working length.

**6.1. Influential factors of the working length determination**

Classical apical constrictions are exist less than half of the cases. The apical part of the root canal usually conical or cylindrical shaped. The constriction sometimes is missing eg.in the case of apical resorption. The classical way of the working length determination is the radiological method (needle control x-ray). The deficiency of this method can cause several suspense. For example the place of the apical constriction usually found 0,5 -1 mm coronal from the foramen anatomicum (changing with age);the foramen apicale more than 60% of the cases is found laterally from the anatomical apex; the orovestibular curvatures of the roots are restricted visible on the x-rays. Because of the detailed reasons, the distance between the radiological apex and the apical foramen should be determined within 0-3,0 mm which is a relatively wide range especially regarding to the circumstance that the apical 3 mm of the root canal is a critical zone in the cases of treating infected root canals.

**6.2. Methods of working length determination**

Accepted, objective methods of working length determination:

- **Radiological method** (preliminary intraoral x-ray and needle control x-ray): used independently or combined with electronic measuring method.
- **Electronic method** (apexlocator): used independently or combined with radiological measuring method.

Alternative methods, providing supplementary information:
• Based on tactile sensation – not accepted.
• Based on the feedback of the patient – not accepted.
• Usage of paper point (overextended/opened apex).

**Figure 3.93. Figure 2. – Usage of paper point to refine the working length. A: needle control xray(internal resorption, wide apical constriction). B: Conducting of the paper point till the working length, the tip of the point is wet. C: Reduction of the working length with 0.5 mm, the end of the point is dry. D: Root canal filling after the refinement of the working length (injectable guttapercha technique)**

6.2.1. **Methods of radiological working length determination (needle control x-ray)**

In the practice the position of the apical constriction are determined 0.5-1 mm coronal far from the radiological apex on the x-ray. This is a compromise leading to proper apical cleaning without over instrumentation in most of the cases.

During the radiological working length determination method the diagnosing of the tooth should be considered. Based on these facts treating of teeth with vital pulp (without infection), teeth with non vital pulp (infectioned teeth), and retreatments can be differentiated.

6.2.1.1. **Radiological working length determination in the case of vital teeth**

There are no microorganisms in the apical region in the case of vital pulp. In the case of pulpitis the infection usually affect on the pulp chamber. Unnecessary preparing near by the formen apicale.

The purpose of the apical preparation is removing the not or slightly infected pulp tissue, and shaping of the root canal.

The working length is determined 2-3 mm coronal from the radiological apex.

Advantages of the method:
• Apical pulp stump developed.
• Prevent the apical extrusion of the root canal filling material.
• Good results.

6.2.1.2. **Radiological working length determination in the case of non vital teeth**

The root canal systems should be considered infected when the diagnoses is gangrena pulpae. The bacterial contamination reach the apical part of the root canal sometimes the periapical area is also infected.

The purpose of the preparation is the removing of the necrotic tissues and elimination of the microbes and with the root canal filling is to close the survival microbes.
The purpose of the apical instrumentation is to reach the whole length of the canal.

The best results are suspectable if the end point of the root canal treatment is determined 0-2.0 mm coronal from the radiological apex.

6.2.1.3. Radiological working length determination in the case of retreatment

In the case of endodontic retreatment compared to the non vital teeth the working length determination is affected by not only the old root canal filling material but the solving materials used for removing of old root canal fillings also. If there is periapical radiolucency the persistent microbes at the apical part of the canal are suggested to support it. The importance of preparation as long length as possible in contrast with the risk of spreading remaining root canal filling material and chemical agents to the periapical area should be considered.

Beside the proper preparation technique (corono-apical) the apical end point of the preparation is important. It is determined 1-2 mm coronal from the radiological apex.

Figure 3.94. Figure 3. – Retreatment of the tooth 37. A: Incomplete root canal filling, unfilled root canal, middle size periapical radiolucent area. B: Pus motion from the sulcus. Guttapercha point put into the periodontal pocket is on the xray. C: The needles are in the correct position on the xray. D: Root canal filling, control xray. E: Control xray 4 years after the root canal filling. Healed periapical area

6.2.1.4. Advantages and disadvantages of the radiological working length determination

Advantages:

• Long term clinical experience.

• The shape of the canals are well defined(1).

• Well documented measurment.

• Visualisation of the preparation mishaps (eg. fals-root).

Disadvantages:

• The foramen physiologicum not visible on the x-ray.

• If the placement of the foramen physiologicum is not within average range: over/under instrumentation.

• Provide two dimensional aspect (the anatomical and the radiological apex are not always matching).

• Wrong, not assessable pictures.

• The end of the smaller size of the instruments (#6, #8, or #10) not always visible on the xray.

• Higher irradiation dose.

• Time (especially making repeated radiographs).
6.2.2. Electric working length determination

The working length determination can be done with electronic measuring instruments (apexlocator).

The resistance between the oral mucosa and the apical periodontal ligament is constant 6.5 kΩ (Suzuki 1942). This data can be used for the determination the position of the needle placed into the root canal (Sunada 1962). The first apex locators are worked with direct current. Later the new instruments are worked with a.c. for the more precise measuring.

Kobayashi (1995) developed an instrument: which measures the impedance on two or more frequency of a.c. (e.g., 400 and 8000 Hz).

The recent instruments calculate the rate of impedance. Precise measurement characterizes this instruments in the case of root canal filled with electrolyte.

The position of the tip of the file used for the measuring is visualized on the display of the apexlocator.

The dentist can decide about working length: shorter (far from the apex) or longer (closer to the apex). The decision based on the diagnosis, anatomical or pathological processes.

Figure 3.95, Figure 4. – Schematic figure of the measuring with apexlocator. The electrodes are in contact with the mucosa and with the needle put into the root canal. The optimal position of the needle is indicated on the screen.

1. video – The progression of the needle in the canal can be followed on the screen. The area of the apical constriction is marked by the „green area.” Over instrumentation is marked by the red spot.

2. video – Measuring process illustrated by extracted tooth with physiologic saline model. The position of the needle is marked precisely by the instrument.

6.2.2.1. The process of electronic working length determination

Circumstances should persist for the proper measurement:

• Absolute isolation (rubber dam).
• Proper access cavity preparation.
• Stable coronal reference point.
• (Calibration of the apexlocator – new generation not a requirement).
• Avoid contact with alloy restorations.
3. Reconstructive dentistry

**Figure 3.96.** Figure 5. – The electronic measurement was unstable because of the treatment was done through the metal ceramic crown of the tooth 13. The working length determined with apexlocator was refined with the needle control xray.

- Root canal filed with electrolyte (rinsing solution).
- Dry pulp chamber (drying with cotton palet/puster).
- Usage of the file with proper diameter (comparable diameter with the diameter of the root canal).
- Reproducible measuring during the treatment if it is necessary.

Aggravating and influencing factors of the measurement:

- Wide apex (resorption, immature apex, previously over instrumentation): unsure measuring result
  - **If the diameter is wider than 0.2mm** the locator will determine the working length more and more far from the foramen apicale as the diameter of the apex is widening.
  - The locator signs too early especially in the case of immature apex.
  - Often required the parallel usage of radiological method.
  - The preliminary x-ray helps to realize the wide apex.
  - The paper point process also recommended for refinement of the measuring results.

- Accessory canals:
  - Signs to early the reaching of the foramen but not signs overinstrumentation.
  - Press more the file the real position of the foramen will determine.
  - Estimated working length based on the preliminary x-ray the measuring fault is become obvious.

- Existing periapical process:
  - Middle size or great periapical mutations could interfere with the measuring. Inflamed periapical resorption is suspect in these cases.

**Figure 3.97.** Figure 6. – Left: Apical external resorption leading to unstable electronic working length determination (on the distal root apex of the tooth 46). Middle: In these distal canals the working length determined with apexlocator was refined with the needle control xray. Right: The definitive root canal filling.
• Shape of the root canal, obstructed root canal
  • The instrument not measures in totally obstructed calcificated canals.
  • Obstruction caused by debris less interfere with the measuring.
  • Usage of crown-down technique and preflareing are suggested to avoid the obstruction.

• Retreatment:
  • Most often occur that the electronic working length determination is not works.
  • Persistence of the root canal filling material inhibit the measuring.
  • If the devise is signs the position of the needle the measurement is acceptable.

6.2.2.2. Advantages and disadvantaged of the electric working length determination

Advantages:
• Determine the position of the tightest point of the root canal (apical constriction).
• Working with small size files.
• Decreased number of x-rays.
• When the patient has severe nausea.
• Reproducible.
• It takes short time.
• Perforation of the rot canal can be determined with the instrument.
• Accuracy is between 75-94%.
• In the case of rotary NiTi instruments it can measure during the preparation.

Disadvantages:
• The accuracy of the measurement only can be checked by the needle control x-ray or with the control x-ray after the root canal filling.
• The shape of the root canal are nit determined with the instrument.
3. Reconstructive dentistry

- In the case of pulpitis, or periapical inflammation the accuracy of the measurement can change but it doesn’t reach the clinically significant level (within 0.5 mm).
- The accuracy can also be influenced by the concentration and the type of the electrolyte.
- Apical resorption, immature apex, hypercementosis, lateral canals could also interfere with the results of the measurement.
- Usually the measurement is not ready to be achieved.
- In the case of retreatment the indication is restricted.
- Contraindicated if the patient has pacemaker.

7. 3.7. Principles of root canal preparation, difficulties and common failures – Dora Ottoffy-Kende

7.1. Principles of root canal preparation

The aims of the root canal preparation are preparation of the root canals in a total length of the canals and widening the canals in the proper rate with the proper endodontic files.

- The remaining soft tissues, the debris, the probably infected content of the root canal, and the probably infected internal dentin layer of the root canal should be removed from the approachable area of the canal during the mechanical preparation.
- Apico-coronally splayed conical shape should be prepared. Making of root canal fillings in a proper quality also requires a conical shaped root canal form.
  - The proper canal shape can be prepared also with hand and rotary files.
  - Widening of the root canal – even in infected teeth – are limited by the thickness of the dentin, because the mechanical stability of the tooth shouldn’t be destroyed by over instrumentation of the canal. The perforation caused by incorrect or over instrumentation of the canal can lead to difficulties during the endodontic treatment and cause decreased success rate.
  - It should be clear that the mechanical preparations shouldn’t be extend to the total of the endodontic space. In itself will be imperfect.

The proper size of the apical widening and the conical shaped preparation form permit of the utilization of the chemical preparation and disinfection ensured by the irrigation.

The highest success rate can be reached by the proper usage of the chemo mechanical preparation method.

The mechanical and chemical preparations are done together simultaneously.

- The mechanical preparation is carried out in a canal filled with irrigation fluid.
- The cleaning effect of the flow of irrigation fluid is belonging to the mechanical cleaning.
- The motion of the preparation instruments conduce to spread the irrigation fluid apical.

The mechanical preparation with preparation instruments, which means the formation of the canal shape, and the chemical preparation and disinfection which is carried out with irrigation fluids, are discussed separately in this curriculum because of didactical reason.

7.1.1. Basic rules of mechanical preparation of the root canal, basic preparation strategies
Figure 3.98. Figure 1. – Improperly prepared trepanation cavity can inhibit the procedure of root canal treatment (tooth 26). A: Removing of the top of the pulp chamber is incomplete. B: Removing the top of the pulp chamber and shaping the trepanation access cavity with Müller bur (Meisinger). C: The MB1, MB2 and DB orifices are localised (from this point of view the orifice of the palatinal canal is not viewable.) D: The pulp chamber after the preparation of the canals

Figure 3.99. Figure 2. – The calcified root canal can inhibit the root canal treatment procedure (tooth 12). A: The obliteration is viewable on the xray. B és C: using of ultrasonic preparation instrument and microscope for destroying the obliterated part of the canal, than the working length was reached with #6 - C Pilot File (VDW) D: definitive root canal filling

Certain rules of the mechanical preparation are standardized, because have a dominant significance in a correct canal preparation:

Figure 3.100. Figure 3. – Correct access cavity preparation, the orifice of the MB2 is unprepared yet (tooth 26)
3. Reconstructive dentistry

Figure 3.101. Figure 4. – Localisation and penetration of the orifice of the MB2 canal with MicroOpenerrel (Dentsply-Maillefer) (tooth 26)

• Creating **straight line access** to the orifice of the canals.

• Checking the penetration of the root canal, and penetrating the canal before the flaring (with small size hand instruments).

• **Figure 3.102. Figure 5.** – Penetration of the root canal with #08 K-file (tooth 26 MB2)
• **Orifice opening** should be done, the **coronal part of the canal should be widening (preflaring)** during the initial part of the preparation leading to straight line access to the more problematic apical area.

• Usually bigger and bigger size of instruments used for widening the root canal. Easy penetrations of the bigger files are permitted of the **glide path** prepared by the smaller instruments.

• Between each files the irrigation of the canal are recommended removing the preparation debris

• After it the recapitulation (usage of small size of instrument at the whole working length or at the non prepared part of the canal) is important avoiding the dentin plug formation.
Rotary or hand instruments or the combination of these can also be used for the canal preparation. The preparation is much easier and quicker with rotary preparation instruments.

Previously - according to the curvature of the canal - curved stainless steel instruments, or NiTi rotary instruments are used to avoid the preparation mishaps. Both of these cases the selective preparation procedure (proper part of the root canal surface is prepared more than the other part of the canal) can be used (e.g.: anticurvature filing technique).

Saving the real form of the root canal is considered important according to any concept. The Balanced force technique and the usage of NiTi rotary instruments are the best from this point of views.

In the case of staged preparation form, the curvature filing of the canal is needed to smooth the surface of the canal during the last step of the preparation.

After reflecting the advantages and disadvantages the dentist should decide about the preparation procedure in other points:

- It is not obvious that the preparation of the canal with a small size(#10, #15) so called patency file till the foramen anatomicum is needed or not. It can help removing the debris, and observe the working length but not always prevents the preparation mishaps.

- There is no consensus regarding to the apical size of the preparation. Wile some of the researchers suggest smaller apical preparation with increased conicity of the canal, others prefer the bigger diameter of the apical preparation permit of the perfect apical penetration of the irrigation fluid.

- The minimal size of the apical preparation is significantly influenced by the diagnosis. In the case of vital (not infected) teeth smaller size of apical preparation is accepted. In the case of gangrena (infected teeth) bigger amount of infected dentin removing is needed (apical minimum#35-#40).

- Apical stop preparation can be prepared according to the apical end of the preparation which could be good to prevent the apical over extrusion of the debris, irrigation fluid, and the root canal filling material. It could be permit of the easier condensation of the root canal filling material. The increased conicity of the root canal thought to be enough to prevent the apical over extrusion.

7.1.2. The principals of chemical preparation and disinfection of the root canal

Chemical preparation and disinfection of the root canal is done during and after the mechanical preparation of the root canal. The irrigation fluids which have debris removing, soft and hard tissue solving and lubricant effect, which helps the preparation instruments during the flaring, affect during the whole preparation procedure. After the preparation procedure a final irrigation protocol should be done for solving of the smear layer and for disinfect the canal with different type of irrigation fluids. Another possibility to elongate the acting time of the desinfection and the tissue solving is to put temporary root canal filling material into the canal which has antiseptic activity (multi-session treatment).

This curriculum deals only with the more often used materials.

Sodium-hypochlorite (NaOCl)

- Disincentive fluid with wide spectrum(baktericid, virucid, fungicid).
- Tissue solvent, proteolitic effect (organic denbris, pulp).
- 0.5-5.25% solution is used. (The antimicrobiotic and necrotic tissue solving effect of the 1% solution is adequate.)
- It is used during the preparation and the part of the final irrigation protocol.
3. Reconstructive dentistry

Figure 3.104. Figure 7. – Endodontic canule and Luer-lock joint syringe for root canal irrigation (#1250 NaviTip és #201 5ml Syringe, Ultradent). The type of the solution should be written on the syringe!

Figure 3.105. Figure 8. – Heated Sodium hypochlorite (e.g. in composite heating oven) the affectivity of the disinfection can be increased (Ena Heat, Micerium)

Ethilen-diamin-tetraacetate

(http://upload.wikimedia.org/wikipedia/commons/8/85/Ethylenediaminetetraacetic.png)

EDTA):

- Chelate formation, demineralization effect.
- Solving of the inorganic debris.
- Usage of ~17% solution of the fluid.
- The part of the final irrigation protocol. Used for lubrication during the mechanical preparation. Alternate with NaOCl.

Chlorhexidin (http://upload.wikimedia.org/wikipedia/commons/5/5b/Chlorhexidine.png) CHX):

- Biguanid type material with wide antimicrobial spectrum(bactericid, fungicid, virucid) Bactericid and bacteriostatic. It causes the lisis of the bacteria.
- Substantive (on the dentin wall, long lasting antimicrobiotic effect.
- 0,2-6% solution is used.
Calcium-hydroxid (Ca(OH)$_2$):

- Produced from the in office mixture of calcium-oxid and sterile distilled water, or physiologic saline, or pre made pastes are used.
- It has slow disincentive effect. It should be put into the canal for 1-2 weeks for disinfection.

**Figure 3.106. Figure 9.** – A: Temporary root canal filling paste mixed from calcium oxid powder and sterile distilled water. B: Lentulo in contra angel hand piece. C: calcium hydroxid paste on the Lentulo ready to put into the root canal

The final irrigation protocol is summarized in the 1. table in the case of infected root canals.

**Figure 3.107. Figure 10.** – Final irrigation protocol used in the case of root canal treatment of infected root canals

<table>
<thead>
<tr>
<th>Step</th>
<th>Solution</th>
<th>Amount/canal</th>
<th>Acting time</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Removing of the preparation debris</td>
<td>NaOCl</td>
<td>2ml</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Solving of the smear layer</td>
<td>EDTA</td>
<td>2ml</td>
<td>1min</td>
<td></td>
</tr>
<tr>
<td>Disinfection</td>
<td>NaOCl</td>
<td>2ml</td>
<td>1min</td>
<td>3x 20 sec ultrasonic activation</td>
</tr>
<tr>
<td>Interruption irrigation</td>
<td>Saline</td>
<td>2ml</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disinfection</td>
<td>CHX</td>
<td>2ml</td>
<td>2min</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 3.108. Figure 11.** – Final irrigation of the root canal, ultrasonic activation of the irrigation fluid (NaOCl)
7.2. Preparation mishaps

During the mechanical preparation sometimes it is not possible to create the required form of the root canal. Preparation mishaps can be occurred. These are more often among difficult anatomic circumstances:

- Grate curvature of the root canal (>30°).

  Figure 3.109. Figure 12. – Determination of the angulation of the root canal curvature \((\alpha=\text{Schneider-angle})\), the green triangle marks the required preparation for the straight line access

- Little radial root canal curvature.

  Figure 3.110. Figure 13. – Curved root canal with small diameter
Figure 3.111. Figure 14. – „J” shaped curvature in the tooth 15

• Double, „S‖ formed root canal curvature.

Figure 3.112. Figure 15. – „S”, shaped curvature on the mesial root of the tooth 37

• Narrow, calcified root canal.
3. Reconstructive dentistry

Figure 3.113. Figure 16. – Narrow, calcified root canal in the tooth 21 (with external root resorption distally)

Figure 3.114. Figure 17. – Strip perforation on the mesial root of the tooth 46, with sealer over extrusion, (complication of removing fractured instrument)

Figure 3.115. Figure 18. – Left: Mesial perforation on the coronal third of the tooth 11. Middle: The perforation and the root canal was opened under microscope. Right: Closing of the perforation with Biodentine (Septodont), than the definitive root canal filling was carried out
3. Reconstructive dentistry

- Translocation of the root canal.

**Figure 3.116. Figure 19.** – Translocation of the root canal. Left: “S” shaped root canal form with severe curvature in two direction. Right: control x-ray from the root canal filling. The root canal translocation is obvious and the apical part of the canal is straightened. The root canal filling is acceptable.

- Debris extrusion to the apical periodontium, over instrumentation.
- Obturation of the apical root canal, Shortening of the working length.

1. animation – Creating of apical perforation (apical „zip” formation).

Most of the preparation mishaps are in connection with the curvature of the root canal. It is important that how (which instruments, preparation procedures) we can decrease the occurrence of these faults.
• Flexible („flex”) stainless steel instruments, or NiTi instruments are safer. (Preparation mishaps occur more often in the case of stainless steel instruments.)

• Work with Crown-down preparation, or at least usage of orifice opening (widening).

• Usage of pre curved stainless steel files according to the curvature of the root canal.

Figure 3.117. Figure 20. – Proper device for bending the stainless steel K-files (Dentsply-Maillefer)

• Usage of anticurvature filing avoiding the „strip“ perforation.

2. animation – Anticurvature filing process.

• Usage of the balanced force preparation technique.

• Avoiding the long lasting preparation in the same level of the root canal during preparation with rotary instruments. The instruments should be moved apico-coronal continuously.

To avoid the debris extrusion and the obstruction of the apical area:

• Working in canals filled with irrigation fluid.

• Often irrigation during the instrumentation.

• Often recapitulation.

• Cleaning the files often during the preparation (sponge).

Figure 3.118. Figure 21. – During the preparation the instruments should be cleaned with a special sponge (Interim Stand, VDW)
8. 3.8. Step-back technique, step-down technique, double-flared technique, balanced-force technique – Edina Lempel

There are several traditional methods for manual and manually performed, combined mechanical preparation of root canals (http://dentallecnotes.blogspot.hu/2011/10/root-canal-preparation-techniques.html) The most well-known methods are summarized briefly below.

Two main preparation techniques are differentiated:

- **Apico-coronal** preparation technique:
  
  - The preparation is started at the entire working length.
  
  - Apical stop is defined.
  
  - While moving in coronal direction the conicity of the root canal is shaped with instruments gradually increasing in size on decreasing working lengths, thus widening the canal.

- **Corono-apical** preparation technique:
  
  - The preparation is started from coronal direction with bigger instruments.
  
  - Smaller and smaller instruments are used to prepare the canal closer to the apex.
  
  - Apical stop is shaped after reaching the working length.
  
  - If necessary, the conicity of the root canal will be increased with further preparation.

8.1. **Step-back technique (Clem 1969)**

The best known apico-coronal technique and it is taught in most practical and theoretical courses. It is used for preparation of mild or moderately curved root canals. Manual stainless steel instruments, Ni-Ti rotary instruments and Gates-Glidden burs are used in combination. The preparation is started at the entire working length.

**Figure 3.119. Figure 1. – Measured and bended instruments for Step-back preparation**
The stepback technique helped to overcome the procedural errors of the standardised technique in slight to moderately curved canals, but in the more severely curved root canals problems still exist.

The "Step-back" method indicates that the dentist works from the bottom of the canal towards the crown. Following the bulk removal of the pulp in the extirpation phase, small hand files are used nearest the apex. The dentist progresses to larger sizes of files working his or her way back up the canal. Each file cuts and removes a little bit more dentin. These advance about one millimeter with each new instrument.

**Steps of the Step-back technique**

**Apical enlargement:** After working length determination the apical part is instrumented first. The first file that binds in the canal at the working length without previous preparation is considered the initial apical file (IAF). The instruments are bended according to the curvature of the root canal. The root canal must be enlarged by circumferential filing through an additional four sizes. If the root canal is strongly curved anticurvature filing method is performed. The last file manipulated to the working length should remove white dentin shavings, and is designated the apical master file (AMF). With the master file the apical stop is defined. Only the K files used in the step-back technique provided a definitive apical stop, an almost round canal cross-section, a very good apical preparation without any indentations, as well as a conical canal shape from apical toward coronal. The minimum size of AMF is 25# in case of irreversible pulpitis and 35# if the root canal is infected.

**Irrigation:** Copious irrigation is important throughout the shaping of the canal walls, as the filing and reaming actions used during cleaning and shaping generate debris, which also can lead to infection and inflammation of the root canal or apical region. Copious irrigation helps to clear the loosened debris out of the canal and also serves to facilitate instrumentation by lubricating the canal walls and by removing materials that can create blockage in the canal.

**Step back phase:** Recall the working length of the canal and the size of the last file (AMF) used. Subtract 1mm increments from the original working length with each larger file size used until the instrument reaches within 4-5 mm of the canal orifice.

**Figure 3.120. Figure 2. – Step back phase of preparation with manual instruments**
3. Reconstructive dentistry

Shape the canal using a combination of reaming and filing actions within the canal. Begin with a reaming action; insert the first shaping file, rotate the file one-quarter turn, pull out of the canal, and repeat: "quarter-turn and out, quarter-turn and out, quarter-turn and out. The emphasis is on the in stroke; the quarter-turns engage the file within the dentin and the file is passively pulled out of the canal. Reaming action permits efficient shaping of the canal walls, and is repeated until the file no longer encounters resistance within the canal. Continue shaping with the starting file using a filing action. Filing action is used to widen the canal and is done circumferentially around the canal walls. This ensures that contact force is applied to the entire surface of the wall, 360 degrees around. Gates Glidden instruments (with increased sizes) are generally reserved for shaping the canal 5mm away from the apical stop (~600rpm, 2-2mm step back).

Repeat filing and recapitulation: In stepback filing, each succeeding file must reach the correct length. If the specified canal length cannot be reached with the corresponding K-file size, do not start shaping at the shorter length, or force the instrument further down into the canal. Go back to the previous instrument and continue to shape using filing action, until the canal is sufficiently loose enough to accept the succeeding file. Repeat steps 2-4 for each successively shorter length until you have reached within 4-5 mm of the canal orifice. After shaping with the last file, verify that the canal walls are smooth and that there is no blockage from the coronal portion down to the apex.

In the step back-phase after each bigger file must go back with the apical master file to remove the debris and control the working length = recapitulation.

Confirm Shaping: Recall the last file (AMF) size used at the working length. Place the AMF in the canal to confirm that the entire length of the canal has been properly shaped. Use this file to also check for patency, to ensure that the principle of resistance form has been met, and the integrity of the apical foramen maintained. The properly shaped canal should have an evenly tapering funnel shape with smooth walls. To check resistance form, place the working length file into the canal and turn it one-quarter turn; the file should engage firmly at the apical constriction without going through it. The stepped wall should be sleeken with the AMF with circumferential filing.

Animation 1. – Step-back technique. The dentist progresses to larger sizes of files working way back up the canal preparing conical shape.

8.2. Step down technique (Goering 1982)

Step down involves cleaning and shaping the canal from the coronal third down to the apical third (Corono-apical technique). The apical third of the canal is approached only after the coronal two thirds is sculpted and disinfected. This technique is suggested for narrow and strongly curved root canals.

The benefits of this technique include the following:

- It eliminates cervical dentin constrictures and reduces canal curvatures, thereby giving the clinician full tactile awareness in the apical third.

- It allows deeper and earlier penetration of the disinfecting irrigating solution into the inner recesses of the canal, thereby effectively cleaning the coronal two thirds of the canal before the apical third is approached.
3. Reconstructive dentistry

- It removes the major portion of the pulp and infecting microbes before the apical third is approached, thereby minimizing the risk of pushing pulpal or microbial irritants into the periapical region.

- The working length is less likely to change during apical instrumentation because canal curvature has been reduced before working length is actually established.

**Steps of Step-down technique**

An estimate of the root length is made from the pre-operative radiograph taken with a parallel technique. However, confirmation of the actual working length is not carried out until the coronal preparation of the canal has been completed, as this may straighten a curved canal which would change a measurement that had been taken too early. Most operators now confirm the actual working length when the step-down preparation has progressed to within 3 or 4 mm of the estimated working length.

The root canal preparation is divided into two parts:

- *coronal preparation*, which permits radicular access for
- *apical instrumentation.*

**Coronal preparation** or radicular access: First, the pulp chamber is copiously irrigated with sodium hypochlorite. To make room for enlargement with Gates-Glidden burs, K-type files 10-15 to 25# are sequentially placed in the canal. Circumferential filing with each instrument followed by *recapitulation* and copious irrigation with NaOCl establishes the pathway for the use of Gates-Glidden burs. Then Gates-Glidden burs are next introduced into the canal (preshaped canal - 1 mm), directed apically and laterally away from the furcation. The larger sizes are introduced first, working sequentially further down the canal with smaller sizes. Some canals will accept a 6# bur, but normally a 4# would be used first, followed by a 3#. Each bur will penetrate 2-3 mm further than the previous one. EDTA paste should be used with each bur, and the canal should be irrigated between each entry. Eventually, in a relatively straight canal, the 2# bur is inserted 10-12 mm into the canal from the occlusal reference point. In a curved canal the pre-operative radiograph should be checked for the maximum straight line penetration of the bur. Gates-Glidden burs should be rotated with constant medium drill speed from the time they enter the canal until removed.

**Apical preparation:** The coronal flaring already carried out makes access to the apical portion of the root easier, as there are no dentinal obstructions and access is more direct. Thus, once the coronal preparation is complete, (flexible) K-type files with safe tips may be selected, perhaps a size 60, the tip dipped into a canal lubricant, and the instrument worked slightly further into the canal. Sequentially smaller files are selected until the canal is prepared to 3-4 mm short of the estimated working length. Now the actual working length must be confirmed by radiograph or apexlocator. Once the working length has been confirmed, the apical preparation can be completed according to the step-back technique.

**Animation 2. – Step down preparation.** The coronal two-third of the root canal is prepared with Gates-Glidden burs, the apical one-third is prepared according to the step back technique.

8.3. **Double-flared technique (Fava 1984)**

The double-flare technique involves coronal widening, beginning with large instruments, and then smaller files are used to progress ever deeper into the canal (*Corono-apical* technique). Then smaller instruments are used for instrumentation from apical toward coronal. Thus the procedure is *double flaring*, from coronal and from apical.

The double-flare technique involves three procedures:

- reverse flaring
• apical instrumentation

• flaring of the entire root canal.

This technique is performed in narrow and mild or moderately curved root canals.

**Steps of Double-flared technique**

Following trepanation and extirpation of the pulpal tissues, the working length is determined radiographically and made permeable with a file 10 #.

*Coronal widening* is performed via “reverse flaring.” To accomplish this, the hand instruments (files) destined for later apical instrumentation are employed in reverse order. After using a #10 K file to determine the entire length and the course of the canal, a #45 K file is used only in the first few millimeters of the coronal portion of the canal; subsequently a #40 file is applied 2 mm deeper in the coronal region, and a #35 K file is used to instrument still 2 mm deeper in the apical direction. 

*Apical instrumentation*: After coronal widening of the area of the canal orifice up to the middle of the canal, the #15 K file is again used to the **full working length** to prepare the apical stop; this is followed by files #17, 20, 22, 25, and 27.

*Flaring of the entire root canal*: After completion of the apical widening up to size minimum 25#, generally 35-40#, a step-back technique is used, but shorter by 0.5 mm. Minimum 4-5 steps back create a conical “flaring” within the apical third of the canal.

**8.4. Balanced-force technique (Roan 1985)**

This **apico-coronal** technique is recommended for preparation of strongly curved root canals. The technique can be described as “positioning and pre-loading an instrument through a clockwise rotation and then shaping the canal with a counterclockwise rotation.

**Steps of Balanced-force technique**

After working length determination, balanced force hand instrumentation begins on the entire working length: placing, cutting, and removing instruments using only rotary motions. Insertion is done with a quarter-turn (90 °) clockwise rotation while slight apical pressure is applied. Cutting is accomplished by making a counterclockwise rotation (120-270 °), again while applying a light apical pressure. The amount of apical pressure must be adjusted to match the file size (ie, very light for fine instruments to fairly heavy for large instruments). Pressure should maintain the instrument at or near its clockwise insertion depth. Then counterclockwise rotation and apical pressure act together to enlarge and shape the canal to the diameter of the instrument. It must rotate the instrument sufficiently to move the next larger cutting edge into the location of the blade that preceded it, in order to shape the full circumference of a canal. A greater degree of rotation is preferred and will more completely shape the canal to provide a diameter equal to or greater than that established by the counterclockwise instrument twisting during manufacture. It is important to understand that clockwise rotation “sets” the instrument, and this motion should not exceed 90 degrees. If excess clockwise rotation is used, the instrument tip can become locked into place and the file may unwind. If continued, when twisted counterclockwise, the file may fail unexpectedly. The process is repeated (clockwise insertion and counterclockwise cutting), and the instrument is advanced toward the working depth in shallow steps. After the working depth is reached, the instrument is freed by one or more counterclockwise rotations made while the depth is held constant. The file is then removed from the canal with a slow clockwise rotation that loads debris into the flutes and elevates it away from the apical foramen. The instruments should always be cleaned.

**Figure 3.121. Figure 3. – The movement of the file in the root canal during balanced force preparation**
Movie 1. – Introduction of balanced force technique. The file loads debris into the flutes and elevates it away from the apical foramen.

The balanced force technique can be used with any stainless steel or Ni-Ti K-type file; however, the shaping and transportation control are maximum when a Flex-R file is used. The Flex-R file design incorporates a guiding plane and removes the transition angles inherent on the tip of standard K-type files. Those angles, if present, enable the tip to cut in an outward direction and give it the ability to cut a ledge into the canal wall. Lacking a sharp transition angle, Flex-R files follow the canal and are prevented from gouging into the walls. The tip design causes a Flex-R file to hug the inside of a curve and prevents tip transport into the external wall of that curve.

**Apical enlargement:** Roane firmly believes in enlarging the *apical area* to sizes larger than generally practiced. He expects a minimum enlargement of size 45, 1.5 mm short of the foramen apicale in curved canals, and size 80 in larger single-rooted teeth. These sizes, of course, depend on root bulk, fragility, and the extent of curvature.

After working length determination the root canal is enlarged at the entire working length. Sabala and Roane also believe in carrying the preparation through to “full length,” the radiographic apex of the root. A step-back in 0.5 mm increments is used with at least two groups of instruments to form a stepped apical stop (control zone). This shaping provides a minimum diameter at a known depth within the canal.

**Figure 3.122. Figure 4. – Stepped apical enlargement in narrow root canal**

*Apical constriction* is formed at a measured depth for *small, medium and large* canals.
A size 45 control zone is shaped for small root canals by first expending a size 15 and 20 file to the periodontal ligament and then reducing the working depth by 0.5 mm for sizes 25, 30, and 35 and completing the apical shape 1 mm short using sizes 40 and 45.

For medium root canals first a size 25 file reaches the radiological apex, followed by 30-40 # then 40-60 # reducing the working depth by 0.5-0.5 mm.

A size 80 control zone is shaped for large root canals by first expending size 30 to the periodontal ligament. Then the working depth is reduced by 0.5 mm for sizes 35, 40, 45, 50, 55 and the apical shape is completed 1 mm short using sizes 60, 70 and finally 80.

Table 3.1. Table 1. – Recommended apical enlargements in balanced force technique

<table>
<thead>
<tr>
<th></th>
<th>Small root canals</th>
<th>Medium root canals</th>
<th>Large root canals</th>
</tr>
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<tbody>
<tr>
<td>Munkahosszon (MH)</td>
<td>25</td>
<td>30</td>
<td>40</td>
</tr>
<tr>
<td>MH – 0.5 mm</td>
<td>35</td>
<td>45</td>
<td>60</td>
</tr>
<tr>
<td>MH – 1.0 mm</td>
<td>45</td>
<td>60</td>
<td>80</td>
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</tbody>
</table>

Coronal preparation: The coronal and mid-thirds of a canal are flared with Gates-Glidden drills (2#-6#), preparing 1-1 mm shorter with each bur.

Preparation is finished with final smoothing. Sodium hypochlorite irrigation is generously used.

9. 3.9. Lateral compaction, vertical compaction.
Removing of root canal filling – Dora Ottoffy-Kende

The goal of the root canal filling is to fill the previously prepared root canal space, and the area which is not reached with the mechanical preparation with different types of root canal filling materials. The unfilled area in the tooth can decreased properly. During the root canal filling procedure the over extrusion of the root canal filling materials should be avoid.

The root canal filling and the final coronal restoration also can inhibit the reinfection.

9.1. Procedures of root canal filling

The permanent root canal filling is prepared from guttapercha and sealer. The condensation of the guttapercha can be coldly (e.g.: cold lateral condensation) or with thermoplastic (warm vertical condensation) technique.

9.1.1. Cold lateral condensation technique

One of the most frequently used root canal filling technique.

Simple technique with simple material and instrumental requirements.

Figure 3.12. Figure 1. – Materials and instruments for the cold lateral condensation technique
It can be used in the case of conical form of the canal with regular round or ovoid shaped cross section, if the dentin wall is not too thin (risk of fracture). It has limited ability to fill the irregularity of the root canal.

**Figure 3.124.** Figure 2. – Mesial root fractured during the lateral condensation. Sealer extrusion is obvious on the xray

The procedure:

- Drying the prepared canal.
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**Figure 3.125. Figure 3. – Drying the root canals with paper points**

- Trying a gutta-percha cone into the dried canal. The size of it is equivalent with the **master file**. If it reach the working length and there is resistance during the removing of the cone this will be the master (primer) gutta-percha cone. Than this cone is should be removed from the canal.

- The wall of the root canal should be covered and **wet** by the **sealer** (with the master point, or with a spreader or a sterile file).

**Figure 3.126. Figure 4. – Canals covered by sealer**

- Insertion of the master gutta-percha cone till it reach the working length.

**Figure 3.127. Figure 5. – Insertion of the master gutta-percha poen till it reach the working length**
• Condensation of the gutta-percha with spreader.
  • The size of the spreader should be the same or 1-2 ISO size smaller than the apical diameter.
  • Marking the deepness of the insertion on the spreader with silicone ring. (working length minus 1 mm).
  • The spreader should be inserted into the root canal beside the gutta-percha cone, then it should be pressed axial direction. (~ 20N).
    • The size of the spreader is correct if it reaches the working length minus 1-1,5 mm.
    • Smaller size of the spreader is needed if it does not reach this length.
    • Bigger size of spreader is needed if it can reach easily the working length.

**Figure 3.128. Figure 6. – Condensation of the mater gutta-percha cone with spreader**
• The pressure should be supported during few seconds, than the spreader should be removed from the canal with alternate quarter rotating movements.

**Figure 3.129. Figure 7. – Created space after removing the spreader**
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- The space done by the spreader should be filled with the accessory (secunder) gutta-percha cone. The size of fit should be the same or one size smaller than the spreader size.

**Figure 3.130. Figure 8. – Insertion of the secondary gutta-percha cone**

- Condensation with the spreader, which can be inserted 1-1.5 mm shorter length than during the first condensation.
- If the spreader can’t reach the proper length smaller size of fit is needed.
- If the spreader can reach easily the same length than during the previous condensation bigger size of it is needed.
• The space done by the spreader should be filled with accessory gutta-percha cone.

• The condensation and the insertion of the secondary points should be repeated till the spreader can penetrate only the coronal 3mm of the canal.

**Figure 3.131, Figure 9. – Filling the root canal with accessory points**

Video 1. – Condensation of the gutta-percha cones with spreader.

• The top of the gutta-percha cones should be removed with a heated instrument (e.g. excavator) at the level of the orifice of the canal.

• At the end of the procedure the coronal part of the root canal filling should be condensed with the plugger.

• The excess of the sealer should be removed from the pulp chamber with alcohol soaked cotton pellet.

**Figure 3.132, Figure 10. – Condensation with plugger, the pulp chamber after the root canal filling is done**
9.1.2. Warm vertical condensation

It is used for the filling of regular or irregular formed root canals. Irregularity of the root canal can be filled with these techniques.

The modifications of the original technique (Schilder 1967) is used. Applied materials and instruments and the details of the procedure could be different according to the knowledge of the dentist. After the apical sealing of the root canal the filling of the remaining part of the canal is carried out with little 3 mm long gutta-percha pieces warmed with flame, condensed with plugger.

Figure 3.133. Figure 11. – Heat carrier plugger (Dentsply-Maillefer). One end of the instrument (heat carrier) can be heated in flame

The work of the dentist can be more simple and faster with the proper instruments. Electronic heat carrier plugger is usually used for made the apical sealing. After this part of the filling injectable gutta-percha technique is used for filling the whole root canal. This modified technique requires the proper instruments (e.g.: pluggers, heat carrier, gutta-percha pistol).

The procedure:

• Drying the root canal.

• Trying a gutta-percha cone (same size like the master file) into the dry canal (master cone). If the preparation is carried out with NiTi rotary instruments the master gutta-percha cone should be matched to these rotary files according to the conicity of it. The poen then should be removed from the canal.

Figure 3.134. Figure 12. – UP: ISO .02- conicity (DiaDent), down more conical gutta-percha poen accordin to the NiTi rotatry instruments (Reciproc, VDW)
• Cutting the 1 mm tip of the master poen is recommended avoiding the over extrusion of the guttapercha.

• Trying the **pluggers** into the root canal. Marking the penetration depth’s of the spreader with silicone ring (in the case of working without microscope).
  
  • The smalest spreader can reach the working length minus 3-4 mm deepness.
  
  • The other plugger can penetrate into the canal gradually (3-4 mm) (2-4 pluggers are usually used, depends on the length of the canal).

• The walls of the canal are covered with sealer (e.g.: with the help of guttapercha poen, spreader or a file).

**Figure 3.135. Figure 13. – Dried root canal, covered by sealer**

• Insertion of the master guttapercha cone into the canal.

**Figure 3.136. Figure 14. – Inserted master guttapercha poen**
• Cutting the coronal excess of the guttapercha with heat carrier plugger.

**Figure 3.137. Figure 15. – Removing of the excess guttapercha with heat carrier**

• Condensation of the guttapercha with heat carrier plugger till reaching the working length minus 4-5 mm.

**Figure 3.138. Figure 16. – Apical condensed and cut guttapercha**
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- Condensation of the plastic gutta-percha with **cold plugger**.

**Figure 3.139. Figure 17. – Apical condensation with plugger et previously measured length, the ready made apical plug**

- Video 2. – Making of apical plug. (First phase of the vertical compaction).

- Estimating the quality of the apical sealing with x-ray.

- Filling up with injectable gutta-percha the other part of the canal.
  - Filling the gutta-percha pistol, setting the proper temperature. (~200°C).
  - Deaerating the canule, pressing a little amount of gutta-percha onto a paper to check the consistency.

**Figure 3.140. Figure 18. – Filling the gutta-percha pistol with pieces of gutapercha. The filling space is marked on the picture. (E&Q Plus Gutta Percha Bur és E&Q Master Gün, META)**
• Inserting the canule till the apical guttapercha plug, filling up the canal with guttapercha. Holding the tip of the canule in the guttapercha fluid during the procedure avoiding the bubble formation.

• Filling procedure can be carried out in one or more steps.

• In the case of injecting one bigger amount of guttapercha the gutapercha should be condensated with a big plugger at the orifice of the canal.

• Usually one or more smaller amount of guttapercha is placed into the canal, condensated separately with plugger.

**Figure 3.141. Figure 19. – Injection of the guttapercha (up) condensation of it (down)**
Video 3. – Injection and condensation of the gutta-percha with plugger. (Modified vertical compaction, second phase of the technique).

- Removing the excess sealer from the pulp chamber.

**Figure 3.142. Figure 20. – MB1 canal is filled**

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**9.1.3. Other thermoplastic root canal filling procedures**

Other techniques exist to fill the root canal but these are not detailed in this curriculum:

- **Carrier-based guttapercha technique** (Thermafil technique).
  - Used for quickly sealing standard formed, canals prepared with NiTi rotary instruments.
  - **Obturators** are equivalent with the file systems.
  - These should be heated in an oven then inserted into the root canal.

**Figure 3.143. Figure 21. – Obturators (GuttaMaster, VDW)**

- Thermomechanical compaction (**McSpadden**).
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- „File – like”, rotary condensation instrument with opposite edge geometry.
- Requires a micro motor and a contra angle hand piece.
- The gutta-percha is softened by the heat produced by the friction during the instrument working with ~ 8000 rpm.
- The softened gutta-percha is condensed apical by the edges of the compactor.

**Figure 3.144. Figure 22. – GuttaCondensor (Dentsply-Maillefer) for the thermomechanic compaction**

**Figure 3.145. Figure 23. – Root canal filling with thermomechanic technique (filling instrument: GuttaCondensor #40, Dentsply-Maillefer; sealer: AH Plus, Dentsply-Maillefer; gutta-percha: Reciproc #50, VDW) root canal filled tooth 46(preparation: Reciproc #50, VDW)**

**Video 4. – Retreatment of the tooth 35. The root canal filling is performed with termomechanic compaction (GuttaCondensor, Dentsply-Maillefer).**

- Warm lateral compaction and lateral compaction with the usage of ultrasonic spreader.
- The gutta-percha can be softened by a heated spreader. Smaller force is needed for the condensation. The irregularities of the canals are filled better with this technique.
- Instead of using spreader heated by electricity or flame ultrasonic spreader can be used with better condensation effect.
9.2. Removing of the root canal filling

In the case of failure of the primer endodontic treatment endodontic revision is needed. If the primer endodontic treatment is proper from clinical and radiological point of view but the root canal filling is incomplete and the tooth is need for prosthodontic treatment (intrapulpal post) endodontic retreatment is also required. Indications of the endodontic retreatment are the following: injured coronal restoration leading to the coronal leakage of the root canal filling (more than 30 days). In these cases the removing of the root canal filling from the canal is required. Removing of the guttapercha can be carried out mechanical preparation (with hand or rotary instruments). This mechanical preparation can be supported by making the root canal filling materials plastic with heat or with solving materials.

It would be better to avoid the usage of the solving materials, but in some cases especially well condensed root canal fillings solving materials are needed. The guttapercha can be made plastic with several solving material.

Usage of different oils (eucalypt or orange oil) and chloroform are suggested.

- Unfortunately the solving materials destroy the rubber dam. The connection of the solving materials with the mucosa should also be avoided.
- Very small amount (few drops) of solving material is needed for each canal.
- Only mechanical preparation is required in the case of overfilled root canal (where the goal of the treatment is the removing of the overfilled guttapercha).
- Removing of the root canal filling is easier with the NiTi rotary instruments.

**Figure 3.146. Figure 24. – Retreatment through PFM crown** Left: small size periapical radiolucency, short but compact root canal filling removed with the help of solving material. Middle: root canal filling control xray. Right: control xray 7 yeras after the treatment. Sound periapical area

**Figure 3.147. Figure 25. – Left: Tooth 35 overextended, incomplete root canal filling, small periapical radiolucency (chronic periapical periodontitis). Middle: Overextended guttapercha poen was removed without solving material. The root canal was filled with CaOH. Right: Definitive root canal filling and temporary filling in the crown**
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Removing of the root canal filling is easier with the NiTi rotary instruments.

Removing totally the root canal filling material is no tan easy procedure. Working with operation microscope the cleaning of the walls of the rot canal will be more precise.

**Figure 3.148. Figure 26.** – Removing of gutta-percha pieces with ultrasonic K-file in the case of the retreatment of tooth 26. Left: Remaining gutta-percha investigated with microscope. Middle: Removing of gutta-percha with ultrasonic file. Right: the palatinal canal is totally cleaned

10. 3.10. Components of dental composites, their effects on color – Edina Lempel

Composites are resin based esthetic dental materials and widespread on every field of dentistry.

**Figure 3.149. Figure 1.** – MOD composite fillings in 15, 16 teeth
In advance of composites the methyl-methacrylate (MMA) was used from 1950. MMA is an aromatic, colorless, evanescent liquid.

**Figure 3.150. Figure 2. – Molecular structure of MMA monomer**

![MMA monomer diagram]

Double bonds are found in the monomer can easily disrupt and the monomers can react with each other or with other molecules. The final product of this reaction is a polymer. The **polymerization** reaction leads to a 18-20% shrinkage. The name of the filling materials are made from MMA is self curing acrylates. However these are unserviceable, because of their several disadvantages.

**10.1. Structure of composites**

The composites are composed of 3 main parts.

**Figure 3.151. Figure 3. – Structure of composite resin materials**

- *Organic polymer matrix,*
3. Reconstructive dentistry

- Inorganic filler particles,
- Coupling agent.

10.1.1. Organic resin matrix

The ingredients of the matrix are oligomers and monomers.

These monomers possess reactive carbon double bonds on each ends of the molecule and are able to polymerize to a network with addition polymerization in the presence of free radicals.

Figure 3.152. Figure 4. – Formula of polymerization

\[ n(CH_2=CH_2) \rightarrow -(CH_2-CH_2) \_ n \]

Generally used monomers are 2,2-bis[p-(hydroxy-3'-methacryloxypropoxy)phenylene]-propane, BisGMA, ethoxylated bisphenol-A dimethacrylate, BisEMA(6) and urethane dimethacrylate, UDMA.

Figure 3.153. Figure 5. – Molecular structure of BisGMA

Figure 3.154. Figure 6. – Molecular structure of BisEMA(6)

Figure 3.155. Figure 7. – Molecular structure of UDMA

BisGMA is a long, rigid, bifunctional molecule. It has an aromatic structure which leads to high compressive strength. The polymerization shrinkage of this molecule is comparatively low (4-6%). BisGMA has a very high viscosity which can amplify the matrix, however leads to slabbiness of the material.

In order to decrease the slabbiness, improve the handling and the polymerization ability of BisGMA, a low molecular weight monomer (TEGDMA) is added to the matrix.

Figure 3.156. Figure 8. – Molecular structure of TEGDMA
Macrofill composites contain 10-35% TEGDMA. The application of TEGDMA makes the matrix more flexible, decreases the fragility of the resin, improves the marginal sealing however decreases the wear resistance. Whereas TEGDMA is a small molecule during polymerization it has higher shrinkage compared with BisGMA and influences the dimensional stability of the material.

Two types of UDMA molecule structure are differentiated: aromatic and aliphatic. Generally the aliphatic type is used because the viscosity is lower thereby additional diluting molecule is not necessary to apply.

In consequence of the high viscosity of the matrix the monomer migration during the polymerization is limited. It leads to the presence of high amount of unreacted monomers after the polymerization process. Temperature rise can intensify the migration of the monomers and consequently increase the degree of conversion (DC) of the monomers to polymer.

10.1.1.1. Relation between the matrix and color

The matrix is a translucent material, therefore can illuminate the light and gives opalescence for the composite. The material appears yellowish-red in transmitted light and blue in the scattered light perpendicular to the transmitted light.

**Figure 3.157. Figure 9. – Opalescence appears when enamel color is used**

**Figure 3.158. Figure 10. – Translucence of matrix is dominant in enamel and translucent colors**
10.1.2. Inorganic filler particles

Inorganic filler particles provide dimensional stability for the soft resin matrix. Fillers have a wide range of its size: less than 0.04 μm – above 100 μm. These inorganic particles decrease the polymerization shrinkage and the thermal coefficient, and increase the hardness.

The material of the filler could be cristalline quartz, pyrolytic silica and different type of glasses (lythium-aluminium-silicate, barium-aluminium-silicate and stroncium-aluminiun-silicate).

Composites are usually classified according to the size and distribution of the fillers.

10.1.2.1. Macrofill composites

The first composites were macrofilled with 60-70 v/v% filler content.

There are two types of macrofills: traditional macrofill (filler size: 20-50 μm), and fine particle macrofill (1-5 μm).

Disadvantages of the traditional type are bad polishability and poor wear resistance which leads to fragmentation of big particles from the surface, therefore the material is prone to discoloration and plaque-accumulation.

Fine particle macrofill has better esthetic features and mechanical properties.

10.1.2.2. Microfill composites

Microfill composites contain 0.04-0.2 μm spherical colloidal silicon-dioxide molecules. The small filler particles make possible to highly polish the restoration, however the mechanical properties are poor because of the relatively low filler content. In contrast the total surface of the fillers is very high, therefore the viscosity is increased. Further increase of filler content can improve the physical properties however it could increase the viscosity as well. To solve this problem the inhomogen microfill composites were introduced, where inorganic fillers (0.04 μm silicon-dioxide) are added to the matrix and polymerized. After grinding to 10-20 particles the
prepolymerized particles are mixed to the resin matrix with separated 0.04 µm filler particles (filler content: 50 v/v%). However poor connection between the prepolymerized parts and the matrix can lead to fracture of the restoration. These materials are used mostly in the front region.

10.1.2.3. Hybrid composites

Hybrid composites are a combination of macro- and microfilled composites where two types of particle are used: 0.04 µm colloidal silica, and 2-15 µm spherical or angular macroparticles. Microparticles fill the space between the macrofills, therefore the filler content could be increased to 70-80 v/v% improving the mechanical properties.

Besides the good mechanical and esthetical features, the material is characterized by decreased polymerization shrinkage.

10.1.2.4. Microhybrid composites

Microhybrid composites contain 0.4-3 µm sized irregular glass (boric-silicate, lithium- or barium-aluminium-silicate, stroncium or zinc glass), quartz, zirconium particles and 5-15 % in size of 0.04-0.2 µm. Smaller parts fill the space between the bigger parts. The filler content is 60-70 v/v%. Microhydrs have excellent esthetical features and increased wear resistance.

Figure 3.159. Figure 11. – Direct restoration from microhybrid composite (before and after)

10.1.2.5. Nanofill composites

Nanofill composites mix the advantageous properties of the microfills and the hybrids. Similarly to microhydrs these have excellent polishability and good mechanical features.

Filler size of nanofills is 0.1-100 nm.

Two types of particle were developed: nanomers are 5-75 nm independent silica particules; „nanoclusters” are conglomerates of nanomers with loose connection between the molecules (0.6-1.4 µm).

Nanofills provide long lasting surface smoothness, because during wear only the small particules fetch away from the surface.

There are advantageous optical features, because the light with high wavelength can illuminate through the material without optical interaction (the size of the particules are smaller, than the wavelength of the light). Excellent translucency, well manipulated opacity and color characterize the material.

Disadvantage of nanofills is the fragility.

Figure 3.160. Figure 12. – Building-up of a peg lateral incisor from nanofill composite (before and after)
10.1.2.6. Nanohybrid composites

A mixture of nano- and microparticles is found in nanohybrid composites.

These materials are characterized by excellent optical and mechanical properties, decreased water sorption and fragility.

10.1.2.7. Association between fillers and color

As fillers have an effect on the scattering optical features are affected by the quantity and the size of the fillers. Scattering is a base of translucency and opalescence.

Increased amount of fillers lead to increased opacity, however there is a plato and above this level the color could be inacceptable green-yellow colored. Above this plato, if we would like to increase the opacity and change the color, have to use pigments.

Figure 3.161. Figure 13. – High filler content affects the opacity of dentin colors
10.1.3. Initiators and accelerators

Composites could be light-cured, chemical-cured and dual-cured.

The most common photoinitiator is the camphorokinone (CQ). However there are new photoinitiators, like TPO (trimetibenzoil-diphenyl-phosphin-oxide) informal name is lucerin, or PPD (1-phenyl-1,2-propendione).

To accelerate the process there is a need of organic amine molecule (carbon-carbon double bonds). During the polymerization process the free radicals attack these double bonds.

A good photoinitiator has high light-absorbing ability with low concentration of molecule.

The physical and chemical properties of the composites are strongly influenced by the conversion rate of monomers to polymer. Low conversion rate leads to degradation, fracture, bad marginal sealing, poor biocompatibility.

Camphorokinone is a diketon which is added to the composite in 0.2-1 %. The light absorption generate higher energy level in the molecule (triplet stage) and can lead to free radical formation. If this triplet staged CQ
interacts with an amine molecule it leads to a higher energy exciplex molecule formation (CQ deprives a hydrogen ion from the amine and leads to a free radical formation).

**Animation 1. – The process of free radical formation and polymerization.**

Free radical formation is influenced by the concentration of CQ. Increased concentration increases the DC which leads to improved mechanical properties however this process is limited.

### 10.1.3.1. Relation between the photoinitiator and color

CQ is a yellow molecule however during the photoreaction it loses its color.

If the light cannot penetrate into the material, or the light-curing is poor, there are remaining unreacted yellow molecules, which affect the color of the restoration and by time their yellow color becomes stronger and stronger.

In order to eliminate the discoloring effect of CQ, colorless photoinitiators were developed, like 1-phenil-1,2-propanedion (PPD) or trimethilbenzoil-diphenil-phosphin oxide (TPO) = lucerin.

These molecules are used alone or in combination with CQ.

In case of PPD, the speed of polymerization is lower than in case of CQ therefore it can decrease the stress-formation in the material during the polymerization process.

However the maximum level of their light absorption is at a lower wavelength, therefore there is a mismatch with the maximal light emission of the curing units. To avoid the poor polymerization it is necessary to increase the curing time.

The absorption maximum of TPO or lucerin is at 380 nm. During photo-absorption two free radicals are formed: acil and phosphonil free radicals. These can act independently from the terciery amine, therefore TPO is more effective and lower concentration is enough (not affect the color). The disadvantage of TPO is the highly accelerated polymerization which leads to increased stress formation and the low depth of cure.

### 10.1.4. Pigments

Pigments give the expected color of a composite. Pigments are inorganic oxides. These yellow or grey, blue or green pigments are mixed with the colorless matrix. The disadvantage of these molecules is their effect on the translucency of the material especially the black titanium-dioxide pigments.

### 10.1.5. Co-iniciators

Terciery-amine is the co-iniciator of composites. It could be aromatic or aliphatic. These are colorless molecules however their color can be changed by the photoreaction (yellowish, brownish, redish discoloration).

### 10.1.6. Inhibitors

Inhibitors avoid the polymerization before the photocuring and increase the manipulation-time. Hydrokinone is the most common inhibitor. This is a colorless molecule however can discolor by time.

### 10.1.7. Coupling agents

3-methacryloxypropyltrimetoxy silane is the coupling agent in composites which can bind the inorganic filler particle to the matrix. It has two reactive ends. The Si end is connected to the filler before mixed with the monomer-oligomer matrix. Methoxy-group hydrolizes to hydroxil-group. It can react with a hydroxil-group of the filler, and with another hydrolized silane molecule. It leads to a homopolymer formation on the filler-surface. Carbon double bonds of silane can react with the matrix. Poor silane connection leads to higher water absorption, which can increase the degradation of the resin.

### 11. 3.11. Polymerization stress, layering techniques in molars – Edina Lempel
11.1. The process of the polymerization

During polymerization free radicals are formed by the interaction of the photoinitiator/co-initiator and the 400-500 nm wavelength blue light. Free radicals attack carbon double bonds helping the monomers to react with each other, forming a network. Inorganic filler particles are embedded into this resin network and can stabilize the composite material.

Figure 3.1. Figure 1. – Formation of polymer network

Polymerization shrinkage of dental resin composites is due to the fact that monomer molecules are converted into a polymer network and, therefore, exchanging van der Waals spaces in covalent bond spaces.

Shrinkage is the highest at the beginning of the polymerization, but the material is weak and able to become deformed. This stage is the gel-phase, where there is a chain formation. There is not cross-linking.

It is continued by the cross-linking (network formation), the material becomes rigid. The shrinkage rate is lower, but the stress formation is higher.

11.1.1. Influencing factors of the polymerization kinetics

- Chemical content of the resin matrix.
- Quantity and quality of the fillers and the coupling agent.
- Viscosity and streaming of the matrix.
- Degree of humidity.
- Curing time and mode.

Important to increase the time of gel-phase, because the composite is able to relax in this stage. Furthermore it is most likely that most of flow takes place before the gel point and that after the gel point most of the contraction stress is being developed.

11.2. Polymerization stress

Polymerization stress development results from the complex interplay of volumetric shrinkage, reaction kinetics, and viscoelastic properties. During setting of the resin composites the polymerization shrinkage induces contraction stress.

Clinical consequences of polymerization stress:

- Stress can cause deformation and cracks on the tooth, it may fracture the unsupported cusp. The higher the loss of dentin, the lower the resistance of the tooth against fracture during or after the polymerization.
3. Reconstructive dentistry

- Stress can lead to marginal leakage formation on the composite-tooth interface.
- It may result micro cracks in the material which lead to fracture during mastication.

**Figure 3.163. Figure 2. – Consequences of polymerization shrinkage**

Degree of shrinkage stress is influenced by the size of the restoration and the thickness of the cavity wall.

**11.2.1. Influencing factors of degree of shrinkage or stress**

**11.2.1.1. Filler load**

Matrix is responsible for shrinkage. The more monomer form network, the higher the shrinkage. Spaces in the network filled with the fillers and can reduce the degree of shrinkage.

**11.2.1.2. Degree of polymerization**

Direct correlation was found between the degree of polymerization and the amount of shrinkage stress. Higher contraction stress was observed with high polymerization rates.

However lower degree of polymerization impair the quality of the filling (mechanical and biocompatible properties are decreased).

**11.2.1.3. Elastic (Young) modulus**

Stiffness is quantified by Young’s modulus of elasticity, which represents the resistance of a material to elastic deformation. The elastic modulus of the resin composite has been found to be an important factor for both the shrinkage and the contraction stress. In vitro test has shown that the setting shrinkage increases with the rigidity of the resin composite. The lower the Young’s modulus of a resin, the greater its flexibility and it has more capacity to reduce remaining contraction stress.

Stress is defined by the shrinkage and multiplicatied by the elastic module (Hooke’s principle).

It has shown that the elastic modulus of resin composite increases with the volume fraction of the inorganic filler content. Thus, flowable composites have the lowest elastic module.

**11.2.1.4. C-factor**

The magnitude of contraction stress has been found to be dependent on the cavity configuration (C-factor), which is the ratio of the bonded to unbonded surface area of the restoration. C-factor theory is originated from Fleizer.

Throughout the entire polymerization process, plastic deformation or flow of the resin composite occurs and may partially compensate for the induced shrinkage stress. This irreversible plastic deformation takes place during the early stage of the setting process, when the contraction stress exceeds the elastic limit of the material. As the setting proceeds, contraction and flow gradually decrease, because stiffness increases. Such compensation through flow is affected by the configuration of the restoration.
Figure 3.164. Figure 3. – The values of configuration factor in cases of different cavities

<table>
<thead>
<tr>
<th>Cavity pattern</th>
<th>C-factor =</th>
<th>Bonded surface</th>
<th>Unbonded surface</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class II., III.</td>
<td></td>
<td></td>
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<tr>
<td>Class V.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class IV.</td>
<td></td>
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</tr>
</tbody>
</table>

The higher is the C-factor, the more rigid is the material in the cavity and leads to higher stress formation.

Layering techniques or applying a low elastic modulus liners between the tooth structure and the resin composite have been proposed to minimize the internal stress and deformation of the tooth structure.

11.2.2. Possibilities to decrease the stress

11.2.2.1. Application of flowable composite or resin modified glass ionomer cements

The rationale behind the usage of liners under resin composite is that the materials will absorb the volumetric changes and can stretch or flow to allow stress relaxation. Because of their low filler content and reduced elastic modulus these materials could act as a „stress breaker” to absorb the forces of the polymerization shrinkage or cyclic loading. The low modulus of elasticity reduces the stiffness and increases the stress absorption capacity of the restoration. The resin modified glass ionomer cements are preferred over the conventional glass ionomer cements because they can chemically copolymerize with the restorative resin composite placed over the intermediate cement layer.

These base materials with the appropriate elastic modulus can reduce the marginal defect in an overlying composite resin restoration.
11.2.2.2. Polymerization strategies (Curing time and mode)

Photo polymerization with high intensity increases the degree of conversion (DC) and improves the physical properties, however with short exposure time increases the shrinkage and the stress as well because insufficient time is allowed for stress relaxation.

Curing with lower intensity can allow the material to relax decreasing the stress formation.

Polymerization process much more influenced by the total energy of the light-curing. The total energy is the product of the light intensity and the curing time. Lower intensity and slower polymerization can lead to higher stress-relaxation.
3. Reconstructive dentistry

Soft start, ramped, delayed and oscillating curing

These techniques of curing provide an initial low rate of polymerization thereby extending the time available the stress relaxation before reaching the gel point.

Soft start polymerization

This involves 100 mW/cm² output for 10 seconds, followed by an immediate jump to 600 mW/cm² output for 30 seconds.

Ramped curing

The intensity is gradually increased or „rumped up“ during the exposure. This ramping consists of either stepwise, linear or exponential modes.

Delayed curing

The restoration is initially incompletely cured at low intensity. The clinician then sculpts and contours the resin to the correct occlusion and later applies a second exposure for the final cure. This delay allows the substantial stress relaxation to take place. Longer the time available for relaxation, the lower the residual stress.

Delayed curing and exponential ramped curing appears to provide the greater reduction in curing stress.

Oscillating or Intermittant curing

Full intensity is applied for 1 second which is followed by a dark period for the same time. This pattern is repeated for 10-20 seconds.

The applied light intensity and efficacy depends on the type of the curing unit. These devices could be different:

Quartz-Volfram-Halogen (QTH) units

Halogen bulbs generate light through the heating of tungsten filaments to high temperatures. A small percentage (< 1 %) of the energy is given off as light, while most of the energy given off is in the form of heat. A drawback of halogen bulb is that this generation of heat causes a degradation of the components of the curing unit over time. The result can be a decline in the irradiance, which compromises the curing ability of the unit. Light output is 300 – 1000 mW/cm². Spectrum of the light is very wide, needs filter to emit the light at 370- 550-nm—wavelength range (adjusted to camphoroquinone = CQ).

Light Emitting Diode (LED) units

LEDs use junctions of doped semiconductors for generating light. Diodes (blue gallium-nitrit) are incensed with electric potential. When return to the base stage emit photons on a special wavelength. The advantages of these units are that the spectral output falls between 400 and 500 nm so that no filters are required. Because the energy generated is not in the form of heat, LEDs have a longer lifetime, with little degradation over time. Capacity of
LEDs is 1000-1400 mW/cm². Emission spectrum is 450-480 nm, it does not need filter. This spectrum is compatible with CQ, however is not with other photo initiators (430 nm).

*Plasma Arc (PAC) units*

Plasma-arc lights are made up of two electrodes in a xenon-filled bulb. Current heats the plasma to several thousand degrees Celsius. The heated plasma gives off light and heat. The light intensity (1300-2000 mW/cm²) given off by plasma-arc units is greater than that for halogen-based units, which can decrease curing time up to 75 percent (3-6 sec). Filters are required. The increase in light intensity and decrease in curing time with plasma-arc lights have been shown to enhance polymerization shrinkage in some studies. It has a narrow spectrum (450-480 nm).

*Argon laser*

‘Light Amplification by Stimulated Emission of Radiation’

Lasers can emit light at specific wavelengths as a result of the excitation of atoms of suitable gases to specific energy levels. Because lasers emit light at specific wavelengths (400-450 nm), there is no need for filters. Lasers are reported to require less time to adequately polymerize composites. However, these units are large and expensive. There are many factors that affect curing of dental materials. The most important of these are intrinsic to the material and the light source. There are, however, steps that the dental office can take to maximize the curing process, such as keeping the light tip clean and free of scratches, positioning the light tip in the correct distance from and the correct orientation to the material, maintaining the bulb and filter in good working order, and establishing appropriate curing times for particular materials.

11.2.2.3. Placement (incremental) techniques

The rationale for applying multilayer technique (oblique increments) is to reduce the overall polymerization stress by increasing the number of increments and giving them an optimal geometry to augment the total free surface area. Incremental layering minimizes the C-factor.

- The main factor to decrease the polymerization stress.
- Small amount of material, maximum 2 mm layer thickness.
- With layering, we can decrease the C-factor (minimal contact with the opposing wall).

*Incremental techniques in molars and premolars*

- *Facio-lingual (vertical) layering*: The composite layers are placed in parallel to the axis of the tooth and the wall, starting from the vestibular wall towards the oral wall. The disadvantage is the height of the layers.

**Figure 3.167. Figure 6. – Vertical layering**
• **Gingivo-occlusal (horizontal) layering:** The layers are perpendicular to the axis of the tooth, the filling up is started from the bottom of the cavity. The disadvantage is that it does not reduce the C-factor acceptably.

**Figure 3.168. Figure 7. – Horizontal layering**

![Horizontal layering](image)

• **Wedge-shape Layering (oblique):** In this technique wedge-shaped composite increments are placed and polymerized only from the occlusal surface.

**Figure 3.169. Figure 8. – Wedge-shape layering**

![Wedge-shape layering](image)

• **Axial bevel technique:** In Class II restorations, the enamel proximal surface is built up first through the application of different wedge-shaped composite increments using an oblique layering technique while being careful to avoid having a single composite increment be in contact with opposing cavity walls. Each composite increment is pulse-cured with a low intensity light for a short duration (depending on the type of composite and depth of the preparation) followed by a waiting time of three minutes to allow for strain relief. During this three-minute waiting time, a thin layer of flowable composite is applied to a single surface in the occlusal pulp floor and axial wall of the preparation to reduce the C-factor and help avoid cusp deflection due to stress from polymerization. At this point, the resin-based composite restoration’s proximal surface and the flowable composite are cured together at once at a higher intensity. Final polymerization of the composite restoration’s proximal surface and the flowable composite is completed at higher intensity.

**Figure 3.170. Figure 9. – Axial-bevel technique**

![Axial-bevel technique](image)
• **Three-sited technique**: This is a layering technique associated with the use of a clear matrix and reflective wedges. First, the curing light is directed through the matrix and wedges to guide the polymerization vectors toward the gingival margin, thus, preventing any gap formation. Then wedge-shaped increments are placed to prevent distortion of cavity walls and reduce the C-factor. This technique is associated with polymerization first through the cavity walls and then from the occlusal surface in order to direct the vectors of polymerization toward the adhesive surface (indirect polymerization technique).

• **Incremental technique**: The first layer fills the proximal box. Next layers are horizontal (2mm). But horizontal layers increase the C-factor.

**Figure 3.171. Figure 10. – Incremental layering**

• **Centripetal Build-up Technique**: This technique was especially developed for class II cavity restorations. An initial vertical composite increment is applied on the cervical margin against the metal matrix. Cavity filling is then completed by horizontally layering the composite. This technique allows transformation of class II cavities into class I cavities.

**Figure 3.172. Figure 11. – Centripetal build-up technique**
• **Successive Cusp Build-up Technique:** In this technique the first composite increment is applied to a single dentin surface without contacting the opposing cavity walls, and the restoration is built up by placing a series of wedge-shaped composite increments to minimize the C-factor in 3-D cavity preparations. Each cusp is then built up separately.

**Figure 3.173. Figure 12. – Direct filling constructed with successive cusp build-up technique**

• **Combination of the axial bevel and the successive cusp built up techniques:** For II. Class cavities the proximal box is layered according to the axial bevel technique, transforming the cavity to I. Class cavity, which is then built up according to the successive cusp built up technique.
Figure 3.174. Figure 13. – Combination of the axial bevel and the successive cusp built up techniques (Building-up of the proximal wall)

Figure 3.175. Figure 14. – Combination of the axial bevel and the successive cusp built up techniques (Building-up of the oblique cusp)
Figure 3.176. Figure 15. – Combination of the axial bevel and the successive cusp built up techniques (The filled-up cavity)
Figure 3.177. Figure 16. – Combination of the axial bevel and the successive cusp built up techniques (The polished restoration)
• *Divided, horizontal layering technique* (for Class I. and V.): As a first step, a horizontal layer is adapted to the occluso-pulpal wall and then it is divided into four parts with a cross-shaped recess before the polymerization. After the polymerization, the cross-shaped notch is filled with composite resin and polymerized. These steps are repeated until the entire cavity is not filled.

**Figure 3.178. Figure 17. – Divided, horizontal technique**
12. 3.12. Direct veneers with different techniques – Edina Lempel

12.1. The features of tooth color

In the 1600s Newton discovered that light composed of particles. It was demonstrated on a prism that white light is a combination of multiple colors (rainbow is a natural prism). The light is different wavelengths of energy. The particles are constantly vibrating with different intensities. The red color has the longest wavelength and the slowest vibration, while the purple has the shortest wavelength and the highest frequency vibration. The 40% of light is only seen.

Ultraviolet light is above the violet and even beyond the X-rays and gamma rays, which have a very short wavelength and the vibration is very intense, thus the energy level is very high. At the other end of the spectrum the red wavelength is located and above to this the infrared, and the electromagnetic radiation. Here, the wavelength increases, the frequency decreases. The radio waves can be found in this range.

12.1.1. The color of objects

The color of an object is determined by the wavelength of the reflected and scattered light. Photons represent energy, energy stimulates the receptors in the retina and the brain creates a visual color depending on the photon wavelength. Blue: 400-500 nm, green: 500-600 nm, red: 600-700 nm. Besides the shade (hue), the intensity (value) of the color is important as well. Each wavelength may appear in different intensities. The black color intensity is 0%, the white is 100%, while the gray is 50%. The color saturation (chroma) refers the weak or strong expression of the color. Munsell (1898) color diagram illustrates the three dimensions of color: the hue, the chroma, and the value.
12.1.2. The color of teeth

According to Vanini (http://www.enahri.com/amministrazione/repository/files/86/01ppav13n1p19.pdf) the color of the teeth has two additional features besides the three dimensions: the intensity and the characterization.

12.1.2.1. Hue és Chroma

The hue is the shade, which is mainly determined by the dentin. The chroma is the saturation, which is the manifestation of dentin’s color modified by enamel.

Figure 3.179. Figure 1. – The hue is determined by dentin

The hue and chroma were named as chromaticity by Vanini which is basically the two dimensions of color. According to spectrophotometric studies the colors of tooth could be only "A" and "B", but the darker versions of these are "C" and "D". "A" is the dominant color (orange-red shades), while the shades of "B" is a bit more rare (yellow-green). "A", "B", "C", "D" colors are the shades of teeth, based on the VITA Classical shade guide.

12.1.2.2. Value

The value is brightness, which is determined by the quality and thickness of enamel. According to the age of enamel, three types of value could be distinguished:

- High value (young enamel),
- Moderate value (middle-aged enamel),
- Low value (matured enamel).
12.1.2.3. Intensities

White specks, irregular, opaque, milky-white discoloration with different manifestation can be observed on natural teeth. Vanini called them as intensities. These white “pigmentations” can be classified into 4 groups:

- spots,
- clouds,
- snowflakes,
- horizontal streaks.

Figure 3.180. Figure 2. – Cloud-like intensities on the incisal edge

Figure 3.181. Figure 3. – Horizontal intensives on the central incisors
12.1.2.4. Opalescence

Enamel is responsible for opalescence, because of its translucent character. The enamel can amplify the short wavelengths of the light, which goes through. Because short wavelengths are responsible for the blue color, the translucent parts of the enamel appear blue-gray. This opalescence is the most pronounced on the incisal edges of the anterior teeth. This is so-called “incisal halo”. Incisal halo can be classified according to its appearance:

- Mamelon-style,

**Figure 3.182. Figure 4. – Incisal halo with mamelon-style**
According to the color tone of the incisal halo and the maturity of enamel, the human eye can distinguish three types of opalescence: gray (adult), blue (children), white or amber (matured). The dentin can also affect the opalescence, since the thicker the enamel the more the reflected wavelengths by dentin.

**12.1.2.5. Characterization**

According to Vanini characterization has five types, which are responsible for the individual appearance of the teeth:

- Mamelon,
- Band,
- Margins,
- Patch,
- Cracking.

The brightness of the incisal halo can be increased by the mamelon-type characterization.

Primarily it is typical in young mouths, where enamel is not abraded.

The band characterization is an intensity feature on the equator of the crown.
The incisal characterization is a white border between the incisal halo and the incisal edge.

The stains and cracks characterize the matured enamel (brown or amber effects).

**12.1.3. Other optical features of the tooth**

**12.1.3.1. Fluorescence**

Fluorescence is the emission of light by a substance that has absorbed light. Dentin-pigments are responsible for this phenomenon.

**12.1.3.2. Opacity**

Opacity is the measure of impenetrability to visible light; the degree to which light is not allowed to travel through.

**12.1.3.3. Transparency**

A physical property of allowing the transmission of light through a material without being scattered.

**Figure 3.183. Figure 5. – The application of enamel color gives transparency to the tooth**

**12.1.3.4. Translucence**

Translucence is a super-set of transparency, allows light to pass through; but the photons can be scattered at either of the two interfaces where there is a change in index of refraction, or internally on particles. The opposite property of translucency is opacity. When light encounters a material, it can interact with it in several different ways. These interactions depend on the wavelength of the light and the nature of the material. Tooth can scatter the short wavelengths (blue light).

**12.1.4. Dentin-effect**

Due to the dentin’s macro-structure there are heavily saturated and partially opaque areas. This is the reason that the hue and saturation is determined by the dentin.

The diameter, the density, the S-shaped curvature and the degree of mineralization of the dentinal tubules and inter tubular parts can affect the absorption and reflection of the light. This is the so-called polychromatic effect. Traditionally, three distinct areas can be distinguished on the crown considering the polychromatic effect: the neck, the middle and the incisal third. However Vanini isolated different polychromatic areas within these bands as well.
12.1.5. Enamel-effect

The enamel transmits, reflects and scatters the light due to the organized running of the enamel rods, the thickness of the enamel and the presence of organic pigments. The thicker the enamel, the more light is scattered and reflected, therefore the higher the value. In addition, the translucence and opalescence can increase the light value.

Figure 3.184. Figure 6. – Restoration of erosion with enamel color (before)

Figure 3.185. Figure 7. – Restoration of erosion with enamel color (after)
### 12.1.6. Dentin-enamel effect

Enamel value is significantly reduced by the opaque dentine, thereby creates a grayish hue. If the enamel is thin and dentin is saturated, then the hue is dominant, which is determined primarily by dentin. This is mainly observed in the cervical region.

As the enamel becomes thicker towards the incisal edge and dentin is undermined, the value of enamel is increased.

The polychromatic effect of dentin could be observed on enamel as well. The value is distinct, different patterns appear on the teeth.

### 12.2. Direct composite veneers

In consequence of continuous improvement of composite materials there is possibility to treat bigger destructions with these restorative materials.

Veneering means: “thin blanket of bad properties”. Besides the esthetic aspect the minimal invasivity, the functionality and the consideration towards the periodontium play an important role in case of veneer preparation. The material of veneer may be ceramic or composite.

The advantage of direct composite veneers is that there is, compared to porcelain veneers, usually less filing required and more conservative preparation is need. In addition, the treatment can be completed in only one session. Direct veneer is cheaper, changeable, time saving, less abrasive and can be replaced at any time by indirect ceramic veneer.

In addition to the advantages, there are, of course, also disadvantages associated with the direct use of composite. Some examples are wear-and-tear and degradation of the material over the course of time, loss of surface shine and cohesive cracks in the material. Direct composite veneers are also difficult concerning the processing and color composition. Adapting and modelling the composite correctly without creating air bubbles – gaps and voids – is important. The longevity of direct composite veneers is 4-6 years.

*Indications for direct composite veneers in the front region:*

- Caries treatment
- Fracture
- Diastema closure
- Correction of shape disorders
- Correction of eroded surfaces
- Correction of mild discoloration or enamel defect.

*Contraindications:*

- Composite allergy
- Para functions
- Malocclusion (especially edge to edge bite)
- Enamel deficiency or serious enamel destruction
- Dark staining.

### 12.2.1. Preparation

A round-ended diamond is used for preparation a labial chamfer in enamel. This chamfer extends gingival, but must keep on enamel, close to the dentin-enamel junction. The mesial and distal proximal borders of the
preparation are stopped just slightly labial to the contact areas. This type of preparation enhances resistance form and results in a clearly defined periphery to which the composite resin can be finished. The depth of the preparation should be 0.3 mm at the gingival, 0.5-0.7 facially and 1 mm incisally. This depth allows for the labial thickness of composite needed to cover most discoloration without significant over contouring. In severely discolored teeth, it allows space for color modifiers to mask stains and alter the shade of the underlying tooth. A straight butt-joint finish line was utilized at the lingual aspect to improve the strength of the margin. In case of fracture a chamfer should be prepared all around the margins.

**Figure 3.186. Figure 8. – Chamfer preparation at the gingival and contact area**

![Chamfer preparation at the gingival and contact area](image)

**12.2.2. Reconstruction techniques for direct veneers**

Several techniques are available to restore directly a tooth deficit with composite resin.

**12.2.2.1. Free-hand technique**

For this technique, the stratification is started centrally and continued towards facially. First of all a dentin core should be constructed and then the enamel should be restored with thin enamel color. In free-hand technique the anatomical layering, color creating build up technique is the best choice to build-up a tooth. In case of anatomical layering the tooth color is created by the dentist using different dentin and enamel colors with different hue and layer thickness according to the original individual optical features. In this technique the layer thickness has an all-important role in color creation. Characterization is accomplished with translucent and effect colors (different white and amber colors).

The next images introduce a direct veneer building-up with free-hand technique. The applied material is Enamel Plus HRi microhybrid composite (Micerium, Italy).

**Figure 3.187. Figure 9. – Before restoration (Both central incisors are fractured obliquely, the right incisor is built-up temporary)**

![Before restoration](image)
3. Reconstructive dentistry

Figure 3.188. Figure 10. – Prepared teeth (The hue, the enamel value, the shape and place of intensities, the incisal opalescence and other features are determined before the rubber dam placement)

Figure 3.189. Figure 11. – Conditioning
Figure 3.190. Figure 12. – Application of bonding agent

Figure 3.191. Figure 13. – Light-curing of the adhesive
Figure 3.192. Figure 14. – Dentin core (1. layer: UD4 color)

Figure 3.193. Figure 15. – Dentin core (2. layer: UD3 color)
Figure 3.194. Figure 16. – Dentin core (3. layer: UD2 color)
Figure 3.195. Figure 17. – Shaping of the mamelons
Figure 3.196. Figure 18. – The dentin core without enamel layer (Transparency and translucence are missing)

Figure 3.197. Figure 19. – Removal of excess material with diamond bur
Figure 3.198. Figure 20. – The application of transparent color to create the incisal halo
Figure 3.199. Figure 21. – UE3 enamel layer
Figure 3.200. Figure 22. – The layering of enamel color (UE3 enamel color can mimic the young enamel)
Figure 3.201. Figure 23. – Restoring of the contact surface with enamel color

Figure 3.202. Figure 24. – Characterization with intensive colors
Figure 3.203. Figure 25. – Modelling the surface texture with diamond bur (The bigger irregularities are removed with red diamond bur and finishing stones.)

Figure 3.204. Figure 26. – Polishing with abrasive paste
Figure 3.205. Figure 27. – Micro texture of the surface is modelled with brushes (High value is provided with polishing rubbers and textile wheels)

Figure 3.206. Figure 28. – The polished direct veneers
Figure 3.207. Figure 29. – The individual characterization is provided with different intensive colors

Figure 3.208. Figure 30. – The appearance of the incisal halo is provided with transparent color
12.2.2.2. Wax-up technique

Wax-up technique usually used in fractured teeth or after removal of old restorations in the front region. An impression is taken from the involved jaw and from the antagonist and plaster casts are prepared. Then the restorable teeth are constructed from wax on the cast, according to anatomic conditions, taking into account other aesthetic aspects.

Figure 3.209. Figure 31. – Before restoration with wax-up technique (Old, discolored restorations and lateral incisor aplasia)

Figure 3.210. Figure 32. – Prepared teeth
Afterwards a silicon stent is taken from the wax model. On the impression the final shape of the palatal surface and incisal edge is fixed.

**Figure 3.212. Figure 34. – Silicon stent**
Enamel color is placed into the silicon stent and than pressed to the etched and bonded palatal surface of the tooth.

**Figure 3.213. Figure 35. – Construction of the palatal surface with the help of silicon stent**

After light-curing the silicon stent is removed and this thin enamel layer is used as a firm base during the building-up of the dentin core to an oro-vestibular direction. Finally a thin layer of enamel color is applied onto the buccal surface.

**Figure 3.214. Figure 36. – The thin palatal enamel layer is act as a base during the placement of the dentin core**
Celluloid strip is used to model the proximal surface. The contour is shaped with free-hand. The central line positioning is helped by the silicon stent. The restoration is finally finished and polished.

**Figure 3.215. Figure 37. – The polished direct veneers**

**12.2.2.3. Silicon build up guide and intraoral mock-up technique**

Composite mock-up is used (without conditioning and adhesives) to establish the length and contour of incisal edges and anterior guidance. Silicon putty stent is utilized the lingual surfaces and incisal edge position of the composite mock-up. Silicon build up guide and intraoral mock-up technique is the analogue of the wax-up technique, but in this case the fractured or abraded teeth are built up firstly with composite. This state is fixed with the silicon stent.

After removal of the mock-up placement of the enamel color on the lingual stent initiates lingual stratification prior to positioning of the stent on the etched and bonded central incisors. Then dentin colors are stratified along
an oro-vestibular direction, and finally thin enamel colors layered on the facial side. Interproximally a thin enamel layer is applied and contoured with a clear interproximal strip and freehand sculpting.

**Figure 3.216. Figure 38. – Before restoration (fracture of tooth 11)**

![Before restoration (fracture of tooth 11)](image)

**Figure 3.217. Figure 39. – Composite mock-up on tooth 11**

![Composite mock-up on tooth 11](image)
Figure 3.218. Figure 40. – Prepared tooth and the silicon stent
Figure 3.219. Figure 41. – The polished direct restorations
13. 3.13. Dental ceramics – Edina Lempel

Aesthetic restorations made of tooth-colored materials, mostly different types of ceramics and composites. Depending on the intraoral or extra oral implementation of the restoration we can talk about direct, semi direct, and indirect techniques.

Direct technique: if the restoration is implemented only with intraoral steps (eg. plastic filling techniques with tooth-colored resin materials).

Semi direct technique: intraoral and chair-side extra oral steps are need (eg. intraoral composite inlay).

Indirect technique: the restoration is made by a dental laboratory (dentist takes an impression from the tooth/teeth which need restoration (eg: ceramic inlay, onlay, overlay).

**Figure 3.220. Figure 1. – Impression has taken to indirect ceramic onlay**
Figure 3.221. Figure 2. – Indirect ceramic onlays on plaster cast
The ceramics are preferred to use in dental practices because of their several advantages:

- Low plaque-accumulation,
- Shape-, color stability,
- Erosion-, abrasion resistance,
- High aesthetic,
- High optical properties,
- Biocompatibility,
- Chemical resistance.

**Figure 3.222. Figure 3.** – Ceramics have shape and color stability (Ceramic onlays are cemented for 10 years)
Ceramics are composed of metallic (Al, Ca, Mg, K, etc) and nonmetallic (Si, O, B, F, etc) elements. Ceramics have two different phases: glass matrix and incorporated crystalls (different crystalls; depend on the type of ceramic).


### 13.1. Classification according to the composition

#### 13.1.1. Silicate ceramics

Silicate ceramics contain minimum 15% leucit.

Types:

- **Conventional feldspathic ceramics**
- **Feldspathic ceramic with leucit reinforcement**
- **Lithium-disilicate glass ceramic with syntherized glass ceramic coverage**
13.1.1.1. Conventional feldspathic ceramics

Big, irregular leucit crystals are incorporated into a silicate-glass matrix. Feldspathic ceramics can be used for coverage of metal-ceramic restorations, for all-ceramic crowns and inlays/onlays. Disadvantages are the fragility and the low elastic module, however the fracture resistance can be increased with adhesive resin cement.

Figure 3.223. Figure 4. – Metal-fused ceramic crown

13.1.1.2. Feldspathic ceramic with leucit reinforcement

This type contains 30-40 % crystallized leucit, instead of 15%. It can increase the fracture resistance. These reinforced ceramics are used for anterior, posterior full-ceramic crowns and for inlays/onlays/overlays.

Figure 3.224. 5. ábra – Figure 5. – Veneers (11, 21) and crown (22) from feldspathic ceramic with leucit reinforcement
13.1.1.3. Lithium-disilicate glass ceramic with syntherized glass ceramic coverage

The frame of this type of ceramic is a lithium disilicate and lithium orthophosphate containing glass ceramic, while for coverage a fluoroapatit containing sintered ceramic is used. These are characterized by high fracture resistance and flexibility, so short-span bridges may be made from them.

13.1.2. Oxide ceramics

Two main types of oxide ceramics can be differentiated: the aluminium-oxide ceramics and the zirconium-oxide ceramics.

13.1.2.1. Aluminium oxide ceramics

As aluminium oxide is incorporated into the glass-matrix, its strength is increased. It is used as a frame. Feldspethic porcelain is offered for the coverage.

There are three different types:

- Glass infiltrated aluminium oxide ceramic: the aluminium oxide frame is made with dry sinterization. The frame is infiltrated with melted glass afterwards. The elasticity is increased (450MPa).

- Syntherized aluminium oxide ceramic: high-purity, densely sinterized alumina ceramic is prepared, which does not contain silicate. Elastic module is 610 Mpa.

- Glass infiltrated Spinell ceramic: contains magnesium oxide (MgAl₂O₄) which can improve the optical properties, however attenuates the frame.

13.1.2.2. Zirconium oxide ceramics

1/3 part of aluminium oxide is substituted with zirconium oxide, thereby the fracture resistance is about 1000 MPa.

Indications of zirconium oxide ceramics:
• bridges (longer span),
• all-ceramic crowns,
• implant abutments,
• endodontic posts.

**Figure 3.225. Figure 6. – Full-ceramic crowns with zirconium oxide ceramic frame**

---

**13.2. Classification according to the odontotechnological procedure**

**13.2.1. Laminated ceramic**

This technique is analogous to the platinum foil technique. A core is burnt to a fireproof working cast from feldspethic ceramic. Afterwards a hydrothermal glass mass is laminated and burnt to this core on a lower temperature.

**Figure 3.226. Figure 7. – Indirect onlay from laminated ceramic**
Figure 3.227. Figure 8. – Ceramic powders in different colors
Figure 3.228. Figure 9. – Ceramic mass used for laminated ceramics
13.2.2. Glass infiltrating procedure

Aluminium-, magnesium-, zircon oxide ceramic mass is laminated to the working cast and after it is burnt. Then this core is infiltrated with lanthanum-glass and it is burnt as well. The core is covered with aluminium oxide ceramic.

13.2.3. Moulded (cast) ceramics

The restoration is modeled from wax on the master cast and it is invested. After the wax is burnt out and the melted glass is cast into the ingot with centrifugal force. The restoration is elaborated and placed into a ceramization investing material where the fluormica crystals build into the ceramic restoration.

Figure 3.229. Figure 10. – Cemented cast ceramic onlays (15, 16)
13.2.4. Pressed ceramics
(http://www.roedentallab.com/downloads/emaxpressdata.pdf)

The restoration is formed from ceramic ingot with high pressure. The restoration is modeled from wax, it is invested into an ingot-mould and the wax is burnt out. After the reinforced glass-ceramic ingot is pressed into the ingot-mould with high pressure in a heat and press furnace. The restoration is colored with special colorific glass-ceramic in order to make it individual.

**Figure 3.230. Figure 11. – Ceramic ingots in different colors**
Figure 3.231. Figure 12. – A2 shade ceramic ingot
Figure 3.232. Figure 13. – Ingot mould (skillet) for investment pressing technique
Figure 3.233. Figure 14. – Making wax model for pressed ceramic crown
Figure 3.234. Figure 15. – Working pressing furnace
Figure 3.235. Figure 16. – Ceramic dye for individualization
13.2.5. CAD/CAM technique

It is possible to choose from a variety of CAD / CAM (Computer Aided Design/Computer Aided Manufacturing) techniques nowadays, but according to the original method, a three-dimensional intraoral camera takes an optical impression from the prepared tooth, and according to the data the burring-engine, guided by computer, mills the restoration from a pre-fabricated ceramic workpiece.

Figure 3.236. Figure 17. – The process of scanning

Figure 3.237. Figure 18. – The planning phase according to the scanned information
Figure 3.238. Figure 19. – CAD/CAM milling chamber
Figure 3.239. Figure 20. – CAD/CAM sintering furnace
3. Reconstructive dentistry
13.2.6. Sonoerosion procedure

The restoration is modeled from wax and two negative copy is formed from a special metal and it is placed into a bromic-carbid suspension. A ceramic quadrant is placed between the two metal copy. After an erosion procedure is induced with ultrasonic system. The excited erosion process reproduces the negative copy of the negative metal copies from ceramic. This is the exact replica of the original wax-up.

13.3. Surface treatment of ceramics

The adhesive cement provides good retention for the restoration, decreases the marginal leakage and increases the fracture resistance of the tooth and the restoration. This requires a clean, rough inner surface. For this purpose, a pretreatment is necessary.

13.3.1. Methods for surface treatment

13.3.1.1. Abrasion with a diamond rotary instrument

This is the simplest, but not very precise method of increasing the inner surface of the ceramic restoration. The diamond abrasive grains - sized 80-100 microns - leave behind a rough surface, but both the matrix and the various crystals are eroded. There is an increased risk for marginal damage.

13.3.1.2. Etching

For acid etching 2.5-10% hydrofluoric acid is used, which selectively dissolves the glass matrix from among the crystals, increasing the surface and by this way the bonding force. The size and location of leucit crystals determine the formation of micro pores. If leucit crystals are fewer, smaller retention area can be created. Acid etching is effective on silicate ceramics, however this surface enhancement method is not able to increase the surface of oxide ceramics.

13.3.1.3. Sandblasting

In case of sandblasting the inner surface of the ceramic restoration is treated with 50-110 microns particle size of Al₂O₃ on 2.5-4 bar pressure. Primarily this method able to increase the surface of silicate ceramics, however the surface of oxide ceramics is not increased to the extent desire, because the Al₂O₃ particles have similar hardness than the oxide ceramic’s hardness. Al₂O₃ particles can erode the softer surfaces. Instead of Al₂O₃, 1-3 microns diamond abrasives are used to handle the oxide ceramic surface.
13.3.2. Treated by silane (Silanization)

The inner surface of ceramic restoration should be treated with silane solution in order to increase the bond strength between the ceramic and composite cementing agent. The silane molecules are bifunctional, since one of the end is silicon dioxide which can attach to the ceramic’s OH group and the other functional group (methacrylate) can contact with the organic matrix of the composite, thereby ensuring a reliable connection between the ceramic and the adhesive cement.

Figure 3.241. Figure 22. – Hydrofluoric acid and silane used for surface treatment of ceramics

13.3.3. Cementing (luting) materials

Ceramic restorations are mostly cemented with dual-cure adhesive cement.

Figure 3.242. Figure 23. – Dual-cured adhesive cements for luting ceramic restorations
The composition of adhesive cements is similar to that of composites. For the initiation of dual curing, 400-500 nm wavelength is required, but in the deeper layers, where light penetration is impeded, the polymerization is continued by self-curing way. Thus, beside the photoinitiator and the amine molecule, benzoyl-peroxide molecule completes the polymerization reaction.

The bond strength of the dual cements is 20-25 MPa to enamel, 15 Mpa to dentin and 40 Mpa to ceramics. The compressive strength is 330-350 MPa and the elastic modulus is 8.5 GPa. The positive effect of the high elastic modulus is that the cement disperses the forces which are mediated by the rigid ceramic and the biting forces.

Low viscosity lets the restoration to fit easy. The layer thickness of the cement is 15-200 microns. The bigger is the gap, the greater is the polymerization stress. During the polymerization stress compensatory motion occurs, the restoration is subsided, the cavity walls are inclined towards the restoration. Therefore - as with any type of restoration - it is important to provide accurate fit.

**Figure 3.243. Figure 24. – Lack of fit (In case of ceramic restorations 100 µm fit accuracy is acceptable. Arrow shows the gingival gap)**
13.3.4. Indications of ceramic restorations

- Significant loss of tooth structure (if the orovestibular diameter of the cavity is between one-third and half according to the cusp-cusp distance, than inlay is indicated, if it is greater, or the cavity walls are too thin (<2 mm), or partially are missing, onlay, overlay preparation is suggested.

- On root canal treated teeth the adhesively fixed onlay/overlay has a splinting effect on the rest tooth structure, and protects against the fracture.

- To reconstruct abraded teeth.

- In case of metal allergy.

- To counteract the formation of local element in case of more metal restorations.

- To restore missing tooth as a bridge.

- Periodontium-saving preparation (preparation in enamel along the margins).

- Good oral hygiene.

13.3.5. Contraindications of ceramic restorations

- High caries disposition, poor oral hygiene.

- The presence of parafunctions.

- Allergy to composite (relative contraindication).

- Young age (wide pulp chamber).
• Periodontal problems (mobility, refractory periodontitis).
• Heavily discolored tooth (relative contraindication).
• Too short clinical crown (<5 mm).
• Marginal enamel deficiency (<0.5 mm).
• Rubber dam placement is not possible.
• If the patient cannot afford the high costs.


14.1. Definition

Inlays, onlays and overlays are indirect tooth coloured restorations cemented into the prepared cavity in solid form restoring the form of the tooth. Indirect tooth coloured restorations are aesthetically and functionally high quality restorations. From this point of view the quality of indirect tooth coloured restorations are higher than direct composite fillings.

Three main types of tooth coloured solid fillings can be defined.

14.1.1. Inlay
Preparation doesn’t cover the top of the cusps. Preparation without cusp reduction.

14.1.2. Onlay
Preparation partially covers the top of the cusps. Preparation with cusp reduction.

14.1.3. Overlay
Overlay: Preparation covers all of the cusps. Preparation with total occlusal surface reduction.

14.2. Indications and contraindications

14.2.1. Indications
Loss of tooth structures caused by trauma or caries

• The orovestibular diameter of the cavity is between the half and one third of the distance between the cusps inlay is indicated. If the orovestibular diameter of the cavity is bigger than the half of distance between the cusps onlay is indicated.

• In the case of smaller cavity inlay could be indicated if the patient chose this type of restoration.

Rehabilitation of occlusal surface because of abrasion.

Abutment of fixed partial denture (FPD).

14.2.2. Contraindications
Extremely heavy occlusal forces (bruxism, clenching habits).

Short clinical crown (<5mm).
0.5 mm enamel is not exist around the margin of the cavity.

In the case of deep subgingival margins when proper isolation is not possible during the cementation.

Rubber dam isolation is not possible because of some reason (eg: intolerance, respiratory insufficiency, and allergy).

**14.3. Advantages and disadvantages of indirect tooth coloured restorations**

**14.3.1. Advantages**

Indirect ceramic and composite restorations have several advantages according to direct composite fillings.

- Indirect restorations have better physical properties because of ideal laboratory conditions during fabrication.
- Several materials (indirect composites, different type of ceramics) with good physical properties can be used for the fabrication of indirect inlays, and onlays.
- Indirect restorations have higher wear resistance. Wear resistance of ceramic restorations are higher than indirect composite restorations. Both of them have higher wear resistance than direct composite fillings.
- Reduced polymerization shrinkage and reduced polymerization stress characterize the indirect restorations. Because of these properties smaller marginal gap, smaller microleackage and less postoperative sensitivity characterize these restorations.
- According to the literature indirect restorations with adhesive cementation are able to strengthen remaining tooth structures.
- Indirect techniques usually provide more precise occlusal and proximal contacts, saving the periodontium and the TMJ.
- Ceramic materials are the most inert and biocompatible tooth colour restorative materials.
- Can save the dentist’s time.

**14.3.2. Disadvantages**

- Increased cost and time (more than one appointment).
- Technique sensitivity. Fabrication of a correct tooth coloured indirect restoration require very precise work of the dental technician and dentist and high level of operator skill.
- In the case of ceramic restorations the most often major failure is fracture because of the brittleness of ceramic material.
- The older ceramic restorations can wear severely the opposing restorations and dentition.
- Resin to resin bonding can be difficult in the case of high cross linked indirect composite resin restorations. Mechanical and chemical surface pre-treatment of the restoration can lead to better adhesion.
- Not enough well controlled clinical studies are available to estimate the long - term durability of these restorations especially the new generation ceramic restorations.
- Indirect ceramic restorations are difficult to repair.
- Difficult to polish the ceramic restorations intraoral.
- High costs.
14.4. Materials and methods of making indirect tooth coloured inlay onlay and overlay

14.4.1. Materials and laboratory process of making indirect ceramic inlays and onlays

Conventional feldspathic ceramic reinforced silicate ceramic and oxid ceramic can be used for fabrication of ceramic inlays (3.14.2. table). There is several laboratory processes for fabrication of indirect ceramic restorations. Three main types of processes are.

14.4.1.1. Conventional layering technique

After the tooth preparation and impression the technician fabricate the split cast master model. This model duplicated and purred with refractory investment (heat-resistant). This will be the refractory die. Ceramic added onto the die in several layers. Fired from layers by layers. The ceramic restoration is removed from the refractory die and seated onto the master die for finishing and polishing.

14.4.1.2. Heat pressed ceramic technique

There are several pressed ceramic techniques. (http://www.ivoclarvivadent.us/en/competences/all-ceramics/ips-emax-system-dentists/ips-e_max-lithium-disilicate)

After the tooth preparation and impression the technician fabricate the split cast master model. In this model the wax pattern of the restoration is made. Then the technician invests the wax pattern and burns it out. Hot ceramic ingot is pressed into the place of wax. It is followed by the final characterization (staining) finishing and polishing, restoration seating the restoration on to the master die.

Figure 3.244. Figure 1. – Additional silicon impression for making indirect ceramic onlay
Figure 3.245. Figure 2. – Split cast model for making ceramic onlay
Figure 3.246. Figure 3. – Split cast model in the articulator
3. Reconstructive dentistry

Figure 3.247. Figure 4. – Wax pattern of a pressed ceramic onlay with the casting tap

Figure 3.248. Figure 5. – Vacuum mixing of the investment material of the e.max ceramic system
Figure 3.249. Figure 6. – Investment of the wax pattern of the e.max pressed ceramic onlay
Figure 3.250. Figure 7. – Burn out of the wax pattern
3. Reconstructive dentistry

Figure 3.251. Figure 8. – e.max ceramic ingot during the pressing process

Figure 3.252. Figure 9. – e.max oven
Figure 3.253. Figure 10. – The pressed ceramic onlay before surface painting on the second model
Figure 3.254. Figure 11. – The pressed ceramic onlay prepared for the surface painting
Figure 3.255. Figure 12. – The surface characterisation of the e.max pressed ceramic onlay

Figure 3.256. Figure 13. – The palette of the stains used for the surface characterisation of the ceramic inlays
Figure 3.257. Figure 14. – Finishing and polishing of the pressed ceramic onlay after the characterisation

Figure 3.258. Figure 15. – The ready-made pressed ceramic onlay on the second model
14.4.1.3. Milling (CAD/CAM techniques)

Several CAD/CAM techniques are exist. (http://discovery.ucl.ac.uk/14476/1/14476.pdf)

Dental CAD/CAM is a process. During this process the model of a prepared tooth is scanned. This scanned data is then used to generate a reproducing design (CAD) which is used to generate a cutting path for manufacturing the restoration (CAM).

These techniques require ceramic blocks with grate mechanical and physical properties made under industrial circumstances. The CAD/CAM systems are consisting of three main parts: the scanner, software (CAD), and the milling unit (CAM). Type of the scanning (intraoral eg: CEREC; extra oral eg: PROCERA) the place of designing (dentist eg: CEREC; laboratory eg: PROCERA; centralized) the place of milling (dentist eg: CEREC; laboratory eg: EVEREST; centralized eg: PROCERA) and the type of the material (presintered – green or densed sintered ceramic blocks) can be different.

Table 3.2. Table 1. – Grouping the ceramic materials according to odontotechnological process

<table>
<thead>
<tr>
<th>Laboratory process (type of ceramic)</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
</table>
| **Layering technique**  (conventional feldspathic ceramic) | excellent aesthetic properties  
                       excellent adhesion properties to resin cement  
                       low elastic modulus (82 GPa) | technique sensitivity  
                       wider marginal gap formation  
                       weaker mechanical properties  
                       severe wearing rate of antagonist dentition |
| **Heat pressed technique**  (silicate ceramic)         | good mechanical properties                                   | good aesthetic properties                                                   |
Laboratory process (type of ceramic) | Advantages | Disadvantages |
--- | --- | --- |
| | good marginal integrity | | 
| | optimal adhesion properties to resin cement | | 
| | medium elastic modulus (95 Gpa *e.max*) | | 

**Milling process (oxid ceramic, reinforced silicate ceramic)** | excellent mechanical properties | unfavourable esthetic properties |
| | excellent marginal integrity | problematic adhesion to resin cement (Zirconia) |
| | | High elastic modulus (210 GPa *Zirconia* 418 Gpa *Alumina*) |

Table 3.3. Table 2. – Grouping the ceramic materials according to clinical usage

<table>
<thead>
<tr>
<th>Type of ceramic</th>
<th>System</th>
<th>Laboratory process</th>
<th>Clinical use</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>glass ceramic</strong> lithium – disilicate</td>
<td>IPS Empress 2 (Ivoclar Vivadent, Schaan, Liechtenstein)</td>
<td>Heat pressed</td>
<td>Crowns, anterior FPDP</td>
</tr>
<tr>
<td>(SiO₂–Li₂O)</td>
<td>IPS e.max Press (Ivoclar Vivadent)</td>
<td>Heat pressed</td>
<td>Onlays, 3/4 crowns, crowns, FPDP</td>
</tr>
<tr>
<td><strong>Leucite</strong> (SiO₂–Al₂O₃–K₂O)</td>
<td>IPS Empress (Ivoclar Vivadent)</td>
<td>Heat pressed</td>
<td>Onlays, 3/4 crowns, crowns</td>
</tr>
<tr>
<td>Optimal Pressable <strong>Ceramic</strong> (JenericPentron, Wallingford, Conn)</td>
<td>IPS ProCAD (Ivoclar Vivadent)</td>
<td>Heat pressed</td>
<td>Onlays, 3/4 crowns, crowns</td>
</tr>
<tr>
<td><strong>Feldspathic</strong> (SiO₂–Al₂O₃–Na₂O–K₂O)</td>
<td>VITABLOCS Mark II (VITA Zahnfabrik, Bad Sackingen, Germany)</td>
<td>Milled</td>
<td>Onlays, 3/4 crowns, crowns</td>
</tr>
<tr>
<td></td>
<td>VITA TriLuxe Bloc (VITA Zahnfabrik)</td>
<td>Milled</td>
<td>Onlays, 3/4 crowns, crowns</td>
</tr>
<tr>
<td></td>
<td>VITABLOCS Esthetic Line (VITA Zahnfabrik)</td>
<td>Milled</td>
<td>Onlays, 3/4 crowns, crowns</td>
</tr>
<tr>
<td><strong>Alumina</strong> Aluminium-oxide (Al₂O₃)</td>
<td>In-Ceram Alumina (VITA Zahnfabrik)</td>
<td>Slip-cast, Milled</td>
<td>Crowns, FPDP</td>
</tr>
<tr>
<td></td>
<td>In-Ceram Spinell (VITA Zahnfabrik)</td>
<td>Milled</td>
<td>Crowns</td>
</tr>
<tr>
<td></td>
<td>Synthoceram (CICERO Dental Systems, Hoorn,</td>
<td>Milled</td>
<td>Onlays, 3/4 crowns, crowns</td>
</tr>
</tbody>
</table>
### 3. Reconstructive dentistry

<table>
<thead>
<tr>
<th>Type of ceramic</th>
<th>System</th>
<th>Laboratory process</th>
<th>Clinical use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zirconia</td>
<td>In-Ceram Zirconia (VITA Zahnfabrik)</td>
<td>Slip-cast, Milled</td>
<td>Crowns, posterior FPDP</td>
</tr>
<tr>
<td></td>
<td>Procer (Nobel Biocare AB, Goteborg, Sweden)</td>
<td>Densely sintered</td>
<td>Veneers, crowns, anterior FPDP</td>
</tr>
<tr>
<td></td>
<td>Lava (3M ESPE, St. Paul, Minn)</td>
<td>Green milled, sintered</td>
<td>Crowns, FPDP</td>
</tr>
<tr>
<td></td>
<td>Cercon (Dentsply Ceramco, York Pa)</td>
<td>Green milled, sintered</td>
<td>Crowns, FPDP</td>
</tr>
<tr>
<td></td>
<td>DC-Zirkon (DCS Dental AG, Allschwil, Switzerland)</td>
<td>Milled</td>
<td>Crowns, FPDP</td>
</tr>
<tr>
<td></td>
<td>Denzir (Decim AB, Skelleftea, Sweden)</td>
<td>Milled</td>
<td>Onlays, 3/4 crowns, crowns</td>
</tr>
<tr>
<td></td>
<td>Procer (Nobel Biocare AB)</td>
<td>Densely sintered, milled</td>
<td>Crowns, FPDP, implant abutments</td>
</tr>
</tbody>
</table>

### 14.4.2. Materials and methods of making indirect composite inlays and onlays

Physical properties of composite resins used for indirect restorations (ICR) are better than resins used for direct restorations because the ICR is free of voids during the polymerization process and the resin matrix of it is well-polymerized during a final curing process. Dental laboratories devices for final polymerization polymerize the composite under pressure, vacuum, inert gas, intense light, and heat and/or combination of these conditions. These circumstances are not available in the mouth.

(http://www.dentalaegis.com/id/2006/12/indirect-composite-resin-systems-a-clinical-material-review)

The compound and physical properties of indirect composite resins are differ from resins used for direct composite filling. Two main generations of the indirect composite resins are exist. The first generations of ICR used for also semi direct and indirect techniques.

Main first generations indirect composite resins are: **SR Isosit, Coltene Brilliant, Visio-Gem** (ESPE), **Concept** (Ivoclar). There were several problems with these materials like: low wear resistance, often fractures, severe marginal gap formation, microleakage, debonding.

To solve these difficulties of the first generation ICR’s the second generations of ICR were developed. Compound of the material (size and ratio of the filler particles) and the type of laboratory process are different from the first generations of ICR materials leading to better mechanical properties. Main second generations indirect composite resins are: **Artglass Heraeus-Kulzer 1995**; **Belleglass HP** Belle de St. Claire 1996; **Sinfony** 3M ESPE; **TargisS Adoro** 1996 (Ivoclar Vivadent); **Soldix** Shofu; **Sculpture plus** (Pentron); **TESCERA ATL** (BISCO); **Paradigm MZ100** (3M ESPE); **Vita ZetaLC** (Vita Zahnfabrik); **Pearleste E2** (Tokuyama Dental Corp); **Estenia C&B** (Kuraray); **Gradia** (GC Corp).

(http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3010022/)

(http://www.worlddental-online.com/index.php?id=2720)

There are two main laboratory process for fabrication of indirect composite resin inlays and onlays.

### 14.4.2.1. Layering technique

After the tooth preparation and impression the technician fabricate the split cast master model. Composite is built up in layers onto this model. Each layer is polymerized with a short exposure with a light curing unit till the whole restoration is built up. After it, the restoration is coated with a special gel blocking out air and preventing formation of an oxygen-inhibited surface layer. During the final cure the restoration is insert into a
special device in which the composite can be polymerized under special circumstances (pressure, vacuum, inert gas, intense light, and heat). The complete composite restoration then seated onto the die for finishing and polishing.

14.4.2.2. Milling (CAD/CAM technique)

In these days CAD/CAM systems are not very often used for fabricating indirect composite restorations. First time usage of this method was permit of the Paradigm MZ100 composite block developed by 3M for the CEREC system. Mechanical properties of these composite blocks produced under industrial circumstances are better than the properties of ICR’s used for layering technique leading to durable and high quality restorations. (http://multimedia.3m.com/mws/mediawebserver?mwsId=66666UF6EVsSyXTl0xM_5xF6EVtQEVs6EVs6EVs6E666666--)

14.5. Comparison of indirect ceramic and composite restorations

As I detailed at the beginning in this chapter the indirect ceramic and composite inlay and onlays have several advantages against direct composite fillings. The 3.14.3 table summarize the attributes, advantages and disadvantages of indirect ceramic and composite inlays and onlays.

The main advantage of the indirect composite restorations according to the latest studies is the low elastic modulus (6-22 GPa). Because of this composite resin restoration has a greater capacity to absorb compressive forces and reduce the impact of the forces more (57%) than porcelain. Composites transmit less force to the underlying tooth structure protecting the adhesive bond at the tooth restoration interface. In the case of implant supported FPD. The indirect composite suprastructure transmit less forces to the implant - bone interface.

Table 3.4. Table 3. – Comparison of indirect ceramic and composite inlays

<table>
<thead>
<tr>
<th>Ceramic</th>
<th>Kompozit</th>
</tr>
</thead>
<tbody>
<tr>
<td>worse intraoral polishability</td>
<td>good intraoral polishability</td>
</tr>
<tr>
<td>high compression strength, low flexural strength</td>
<td>high flexural strength</td>
</tr>
<tr>
<td>ceramic is harder than the natural enamel can wear severe the antagonist dentition</td>
<td>not wear severe the antagonist dentition</td>
</tr>
<tr>
<td>wider micro gap formation at the margins of the restoration (different thermal expansion than the resin cement)</td>
<td>smaller micro gap formation at the margins of the restoration (similar thermal expansion than the resin cement)</td>
</tr>
<tr>
<td>difficult repairing in the mouth</td>
<td>good repairing properties</td>
</tr>
<tr>
<td>no potential for impact absorption (high elastic modulus)</td>
<td>greater capacity to absorb impact forces (low elastic modulus)</td>
</tr>
<tr>
<td>CAD/CAM technology is available excellent wear resistance color and gloss stability</td>
<td>CAD/CAM technology is not common color stability and wear resistance are questionable?</td>
</tr>
<tr>
<td>widely used systems need proper surface pre-treatment (mechanical and chemical) to achieve good adhesion to resin cement</td>
<td>less common recently used systems need proper surface pre-treatment (mechanical and chemical) to achieve good adhesion to resin cement</td>
</tr>
</tbody>
</table>

(http://www.moderndentistrymedia.com/nov_dec2007/terry2.pdf)

14.6. Success rate of indirect ceramic and composite inlays and onlays. Often failures and options of correction

Several studies investigate the success rate of indirect and direct tooth coloured restorations. Based on the literature the average (10 years) survival rate of direct composite fillings is 73%; the indirect composite inlays and onlays is 88%, and the indirect ceramic inlays and onlays is 90%.
Major and minor failures can be separated.

In the case of major failures the restoration should be changed. In the case of ceramic inlays and onlays the most common major failures are the bulk fracture (caused by not enough tooth preparation), tooth fracture, root fracture, secondary caries.

In the case of minor failures the restoration is reparable. In the case of ceramic inlays and onlays the most common minor failures are smaller cracks and fractures at the margins of the restoration (caused by the different thermal expansion and lower wear resistance of the luting cement), debonding, if root canal treatment is needed after the restoration. Repairing of the ceramic restorations is not good and not easy.

Mode of the repair is to restore the form of the restoration with direct composite „filling”.

### 14.6.1. Steps of the repair of ceramic inlays

- Mechanical roughening of the involved surfaces (ceramic, tooth) with a diamond bur.
- Put on the rubber dam isolation.
- Etching the surface of the ceramic with 10% HF acid (2 minutes).
- Application of silane onto the surface of the ceramic (5 minutes).
- Etching and bonding of the tooth surface with 37% orthophosphoric acid.
- Application of the composite resin.
- Fotopolimerization.

Most often failures occurred in the case of indirect composite restorations are the fracture of the restoration, wearing the proximal and occlusal surface of the restoration leading to opened contact area, secondary caries. Repair of the composite inlay and onlays are easier than ceramic ones. The steps of the repair are nearly the same.

### 14.6.2. Steps of the repair of composite inlays

- Mechanical roughening of the involved surfaces (ceramic, tooth) with a diamond bur.
- Put on the rubber dam isolation.
- Application of silane onto the surface of the composite (5 minutes).
- Etching and bonding of the tooth surface with 37% orthophosphoric acid.
- Application of the composite resin.
- Fotopolimerization.

### 14.7. Making of indirect ceramic and composite inlays and onlays clinical steps and laboratory process

#### 14.7.1. Preparation

The main preparation rules are the same during preparation of ceramic and composite inlays and onlays.

Preparations for indirect tooth-coloured inlays and onlays basically are meant to provide adequate thickness for the restorative material and a passive insertion pattern. The main rule of the inlay preparation that after the preparation no undercuts are allowed. If to maintain this rule too much tooth structure should be removed leading to weak tooth structure the undercuts can be blocked with glasionomer cement or composite resin after removing the carious tooth structure.
Figure 3.259. Figure 16. – Blocked out the undercutted area with glasionomer cement

14.7.1.1. Rules of inlay preparation

• 6-12°divergtion of the axial walls (maintain the adequate retention and the path of insertion).
• Conversation of the mesio and distoaxiopulpal walls (maintain the path of insertion).
• At least 2 mm depth of the preparation (maintain the mechanical stability of the restoration).
• Minimum 2mm wideness of the isthmus (maintain the mechanical stability of the restoration).
• All margins should have a 90-degree butt-joint cavosurface angle (ensure marginal integrity and strength of the restoration).
• All line and point angles, internal and external, should be rounded (avoid stress concentrations in the restoration and tooth, reducing the potential fractures).
• Inside and margins of the preparation should be smooth and polished (ensure the precise marginal integrity of the restoration).
• Supragingival preparation margins (ensure the good isolation and cementation circumstances).
• The occlusal margin of the preparation shouldn’t be in direct contact of opposing occlusion (ensure the marginal integrity).
• At least 0,5 – 1,0 mm clearance at the axial and gingival margins of the restoration (ensure precise marginal and approximal integrity of the restoration).

Figure 3.260. Figure 17. – Schematic figure of the inlay preparation
14.7.1.2. Onlay preparation rules

If the wideness of the remaining cusp less than 2.0 – 2.5 mm the cusp reduction is needed.

The rules of the preparation are nearly the same with the following supplements:

- Cusp reduction: in the case of supporting cusp: 1.5 -2mm, in the case of functional cusp: 2.0 -2.5mm (ensure the mechanical stability of the restoration and the remaining tooth structure.)

- If it is needed 0.8 -1.5 mm width rounded chamfer with but joint (90°) margins, and with minimum 1.0-1.5 mm axial reduction (ensure the proper retention and the proper mechanical stability of the remaining tooth structure and the restoration).

Figure 3.261. Figure 18. – Schematic figure of the onlay preparation

14.7.2. Try in and correction

Preliminary inspection

- Check the fit of inlay/onlay on the die.
- Check the contacts, marginal integrity, potential fractures and micro cracks (with polymerization lamp).

Try-in

- Remove the provisional and thoroughly clean the preparations.
- Try-in restoration. Verify the fitting (marginal internal) and shade.
- Check and adjust the proximal portion of the restoration (with soft diamond, ceramic polishing instruments, articulating paper, floss).
3. Reconstructive dentistry

- Do not check the occlusion before cementation.

14.7.3. Cementation

Put on rubber dam isolation.

Figure 3.262. Figure 19. – Rubber dam isolation put on before cementation of the inlay

14.7.3.1. Preparing the inner surface of the restoration for the cementation

- Clean the preparation cavity with pumice. Wash and dry.

- In the case of ceramic inlay etch the internal surface of the inlay/onlay with HF acid (5 %) for 5 minutes. Rinse and dry. Paint silane onto the etched porcelain to enhance adhesion of the resin. Allow to air-dry.

Figure 3.263. Figure 20. – The unglazed etched inner surface of the ceramic inlay
• In the case of composite inlays the inner surface of the restoration need mechanical (with soft diamond, sand-blasting) and chemical (special fluid) pre-treatment.

14.7.3.2. Preparing the tooth surface for the cementation

• Place teflon tape interproximally to protect adjacent teeth.

• Etch the tooth with 37% phosphoric acid for 15 seconds. Rinse and dry.
14.7.3.3. Cementation

- Apply bonding agent to etched tooth surface and to the restoration.
- Apply dual-cure resin luting agent into the cavity and/or to the restoration.
- Position the inlay/onlay until fully seated.
- Remove excess luting agent with a brush and with probe and with floss.
- Hold restoration in place during light-curing the resin cement.
- Remove the excess resin cement before full curing, but be careful not to pull cement out from the margins.
- Light cure for 40-60 seconds from several directions for 2-3 cumulative minutes.

Figure 3.265. Figure 22. – The cemented onlay before finishing and polishing

14.7.3.4. Finishing and polishing

- Check and correct the occlusion with articulation paper and soft diamonds.
- Finishing and polishing the restoration with special ceramic finishing and polishing burs and rubbers.
- Polishing of the proximal area with proximal polishing strips.
- Control X-Ray for checking the excess cement under the contact area.

Figure 3.266. Figure 23. – Controlling of the exactitude of the cementation on the RVG
• Finally check the marginal closing of the restoration with probe and with floss proximally.

Figure 3.267. Figure 24. – Cemented ceramic onlay in the mouth (tooth:14,15)
14.8. Summary

Ceramic inlays and onlays provide a good alternative for the aesthetic restoration of posterior teeth. The longevity of these restorations depends greatly on operator attention to detail.

Restoring premolars the second generations ICR inlays and onlays are good options.

In the case of molars, ceramic restorations (especially the new generation of silicate ceramics processed by heat pressed, or milled technique) provide a better solution because of better mechanical properties of the material.

The colour stability and optical properties which are more similar to natural teeth (eg: transparency) of the ceramic restorations are better than composite restorations. These are more aesthetic restorations.

15. 3.15. Ceramic veneers – Dora Ottoffy-Kende

15.1. Definition

The veneer is a thin layer of material placed over a tooth, to improve the aesthetic and functional properties of a tooth or to protect the tooth surface from damage. There are two main types of material used to fabricate a veneer: composit and ceramic. A composite veneer can be placed directly to the tooth surface in the mouth or can be made indirectly by the technician. The ceramic veneer always is done indirectly in a dental laboratory. Several ceramic materials and processes are exist to fabricate ceramic veneer.

15.2. Indications and contraindications of making ceramic veneers

15.2.1. Indications

Discoloration: single discolored teeth but not in very severe case.

Masking of little enamel defects.

Diasthema closing.
3. Reconstructive dentistry

Malpositioned Teeth: Little changing in tooth shape position size and surface appearance can be created with veneers if the patient doesn’t want the orthodontic treatment.

Poor Restoration: Teeth with multiple but not very big unaesthetic restorations on labial surfaces can be removed and restore the tooth with veneer.

Labial erosions and front teeth with cervical restorations.

Masking of the „age of the tooth”: During the aging process teeth usually become darker and weared. These changing can be corrected with veneers.

Weared teeth: veneer can change the shape and correct the function of a not very severe weared tooth.

Agenesis of the lateral Incisor: In this case if the canine are exist, the shape of the canine can be changed to a lateral incisor’s shape with veneer.

Peg lateral: The shape of the lateral incisor can be corrected with veneer.

Fractured teeth: the normal shape of the tooth can be restored with veneer.

Region caused by acid erosion (eg: because anorexia, bulimia nervosa) can be restored with veneer.

15.2.2. Contraindications

There is not enough enamel around the margins of the restorations leading to not enough bonding strength. If there is not enough enamel support crown is indicated.

The etching properties of the enamel are not good: eg: severe form of fluorosis.

Bad habits: bruxism, parafunctional movements.

Patients with poor oral hygiene: long margins of the restorations.

Patientes with poor periodontal health.

Endodontically treated teeth: a crown is indicated.

Teeth with extensive fillings: crown is indicated.

Extensive malocclusion: severe deep bite or edge to edge occlusion.

Morphological compromised teeth: if there is not enough surface of the tooth to maintain the adhesion of the veneer (eg: triangular shaped mandibular incisor).

Severe discoloration of the teeth.

15.3. Materials and laboratory process producing ceramic veneers

The veneers are usually made of ceramic and composit material. There are two types of esthetic veneers: partial veneers and full veneers. Localized defects and discolorations can be restored by partial veneers. Partial and full veneers can be made of composite with direct and indirect technique. The main indication of direct composite veneer is the fractured tooth of young patientes

Making of ceramic veneers only possible with indirect technique.

Direct and indirect veneers have a special indication fileds with several advantages and disadvantages.

15.3.1. The attributes of indirect veneer technique

It needs at least two appointments.
In the case of multiple veneering indirect technique usually used because of faster and more precise result.

The final result not strictly depends on the esthetic expertise of the dentist. Indirect veneers are typically more aesthetic.

Based on the latest literature indirect veneers are more long lasting restorations than direct veneers.

### Table 3.5. Table 1. – Advantages and diadvantages of making indirect ceramic veneers against composite veneers

<table>
<thead>
<tr>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Better color stability</td>
<td>Time consuming</td>
</tr>
<tr>
<td>Better bond strength to the enamel with</td>
<td>Very technique sensitivity</td>
</tr>
<tr>
<td>adhesive cementation technique</td>
<td></td>
</tr>
<tr>
<td>Less plaque accumulation because of glaze</td>
<td>Repairing the veneer is difficult</td>
</tr>
<tr>
<td>surface</td>
<td></td>
</tr>
<tr>
<td>Better wear resistance</td>
<td>Color correction of the ready made restoration is not possible</td>
</tr>
<tr>
<td>Luted veneer has good share and tensile</td>
<td>Usually needs more tooth structure removing</td>
</tr>
<tr>
<td>strengths</td>
<td></td>
</tr>
<tr>
<td>Less fluid absorption capacity</td>
<td>Ceramic is brittle, the technique requires very precise technical and</td>
</tr>
<tr>
<td></td>
<td>cementation work</td>
</tr>
<tr>
<td>Better esthetic properties</td>
<td>High cost</td>
</tr>
<tr>
<td>Temporary restorations not always required</td>
<td>The thickness and the color of the luting cement has a great affect on the</td>
</tr>
<tr>
<td>especially in the case of non prep veneers</td>
<td>final color of the restoration</td>
</tr>
<tr>
<td></td>
<td>Conventional veneer technique usually requires temporary veneers (time</td>
</tr>
<tr>
<td></td>
<td>consuming)</td>
</tr>
</tbody>
</table>

15.3.2. Materials for making ceramic veneers

Conventional feldspathic and ceramic reinforced silicate ceramic and oxid ceramic can be used for fabrication of ceramic inlays (3.14.2. table). There are several laboratory processes for fabrication of indirect ceramic restorations. Three main types of processes are: conventional layering technique, heat pressed ceramic technique, milling (CAD/CAM) techniques. All of the techniques and materials have advantages and disadvantages.

The conventional feldspathic ceramic processed by layering technique results excellent esthetic properties of the restoration. It requires very precise laboratory work with a great expert. The adhesive properties of etched ceramic veneers are very good if the preparation remain in the enamel. The main disadvantages of this method are that the feldspathic ceramic is very brittle with weak mechanical properties. The material is very transparent. It needs lots of tooth structure reduction leaving space for opaque layers to mask severe discolorations.

Restorations with great mechanical properties and very good marginal fitting can be made of different oxid ceramics processed by sintering (Alumina) or milling (Zirconia) process. The disadvantages of these materials are the very high elastic modulus and the great opacity. These restorations are very opaque and very white (Zirconia). These restorations have worse adhesive properties.

Very precise ceramic veneers with good marginal integrity, good mechanical and esthetic properties are made of the latest developed reinforced silicate ceramics processed by milling or heat pressed technique.

The latest system are already eliminated the problem regarding to the high transparency of the material (IPS Empress) with bad masking effect. Ceramic ignots with different transparency are already exist. These materials are the best for making ceramic veneers. (http://www.ivoclarvivadent.us/en/competences/all-ceramics/ips-emax-system-dentists/ips-e_max-lithium-disilicate)
15.4. Options of preparation of ceramic veneers

15.4.1. Classification according to preparation depth

We can define non prep veneers without preparation, Tenuia veneers with minimal (0.3-0.5 mm) preparation and conventional veneers with conventional (0.5-0.9 mm) preparation. All of the preparation types can be used in proper indications. The chosen preparation type depend on he desired tooth color, tooth shape and position. A veneer requires about 0.2 - 0.3 mm thickness for each shade change. For example, to change the tooth color from A3 to A0 requires about 0.6-0.9 mm thick veneer. A recent study shows that 0.3-mm-thick veneers showed some cracking during polymerization when they wrapped around the incisal edge. Based on these data veneers prepared with incisal wrapped should be at least 0.5 mm thick.

In case all of the preparation type it is important that the preparation should remain in the enamel. Because dentin has lower modulus of elastic. It is more flexible than ceramic and than enamel. It bends more under a given load, causing in the veneered porcelain higher tensile and shear stresses leading microgap and finally debonding.

15.4.1.1. Non-Prep veneers

Indications of non-prep veneers are very little. This type of restoration is indicated in the case of minimal shape and color change is required if the restored tooth is a little under contoured, avoiding overcontouring casuing gingivitis and unesthetic result. Main putative advantage of non-prep veneer is the reversibility of fit. In the practice removing of fixed non prep veneer is very difficult without enamel injury. Debonding is more often occur because the upper layer of the enamel has high fluoride content ledaing to difficulties regarding to etching ability of the enamel.

There are several non-prep veneer systems on the market (Lumineers, Durathin, Vivaneers).

15.4.1.2. Thin (Tenuia) veneers

Development of new ceramic materilas and process enables to use the thin veneers safely and successful. Thin veneers are indicated in the case of moderate shape and color change required.

15.4.1.3. Conventional veneers

These are indicated in the case of severe shape and color change is required.

15.4.2. Classification according to preparation design

The preparation design is determined by the gingival, approximal and incisal extent of the preparation.

15.4.2.1. Advantages and disadvantages of the preparation design of the incisal edge

Table 3.6. Table 2. – Preparation design of the incisal edge

<table>
<thead>
<tr>
<th>Preparation design</th>
<th>Advantages</th>
<th>Disadvantages</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Window preparatio n</td>
<td>Incisal edge is not prepared Fracture resistance of the restoration is better</td>
<td>Weaken incisal edge Margins are placed on visible surface Length correction is not possible Stress concentration mainly at the</td>
<td>Minimal shade and shape correction without length correction</td>
</tr>
</tbody>
</table>
## 3. Reconstructive dentistry

<table>
<thead>
<tr>
<th>Preparation design</th>
<th>Advantages</th>
<th>Disadvantages</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incisal edge preparation</strong>&lt;br&gt;Incisal edge preparation no incisal edge coverage</td>
<td>Easiest to manage&lt;br&gt;No need for provisionalization</td>
<td>Weaken incisal edge&lt;br&gt;Weaken incisal part of the veneer</td>
<td>Minimal shade and shape correction without length correction</td>
</tr>
<tr>
<td><strong>Horizontal preparation of the incisal edge</strong></td>
<td>Length correction is possible&lt;br&gt;Esthetic characterisation of the incisal edge is better&lt;brMargins of the preparation are placed at invisible surface&lt;brIncreased bonding surface</td>
<td>Requires more tooth structure removing&lt;brNeed provisionalization</td>
<td>Length shape and color correction</td>
</tr>
<tr>
<td><strong>Incisal wrapped Incisal edge preparation with palatinal chamfers</strong></td>
<td>Length correction is possible&lt;br&gt;Esthetic characterisation of the incisal edge is better&lt;brMargins of the preparation are placed at invisible surface&lt;brIncreased bonding surface&lt;brPositive seat during cementation&lt;brSpreading stress at the surface of the restoration&lt;brNot very severe stress concentration at the incisal edge of the restoration&lt;brBetter mechanical</td>
<td>Requires significant tooth structure removing&lt;brPalatinal chamfer should be positioned far from occlusion points&lt;brOtherwise fracture of the restoration is possible&lt;brNeed provisionalization</td>
<td>Length shape and color correction&lt;brIn the case of severe worn teeth&lt;brBucco-lingual width is thick</td>
</tr>
</tbody>
</table>
3. Reconstructive dentistry

<table>
<thead>
<tr>
<th>Preparation design</th>
<th>Advantages</th>
<th>Disadvantages</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>properties of the restoration</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 3.268. Figure 1. – Window preparation

![Figure 1. Window preparation](image1)

Figure 3.269. Figure 2. – Incisal edge preparation

![Figure 2. Incisal edge preparation](image2)

Figure 3.270. Figure 3. – Horizontal preparation of the incisal edge

![Figure 3. Horizontal preparation](image3)
3. Reconstructive dentistry

Figure 3.271. Figure 4. – Incisal edge preparation with palatinal chamfer

15.4.2.2. Preparation design of the gingival margin

It is the best if the gingival margin of the preparation is at or slightly below the free gingival margin. Impression and cementation procedure are easier to manage when the margin of the preparation is positioned supragingivally. Subgingival preparation design is only necessary if there is caries or cervical abrasion or severe discoloration under the gingival level, or if the previous preparation design was subgingivally. The bonding conditions are not ideal in the case of subgingival margins.

15.4.2.3. Preparation design of the approximal area

At the approximal region slightly chamfer is positioned. Breaking the contact usually is not necessary. Breaking the contact is only necessary when it is required by the shape and position correction. Slice preparation design
used in the case of diasthema closure. The proximal subcontact area only prepared when bigger color change is required.

**Figure 3.272.** Figure 5. – The proximal extension of the preparation without breaking the contact point

**Figure 3.273.** Figure 6. – The proximal extension of the preparation in the case of diatema closure

**Figure 3.274.** Figure 7. – Preparation of the proximal subcontact area when color changing is not needed

**Figure 3.275.** Figure 8. – Preparation of the proximal subcontact area when severe color changing is required
15.4.3. Conventional veneer preparation step by step

The aims of tooth preparation are:

• Providing enough thickness for the porcelain (maintain sufficient fracture resistance and avoid overcontouring of the final restoration).

• Providing a clear margin, leading to a definite finishing line (maintain the correct contour of the restoration at the margins).

• Maintaining the preparation within enamel as much as possible.

• Providing a finished preparation, which is smooth without any sharp internal line-angles, may causing stress concentration within the ceramic.

• Providing definite insertion pathway making proper seating of the veneer.

There are several methods to ensure the desired tooth reduction:

1. Freehand technique – usually led to under preparation and overcontoured restoration.

2. Use of depth cuts or grooves with surface staining. Usually lead to over preparation (~0.1 mm).

3. Use of silicone putty index based on the diagnostic way – up. Usually lead to overpreparation not as much as preparation with depth cuts.

Preparation steps

Labial preparation

The preparation of the buccal surface should be carried out according to the plane of the buccal surface of the incisors (which are convex). The axis of the preparation should follow the cervical, middle, and incisal plane of the buccal surface. Gingival reduction should be about 0.3-0.4 mm. Labial reduction should be about 0.4-0.9 mm.

For the preparation round and tapered diamond bur is used.

Incisal preparation

1.0 – 1.5 mm tooth structure removing from the incisal edge if this preparation design is indicated.

Palatal preparation

Slight palatal chamfer preparation if this design is indicated.

Placed the retraction cord.

Correct the finishing line.
Smooth the preparation with soft diamond and polishing rubbers.

Clean the proximal area with polishing strips.

15.5. Success rate of ceramic veneers and the influencing factors

Several studies investigated the success rate of direct and indirect composite and ceramic veneers. Based on the results of the latest studies the success rate of ceramic veneers after 10 years is ~92%. This value in the case of indirect composite veneers is ~90%, in the case of direct composite veneers is ~74%.

The most frequent failures are fracture, microleakage and gap formation and debonding.

15.5.1. Influencing factors of success rate

Thickness of the veneer

In the case of window preparation the minimal thickness of the ceramic which is required is 0.5 mm. (KH én)

Bonded surface of the tooth structure

In the case of bonding the veneer to the dentin surface (because of overpreparation) the low elastic modulus of the dentin leads to high tensile and shear stress at the veneer/tooth interface.

Isolation

Without proper isolation during cementation microleakage and debonding will occur.

Thickness of the Veneer/ thickness of the luting cement ratio (ideally >3)

Not proper fitting of the restoration results thick luting cement which interfere the proper stress distribution at the surface of the veneer and at the bonding surface.

Direction and magnitude of forces affect on the restoration

The palatal concavity and the incisal areas of maxillary anterior teeth are considered to be high stress concentration areas during tooth function. This incisal load is also affect on the incisal part of the ceramic veneer.

In the case of patients with parafunctional movements this loading is much more severe.

Preparation

Not proper preparation (little undercuts or too parallel preparation) can cause tensions into the restoration.

15.6. Making of ceramic veneer

15.6.1. Steps of imaging

1. Recording the general and dental medical history of the patient, extra and intraoral examination, recording the state of the teeth, professional oral hygienic treatment.

2. Planning the smile with computer imaging system (based on photographs). (KH én smile design fejezet)

3. Making of study model, bite registration, making of diagnostic wax up (based on previously planned smile) (KH Én 2. pont e fejezet)

4. Making of silicone putty index for the preparation based on the diagnostic wax up.

15.6.2. Preparation (conventional)

Figure 3.276. Figure 9. – Prepared two upper incisors for ceramic veneers because of diastema closure
15.6.3. Shade selection, bite registration

Prepared tooth shade and the desired shade should also be selected.

Photographs of the teeth with the shade tabs can help the technician determining the type and shade of ceramic required to achieve the desired result.

Final orientation of the incisal plane should be communicated clearly to the technician. The midline and the interpupillary or intercomissural line should be registered on the bite registration (e.g., with sticks).

15.6.4. Making of veneer with layering or heat pressed ceramic process based on conventional impression

15.6.4.1. Impression

Silicone or polyether impression material are used. Placing soft wax in the lingual embrasures before the impression will minimize tearing of the impression in these areas.

15.6.4.2. Steps of making temporary veneers based on diagnostic wax up

Making silicone impression of the diagnostic wax up extraorally.

Removing the excess from the impression which is interfere to put the silicone into the mouth.

Put the temporary acrylic resin into the silicone block. Put the silicone into the mouth.

Remove the silicone from the mouth and remove the excess temporary material from the undercuts before the total setting.

Shaping and clean the margins of the temporary veneers.

Fix the temporary veneers with spot etch technique or with eugenol free emporary cement.

In the laboratory the wax pattern of the final restoration (heat pressed technique) or the final ceramic restoration (layering technique) is made with the help of silicone index based on the diagnostic wax up.
15.6.5. **Making of ceramic veneer with CAD/CAM technique**

After the scanning of the diagnostic wax up the computer can designed the virtual 3D model of the final ceramic veneer restoration. The veneers can be milled out of a ceramic block.

(http://www.zfx-dental.com/sites/default/files/zfx_03_11_cad_veneers.pdf)

15.6.6. **Veneer assessment on casts**

Veneers are somewhat fragile, and should be handled carefully.

- Check accuracy of fit - the gingival margin is critical and must fit properly.
- Check inside of veneer for uniformity of etch. - The etched surface should look frosty.
- Check proximal contacts - The proximal contacts are checked with all veneers in place.
- Check marginal contours - The contours are evaluated particularly at the gingival area to avoid cementing an overconoured restoration.

15.6.7. **Try in**

Remove the temporary restoration and the temporary cement with polishing brush.

- Check the same parameters which were assessed on the cast.
- Evaluate the fit and esthetic using water solvable try in paste.
- Remove and clean the veneers with water and air and acetone.

- Clean the tooth surface with polishing brush.
- Isolation (Optra dam and sulcus retainer (size 00 or 0), teflon strip onto the neighboring teeth).
- Surface pretreatment of the ceramic.
- Surface pretreatment of the tooth.

Bonding of the restoration and the tooth surface.

15.6.8. **Cementation**

In the case of thin veneers it is the best solution to use light curing (not dual curing) luting material. It allows much more time for the placement of the restoration and to remove the excess.

15.6.8.1. **Steps of the cementation**

- Apply the cement to the preparation and the surface of the veneer with a brush or with plastic instrument.
- Seat the restoration with slight finger pressure or with a special sticky instrument and hold in place while the excess cement is removed with a brush and with floss.
- Short curing (1 sec from each side).
- Removing the excess with blade.
- Light curing till 60 seconds from each side.

**Figure 3.277. Figure 10. – Water solvent try-in paste used for the ceramic veneers**
15.6.9. Finishing and polishing

Finishing the margins with soft diamond and polishing the restoration with ceramic polishing rubbers. Approximately polishing the restoration with polishing strips.
15.7. Summary

Making of long lasting durable and esthetic ceramic veneer requires very precise imaging procedure and high quality work from the dentist and the dental technician. Several preparation technique are exist. The preparation which remain in the enamel with incisal overlap and palatal chamfer with slight supragingival chamfer at the gingival margin, considered the best solution regarding the durability of the veneer. The best materials for making ceramic veneer are the reinforced silicate ceramic processed by heat pressed or CAD/CAM technique.

16. 3.16. Smile design – Dora Ottoffy-Kende

16.1. Definition

The „smile design” is a modern and relatively new branch of the esthetic dentistry. During the procedure of smile design the specialist should make changes in soft and hard tissues affecting positively to the harmony of the face and smile of the patient and create more harmoniously general look of the patient leading to more harmonious personality. Creating an esthetics smile requires the integration of facial and dental composition.

The main goal of the smile design is to create a smile which is harmonized with the patient face, lips, teeth and personality. The prepared restoration based on the smile design requires very precise design and high quality artistic work from the dentist and the technician.

16.2. Principles of smile design

4 main factors should be considered during the procedure of the smile design.

16.2.1. Psychological factors

What the beauty is? Several cultural, social factors have an effect on the concept of beauty in a society. Beside of these several factors depending on the different personality of the people effects the concept of beauty or what is desired beauty according to the people opinion. Each people have a different opinion about the concept
of beauty. These opinions based on the previous experiences and sensations of the people heavily affected by
the personality of them. The „different type” of personalities should be considered during the initial part of the
smile design. This procedure is called typology of the personality.

16.2.2. Physiological factors

The general dentogingival health of the patient has a significant effect on the treatment plan of smile design.
Some general and dental diseases can be contraindications of the treatment. Asking the patient about the general
and dental health history, making the oral record, and x-rays, checking the vitality of the teeth, analyzing the
occlusion are should be the initial part of the smile design procedure.

16.2.3. Functional factors

The functional factors should also be considered during the procedure. The occlusion, articulation, functional
and parafunctional movements, movements of the temporomandibular joints and the speaking properties of the
patient should be checked before the treatment. The recognized irregularities should be treated during the smile
design procedure.

16.2.4. Esthetic factors

Within the esthetic factors macro mini and micro esthetics should be differentiated. All of it are investigated
during the proper phase of smile design procedure.

16.2.4.1. Macroesthetics

The concept macroesthetics means the harmony of the face. It deals with the relation of the face and the smile. It
can be investigated 1.5 m far from the patient. The evaluation of the macroesthetics based on different
measurements on the face by the help of proper reference points. These measurements should be carried out on the
special photo of the patients.

16.2.4.2. Miniesthetics

The concept miniesthetics means the harmony of the smile. It deals with the relation of the lips, teeth and the
gingiva in a rest position (M position) and during the smile (E position). It can be investigated about 60 cm far
from the patient. The frontal vertical and horizontal parameters of the smile are evaluated during the
miniesthetic analysis. These measurements should be carried out on the special photo of the patients.

16.2.4.3. Microesthetics

The microesthetics deal with the harmony of the tooth and the gingiva. It investigates the pattern and the texture
of the teeth and the gingiva. It can be investigated on intraoral macro photos. The microesthetic analysis
contains the intradental, interdental and the dentogingival analysis.

16.3. The procedure of the smile design

16.3.1. Typology of the personality

The patients can be classified into 4 main groups based on a simplified concept. This classification can be useful
for the evaluation of the most harmonious smile of the patient from the personality point of view. Four main
types of personality are exist based on this model.

16.3.1.1. Sangvinic

The sangvinic personality can be characterized by playful, emotional, active behavior. They usually not deal
with the details. They usually want to know the main reason of the treatment. They usually have sporty, sexy, or
sassy smile type. Natural colors (A2), nice, natural, long angled forms are the best for them.

16.3.1.2. Melancholic
The melancholic personality can be characterized by introversion, sensitivity, pessimism. These patients know what they want but cannot tell and define it clearly. The function is the most important for them. They want to know the details. They usually have a sophisticated smile with natural forms and colors.

16.3.1.3. Choleric

The choleric personality can be characterized by quick, temperamental, purposive behavior. These patients usually work in a leader position. They require that the dentist must do what they want. They deal with the details. They know and say what they want. They usually have a sophisticated smile with bleached colors and regular “nice” forms.

16.3.1.4. Flegmatic

The flegmatic personality can be characterized by calmness and internal balance. They don’t like long and difficult treatments and discomforts. They want to know just the substance of the treatment especially from the functional point of view. They usually have sportic or sassy smile. Extreme forms and colors (A4, B0) can be used.

16.3.2. Analysis of the functional, physiological and esthetic factors

Investigation of the personality is followed by the real chair side work contains the analysis of the functional, physiological and esthetic factors. This curriculum deals with the esthetic factors.

Materials and methods used for the analysis of the esthetic factors:

1. Extra and intraoral photographs: face and profile photos made 1 m far from the patient; mouth photos made about 60 cm far from the patient and macro photos of the teeth and the gingiva.

   Figure 3.280. Figure 1. – Face photo
Figure 3.281. Figure 2. – Profile photo
Figure 3.282. Figure 3. – Mouth photo
3. Reconstructive dentistry

2. Smile design computer software (computer imaging).

3. Diagnostic impressions and models, making of wax-up and mock-up models.

4. **Figure 3.284. Figure 5. – Wax-up model**
5. Making of temporary restorations according to the desired forms.

16.3.3. Principels of esthetic rules

16.3.3.1. Rules of macroesthetics (*harmony of the face*)

The harmonious face can be characterized by proper reference points on the face and by the ratio between the connective lines of these points. The main rules are the following:

**Horizontal dimension of the face**

a. The width of the face should be the width of five "eyes".

b. The distance between the eyebrow and chin should be equal to the width of the face.

c. ([http://www.jcd.org.in/article.asp?issn=0972-0707;year=2010;volume=13;issue=4;spage=225;epage=232;aulast=Bhuvaneswaran](http://www.jcd.org.in/article.asp?issn=0972-0707;year=2010;volume=13;issue=4;spage=225;epage=232;aulast=Bhuvaneswaran))

**Vertical dimension of the face**

a. The height of the face can be divided into three equal parts from the fore head to the eyebrow line, from the eyebrow line to the base of the nose and from the base of the nose to the base of the chin.

b. The full face is divided into two main parts, eyes are the midline.

c. The lower part of the face from the base of the nose to the chin is divided into two parts, the upper lip creates the one-third of it and the lower lip and the chin two-thirds of it.

**Figure 3.285. Figure 6. – The ratios of the vertical dimension of the face**
The ratios between the vertical and horizontal main reference lines of the face

During the smile design (width of the smile, the length and the width of the incisoros, vertical dimension of the occlusion) these ratios should be considered and harmonized.

Equal distances signed by the following lines: (blue lines on the 3.16.001.animation):

Distance between the mesial margins of the eyebrows, distance between the inner part of the eyes, distance between the nasal flaps, distance between the philtrum and the comissura, distance between the comissura and the side face, distance between the canines, distance between the connecting line of the chantus and the connective line of the nasal flaps.

Equal distances signed by the following lines: (red lines on the 3.16.001.animation): distance between the pupils, distance between the comissuras, distance between the pupil and the comissura, distance between the mentum and the middle point of the upper curvature of the upper lip.
3. Reconstructive dentistry

Animation 1. – Ratios o the vertical and horizontal reference lines of the face.

**Rules of the vertical lines of the face**

*Facial midline*

Facial midline is a line determined by the glabella, tip of the nose, philtrum, tip of the chin. In the case of harmony it is parallel with the dental midline and it is perpendicular to the main horizontal lines (bipupillar line, intercomissural line).

The facial midline could be straight, inclined and curved. During the smile design the inclination of the first incisors should follow the inclination of the midline and the interincisal line should be parallel with the facial midline.

**Rules of the horizontal lines of the face**

The *bipupillar line* and the *intercomissural line* are parallel with each other and perpendicular to the facial midline.
Shape of the face

The area bounded by the scalp, side face, and the tip of the chin is called face. Different type of face forms can be differentiated: a. round, b. oval, c. square, d. rectangle, e. trapezoid, f. tapering

During the smile design the shape of the face should be harmonized with the shape of the teeth.

Figure 3.286. Figure 7. – Shape of the face

Curvature of the lower lip

During smiling the lower lip can be straight, slightly and severe curved. During the smile design the incisal line should be parallel to the curvature of the lower lip, except in the case of extremely curved lower lip.

16.3.3.2. Rules of miniesthetic (harmony of smile)

During the evaluation of the harmony of the smile the position of the incisal edge of the upper incisors are investigated according to the upper and lower lip, to the soft tissues and to the main reference lines of the face.

Incisal line

The line which connects and goes through the first upper incisors’ edges. It is parallel with the horizontal reference lines (bipupillar, intercomissural). It is perpendicular to the facial midline.

Interincisal line (dental midline)

The midline refers to the vertical contact interface between two maxillary centrals.

It should be parallel to the facial midline. It is perpendicular to the horizontal lines especially to the intercomissural line and the incisal line. It should cover the facial midline. The maximum allowed discrepancy
is 2 mm. Greater than 2 mm discrepancy is esthetically acceptable if the dental midline is perpendicular to the bipupillar line.

The center of the philtrum should match the papilla between the centrals. If these two structures match and the midline is incorrect, then the problem is usually incisal inclination. If the papilla and philtrum do not cover each other, the problem is a true midline deviation.

**Incisal curvature**

It is a line which is go through the edge of the incisoros and the tip of the cusps of the posteriors. It could be straigth, slightly and severe curved and reversed. During smile design it should be harmonized with the curvature of the lower lip. Except in the case of reverse smile. In this situation the incisal curvature should be harmonized with the curvature of the upper lip.

**Figure 3.287. Figure 8. – Incisal line (blue), interincisal line (red), intercomissural line (yellow), incisal curvature (green)**

![Incisal curvature diagram]

**Tooth exposition at rest (M position)**

Viewable part of the upper first incisor at rest position of the upper lip. It decreases with the age. In a young patient it is 4-6 mm. In a middle aged patient 2-4 mm, in an old patient 1-2 mm of the upper incisors are viewable.

**Figure 3.288. Figure 9. – Tooth exposition at rest position in a young patient**

![Tooth exposition at rest image]

**Tooth exposition during smile (Eposition)**
Viewable part of the upper first incisor during smiling (cheese, E position of the upper lip). It decreases also with the age. The ratio is 75-100% in young patient; .50% in a middle aged patient and 10-20% in the old patientes. The smile can be created younger if the tooth exposition is increased.

**Figure 3.289. Figure 10. – Tooth exposition during smile (E position) in a young patient**

**Lip touching**

The relation between the lower and the upper lip during a slight smile. Smile without lip touching, smile with lip touching and smile behind the lower lip can be differentiated. The most harmonic is the smile with lip touching.

**Incisal profile**

The relation between the upper first incisors and the Vermillon fissure. In the case of harmony the upper incisors touch the lower lip at the Vermillon fissure. During the smile design it is important because of the evaluation of the bucco palatinal position of the incisors.

**Smile line**

The relation between the upper lips and the upper teeth and the gingiva. The smile line could be deep (the upper lip cut the upper teeth upper one third of these), average (the upper lip cut the papillas and the upper teeth at the level of half papilla), high (The lower lip cut the gingiva over the teeth, 1-2 mm gingiva is viewable during smile), gummy smile (more than 1 3-4 mm gingiva is viewable during smile). During the smile design the average type of smile line should be created in the case of harmony.

**Widness of the smile**

The number of viewable teeth from frontal view during cheese smile.

**Labial corridor**

Buccal corridor is dark space during smile formation between the corners of the mouth and the buccal surfaces of the maxillary teeth. It could be harmonious (1-2 mm), wide (>2 mm), and missing.

**Rule of perspective**

The viewable parts of the posteriors are gradually decreased toward the comissural part of the lips.

**16.3.3.3. Rules of micro esthetics (harmony of the teeth and the gingiva)**
3. Reconstructive dentistry

Rules of the teth harmony (intra and interdental relations)

Interdental features

The proportion between the width of the upper incisors and canines

In the case of a harmonious smile the proportions should follow the rules of the golden proportion.

Figure 3.290. Figure 11. – The proportion between the width of the upper incisors and canines

The vertical position of the upper incisors and canines

Regularly in the case of a harmonious smile the laterak incisors are 2.5 mm shorter, the canines approximately 0.5 mm shorter than the central incisors. There are some differentiations in the case for different smile types. Recognition and characterization of a smile is easy with different smile types. Four main smile types can be separated based on the vertical position of the central, lateral incisors and the canines and the premolars according to the Smylist® smile design. The different smile types can also be recognized with straight, slightly or severe curved or reverse incisal curvature.

1. Type I. (sofistic) smile

Each group of the teeth reach and touch the incisal curvature created by the edge of the incisors, the cuspid of the canines, premolars and molars.

Figure 3.291. Figure 12. – Sofistic smile

2. Type II. (sexy or attractive) smile

Between the central and lateral incisors a little step can be recognized (staircase effect). The lateral incisors are placed little (1-1.5 mm) apical from the incisal curvature.

Figure 3.292. Figure 13. – Attractive smile
3. Type III. (sporty) smile

The central and lateral incisors not reach the incisal curvatura, placed 0,5-1,0 mm apical from it. The staircase effect is also could occur. The canines and the premolars are the dominant in this smile type.

**Figure 3.293. Figure 14. – Sporty smile**

4. Type IV. (sassy) smile

This smile is characterized by diasthema, which usually occur between the central incisors or between al the teeth. The smile – according to the vertical position of the teeth – could be Snylist® type I–III.

**Figure 3.294. Figure 15. – Sassy smile**
Gingival zenith points

Zenith points are the most apical position of the cervical tooth margin where the gingiva is most scalloped. It is located slightly distal to the vertical line drawn down the center of the tooth. The zenith point of the lateral incisor is could be centrally located.

Inclination of the anteriors

Axial inclination compares the vertical alignment of maxillary teeth, to central vertical midline. Mesial inclination of the anteriors is increased from the central (which has nearly straight axis) to the canines.

Interdental contact area (ICA) and contact point (ICP)

ICA is defined as the broad zone in which two adjacent teeth touch. ICP is the most incisal part of the ICA. The ratio between the ICA and the other part of the tooth margin decreased from the central to the canine. As a general rule, the ICP moves apically, as we move from central to canine. The line drawn through the contact points is parallel with the incisal curvature.

Figure 3.295. Figure 16. – Ratio of the interdental contact areas and connecting line of the contact points

Intradental features

The size of the upper first incisors
Approximate length of the central should be 10-11 mm and the width is calculated accordingly so that the ratio falls between 75 and 80% because the width length ratio if the first incisor is about 79% in a harmonious smile.

If this ratio is differ from this the upper incisor can be define as long ar short incisor.

**Shape of the first incisors**

The shape of the first incisors should be harmonized with the shape of the face. It could be trapezoid, oval, or squared.

**Shape of the laterals**

These are the playful part of the smile. They provide individuality, are never symmetrical and influence the characteristic of the smile. The shape of them could be french type, sporty, and normal.

**Figure 3.296. Figure 17. – Normal lateral incisor**

![Normal lateral incisor](image1)

**Figure 3.297. Figure 18. – French type lateral**

![French type lateral](image2)
The shape of the canine

They play a critical point in creating a harmonious smile.

The canines are keep the junction between the anterior and posterior dental segments; hence, only the mesial half of the canine is visible from the frontal view when the patient smiles. The shape of the canine could be cuspid, rounded, and flat.

Principels of gingival harmony

Connecting line of the papilla

The papilla is the apical border of ICA. The connecting line of the papilla is paralell to the incisal curvature and the connecting line of the contact points.
3. Reconstructive dentistry

The relation of the gingival zenith points

The zenith points of the lateral incisors can reach the line drawn through the zenith points of the canines and the first incisors (American type) or it could be placed a little bit (1.25 mm) incisally from it (European style).

16.3.4. The main parameters of the smile

The anatomical structure of the face should be considered during the smile design. The advantages of the Smylist® smile design that the genetical smile (nomenclatura of dr. Csillag Mária) can be detected based on the evaluation of the characteristic feature of the face even if the teeth are missing.

The five main parameters of the smile which influence the esthetic result are the following according to the rules of the Smylist smile design:

- The shape of the central incisors.
- The smile type (the vertical dimension of the anterior teeth related to each other).
- The type of the incisal curvature.
- The inclination of the premolars and molars.
- The incisal characteristic of the incisors.

During the refinement of the shape system of the smile the other determining factors are the following:

- The shape of the lateral incisors.
- The character of the canines.
- The orovestibular dimension of the anteriors.

16.3.5. The procedure of the smile design from step by step

Based on the rules mentioned above the first step is the designing of the smile.

- Based on the designed smile making of wax up models.
- Based on the wax-up modell making of silicone index.
- Making of temporary restoration, or mock-up modells with the help of the silicone index (after the required preparations).
- Created the final restoration with the following techniques.

16.3.5.1. Recontouring technique

This is a special minimal invasive technique which works with the recontouring and polishing of the teeth. For this procedure the dentist use fine finishing and polishing diamonds and polishing discs. The goals of the procedure are the following: polishing of the overcontoured forms, rounding the heavy angles, smoothing the fractured enamel margins, decresion of the disharmony caused by crowded teeth.

16.3.5.2. Direct build up of the teeth according to the rules of the „natural layering concept”

This is also a minimal invasive technique. For this procedure the dentist use special composite materials, fine finishing and polishing diamonds and polishing discs. This technique is good for building up small fractures of
the teeth, closing diastemas, corrigation of the length and the shape of the teeth, and corrigation of the position of slightly covered teeth.

16.3.5.3. Making of indirect veneers and crowns

If more severe shape correction is needed the creation of the designed smile is carried out with indirect restorations (usually full ceramic restorations).

16.3.5.4. Orthodontics

The smile design based orthodontic treatment can reach a much more esthetic result regarding to the incisal curvature, the vertical position of the anteriors, the inclination of the teeth with the good positioning of the brackets.

16.4. Summary

Figure 3.299. Figure 20. – Patient before smile design
Figure 3.300. Figure 21. – Patient after smile design
17. 3.17. Classification of periodontal diseases – Ivan Mandel

17.1. Diagnosis of periodontal diseases

17.1.1. Medical history

The first step towards a proper diagnosis is taking medical history. On the one hand, it helps us to identify systemic risk factors of periodontal diseases. However, physical examination may cause bacteraemia and bleeding therefore the pretreatment of patients is necessary in certain conditions.

Table 3.7. Table 1. – Medical conditions, their risks and pretreatment of patients

<table>
<thead>
<tr>
<th>MEDICAL CASE HISTORY</th>
<th>RISKS</th>
<th>PRETREATMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>bleeding disorder</td>
<td>heavy bleeding that is difficult to control</td>
<td>consultation with a hematologist (factor or thrombocyte replacement)</td>
</tr>
<tr>
<td>anticoagulant therapy (coumarins)</td>
<td>heavy bleeding that is difficult to control</td>
<td>consultation with an internist to switch temporarily to LMWH treatment (INR below 1.7 is ideal for treatments)</td>
</tr>
<tr>
<td>acute infectious disease (upper respiratory tract, herpes, etc.)</td>
<td>cross infection</td>
<td>postpone dental treatment until recovery</td>
</tr>
<tr>
<td>chronic infectious disease (viral hepatitis, tuberculosis, etc.)</td>
<td>cross infection</td>
<td>additional precautions, schedule the patient at the end of the day</td>
</tr>
<tr>
<td>Bisphosphonate therapy</td>
<td>bisphosphonate induced osteonecrosis especially when administered intravenously</td>
<td>antibiotic prophylaxis</td>
</tr>
</tbody>
</table>
### MEDICAL CASE HISTORY

<table>
<thead>
<tr>
<th>RISKS</th>
<th>PRETREATMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>immunosuppressed state (chemotherapy, organ transplant recipient, etc.)</td>
<td>bacteremia may lead to infection elsewhere in the body</td>
</tr>
<tr>
<td>circulatory problems causing turbulent blood flow (heart valve insufficiency, AV shunt, Cimino fistula, etc.)</td>
<td>bacteremia may lead to infection elsewhere in the body</td>
</tr>
<tr>
<td>orthopedic and cardiovascular implants (artificial joints, stents, artificial heart valves and blood vessels)</td>
<td>bacteremia may lead to infection of the implant</td>
</tr>
</tbody>
</table>

Protocol of antibiotic prophylaxis:

- First choice is Penicillin derivative, 2000mg 1 hour before treatment,
- or Clindamycin, 600 mg 1 hour before treatment,
- or Azithromycin 500 mg 1 hour before treatment.

### 17.1.2. Physical examination

#### 17.1.2.1. Periodontal probes

Physical examination of the periodontium is performed using periodontal probes. Their common characteristic is that they have blunt tips and millimeter markings.

#### Table 3.8. Table 2. – Types of periodontal probes

<table>
<thead>
<tr>
<th>PROBE NAME</th>
<th>DESCRIPTION</th>
<th>INCREMENTS (mm)</th>
<th>CLINICAL USE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Williams</td>
<td>rounded cross-section, 10 mm long, no markings at 4 and 6 mm</td>
<td>1-2-3-5-7-8-9-10</td>
<td>measurement of probing depth, recession and clinical attachment level</td>
</tr>
<tr>
<td>Goldman-Fox</td>
<td>rectangular cross-section (flat), otherwise similar to the Williams probe</td>
<td>1-2-3-5-7-8-9-10</td>
<td></td>
</tr>
<tr>
<td>UNC (University of North Carolina)</td>
<td>rounded cross-section, 15 mm long, sections between 4-5, 9-10 and 14-15 are colored</td>
<td>1-2-3-4-5-6-7-8-9-10-11-12-13-14-15</td>
<td></td>
</tr>
<tr>
<td>CPITN (WHO)</td>
<td>rounded cross-section, ball-end, colored band from 3.5 to 5.5, marks at 8.5 and 11.5 mm</td>
<td>3.5-5.5-8.5-11.5</td>
<td></td>
</tr>
<tr>
<td>Nabers (furcation probe)</td>
<td>rounded cross-section, curved, colored bands between 3-6 and 9-12 mm</td>
<td>3-6-9-12</td>
<td>measurement of furcation involvement</td>
</tr>
</tbody>
</table>

Figure 3.301. Figure 1. – Periodontal probes (from left to right: Williams, WHO, UNC 15, Goldman-Fox, Nabers). Source: (http://odeicohort10.wikispaces.com/file/view/Periodontal%20Probes.jpg/348192944/Periodontal%20Probes.jpg)
17.1.2.2. Periodontal probing

The probe is inserted into the periodontal sulcus parallel to the long axis of the tooth while the tip of the probe is held against the tooth surface.

**Figure 3.302. Figure 2. – Proper positioning of a periodontal probe**

The probe is inserted applying gentle pressure until resistance occurs (0.2 N/mm² - equal to the pressure to blanch a fingernail with a probe). Conventional or manual probes are the first generation of periodontal probes. The need for standardized measurements however led to the development of pressure-sensitive (second generation) and electronic probes (third generation – Foster Miller probe, Florida probe, Interprobe). Fourth and fifth generation probes are capable of 3D and non-mechanical (e.g.: ultrasound waves) measurements respectively and are currently under development. The probe is moved with a walking stroke: 1 mm bobbing movements without taking the probe out of the sulcus and lateral movements in 1 mm increments.

**Video 1. – Periodontal probing**

Measured values are recorded for 6 sites around each tooth: mesiovestibular-vestibular-distovestibular-distoooral-oral-mesiooral.

**Figure 3.303. Figure 3. – Measurement sites around the tooth**
Each of these sextants is characterized by the deepest reading in that sextant. The sequence of recording follows the above mentioned order of sextants. Only whole numbers are recorded, readings are rounded to the next higher whole number. The following parameters are recorded during probing.

### Table 3.9. Periodontal parameters recorded with the probe

<table>
<thead>
<tr>
<th>PERIODONTAL VALUES and their ABBREVIATIONS</th>
<th>DEFINITION</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Probing (pocket) Depth - PD</td>
<td>distance in mm from the basis of the sulcus (pocket) to the gingival margin</td>
<td>true pocket: epithelial attachment is apical to the cemento-enamel junction (CEJ), pseudo pocket: attachment is at the level of CEJ, but gingival margin is in a more coronal position compared to normal</td>
</tr>
<tr>
<td>Recession - R</td>
<td>distance of gingival margin from CEJ in mm</td>
<td>recessions are more precisely described by the Miller classification</td>
</tr>
<tr>
<td>Clinical Attachment Level - CAL</td>
<td>distance in mm from the basis of the sulcus (pocket) to the CEJ (CAL=PD+R), above the clinically tolerable probing depth (0-3 mm)</td>
<td>Derived value from PD and R. 1-2 mm: mild 3-4 mm: moderate 5 mm or more: severe</td>
</tr>
<tr>
<td>Bleeding On Probing - BOP</td>
<td>if bleeding occurs in 15 seconds after probing than its value is 1, and the value is 0 if no bleeding is detected</td>
<td>the most important indicator of disease activity</td>
</tr>
<tr>
<td>Furcation involvement</td>
<td>probing depth of the furcation in relation to the vestibulo-oral width of the tooth Grade I.: can be probed up to 1/3 of the tooth Grade II.: can be probed between 1/3-2/3 of the width Grade III.: through-and-through lesion</td>
<td>measured with a special furcation probe</td>
</tr>
<tr>
<td>Mobility</td>
<td>looseness of the tooth in horizontal and vertical direction in mm I.: 0.2-1mm horizontal mobility II.: horizontal mobility greater than 1 mm III.: vertical mobility</td>
<td>measured tooth is held between the handles of two hand instruments</td>
</tr>
</tbody>
</table>

### 17.1.2.3. Periodontal charts

Measured values are recorded in periodontal charts. A detailed chart represents all the measured values which is advantageous. The disadvantage is however that the recording is relatively time consuming and the massive amounts of data are difficult to analyze.

Figure 3.304. Figure 5. – A detailed periodontal chart. Source: (http://www.periodontalchart-online.com/uk/)
Periodontal indices are easier to record, however they do not represent all periodontal values at once. Therefore these are used to screen large populations for epidemiological studies.

Table 3.10. Table 4. – Periodontal indices
### 3. Reconstructive dentistry

#### plaque indices

<table>
<thead>
<tr>
<th>INDEX</th>
<th>DESCRIPTION</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral Hygiene Index - Simplified (OHI-S) (Green Vermillion)</td>
<td>Measurement of the amount of plaque and calculus on vestibular surface of teeth 16,11,26,31 and lingual surface of teeth 36,46 on a scale from 0 to 3 Average of the recordings gives plaque index (PI-S) and calculus index (CI-S) OHI-S = PI-S + CI-S</td>
<td>Plaque in the gingival third of the crown is more relevant regarding pathogenicity. Thickness of plaque correlates better with pathogenicity than with the extent. Stained plaque is easier to measure</td>
</tr>
<tr>
<td>Silness-Löe Plaque Index (PI)</td>
<td>Measurement of the thickness of plaque in the gingival third of the crowns of teeth 16,124,36,32,44 on a scale from 0 to 3</td>
<td></td>
</tr>
<tr>
<td>Quigley-Hein Plaque Index</td>
<td>Measurement of the extent of plaque on front teeth on a scale from 0 to 5 after plaque staining</td>
<td></td>
</tr>
<tr>
<td>O’Leary Plaque Control Record</td>
<td>Percentage of surfaces covered by plaque. Measured after plaque staining</td>
<td></td>
</tr>
</tbody>
</table>

#### Gingival indices

<table>
<thead>
<tr>
<th>INDEX</th>
<th>DESCRIPTION</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Löe-Silness Gingival Index (GI)</td>
<td>Measurement of signs of inflammation around all teeth on a scale of 0 to 3</td>
<td>Bleeding index is most commonly used</td>
</tr>
<tr>
<td>Bleeding On Probing (Ainamo-Bay)</td>
<td>Dichotomic measurement of bleeding that occurs in 15 seconds following probing. Percentage of bleeding surfaces is the bleeding index</td>
<td></td>
</tr>
<tr>
<td>Papilla Bleeding Index (Mühlemann)</td>
<td>Measurement of bleeding from the interdental papillae 20-30 seconds after probing on a scale of 0 to 4</td>
<td></td>
</tr>
</tbody>
</table>

#### Periodontal indices

<table>
<thead>
<tr>
<th>INDEX</th>
<th>DESCRIPTION</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Russell Periodontal index</td>
<td>Measurement of signs of inflammation and pocket depth (values: 0-1-2-6-8)</td>
<td>They are now replaced by the detailed periodontal chart</td>
</tr>
<tr>
<td>Ramfjord Periodontal Index</td>
<td>Measurement of inflammation and attachment loss on a scale from 0 to 6</td>
<td></td>
</tr>
</tbody>
</table>

#### Index investigating the need for treatment

<table>
<thead>
<tr>
<th>INDEX</th>
<th>DESCRIPTION</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Community Periodontal Index of Treatment Needs (CPITN)</td>
<td>Measurement of probing depth, bleeding, and plaque/calculus on a scale from 0 to 4</td>
<td>Does not measure attachment loss</td>
</tr>
</tbody>
</table>

### 17.1.3. X-ray examination

If attachment loss is measured on a chart, an x-ray examination of the periodontium is necessary as well. Besides OPG, intraoral periapical radiographs are produced using paralleling or bitewing techniques to get an optimal projection of periodontal structures. Digital subtraction radiography is suitable for long term follow-up of changes in bone density and morphology. A CBCT scan is not recommended as a primary diagnostic method, however it might be useful in the examination of lesions that are difficult to assess on conventional radiographs (buccal and lingual bone plate, intrabony defects).

### 17.1.4. Laboratory tests

Laboratory tests are useful in the identification of specific microorganisms and/or genetic risk factors of periodontal diseases in order to establish a more accurate diagnosis.

### Table 3.11. Table 5. – Laboratory tests

<table>
<thead>
<tr>
<th>INDICATION FOR EXAMINATION</th>
<th>TYPE OF EXAMINATION</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>If hematological disorder is suspected in the background of the disease (e.g.: leukemia, granulocytopenia)</td>
<td>Full blood test</td>
<td>Detailed hematological examination of the patient is required if any alterations are detected</td>
</tr>
<tr>
<td>If a specific microorganism is suspected in the background of the disease or the</td>
<td>bacterial culturing</td>
<td>Obtaining and transporting live bacteria may be difficult and almost 50% of oral</td>
</tr>
</tbody>
</table>
### 3. Reconstructive dentistry

#### Table: Examination Indications and Methods

<table>
<thead>
<tr>
<th>INDICATION FOR EXAMINATION</th>
<th>TYPE OF EXAMINATION</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>efficiency of the treatment needs to be measured or before implant placement</td>
<td>Microbiological enzymatic reaction (BANA test)</td>
<td>Benzoyl-DL-arginin-naphthylamide (BANA) is enzymatically degraded by Tannerella forsythia, Treponema denticola and Porphyromonas gingivalis. The breakdown produces a color reaction (blue). Advantages: easy, fast, cheap, chairside. Disadvantage: only these 3 bacteria can be detected (e.g.: PerioScan®)</td>
</tr>
<tr>
<td></td>
<td>Analysis of bacterial DNA</td>
<td>Bacterial DNA fragments can be detected from plaque samples using PCR technique. It is very accurate and sensitive, chairside test is available (e.g.: Omnimed®)</td>
</tr>
<tr>
<td></td>
<td>Immunological tests</td>
<td>Bacterial proteins can be detected from plaque samples using ELISA technique. Advantages and disadvantages are the same as for the PCR test. Chairside version is available (e.g.: EvaluSite®)</td>
</tr>
<tr>
<td>If decreased immune response of the patient is suspected in the background of the disease</td>
<td>e.g.: examination of interleukin-1 gene polymorphism</td>
<td>IL-1 gene polymorphism leads to hyperreactive monocyte phenotype (altered monocyte function). This can be detected from a mucosal brush cytology sample with PCR technique. Chairside version is available (e.g.: PST®)</td>
</tr>
</tbody>
</table>

### 17.2. Classification of periodontal diseases

The classification of periodontal diseases has changed several times during the past decades. The currently used system was developed by the International Workshop For a Classification of Periodontal Diseases and Conditions in 1999 with the following categories.

#### 17.2.1. Gingival diseases

Most gingival diseases are of inflammatory origin (gingivitis). Gingivitis only affects gingival tissues and never causes periodontal attachment loss. However, gingivitis might occur on teeth that previously underwent attachment loss. The main subcategories of gingivitis include **dental plaque-induced gingivitis** which is more common and might progress to periodontitis and **non-plaque-induced gingivitis** which can be of fungal, viral, specific bacterial, genetic or traumatic origin or may be related to systemic diseases or foreign body reactions.

**Figure 3.305. Figure 5. – Clinical appearance of chronic, plaque-induced gingivitis**
17.2.2. Chronic periodontitis

A dental plaque induced inflammatory disease that leads to progressive and irreversible periodontal attachment loss (periodontitis). It has a chronic course and the extent of attachment loss is always consistent with the amount of dental plaque and local irritating factors. It has no systemic background. It usually occurs in adulthood (over the age of 35), its localized form affects less than 30% of teeth, while in its generalized form more than 30% of teeth are involved.

Figure 3.306. Figure 6. – Clinical manifestation and x-ray of generalized chronic periodontitis
17.2.3. Aggressive periodontitis

Aggressive periodontitis is characterized by a more rapid attachment loss as compared to chronic periodontitis and the amount of dental plaque is never consistent with the amount of tissue loss. Minor defects in immune functions (e.g.: hyperreactive monocyte phenotype) of the individual lead to this condition while other organs are not affected. The patient is otherwise healthy. Familial aggregation is characteristic. The localized form of the disease affects less than 30% of teeth (predilection sites are incisors and first molars) and it has a strong association with the presence of high numbers of Aggregatibacter actinomycetemcomitans. The generalized form affects more than 30% of teeth (without predilection site) and is caused by a mixed bacterial flora. Therefore these clinical forms can be considered as different disease entities.

Figure 3.307. Figure 7. – Clinical manifestation and x-ray of generalized aggressive periodontitis
17.2.4. Periodontitis as a manifestation of systemic diseases

Clinically it is similar to the manifestation of generalized aggressive periodontitis, but the underlying cause is always a more severe immune deficiency that also affects other organs such as in haematological disorders (e.g.: leukaemias, neutropenias) and genetic disorders (e.g.: Down syndrome, Papillon-Lefèvre syndrome, Chediak-Higashi syndrome) and acquired immune deficiencies (diabetes mellitus, HIV/AIDS).

17.2.5. Necrotizing periodontal diseases

An ulcerative necrosis of tissues that primarily affects the inter-dental papillae. It is characterized by a sudden onset and acute course occurring mainly in young adulthood with pain, swollen lymph nodes, low fever and halitosis. It usually affects only gingival tissues (acute necrotizing ulcerative gingivitis – ANUG) or it may progress to periodontal ligament and bone (acute ulcerative necrotizing periodontitis – ANUP).

Figure 3.308. Figure 8. – Clinical appearance of Acute Necrotizing Ulcerative Gingivitis (ANUG)
17.2.6. Abscesses of the periodontium

An abscess is a localized purulent inflammation of periodontal tissues. Abscesses are primarily classified according to their localization (gingival, periodontal, pericoronal). They can also be classified based on the course of the disease (acute, chronic), the number of abscesses formed (single – local cause, multiple – systemic cause) and the presence of prior attachment loss. Without attachment loss an abscess may be formed because of foreign body impaction, root fracture/resorption or altered tooth anatomy. Closure of the coronal part of the pocket and inadequate administration of antibiotics (without subgingival debridement) are common causes of abscess formation around teeth with prior attachment loss. Another important question is at which stage of the treatment an abscess occurs (before or after causal treatment, after correctional treatment, supportive treatment). These factors are important to assess tooth prognosis and treatment, therefore the establishment of a proper differential diagnosis is essential.

Figure 3.309. Figure 9. – A periodontal abscess on the attached gingiva originating from chronic periodontitis
17.2.7. Periodontitis associated with endodontic lesions

Subclasses of these lesions include: 1: **Primary endodontic lesions** with secondary periodontitis; 2: **Primary periodontal lesions** with secondary endodontic lesion; 3: **Combined lesions**, where endodontic and periodontal lesions occur simultaneously.

**Figure 3.310. Figure 10.** – A Primary Endo-periodontal lesion developed from periapical lesion of tooth 43
17.2.8. Developmental or acquired deformities and conditions

These are localized tooth-related or mucogingival factors that are not caused by dental plaque. However, these deformities and conditions are important modifiers of the susceptibility to periodontal diseases. **Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis** (tooth anatomic factors, dental restorations/appliances, root fractures, cervical root resorption and cemental tears), **mucogingival deformities and conditions around teeth** (gingival recession, lack of keratinized gingiva, decreased vestibular depth, aberrant frenum, gingival excess, abnormal color), **mucogingival deformities and conditions on edentulous ridges** (vertical/horizontal ridge deficiency, lack of keratinized tissue, soft tissue enlargement, aberrant frenum, decreased vestibular depth, abnormal color), **occlusal trauma** (primary, secondary).

Figure 3.11. Figure 11. – An aberrant frenum and lack of keratinized gingiva that led to gingival recession
18. 3.18. Microbiology and immunopathogenesis of the periodontal inflammation – Ivan Mandel

Research on experimental gingivitis (Löe, 1965) and research on host-parasite interactions (Page & Schroeder 1976, 1981) proved that the main causative factor of periodontal inflammation was bacterial plaque. Since then different plaque theories have been developed to explain the role of dental plaque.

Table 3.12. Table 1. – Plaque theories

<table>
<thead>
<tr>
<th>HYPOTHESIS</th>
<th>FIRST DESCRIPTION</th>
<th>PRINCIPLE</th>
<th>EXPLANATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-specific plaque</td>
<td>Miller (1890),</td>
<td>The amount of plaque determines pathogenicity, regardless of its composition</td>
<td>It states that all plaque can be pathogenic if present in a certain amount. But plaque accumulation however does not lead to attachment loss in all individuals, while in some cases severe attachment loss can be seen without significant amounts of plaque (aggressive periodontitis).</td>
</tr>
<tr>
<td>hypothesis</td>
<td>Löe (1965),</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Loesche (1976)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>specific plaque</td>
<td>Loesche (1976)</td>
<td>Composition of plaque determines pathogenicity</td>
<td>It states that the presence of certain bacteria leads to attachment loss, but periodontitis is usually not a result of an exogenous infection.</td>
</tr>
<tr>
<td>hypothesis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ecological plaque</td>
<td>Marsh (1991)</td>
<td>Interactions between bacteria and the host determine pathogenicity</td>
<td>The currently accepted theory. Changes in the amount and/or composition of plaque together with changes in host response lead to disease. Periodontitis is an opportunistic infection.</td>
</tr>
<tr>
<td>hypothesis</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Although more than 700 bacterial species live in the oral cavity and approximately 150 species of them are capable of colonizing subgingival areas, only a dozen of them can be considered as directly periodontopathogenic.

### Table 3.13. Table 2. – Periodontopathogenic bacteria

<table>
<thead>
<tr>
<th>BACTERIUM</th>
<th>CHARACTERISTICS</th>
<th>TYPICAL DISEASES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aggregatibacter actinomycetemcomitans</td>
<td>Gram negative, non-motile, saccharolytic, capnophil, rounded rod</td>
<td>aggressive periodontitis, mainly the localised form</td>
</tr>
<tr>
<td>Porphyromonas gingivalis</td>
<td>Gram negative, non-motile, asaccharolytic, anaerobic rod</td>
<td>aggressive periodontitis, mainly the generalized form</td>
</tr>
<tr>
<td>Tannerella forsythia</td>
<td>Gram negative, non-motile, anaerobic rod</td>
<td>deep pockets, active lesions, refractory periodontitis</td>
</tr>
<tr>
<td>Treponema denticola</td>
<td>Gram negative, motile, spiral shaped, anaerobic bacterium</td>
<td>ANUG, active lesions</td>
</tr>
<tr>
<td>Prevotella intermedia</td>
<td>Gram negative, anaerobic, short, rounded rod</td>
<td>pregnancy gingivitis, progressive chronic periodontitis</td>
</tr>
<tr>
<td>Prevotella nigrescens</td>
<td>Gram negative, anaerobic, short, rounded rod</td>
<td>generalized aggressive periodontitis and progressive chronic periodontitis</td>
</tr>
<tr>
<td>Fusobacterium nucleatum</td>
<td>Gram negative, anaerobic rod</td>
<td>ANUG, active lesions</td>
</tr>
<tr>
<td>Campylobacter rectus</td>
<td>Gram negative, motile, anaerobic, short rod</td>
<td>active lesions</td>
</tr>
<tr>
<td>Peptostreptococcus micros (Parvimonas micra)</td>
<td>Gram positive, anaerobic, asaccharolytic coccus</td>
<td>destructive, generalized periodontitis</td>
</tr>
<tr>
<td>Eikenella corrodens</td>
<td>Gram negative, facultative anaerobic, capnophilic, saccharolytic rod</td>
<td>active lesions, refractory periodontitis</td>
</tr>
<tr>
<td>Selenomonas spp.</td>
<td>Gram negative, saccharolytic, motile, curved rod</td>
<td>destructive periodontitis</td>
</tr>
<tr>
<td>Eubacterium spp.</td>
<td>Gram positive, anaerobic rod</td>
<td>destructive periodontitis</td>
</tr>
<tr>
<td>Streptococcus intermedius</td>
<td>Gram positive coccus</td>
<td>refractory periodontitis</td>
</tr>
</tbody>
</table>

Some bacteria that are not part of the healthy oral flora (Klebsiella spp, Enterobacter spp, Pseudomonas aeruginosa, Enterococcus spp.), also have an etiological role in periodontitis. Viruses (Epstein-Barr virus, Human Cytomegalovirus, Herpes simplex virus, Human Papillomavirus) may also contribute to the development of periodontitis through altering immune functions.

Periodontitis is always the result of a mixed bacterial infection, involving more than one species. Bacteria in the oral cavity do not exist in isolated, soluble form. They colonize subgingival areas as part of a community, forming a biofilm. The biofilm structure provides for a stronger attachment to tooth surfaces, a more effective nutritional supply, a more favourable environment (O₂ and CO₂ tension, pH) and a more effective protection against host antibacterial mechanisms. It’s a complex ecological system in which bacteria regulate each other through synergistic and antagonistic interactions.

The first step in biofilm development is the formation of the **acquired pellicle** (1) on tooth surfaces from salivary glycoproteins. Early colonizer bacteria (Gram positive cocci) attach to the pellicle first with reversible and later with irreversible **adhesion** (2). With the **multiplication** (3) of bacteria the biofilm thickens. Ecological changes in the thickening biofilm favour the **co-aggregation** (4) of new bacterial species, which leads to further ecological changes. The 3D structure of the biofilm is provided by an extracellular matrix (glucan, fructan, heteropolymers). At the end of this **maturation** (5) process the biofilm is mainly composed of Gram negative anaerobic species.

Six ecological complexes can be identified in subgingival dental plaque (Socransky, 1998). Most bacteria are part of one of the complexes while few species can be found outside the complexes.

**Figure 3.312. Figure 1. – Bacterial complexes in the biofilm**
During the growth and the maturation of the biofilm the occurrence of the complexes follows a specific spatial and chronological order. The early colonizers are the Actinomyces species, the Yellow-, Purple- and Green complexes. These attach to supragingival surfaces and can be found in relatively shallow pocket areas. Orange complex bacteria are the next to attach in deeper areas. The Red complex can be found in the deepest pockets and these bacteria are called late colonizers.

Figure 3.313. Figure 2. – Spatial structure of oral biofilm and interbacterial relationships
The increasing mass of plaque and the increase in the number of pathogenic bacteria in the plaque lead to gradual activation of antibacterial mechanisms of the host immune system.

**0th line of defense: saliva**

An addition to its washing, diluting and pH regulating effect saliva contains antibacterial compounds such as thiocyanate, lactoferrin, lysozyme, lactoperoxidase and secretory IgA.

**1st line of defense: gingival sulcus**

The sulcular epithelium is not only a passive barrier but it secretes antibacterial compounds (alpha- and beta-defensin, complement system) to the gingival sulcus and also actively participates in antibacterial mechanisms through the production of prostaglandins, leukotrienes, cytokines and chemokines. Langerhans cells are resident cells in the epithelium whereas PMN leukocytes are able to migrate through epithelial layers to the sulcus.

**2nd line of defense: connective tissue of the gingiva**

Several cells of the immune system are resident in the gingival connective tissue while morphological and permeability changes in blood vessels enable other immune cells to immigrate to gingival tissues.

**3rd line of defense: systemic immune protection**

The clonal selection and expansion of cellular components of adaptive immunity takes place in the regional lymphatic tissues.
Interactions between bacteria and the host immune functions lead to histological changes in the periodontium. These changes can be categorized into four distinct phases.

**Table 3.14. Table 3. – Stages of inflammation in the periodontal lesion**

<table>
<thead>
<tr>
<th>PHASE</th>
<th>TIME INTERVAL</th>
<th>HYSTOLOGICAL CHANGES</th>
<th>CLINICAL PICTURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial lesion (subclinical gingivitis)</td>
<td>2-4 days</td>
<td>dilation and winding of capillaries, elevated numbers of PMN leukocytes, increased amount of crevicular fluid</td>
<td>no difference from healthy gingiva</td>
</tr>
<tr>
<td>Early lesion ('acute gingivitis')</td>
<td>4-7 days</td>
<td>initial inflammation manifestations increase, emigration of macrophages and lymphocytes, invaginations of junctional epithelium, ulcerations of sulcular epithelium</td>
<td>swelling, redness, bleeding on probing, loss of tissue tone</td>
</tr>
<tr>
<td>Established lesion (chronic gingivitis)</td>
<td>14+ days</td>
<td>early lesion manifestations increase, plasma cells predominate, rete peg formation of the epithelium, increasing ulceration of epithelium, connective tissue breakdown</td>
<td>moderate/severe inflammation, bluish-reddish color, changes in consistency occur, spontaneous bleeding</td>
</tr>
<tr>
<td>Advanced lesion (periodontitis)</td>
<td>dependent upon host response</td>
<td>Continuation of changes in the established lesion, inflammation extends to periodontal bone and connective tissue, destruction of periodontal attachment</td>
<td>periodontal pocket formation, attachment loss, bone loss</td>
</tr>
</tbody>
</table>

Animation 1. – Development of periodontal inflammation.

Although certain bacteria produce factors that are capable of triggering direct tissue-damage, the main tissue destruction is paradoxically caused by host immune mechanisms. However, bacteria can distract the immune response, hide from it or inhibit it, therefore play an important role in the development of a pathologic immune reaction.

**Table 3.15. Table 4. – Virulence factors of periodontopathogenic bacteria**
<table>
<thead>
<tr>
<th>GOAL</th>
<th>FACTOR</th>
</tr>
</thead>
<tbody>
<tr>
<td>adhesion to tissues</td>
<td>fimbriae</td>
</tr>
<tr>
<td></td>
<td>pili</td>
</tr>
<tr>
<td></td>
<td>adhesins</td>
</tr>
<tr>
<td>colonisation, proliferation</td>
<td>inhibition of other bacteria (bakteriocin)</td>
</tr>
<tr>
<td></td>
<td>inhibition of inhibitory factors</td>
</tr>
<tr>
<td></td>
<td>establishment of food chain</td>
</tr>
<tr>
<td>inhibition, modification, distraction of</td>
<td>cell wall antigens</td>
</tr>
<tr>
<td>immune response</td>
<td>PMN chemotaxis inhibitor factor</td>
</tr>
<tr>
<td></td>
<td>leukotoxine</td>
</tr>
<tr>
<td></td>
<td>proteases that degrade immunoglobulins and</td>
</tr>
<tr>
<td></td>
<td>proteins of the complement system</td>
</tr>
<tr>
<td>tissue invasion</td>
<td>invasins</td>
</tr>
<tr>
<td>direct tissue damage</td>
<td>enzymes</td>
</tr>
<tr>
<td></td>
<td>collagenase</td>
</tr>
<tr>
<td></td>
<td>hyaluronidase</td>
</tr>
<tr>
<td></td>
<td>trypsin-like protease</td>
</tr>
<tr>
<td>bone resorption</td>
<td>LPS</td>
</tr>
<tr>
<td></td>
<td>components of the cell wall</td>
</tr>
<tr>
<td>toxic agents</td>
<td>butyric acid</td>
</tr>
<tr>
<td></td>
<td>propionic acid</td>
</tr>
<tr>
<td></td>
<td>indole</td>
</tr>
<tr>
<td></td>
<td>ammonia</td>
</tr>
<tr>
<td></td>
<td>hydrogen sulfide</td>
</tr>
</tbody>
</table>

Periodontopathic bacteria activate resident cells of the innate immune system mainly through the activation of Toll-like receptors (TLR). These receptors recognize characteristic pathogen associated molecular patterns (PAMPs) like lipopolysaccharide (LPS) in the cell wall or fimbriae. Some resident cells (epithelial cells, fibroblasts, Langerhans cells, dendritic cells, endothelial cells) only recognize, process and present antigens therefore they are called antigen presenting cells (APC). They also activate other cells via secretion of cytokines and chemokines. Other resident cells (macrophages, mast cells) and immigrating cells (monocytes, neutrophil granulocytes, NK cells) have effector functions as well and are directly antibacterial through phagocytosis and the secretion of cytotoxic agents. Parallel to the immediate and nonspecific antibacterial response of the innate immune system, the relatively slow activation of the cells of adaptive immunity begins. Clonal selection and expansion of T- and B lymphocytes leads to pathogen specific immune response. Innate and adaptive immune response develops simultaneously in time and space. The nature of the adaptive response largely depends on the differentiation of naïve T cells to effector cells: cellular immune response is regulated by Th1 cells and it is characteristic in stagnating lesions, while predominance of humoral immune response regulated by Th2 cells is seen in progressive lesions. Th17 cells that have an important role in osteoclastogenesis and subsequent bone resorption are also characteristic in progressive lesions. Regulatory T cells are responsible for the suppression of immune response.
The immune mediated destruction of periodontal tissues creates a niche for further bacterial invasion which leads to a more pronounced immune response therefore the periodontal host-pathogen relationship is best described by a circular model.

Figure 3.315. Figure 4.  – Model of the pathogenesis of periodontal inflammation (Offenbacher, 1996)
19. 3.19. The role of local and systemic risk factors in periodontitis – Ivan Mandel

19.1. Definition of a risk factor

Primary etiologic factors (causative factors) of periodontal diseases are bacteria. They are necessary and sufficient to initiate an inflammation. Risk factors contribute to the process initiated by causative factors through increasing the likelihood of disease occurrence or through modifying the speed and severity of disease development. These can be divided into two main subgroups: local and systemic factors. Local factors can be of natural origin or iatrogenic.

19.2. Local factors

19.2.1. Iatrogenic risk factors

Deficiencies in the quality of dental restorations or prostheses are plaque retentive factors. Overhanging margins of restorations, overcontoured crowns, contact areas of inappropriate size or position, lack of contact areas, insufficient width of embrasure areas under pontics, insufficient space provision for interdental papillae between crowns and pontics, insufficient space provision between extracoronal attachments and the marginal gingiva and inadequate RPD design increase dental plaque accumulation and make oral hygiene procedures more difficult.

Biological width is defined as the amount of soft tissue attached to the tooth surface above the crest of the alveolar bone. It is approximately 2mm, composed of ~1mm epithelial attachment and ~1mm connective tissue attachment. This 2 mm band of soft tissue is always maintained by the body: chronic iatrogenic irritation and the subsequent apical reposition of the epithelial attachment cause equal amount of bone resorption.

Fixed orthodontic appliances are also plaque retentive factors.

Figure 3.316. Figure 1. – An overhanging crown margin and drug induced gingival hyperplasia
Figure 3.317. Figure 2. – Condition after correction of the overhang and flap surgery. Oral hygiene is excellent

![Image](image1.png)

Figure 3.318. Figure 3. – Gingivitis around low quality class 5 fillings

![Image](image2.png)

Figure 3.319. Figure 4. – Plaque accumulation around overhanging buccal filling of tooth 44

![Image](image3.png)
19.2.2. Natural risk factors

19.2.2.1. Anatomical tooth abnormalities

Enamel pearls or cervical enamel projections are surface deformities often found in furcation areas of molar teeth. As epithelial attachment follows these irregularities of enamel development, gingival recession or attachment loss may occur. Pseudofurcation is a shallow groove along the buccal surface of the neck and root of maxillary incisors that often contributes to attachment loss.

Mesial and distal depressions are often seen on the cervical areas of the premolar teeth. These concave surfaces are almost impossible to clean with a toothbrush or with dental floss therefore the use of interdental brushes is recommended.

In microdontia diastemas often cause food impaction in the interdental region.

Figure 3.320. Figure 5. – Diastemas
Macrodontia leads to crowding of teeth.

19.2.2.2. Abnormalities in the position and number of teeth

Ectopia of teeth may lead to dehiscence, fenestration and crowding. Eccentric bite load can be a cause of trauma from occlusion.

Resorption of the interdental septum may be seen in crowding if the distance between the roots of neighboring teeth is narrower than 1.5 mm.

Figure 3.321. Figure 6. – Crowding of teeth makes it difficult to maintain proper oral hygiene
Partial anodontia is associated with the lack of contact areas, elongation of antagonistic teeth. It often leads to traumatic occlusion: widening of the periodontal ligament, increased mobility, drifting and migration of teeth. Trauma from occlusion alone does not result in attachment loss, however it may enhance the rate of progression of attachment loss around teeth with periodontitis.

19.2.2.3. Furcation involvement

Denuded furcation areas of teeth with attachment loss as a result of periodontitis are highly plaque retentive. Furcation involvement worsens the long term prognosis of teeth, especially that of the upper molars.

Figure 3.322. Figure 7. – Class I. furcation involvement
19.2.2.4. Abrasion, abfraction

Loss of hard tissues in cervical areas of teeth may occur as a result of mechanical trauma from abrasive agents (e.g.: improper toothbrushing – scrubbing), which is called abrasion, or due to result of excessive bite load, which is called abfraction. These cervical notches are plaque retentive.

19.2.2.5. Root caries, circular caries, approximal caries

Carious lesions with cavitation in the cervical area of teeth are highly plaque retentive.

**Figure 3.323. Figure 8.** – Carious lesions at the level of cemento-enamel junction with irregular surface
19.2.2.6. Calculus

Research has shown, that sterile calculus does not cause inflammation. However its irregular surface is always covered with dental plaque, which is the main causative factor of periodontal inflammation.

Figure 3.324. Figure 9. – An excessive amount of supragingival calculus on the lingual surface of lower incisors

19.2.2.7. Gingival recession
Denuded root surfaces are more difficult to clean especially in the interdental region (Miller class III. and IV.).

**Figure 3.325. Figure 10. – Interdental areas with plaque retention**

19.2.2.8. **Mouth breathing**

Constant obstruction of nasal airways caused by adenoid vegetation or allergic rhinitis produces breathing through the mouth with subsequent drying of mucosal surfaces resulting in increased susceptibility to infections.

19.2.2.9. **Aberrant frenum**

Aberrant frenum pull caused by a frenum attached close to the marginal gingiva often produces shallow vestibule and gingival recession.

**Figure 3.326. Figure 11. – An aberrant frenum and a shallow vestibule**
19.3. Systemic risk factors

Systemic risk factors of periodontal disease can further be divided into modifiable and non-modifiable factors.

19.3.1. Non-modifiable systemic risk factors

19.3.1.1. Immunodeficient conditions
The main cause of tissue damage in periodontal inflammation is related to host immunological reactions. Both hyper-responsiveness and decreased response of the immune system may lead to a more severe tissue loss.

**Decreased number** (familiar chronic benign neutropenia, cyclic neutropenia, Kostmann-syndrome) or **altered function of PMN leukocytes** (leukocyte adhesion deficiency type I. and II., lazy leukocyte syndrome, Papillon-LeFevre syndrome, Chediak-Higashi syndrome, myeloperoxidase deficiency, acatalasia) leads to severe early onset destructive periodontitis.

Ulcerative gingival and periodontal lesions occur in **AIDS Related Complex (ARC)**. Therefore laboratory testing (blood test) of all patients with clinical manifestations similar to necrotizing ulcerative gingivitis/periodontitis is mandatory.

### 19.3.1.2. Hormonal conditions

Sexual hormones have an effect on capillary morphology and permeability of periodontal tissues as well as on biofilm bacteria. These hormones play a crucial role in pubertal, menstrualional and pregnancy gingivitis.

### 19.3.1.3. Diabetes Mellitus

Badly managed diabetes mellitus patients have a 3.5-4 fold increase in the risk for developing periodontitis. Osmotic damage of cells caused by hyperglycemia and alterations in protein functions due to glycation lead to altered immune functions (increased inflammatory cytokine and interleukine production) and decreased regenerative capacity of tissues. Periodontal inflammation also affects diabetes by increasing insulin resistance and by aggravating the progression of complications of DM. Therefore diabetes and periodontitis are in bi-directional relationship.

### 19.3.1.4. Ethnicity

Research has shown that localized aggressive periodontitis is significantly more common and the occurrence of pockets deeper than 5 mm is 3 times higher in the African-American population than in the Caucasian population in the USA. On the contrary, ulcerative periodontal lesions are more frequent in Caucasian Americans.

### 19.3.1.5. Age

The prevalence and severity of periodontal lesions increases with age.

### 19.3.2. Modifiable systemic risk factors

#### 19.3.2.1. Smoking

Smoking is the most important behavioral risk factor of periodontitis. Tooth loss and attachment loss due to periodontitis proportionately increases with the number of years and number of cigarettes the patient has smoked. Smoking alters gingival blood circulation (decreased BOP on the periodontal chart), decreases O2 tension in pockets (red complex bacteria occur in pockets shallower than normal), alters immune response, and reduces the growth potential of periodontal ligament fibroblasts.

#### 19.3.2.2. Nutrition

Decreased protein intake and vitamin C deficiency are risk factors for periodontal disease. Vitamin C supplementation is especially important for smokers. The use of probiotics decreases the number of pathogenic bacteria in both the supra- and subgingival flora.

#### 19.3.2.3. Socio-economic factors

Lower socio-economic status is a risk factor for periodontal disease. This can be related to more stress, higher prevalence of smoking, poor oral hygiene and fewer dental visits of people living in lower socio-economic conditions.

### 20. 3.20. Morphology of periodontal attachment loss – Ivan Mandel
20.1. Examination of attachment loss

With the progression of periodontal inflammation various soft- and hard tissue defects occur. Proper measurement of the extent and shape of these defects is of fundamental importance to determine tooth prognosis and treatment. Besides physical examination x-ray analysis is also necessary. Although panoramic x-rays give a good overview of periodontal bone, intraoral x-rays are required to precisely measure bony conditions. Paralleling and bitewing radiographs give the best radiographic projection for measurements. Reproducibility of radiographs is also an important factor in the long term follow-up of lesions, which can be achieved by special (individually fabricated) aiming devices.

A nonradiological examination of the bone level is called bone sounding. Anaesthetized tissues are probed with a periodontal probe to establish the level of underlying alveolar bone.

Bone level in health is situated 1-2 mm apical to the cemento-enamel junction. The presence of lamina densa is an indicator of disease activity.

20.2. Gingivitis

Gingivitis does not lead to attachment loss. No infraalveolar changes can be detected except for decreased density of the crest of interdental septa due to inflammation-caused hypomineralisation. This change is temporary, bone density is restored after the resolution of inflammation.

20.3. Chronic periodontitis

Chronic periodontitis is associated with attachment loss. With the relatively slow disease progression horizontal bone loss is a characteristic feature with the formation of supracrestal soft tissue pockets. The alveolar crest is more than 2 mm from the CEJ at an approximately equal level around neighboring teeth, parallel to the occlusal plane. In the case of active inflammation the lamina densa is hard to detect on the crest of septum.

The Schei ruler is a device to measure the percentage of horizontal bone loss in 10% steps in relation to the overall length of the root (between CEJ and tip of the root).

Figure 3.328. Figure 1. – Horizontal bone loss in generalized chronic periodontitis

Figure 3.329. Figure 2. – Measurement with the Schei ruler on tooth 41 (50% mesial and 30% distal bone loss)
20.4. Aggressive periodontitis

Aggressive periodontitis is characterized by rapid attachment loss. This leads to the formation of vertical bone loss with infraalveolar (osseous) pockets. In vertical bone loss the bone level adjacent to teeth is not equal to the highest point of the alveolar crest. With the fusion of the neighboring osseous pockets horizontalisation may occur.

20.4.1. Types of osseous defects

Vertical osseous defects are triangular radiolucencies on x-rays. These infraalveolar defects are classified according to the number of bony walls which together with the root surface enclose the pocket. The number of bony walls depends on the amount of bone present prior to inflammation and also on the extent of the lesion. The prognosis of the affected tooth largely depends on the number of remaining bony walls and on the angulation of these walls in relation to the root surface (good prognosis under 25°, bad prognosis over 35°).
20.4.1.1. Hemiseptum

A hemiseptum is a one-walled osseous defect.

Figure 3.330. Figure 3. – Model of a one walled osseous defect

Figure 3.331. Figure 4. – Hemiseptum on the distal septum of tooth 42
20.4.1.2. Two-walled defect

It represents a hemiseptum combined with one more bony walls (oral or vestibular).

A special type of two-walled lesions is called interdental crater if it is enclosed by vestibular and oral bony walls and two adjacent root surfaces.

Figure 3.332. Figure 5. – Clinical manifestation of an interdental crater
Figure 3.333. Figure 6. – Model of an interdental crater

Figure 3.334. Figure 7. – A radigraph of a two-walled osseous defect (radiographs alone are not suitable for the estimation of the number of bone walls)
Figure 3.335. Figure 8. – Model of a two walled osseous defect
20.4.1.3. Three-walled defect

Such a defect is enclosed by a hemiseptum and two more bony walls (vestibular and oral).

**Figure 3.336. Figure 9. – Model of a three-walled intrabony defect**
Figure 3.337. Figure 10. – Clinical manifestation of a three-walled osseous defect
20.4.1.4. Four-walled (funnel-shaped) osseous defect

It is a circular defect around a tooth with oral, vestibular and two interdental bony walls.

Figure 3.338. Figure 11. – Model of a four-walled osseous defect
Figure 3.339. Figure 12. – A radiograph of four-walled osseous defects
3. Reconstructive dentistry

Figure 3.340. Figure 13. – Clinical manifestation of funnel-shaped osseous defects

Table 3.16. Table 1. – Classification and characteristics of periodontal pockets

<table>
<thead>
<tr>
<th>TYPE OF POCKET</th>
<th>LOCALISATION</th>
<th>SUBTYPES</th>
<th>CHARACTERISTICS</th>
<th>X-RAY FINDINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tr>
</tbody>
</table>

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Created by XMLmind XSL-FO Converter.
3. Reconstructive dentistry

<table>
<thead>
<tr>
<th>TYPE OF POCKET</th>
<th>LOCALISATION</th>
<th>SUBTYPES</th>
<th>CHARACTERISTICS</th>
<th>X-RAY FINDINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td>soft tissue pocket</td>
<td>supraosseal,</td>
<td>1-walled, 2-walled, 3-walled, 4-walled</td>
<td>chronic periodontitis and advanced generalised aggressive periodontitis</td>
<td>horizontal bone loss</td>
</tr>
<tr>
<td></td>
<td>supracrestal,</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>supraalveolar</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>infraosseal</td>
<td>intraosseal,</td>
<td></td>
<td>aggressive periodontitis and advanced chronic periodontitis</td>
<td>angular defect</td>
</tr>
<tr>
<td></td>
<td>infraalveolar</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

20.5. Furcation involvement

Furcation defects occur if attachment loss reaches the furcation area of multi-rooted teeth. These lesions can be classified according to their oro-vestibular extent in relation to the oro-vestibular width of the tooth (measured with a Nabers-probe) or according to the vertical distance between the crest of remaining interradicular septum and the furcation fornix in millimeters.

Table 3.17. Table 2. – Grading of furcation involvement

<table>
<thead>
<tr>
<th>GRADE OF FURCATION INVOLVEMENT</th>
<th>ORO-VESTIBULAR EXTENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I.</td>
<td>less than 1/3 of the oro-vestibular width of the tooth</td>
</tr>
<tr>
<td>Grade II.</td>
<td>more than 1/3, but less than 2/3 of the oro-vestibular width of the tooth</td>
</tr>
<tr>
<td>Grade III.</td>
<td>through-and-through tunnel formation in the furcation area</td>
</tr>
</tbody>
</table>

Figure 3.341. Figure 14. – A Grade I. furcation lesion on the buccal aspect of upper molars

Figure 3.342. Figure 15. – A Grade II. furcation lesion of lower molars
20.6. Gingival recession

A position of the gingival margin apical to the CEJ is called gingival recession. The most commonly used classification system is the Miller classification. It describes buccal recessions and does not classify recessions of the interdental papillae in detail. Newer classification systems include descriptions of palatal and papillary recessions. The Nordland-Tarnow classification of papillary recessions is based on the relative position of the tip of the interdental papilla from the contact point, interproximal CEJ and the vestibular CEJ.
Table 3.18. Table 3. – Miller classification of gingival recessions

<table>
<thead>
<tr>
<th>MILLER CLASS</th>
<th>EXTENT OF LESION</th>
<th>PROGNOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I.</td>
<td>does not extend to the mucogingival junction, papillae are intact</td>
<td>complete root coverage is achievable</td>
</tr>
<tr>
<td>Class II.</td>
<td>extend to or beyond mucogingival junction, papillae are intact</td>
<td>complete root coverage is achievable</td>
</tr>
<tr>
<td>Class III.</td>
<td>extend to or beyond mucogingival junction with moderate interdental attachment loss</td>
<td>only partial root coverage is achievable</td>
</tr>
<tr>
<td>Class IV.</td>
<td>extend to or beyond mucogingival junction with severe interdental attachment loss</td>
<td>even partial root coverage is unpredictable</td>
</tr>
</tbody>
</table>

Figure 3.344. Figure 17. – Miller I recessions

Figure 3.345. Figure 18. – A Miller II. recession

Figure 3.346. Figure 19. – Miller III. recessions
Dehiscence and fenestration

These represent isolated bone loss on the vestibular or oral surfaces of teeth. The root surface denuded of bone is covered only with soft tissues. Dehiscence occurs when bone loss affects the marginal bone while in fenestration the marginal bone is intact.

21.3.21. Repair and regeneration of the periodontium – Ivan Mandel

Following periodontal therapy the root surface may be repopulated from cells of the (1) gingival epithelium, (2) gingival connective tissue, (3) alveolar bone and (4) periodontal ligament or a combination of these. As a result of this repopulation new tissue is formed which can be different from or identical to normal periodontal tissues. Based on the outcome of this process two main ways of periodontal healing exist:

**Repair**: replacement of lost structures by tissues different from the original (scar formation). These reparative tissues are of inferior functional quality compared to normal tissues.

**Regeneration**: replacement of lost structures by tissues identical to the original. Fully regenerated tissues have the same functional quality as the original tissues.

21.1. Periodontal repair

Gingival epithelium and gingival connective tissue have a faster turnover rate than periodontal bone while cementum is not subject to continuous turnover. It means that cells of gingival origin are more likely to repopulate periodontal pockets and root surfaces - thus leading to repair.

Types of periodontal repair include:
1. **Deep sulcus**: Only a few and most apically situated Sharpey’s fibers reattach to cementum. Gingival epithelium grows down along pocket wall and only attaches to tooth surface at the very bottom of the pocket.

2. **Long epithelial attachment**: Epithelial cells grow down the pocket wall and attach widely to root surface with hemidesmosomes.

3. **Tight adaptation of connective tissue**: Fibers from gingival connective tissue attach to root surfaces.

Although histologically long epithelial attachment and connective tissue reattachment cannot be considered as real attachment gain, clinically significant reduction of pockets can be seen on gentle probing after these healing processes. These forms of attachment are more vulnerable to future noxae than the original tissues, however they have good long-term stability if oral hygiene is good and the periodontium is free of inflammation.

### 21.2. Periodontal regeneration

Periodontal regeneration means the formation of a **new attachment**: newly formed periodontal ligament fibers connect the newly formed alveolar bone and cementum. Histologically regeneration means the new formation of all three of these tissues. Because of the above mentioned differences in growth potential between different periodontal tissues the likelihood of spontaneous regeneration after conventional periodontal therapy is low and may only occur under certain circumstances.

#### 21.2.1. Criteria required for periodontal regeneration

1. **Prevention of apical growth of the epithelium**: The rapid apical downgrowth of epithelial cells separates root surface from connective tissue elements thus preventing the formation of new attachment.

2. **Presence of periodontal ligament fibroblasts**: The formation of new cementum on root surfaces is essential for periodontal regeneration. Only periodontal ligament fibroblasts can transform into cementum-forming cells and they have to migrate coronally from the very apical parts of the periodontal pocket.

3. **Stable blood clot during wound healing**: The formation of new bone starts in the blood clot formed in the periodontal pocket after periodontal treatment. Therefore the maintenance of blood clot integrity and stability is essential.

4. **Correction of root surface alterations that result from disease**: Complete removal of all deposits from the root surface and root planing. Mechanical cleaning (and chemical conditioning) of the surface in order to achieve a clean dentine surface where the formation of new cementum can begin.

The understanding of these criteria has led to the development of new pocket treatment modalities called Guided Tissue Regeneration (GTR) techniques. The underlying principle of these techniques is the use of a barrier to prevent the growth of epithelial cells into the pocket, so that mesenchymal cells can migrate to the root surface to populate it - forming new periodontal ligament tissues.

#### 21.2.2. Guided Tissue Regeneration techniques

Regeneration can be guided by the use of mechanical **barrier membranes** or by the use of **enamel matrix derivative proteins**.

**1. Barrier membranes**:

The application of a membrane with cell occluding properties under the surgical flap around the neck of the tooth creates a barrier for epithelial cells. Since these membranes have limited mechanical stability (especially resorbable types), bony pockets have to be filled with a bone graft or bone filler material in order to stabilize the blood clot and to mechanically support the membrane. Non-resorbable membranes have to be removed by a second surgery after healing, which presents a great disadvantage over resorbable membranes.

<table>
<thead>
<tr>
<th>bioresorbable membranes</th>
<th>natural</th>
<th>collagen</th>
</tr>
</thead>
<tbody>
<tr>
<td>chitosan or chitosan-collagen</td>
<td>hybride</td>
<td></td>
</tr>
</tbody>
</table>
3. Reconstructive dentistry

<table>
<thead>
<tr>
<th>Materials</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>synthetic</td>
<td>aliphatic polyesters (poly-lactic acid, poly-glicolic acid, polydioxanone)</td>
</tr>
<tr>
<td>non-resorbable membranes</td>
<td>expanded polytetrafluoroethylene (ePTFE)</td>
</tr>
<tr>
<td></td>
<td>titanium mesh</td>
</tr>
</tbody>
</table>

**Table 3.20. Table 2. – Types of bone grafting materials used in periodontology**

<table>
<thead>
<tr>
<th>Types of Bone Grafting Materials</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>autografts</td>
<td>intraoral</td>
</tr>
<tr>
<td>(obtained from the same individual from another site)</td>
<td>extraoral</td>
</tr>
<tr>
<td>allografts</td>
<td>freeze-dried bone allograft (FDBA)</td>
</tr>
<tr>
<td>(obtained from other individuals of the same species)</td>
<td>demineralized freeze-dried bone allograft (DFDBA)</td>
</tr>
<tr>
<td>xenografts</td>
<td>inorganic bovine-derived</td>
</tr>
<tr>
<td>(obtained from other species)</td>
<td>inorganic porcine-derived</td>
</tr>
<tr>
<td>alloplasts</td>
<td>coralline calcium carbonate</td>
</tr>
<tr>
<td>(of synthetic origin)</td>
<td>hydroxylapatite</td>
</tr>
<tr>
<td></td>
<td>calcium phosphate cement</td>
</tr>
<tr>
<td></td>
<td>β-tricalcium phosphate</td>
</tr>
<tr>
<td></td>
<td>bioactive glasses</td>
</tr>
</tbody>
</table>

**Figure 3.348. Figure 1. – Bio-Oss® (inorganic bovine) bone grafting material**

**Figure 3.349. Figure 2. – Infrabony defect**
Figure 3. Bone defect filled with Bio-Oss® bone graft.
Figure 3.351. Figure 4. – Shaping of the barrier membrane (resorbable bovine collagen)
Figure 3.352. Figure 5. – Tension-free suturing of the flap over the membrane
Figure 3.353. Figure 6. – A preoperative radiograph of the bone lesion
3. Reconstructive dentistry

Figure 3.354. Figure 7. – A postoperative radiograph showing bony fill of the lesion

2. Biological mediators – enamel matrix derivatives:
Enamel matrix derivative (EMD) is mainly composed of amelogenins, proteins that are naturally synthetized by the Hertwig’s epithelial root sheath during tooth development. EMD has significant role in regeneration through stimulating the growth of periodontal ligament, cementum, bone and vascular components, while it inhibits epithelial growth and therefore acts as a barrier. It also improves healing of surgical wounds and inhibits the growth of certain bacteria which characteristic is related to its vehicle (propylene glycol alginate). Porcine EMD is marketed under the name Emdogain® (Straumann, Switzerland).

**Figure 3.355.** Figure 8. – Chemical conditioning of the root surface before application of Emdogain®

**Figure 3.356.** Figure 9. – Application of Emdogain®
Figure 3.357. Figure 10. – The sutured wound

Figure 3.358. Figure 11. – The reoperative radiograph of the lesion
Figure 3.359. Figure 12. – The postoperative radiograph showing bony fill of the lesion
22. 3.22. Guidelines for the treatment of periodontal diseases – Ivan Mandel

The main goal of periodontal treatments is to achieve periodontal conditions that can be maintained over a long period of time with individual and professional oral hygiene. These treatments are also aimed at eliminating causal factors of periodontal disease and at stopping further attachment loss. A further goal is to restore tissues lost due to inflammation and to restore the periodontium both functionally and esthetically. Finally, the maintenance of results achieved is also aimed at.

22.1. Diagnosis based treatment plan

The diagnosis is established after a careful physical examination. It includes medical and dental history taking, extra- and intraoral examinations, occlusal analysis, measurement of periodontal parameters, additional radiological, microbiological and laboratory examinations and tests as well as photo documentation. The establishment of the diagnosis is followed by the determination of the prognosis for each tooth. The general prognosis of the case also has to be determined before making a treatment plan because the type of treatment - conservative, radical or palliative - will depend on it.

Table 3.21. Table 1. – Factors influencing prognosis (individual risk analysis)

<table>
<thead>
<tr>
<th>General and systemic factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>• general health, immune status,</td>
</tr>
<tr>
<td>• genetic factors,</td>
</tr>
<tr>
<td>• age,</td>
</tr>
<tr>
<td>• clinical form and severity of disease,</td>
</tr>
</tbody>
</table>
• cooperation of the patient, frequency of recalls,
• individual plaque control, motivation,
• smoking

<table>
<thead>
<tr>
<th>Local factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>• composition of subgingival bacterial flora,</td>
</tr>
<tr>
<td>• plaque retentive factors,</td>
</tr>
<tr>
<td>• localisation and depth of pockets,</td>
</tr>
<tr>
<td>• furcation involvement,</td>
</tr>
<tr>
<td>• number of active sites,</td>
</tr>
<tr>
<td>• degree of attachment loss,</td>
</tr>
<tr>
<td>• morphology and degree of bone loss,</td>
</tr>
<tr>
<td>• root morphology,</td>
</tr>
<tr>
<td>• tooth mobility,</td>
</tr>
<tr>
<td>• occlusal forces</td>
</tr>
</tbody>
</table>

A properly established diagnosis is the basis of a successful treatment. However, the preliminary treatment plan may change according to patient related factors such as cooperation and responsiveness to treatment.

Treatment of periodontal diseases can be divided into four major consecutive steps:

1. Treatment of acute problems.
2. Causal therapy (non-surgical therapy).
3. Correctional therapy (surgical therapy).
4. Maintenance therapy (supportive therapy).

Not all periodontal conditions require this full sequence of treatment.

**Figure 3.360.** Figure 1. – Immediate extractions are needed in the case of end-stage periodontitis
3. Reconstructive dentistry

Figure 3.361. Figure 2. – Crowding and recessions make it difficult to maintain good oral hygiene

22.2. Causal therapy

The establishment of diagnosis is followed by causal periodontal therapy. These causal treatments are non-surgical. The removal of supra- and subgingival calculus and biofilm, polishing, root planing and elimination of local plaque-retentive factors (ill-fitted restorations, carious lesions, open contact points, small anatomical alterations) are essential. The complete removal of the biofilm from surfaces is aimed at, however it is almost impossible to carry out in practice.
In the case of occlusal disharmony an occlusal analysis has to be performed. Increased tooth mobility is an adaptive reaction of periodontal structures to traumatizing occlusal forces. Primary occlusal trauma occurs on teeth without inflammatory attachment loss, while secondary occlusal trauma affects teeth with progressive periodontal inflammation and attachment loss. The treatment sequence of the two types is different from each other.

Table 3.22. Table 2. – Treatment of primary and secondary occlusal trauma

<table>
<thead>
<tr>
<th>PRIMARY OCCLUSAL TRAUMA</th>
<th>SECONDARY OCCLUSAL TRAUMA</th>
</tr>
</thead>
<tbody>
<tr>
<td>• selective grinding,</td>
<td>• splinting,</td>
</tr>
<tr>
<td>• elimination of habits,</td>
<td>• selective grinding,</td>
</tr>
<tr>
<td>• orthodontic treatment,</td>
<td>• orthodontic treatment</td>
</tr>
<tr>
<td>• night guard</td>
<td></td>
</tr>
</tbody>
</table>

Since good oral hygiene is crucial for the healing process, oral hygiene instructions and patient motivation are essential.

Figure 3.362. Figure 3. – Before causal treatment

Figure 3.363. Figure 4. – Removal of supra- and subgingival calculus using ultrasonic scalers
Figure 3.364. Figure 5. – Subgingival scaling and root planing with hand instruments
Figure 3.365. Figure 6. – Polishing
Figure 3.366. Figure 7. – After initial therapy
Since initial therapy is a complex series of treatments it may take several sessions to complete. Four to six weeks after completion a reassessment is needed. Patients’ motivation, oral hygiene should be checked. A new periodontal chart needs to be recorded and the prognosis for teeth has to be reevaluated.

Causal therapy is fully successful if:

- Individual oral hygiene is good (PI < 20-25%).
- Bleeding index is within normal range (no active inflammation present, BOP < 20%).
- Probing pocket depth is decreased.
- No plaque retentive factors are present.

Based on these criteria a decision can be made concerning the following steps of treatment: repeating causal therapy, moving on to maintenance therapy or necessity for correctional therapy.

22.3. Correctional therapy

Correctional therapy is needed if any of the following conditions is present.

Table 3.23. Table 3. – Indications for correctional periodontal therapy

<table>
<thead>
<tr>
<th>Areas that are difficult to reach with individual oral hygiene</th>
</tr>
</thead>
<tbody>
<tr>
<td>• deep pockets,</td>
</tr>
</tbody>
</table>

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</tr>
</thead>
<tbody>
<tr>
<td>• deep pockets,</td>
</tr>
</tbody>
</table>
• wide approximal root surfaces,
• furcation involvement,
• concave root surfaces.

**Deformities that make individual oral hygiene difficult**

• thick, fibrotic marginal gingiva,
• gingival craters,
• shallow vestibule,
• deep gingival recession,
• lack of keratinized gingiva.

**Deep infrabony pockets, irregular alveolar bone crest, interdental bony craters**

Lack of patient cooperation, lack of good oral hygiene, and the presence of certain systemic conditions are contraindications for correctional (surgical) treatment.

Correctional treatments include periodontal surgery, orthodontic and prosthetic treatments.

Destructive periodontitis often leads to tooth loss and the remaining teeth may have a significantly reduced load bearing capacity due to attachment loss. The treatment of partial edentulism in such conditions may be difficult. The success of a periodontal prosthesis will ultimately be measured in terms of long term plaque control and successful periodontal maintenance. Fixed prostheses are preferred over removable ones because they offer less plaque retention and greater splinting effect for periodontally affected teeth. The use of osseointegrated dental implants has many benefits in prosthetic rehabilitation of periodontal patients, because it reduces the risk of using teeth with reduced load bearing capacity as abutments, and reduces the need for extensive and complex fixed prostheses.

Orthodontic treatments should be performed after the completion of causal surgical procedures and before regenerative surgical procedures.

After completion of correctional treatment another phase of observation is necessary with the reassessment of prognosis for teeth. Reassessment is made 1 to 6 months after surgery depending on the type of procedure.

**Figure 3.367. Figure 8. – Before treatment**
Figure 3.368. Figure 9. – After completion of causal therapy

Figure 3.369. Figure 10. – Correctional treatment
The final goal of a complex therapy is the long term maintenance of results achieved by causal and correctional therapy. Supportive therapy needs to be designed individually, depending on all patient related factors. Periodontal risk assessment test of patients encounters all these values and helps to define weather a patient has low or high risk for further periodontal progress. Low risk patients are recalled every 6 months while high risk patients need a 3 month follow up.
3. Reconstructive dentistry

Figure 3.371. Figure 12. – Periodontal Risk Assessment (http://www.periotools.com/pra/en/index.asp)

Table 3.24. Table 4. – Supportive therapy

<table>
<thead>
<tr>
<th>Examinations</th>
</tr>
</thead>
<tbody>
<tr>
<td>• during every recall visit</td>
</tr>
<tr>
<td>• condition of the gingiva (GI, BOP),</td>
</tr>
<tr>
<td>• presence of dental plaque (plaque indices)</td>
</tr>
<tr>
<td>• every 6-12 months in addition to the previous examinations</td>
</tr>
<tr>
<td>• measurement of periodontal parameters,</td>
</tr>
<tr>
<td>• occlusal evaluation,</td>
</tr>
<tr>
<td>• cariologic evaluation,</td>
</tr>
<tr>
<td>• prosthetic evaluation</td>
</tr>
<tr>
<td>• every 3-4 years</td>
</tr>
<tr>
<td>• full mouth series of periapical x-rays</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Treatments</th>
</tr>
</thead>
<tbody>
<tr>
<td>• during every recall visit</td>
</tr>
<tr>
<td>• oral hygiene instructions and motivation,</td>
</tr>
<tr>
<td>• professional oral hygiene treatments,</td>
</tr>
<tr>
<td>• full causal treatment (in case of relapse).</td>
</tr>
</tbody>
</table>
22.5. Antibiotics in periodontal therapy

Most periodontal inflammatory conditions respond well to mechanical therapy however the adjunctive use of antibiotics may be necessary in some cases. Antibiotics may be administered systemically and locally.

22.5.1. Systemic antibiotics

In order to reduce side effects and to lower the chance of antibiotic resistance the use of systemic antibiotics should be limited to certain conditions and disease forms. The use of antibiotics cannot replace mechanical disruption of the biofilm - their use is only adjunctive to mechanical therapy. A full mouth mechanical biofilm disruption should be performed in the shortest time possible (a one session treatment is preferred and multiple sessions should be completed within a few days). The administration of antibiotics should start immediately after the completion of mechanical cleaning.

Plaque-induced gingivitis

- Antibiotics are only recommended in the presence of a severe underlying systemic disease.

Chronic periodontitis

- Chronic periodontitis in general responds well to mechanical treatment alone therefore the use of antibiotics should be limited to certain cases (refractory periodontitis).

- Metronidazole+Amoxicillin, Macrolides.

Aggressive periodontitis

- The use of systemic antibiotics in the causal therapy is necessary in aggressive periodontitis due to the presence of tissue-invasive bacteria.

- Metronidazole+Amoxicillin, Doxycycline, Macrolides.

Periodontitis as a manifestation of systemic diseases

- The protocol is the same as that for aggressive periodontitis.

Necrotizing periodontal diseases

- Metronidazole is recommended as an adjunct to mechanical therapy.

Periodontal abscesses

- The administration of antibiotics should be considered on an individual basis based on the extent of the lesion, systemic symptoms and general health status of the patient.

22.5.2. Local antibiotics

With the use of local antibiotics systemic side effects can be reduced and a higher local concentration of antibiotics is achievable. Their use is recommended in localized periodontal lesions. Recommended devices for clinical use: Actisite (fiber loaded with 25% tetracycline), Minocycline (2% cream), Atridox (10% doxycycline), Elyzol (25% metronidazole), Ebrimycin gel (20% primycin).

Figure 3.372. Figure 13. – Decision tree of periodontal treatments
Figure 3.373. Figure 14. – Decision tree of plaque-induced gingivitis

Figure 3.374. Figure 15. – Decision tree of chronic periodontitis
The main objective of periodontal surgery is to help the long-term preservation of periodontal health. Hard and soft tissue defects caused by destructive periodontitis may remain after successful causal therapy of the disease. The goal of the treatment of these defects is to create accessibility to professional supra- and subgingival cleaning, to create gingival morphology that helps individual oral hygiene measures, and – if possible – to achieve regeneration of periodontal tissues.

The two main types of surgical procedures include periodontal pocket surgery and periodontal plastic surgery.

Table 3.25. Table 1. – Indications and contraindications for periodontal surgery (Lindhe, 2003)
### 23.1. Basic surgical terminology

#### 23.1.1. Types of incisions

1. **External bevel incision**: An incision on the attached gingiva parallel to the gingival margin at the level of the base of the pocket. The scalpel is held at a 45 degree angle to the root surface, pointing in coronal direction.

   **Figure 3.376. Figure 1. – External bevel incision**
2. **Internal bevel incision**: An incision at the gingival margin following its scallop with a scalpel pointing in apical direction. Subtypes include intrasulcular, marginal and paramarginal.

**Figure 3.377. Figure 2. – Internal bevel incision** (1: intrasulcular 2: marginal 3: paramarginal)
3. **Vertical releasing incision**: An incision perpendicular to the gingival margin to help mobilization of a flap.

**Figure 3.378. Figure 3. – Vertical releasing incision**
23.1.2. Types of surgical flaps

According to thickness:

1. **Full thickness flap**: The mucosa, tunica propria and the periosteum are elevated together from the root surface with blunt dissection (with periosteal elevators).

   **Figure 3.379. Figure 4. — Full thickness flap**

2. **Split thickness flap**: The mucosa and mucosal part of the tunica propria are elevated from the periosteal part of the tunica propria and periosteum with sharp dissection (scalpel).

   **Figure 3.380. Figure 5. — Split thickness flap**
In relation to the interdental papilla:

1. **Conventional flap**: Oral and vestibular parts of the papilla are separated interdentally.

**Figure 3.381. Figure 6. – Conventional papilla incision**
2. **Papilla preservation flap**: Incision is made at the base of the papilla, this way the whole papilla becomes part of either the oral or the vestibular flap.

**Figure 3.382. Figure 7. – Incision for papilla preservation flap**

3. **Tunnel preparation**: Papilla is not separated, but is undermined from intrasulcular incisions.

**Figure 3.383. Figure 8. – Tunnel preparation**
According to positioning:

1. **Repositioned flap**: The flap is sutured in its original position.

2. **Apically/coronally/laterally positioned flap**: The flap is sutured in a position different from the original.

Depending on their aim, periodontal surgical treatments are divided into two main classes.

### 23.2. Periodontal pocket surgery

These procedures are aimed at the elimination of periodontal pockets caused by inflammatory tissue loss in order to prevent the deposition of dental plaque on these surfaces that are hard to reach for oral hygiene. These procedures may also be aimed at promoting the formation of new attachment or periodontal regeneration.

**Table 3.26. Table 2. – Types of periodontal pocket surgery**

<table>
<thead>
<tr>
<th>TYPE</th>
<th>TECHNIQUE</th>
<th>FIELD OF INDICATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>resective surgery</td>
<td>gingivectomy</td>
<td>suprabony pockets</td>
</tr>
<tr>
<td></td>
<td>apically repositioned flap surgery</td>
<td>suprabony pockets</td>
</tr>
<tr>
<td></td>
<td>resective surgery of teeth (hemisection, trisection, root amputation, premolarization)</td>
<td>supra- and infrabony pockets</td>
</tr>
<tr>
<td>new attachment techniques</td>
<td>subgingival curette</td>
<td>suprabony pockets</td>
</tr>
<tr>
<td></td>
<td>excisional new attachment procedure (ENAP)</td>
<td>suprabony pockets</td>
</tr>
<tr>
<td></td>
<td>modified ENAP</td>
<td>suprabony pockets</td>
</tr>
<tr>
<td></td>
<td>modified Widman flap surgery</td>
<td>supra- and infrabony pockets</td>
</tr>
<tr>
<td>regenerative surgery</td>
<td>GTR</td>
<td>infrabony pockets</td>
</tr>
<tr>
<td></td>
<td>biological mediators</td>
<td>infrabony pockets</td>
</tr>
</tbody>
</table>

#### 23.2.1. Gingivectomy/gingivoplasty

Soft tissue pocket is removed with an external bevel incision and the wound is covered with a periodontal dressing. The removal of excess tissue is called gingivectomy, while recontouring of the gingival margin is called gingivoplasty. Fields of indication are gingival corrections required for making proper dental restorations and treatment of gingival enlargement, while contraindications include the presence of a bony pocket, if less than 2mm of keratinized gingiva remains after procedure, if the result is esthetically unacceptable.
Figure 3.384. Figure 9. – Gingival enlargement (interdental papillae)

Figure 3.385. Figure 10. – After laser gingivoplasty

Figure 3.386. Figure 11. – 2 weeks after the procedure

23.2.2. Apically repositioned flap surgery

A full thickness flap is elevated from a marginal internal bevel incision. Following debridement of root surfaces and osteoplasty (if needed), the flap is sutured at the level of the marginal alveolar bone. Its advantage is the
radical and immediate pocket reduction, however denuded root surfaces and interdental spaces are harder to keep plaque free and the esthetic result and root hypersensitivity may be unacceptable for the patient.

**Figure 3.387. Figure 12. – Severe periodontitis with horizontal bone loss**

**Figure 3.388. Figure 13. – Flap apically positioned**

**Figure 3.389. Figure 14. – Preserved attached gingiva and complete elimination of pockets**
23.2.3. Root resection surgery

Radical resective surgery of multi-rooted teeth with advanced (class II. and III.) furcation lesions is possible with the surgical separation of roots. The prerequisite of such a procedure is the endodontic treatment of the tooth. It is called hemisection in the case of the lower molars and trisection in the case of the upper molars. Separated roots can be handled as premolars (premolarization), or one of them can be removed (root amputation).

Figure 3.390. Figure 15. – Amputation of DB root of an upper molar

Figure 3.391. Figure 16. – Removal of DB root
23.2.4. Subgingival curettage (‘closed curettage’)

Subgingival curettage is the removal of the inner pocket wall (ulcerated pocket epithelium and granulation tissue) with a periodontal curette performed in the same session as subgingival scaling and root planing (SRP). Although it is aimed at developing new attachment, it does not improve the results achievable with SRP alone. It may be recommended if the excessive amount of granulation tissue causes thick gingival margin that makes oral hygiene difficult.

Figure 3.392. Figure 17. – Removal of the inner pocket wall with a periodontal curette
23.2.5. Excisional new attachment procedure (ENAP)

The field of indication, extent and success rate is identical to that of the subgingival curettage. The difference is that the pocket wall is removed using a sharp dissection (scalpel) instead of a blunt dissection (curette). Excision is always followed by suturing of the flap around teeth. In the case of a modified ENAP technique the incision is made down to the alveolar bone crest.

Figure 3.393. Figure 18. – Removal of the inner pocket wall with a scalpel (ENAP)

23.2.6. Modified Widman flap (‘open curettage’, ‘access flap’)

The inner pocket wall is removed with a combination of paramarginal inner beveled, intrasulcular and horizontal incisions and a full thickness flap is elevated. This is followed by debridement of root surfaces and bone recontouring. If appropriate width of the interdental papilla is present, the flap can be elevated using the papilla preservation technique. The flap is then repositioned and fixed with sutures. The aim of this procedure is the debridement of deep subgingival areas under visual control with minimal postsurgical tissue loss. However it is not aimed at pocket reduction or elimination of bony pockets. Because of this it is more like a causal than a correctional treatment. Regeneration of periodontal structures may be seen in the apical portions of pockets treated using the modified Widman technique.

Figure 3.394. Figure 19. – Incisions for a Widman flap
23.2.7. Regenerative periodontal surgery

The above mentioned techniques result in periodontal repair. The prerequisite of periodontal regeneration is the repopulation of root surfaces by cells originating from the periodontal ligament. This can be achieved by the use of barrier membranes or by enhancing the connection between the wound and root surfaces. Primary wound closure is essential for blood clot stability and successful regeneration, therefore a full thickness flap is elevated from an intrasulcular incision without the removal of the pocket wall. After careful debridement of the root surface and the bony pocket, bone graft and membrane or Emdogain or the combination of bone graft and Emdogain are applied and the wound is closed with sutures.

Figure 3.395. Figure 20. – Extensive vertical bone defect after debridement

Figure 3.396. Figure 21. – The defect filled with bone graft (bovine xenograft)
23.3. Periodontal plastic surgery

The aim of mucogingival surgical procedures is the correction of developmental or acquired deformities of soft tissues. These defects make oral hygiene difficult to maintain, therefore they act as contributing factors to periodontal inflammation and may cause functional and esthetic problems.

Table 3.27. Table 3. – Mucogingival defects and their treatments

<table>
<thead>
<tr>
<th>MUCOGINGIVAL DEFECT</th>
<th>PROBLEM</th>
<th>TREATMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>gingival recession</td>
<td>esthetic problem</td>
<td>surgical covering of recession</td>
</tr>
<tr>
<td></td>
<td>hypersensitivity and abrasion of denuded root surfaces</td>
<td></td>
</tr>
<tr>
<td></td>
<td>makes oral hygiene more difficult</td>
<td></td>
</tr>
<tr>
<td>lack of attached gingiva</td>
<td>susceptibility to gingival recession or pocket formation especially if combined with local irritating factors</td>
<td>widening of keratinized gingiva</td>
</tr>
<tr>
<td>excessive gingival display,</td>
<td>esthetic problem</td>
<td>gingivectomy/ gingivoplasty</td>
</tr>
</tbody>
</table>
23.3.1. Surgical recession covering

Recessions are covered with neighboring gingival tissues that are mobilized with pedicle or tunnel flaps. Additional transplantation of connective tissue grafts may be necessary to thicken/widen the attached gingiva. Complete root coverage can only be achieved for Miller class I. and II. recessions, while only partial root coverage is possible for Miller class III. and IV. recessions. Although several surgical techniques have been developed for recession coverage, many of them have a limited field of application or success rate. There are two main techniques that are widely used in practice: the coronally advance flap technique (with or without a connective tissue graft), and the tunnel technique combined with connective tissue graft transplantation. Both techniques are suitable for the coverage of Miller class I. and II. recessions, while one stage coverage of Miller III. recessions can be achieved using the tunnel technique.

23.3.1.1. Coronally advanced flap technique

A flap with variable thickness (full-split-full) is elevated apically from the basis of papillae. It may be combined with vertical incisions for single recessions or it can be used without vertical incisions for multiple recessions. The flap margin is placed on the CEJ and fixed with sutures to the deepithelized papillae. This technique may be combined with the transplantation of connective tissue graft if keratinized gingiva of inappropriate width and/or thickness is present.

Figure 3.398. Figure 23. – Miller class I. recessions

Figure 3.399. Figure 24. – Incisions for the flap (Zucchelli and De Sanctis)
23.3.1.2. Tunnel technique

A tunnel flap is elevated with a special microsurgical elevator (tunnel-knife) from intrasulcular incision without papilla incision. The preparation should extend beyond the mucogingival junction to allow mobilization of the flap. Connective tissue graft is then placed under the tunnel flap and the flap margin is fixed with sutures around teeth slightly coronal to the level of CEJ.

Figure 3.402. Figure 27. – Miller class III. recession
Figure 3.403. Figure 28. – Tunnel flap is elevated

Figure 3.404. Figure 29. – Connective tissue graft harvested from the palate

Figure 3.405. Figure 30. – Graft placed under the flap and fixed with sutures
23.3.2. Widening of the attached gingiva

It can be performed with vestibuloplasty or transplantation of connective tissue graft or with the combination of these.

23.3.3. Frenectomy

The aberrant frenum is completely excised and the wound edges are sutured up. It may be combined with connective tissue transplantation if necessary.

Figure 3.406. Figure 31. – Aberrant labial frenum

Figure 3.407. Figure 32. – Wound edges sutured after excision
23.3.4. Papilla augmentation

The height of the interdental papilla and the papilla fill of the interdental space largely depends on the distance from the contact point to the crest of interdental septum of alveolar bone. If this distance is greater than 5 mm predictabe papilla fill cannot be achieved due to insufficient blood supply. Among several techniques used for papilla augmentation the tunnel technique combined with a connective tissue graft gives the most predictable results (Azzi-technique).

23.3.5. Surgical crown lengthening

The indications for surgical crown lengthening include inadequate height of clinical crown for retention of dental restorations or placement of the restoration margin can lead to violation of the biologic width, or to the presence of excessive gingival display. Surgical technique is chosen taking into consideration the following guidelines: Appropriate width (> 2 mm) of residual keratinized tissue should be present after treatment, and the preservation of the biologic width.

Figure 3.408. Figure 33. – Indications and techniques for surgical crown lengthening

23.3.6. Correction of edentulous ridge defects

Soft- and hard tissues at tooth extraction sites undergo morphological changes (horizontal and/or vertical dimension, height and shape of the interdental papilla) during the healing process. The extent of change depends on factors such as the original anatomical situation (presence and thickness of buccal bone plate) mode of tooth extraction (atraumatic or not, with or without flap elevation) and the gingival biotype (thin or thick). These deformities are described by the Seibert classification: Class I. a buccolingual defect with preserved vertical dimension, Class II. a vertical defect with preserved buccolingual dimension, Class III. a combined vertical and horizontal defect. Promotion of ridge preservation is possible at the time of tooth extraction applying an
atraumatic tooth removal without flap elevation, complete closure of the wound or by the use of connective tissue and/or bone grafts, or by the use of barrier membranes. Soft tissues at the already healed edentulous ridge can be augmented by pedicle grafts, free connective tissue grafts (inlay/onlay) or by alloplastic grafts. The augmentation is followed by shaping of the tissues by temporary pontics to achieve an esthetically favorable concave gingival contour. Large sized defects (advanced Class I. and most Class II. and III.) require bone augmentation with guided bone regeneration (GBR), with inlay or onlay autologous bone grafts, or with distraction osteogenesis.

24. 3.24. Oral mucosa diseases I. (Classification, patient examination, differential diagnosis) – Ivan Mandel

All surfaces in the oral cavity are covered by mucosa except for the clinical crown of teeth. Mucosa covering gingiva, lips, buccal surfaces and the tongue may be involved in diseases one by one or all together. Diseases may affect only mucosal surfaces or may be manifestations of other systemic (internal organs, hematological, dermato pathological, autoimmune) conditions. Therefore care should be taken on cooperation of different fields of medicine in the treatment of these diseases. Importance of thorough examination of all patients has to be emphasized: questioning of the patient has to extend to symptoms elsewhere in the body and stomato-oncological screening of all patients is mandatory since oral cancer is a growing healthcare problem throughout the world including Hungary.


24.1. Classification

1. Developmental and genetic disorders that cause mucosal symptoms.
2. Physical, chemical and iatrogenic injuries.
3. Infectious diseases.
4. Diseases of the lips.
5. Diseases of the tongue.
6. Immunological disorders.
7. Vesiculobullous and granulomatous skin- and oral diseases.
8. Oral manifestations of diseases of organs and organ systems.
9. Precancerous lesions, conditions and color changes of the oral mucosa.
11. Malignant tumours.

24.2. Steps of establishing a diagnosis

Proper medical history, thorough patient examination, and additional examinations such as blood test, histological examination, microbiological, immunological, genetic- and electron microscopic examinations are needed for a correct and accurate diagnosis.

1. Patient examination
2. Taking medical history
Medical history of the patient can be taken with oral questioning or with a questionnaire or with the combination of both. Building good doctor-patient relationship is essential for the exploration of certain diseases (HIV, hepatitis, sexually transmitted diseases).

Socio-economic background of patients should be investigated as well as already known systemic diseases and previous medications, because certain types of medications may cause oral mucosal disorders or changes in salivary secretion. Alcohol and drug use, smoking and other bad habits should be recorded as well as family medical history which might reveal inherited causes behind certain diseases.

3. Detailed patient examination

The patient’s current complaints should be examined together with a stomato-oncological screening.

• Intraoral examination: (observation, palpation, diascopy, examination of function)
  • Lips,
  • Buccal mucosa,
  • Tongue,
  • Sublingual area,
  • Hard- and soft palate.

• Extraoral examination:
  • Orofacial region,
  • Submandibular, submental and perimandibular regions,
  • Periauricular region,
  • Occipital region,
  • Neck region,
  • Supraclavicular region.

  Video 1. – Extra- and intraoral examination.

4. Differential diagnosis

After the detailed patient examination all the hypothetical diagnoses are summarized. These hypotheses are ranked by different aspects. The number of possibilities is narrowed by exclusion of the less probable diagnoses. Additional examinations may be necessary to establish the definitive diagnosis.

5. Blood test

Hematological disorders, deficiency states, inflammatory processes and metabolic disorders can be tested by a full blood test.

6. Microbiological examination

  Bacterial diagnostics: culturing, molecular biologic methods.

  Viral diagnostics: isolation, electron microscopic examination, immunofluorescence, immunoperoxidase method, ELISA and other molecular biologic methods.

  Mycological diagnostics: culturing, serological, microscopic and electronmicroscopic, molecular biologic methods.

7. Histologic examination
Biopsy and histologic examination is necessary in the following cases: chronic ulcers with unknown etiology and without any tendency to heal, chronic inflammations without tendency to heal, white lesions, changes in colour, abnormal masses of tissue, supposedly premalignant or malignant lesions. Biopsy has to be carried out in medical facilities where definitive treatment is also possible.

Types of biopsies:

- Incisional biopsy: Incisional biopsy is taken if the diameter of the lesion is greater than 1.5-2 cm. A small part of the lesion is removed.

**Figure 3.409. Figure 1. – Excisional biopsy**

- Excisional biopsy: small lesions are removed in total.

**Figure 3.410. Figure 2. – Incisional biopsy**

- Trepan (“punch”) biopsy: suitable for the examination of superficial premalignant lesions and for the examination of hard to reach areas.
- Needle aspiration biopsy: suitable for the removal of a small sample from deep tissue areas.
- Exploration biopsy: tissue sample is removed after surgical exploration of the area.

Tissue samples are processed with various methods and histologic sections are prepared.
8. **Cytologic examination**

Samples obtained with a spatula contain superficial cells, while samples obtained with a brush contain cells from the basal layer as well. Cytobrush technique is suitable for long term follow-up of leukoplakia lesions.

9. **Allergological examinations**

Antigens can be detected with an epicutaneous test or an intracutaneous test.

10. **Immunological examinations**

Abnormal amounts of antibodies, immunocomplexes can be detected with immunofluorescent and ELISA methods.

11. **Molecular biological and genetic methods**

Detection of genetic disorders, identification of bacteria, detection of micrometastases in tumour diagnostics.

RNA detection with hybridization technique:

- Northern-blot,
- Differential hybridization,
- DNA-chip,
- Microarray in situ hybridization.

Detection of proteins:

- Western-blot with antibodies,
- In situ detection with antibodies,
- Protein microarray,
- Detection of protein phosphorylation,
- Detection of enzyme activity,
• Reporter gene assays, transgenic animals.

12. **Imaging diagnostics**
   • Conventional x-ray analysis,
   • Ultrasound tomography,
   • CT scan,
   • MRI scan,
   • PET scan.

13. **Examination of the salivary glands**
   • Sialometry,
   • Sialochemistry,
   • Sialography,
   • Labial salivary gland biopsy.

14. **Patient documentation**

   Besides personal data of the patient all examination results, diagnoses, treatment plan, treatments and the course of the disease have to be recorded. Photo and video documentation can also be helpful. Accurate documentation is also necessary for legal and forensic issues.

25. **3.25. Oral mucosa diseases II. (Ulcerative, vesiculobullous and infectious diseases) – Ivan Mandel**

25.1. **Ulcerative lesions of the oral mucosa**

Ulcerative lesions have two main subgroups:

• Primary ulcers: the first lesion to occur is an ulcer.

• Secondary ulcers: they develop from primary mucosal lesions (e.g.: rupture of a vesicle/bulla).

25.1.1. **Primary ulcers**

• Aphtae,

• Behçet disease,

• Infective ulcers,

• Ulcers as manifestations of systemic diseases.

**Recurrent aphtous stomatitis:**

Predisposing factors:

• Genetic predisposition,

• Local trauma,

• Hormonal influences,
• Gastrointestinal disorders,
• Vitamin deficiencies,
• Hematological disorders,
• Viral and bacterial infections,
• Autoimmunity,
• Stress.

Clinical forms:
• Minor aphtous ulceration (Mikulicz’s aphta),
• Major aphtous ulceration (Sutton aphta, Periadenitis mucosae necrotica recurrens),
• Herpetiform ulceration (Cooke’s aphta),

Minor aphtous ulceration: One or few painful ulcers less than 10mm in diameter (lentil sized) with sharp borders, surrounded by erythematous area. Healing takes 7 to 10 days and leaves no scar. Recurrence varies individually. Predilection site: non-keratinized mucosa

Differential diagnosis: herpetic stomatitis.

Major aphtous ulceration: A very painful, crater-like, usually single ulcer with a diameter from 1 to 4 cm, covered by a yellowish-grayish pseudomembrane. They are hard on palpation because they penetrate to submucosa and therefore heal with scarring. Predilection site: mainly non-keratinized mucosa (soft palate bucca), but may occur on keratinized mucosa as well.

Differential diagnosis:
• Cancerous ulceration,
• Decubitus ulcer,
• Erythema exsudativum multiforme,
• Tuberculosis ulcer,
• Syphilitic ulcer.

Figure 3.412. Figure 1. – Major aphtous ulceration
Herpetiform ulceration: Multiple, pinhead-sized, superficial, pseudomembrane covered ulcers that may occur anywhere in the oral cavity.

Differential diagnosis:
- Herpetic gingivostomatitis,
- Herpangina,
- Allergic stomatitis.

Therapy: There is no known causal treatment, iron and folic acid supplementation, levamisole, local antiseptics.

25.1.2. Behcet's syndrome

Symptoms: reoccurring oral aphthous ulcers, genital ulcers, uveitis with hypopyon. It may also have gastrointestinal, skin and neurological symptoms and may lead to thrombosis.

Diagnosis: There is no specific diagnostic test. The diagnosis is established in the presence of oral symptoms combined with at least two other symptoms.

Differential diagnosis:
- Erythema exudativum multiforme,
- Reiter’s syndrome,
- Aphthous stomatitis.

25.2. Vesiculobullous diseases

25.2.1. Erythema Exudativum Multiforme (EEM)

EEM is a dermatitis with an unknown cause (possibly an immune mediated hypersensitivity), with bullous lesions on the oral mucosa. It has an acute course and tendency to reoccur.
Immunological background:

- Deposition of antigen-antibody immune complexes that cause microvascular damage in the dermis and submucosa.

- Allergens:
  - drugs (barbiturates, phenytoin, penicillins, sulfonamides),
  - bacterial, viral, fungal, parasitic antigens,
  - heteroantigens.

Oral mucosal symptoms: erythematous plaques, subepithelial vesicles and bullae and subsequent erosions, swollen and eroded lips covered by crusts, halitosis, increased salivation, swallowing difficulty, pain, fever. Iris- or target-like skin lesions are common.

Stevens - Johnson syndrome (Ectodermosis erosiva pluriorificialis):

A more severe form of EEM in which symptoms are more pronounced and involve the esophagus, larynx, eyes (conjunctivitis, corneal ulcers) and the genital areas.

Lyell syndrome (toxic epidermal necrolysis):

The most severe form of EEM with extensive mucosal and skin lesions all over the body. A life-threatening condition.

Diagnosis: clinical picture, blood test, histological examination.

Differential diagnosis:

- Pemphigus,
- Pemphigoid,
- Bullous lichen planus,
- SLE,
- Behcet’s syndrome,
- Reiter’s syndrome.

Therapy: local administration of antiseptics and steroids, systemic administration of antihistamines, oxytetracycline, immunostimulants.

**Figure 3.413. Figure 2. – Erythema multiforme**
3. Reconstructive dentistry

Figure 3.414. Figure 3. – Erythema multiforme

Figure 3.415. Figure 4. – Erythema multiforme
25.2.2. Pemphigus vulgaris

A severe skin and mucosal disease characterized by the formation of intraepithelial vesicles and bullae.

Etiological factors: An autoimmune disease. Immunoglobulin G type autoantibodies are produced against glycoproteins of the cell membrane of epithelial cells.

Oral mucosal symptoms: oral lesions precede skin lesions in 60% of cases. Fragile bullae are formed without inflammatory border on the palate, tongue and bucca, sized 0.5-4 cm in diameter. As these bullae rupture they leave behind a painful superficial ulcer.

Skin lesions: Bullae filled with clear fluid are characteristic with 1-2 cm in diameter all over the body.

Diagnosis: clinical picture (positive Nikolsky’s sign), histological picture (presence of Tzanck cells), immunofluorescent microscopic examination.

Differential diagnosis:
- EEM,
- Pemphigoid,
- Bullous lichen planus,
- SLE,
- Behcet’s syndrome,
- Reiter’s syndrome.

Therapy: systemic administration of high dose corticosteroids and other immunosuppressants, local antiseptics and corticosteroids.

Figure 3.416. Figure 5. – Pemphigus vulgaris (bucca)
Figure 3.417. Figure 6. – Pemphigus vulgaris (hard and soft palate)

Figure 3.418. Figure 7. – Pemphigus vulgaris (immunofluorescent: C3)
3. Reconstructive dentistry

Figure 3.419. Figure 8. – Pemphigus vulgaris (immunofluorescent: IgG)

25.2.3. Pemphigus vegetans
A more benign, granulomatous form of pemphigus.

Oral symptoms: formation of a whitish mass of tissue around the angle of the lip and on the buccal.

Skin lesions: Bullae and erosions of intertriginous areas.

Differential diagnosis:

- Leukoplakia,
- Candida infection,
- Cancerous ulceration.

Therapy: systemic and topical corticosteroids.

**Figure 3.420. Figure 9. – Pemphigus vegetans (tongue)**

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**25.2.4. Bullous Pemphigoid**

An autoimmune skin and mucosal disease characterized by the formation of subepithelial bullae. Usually manifests over the age 60. Autoantibodies are produced against the basement membrane.

Mucosal lesions: Bullae filled with yellowish fluid on the gingiva in 20–40% of cases.

Skin lesions: bullae filled with fluid.

Diagnosis: clinical picture (positive Nikolsky’s sign), histologic examination (negative Tzanck test), immunofluorescent microscopic examination.

Differential diagnosis:

- Pemphigus vulgaris,
- Bullous and erosive lichen,
• EEM.

Therapy: local antiseptics, topical corticosteroids.

25.3. Infectious diseases

25.3.1. The most common bacterial infections

25.3.1.1. Ulcerative gingivostomatitis

Pathogens: Fusobacteria and Spirochetes.

Predisposing factors: smoking, immune deficiency, lack of oral hygiene, stress, malnutrition.

Mucosal lesions: Necrotic ulcers primarily on the interdental papilla that progress along the attached gingiva towards the oral mucosa, covered with a whitish-grayish pseudomembrane. Contact ulcers on the lips and buccal mucosa. Halitosis.

Differential diagnosis:

• Herpetic gingivostomatitis,
• Pyostomatitis vegetans,
• Leukaemia,
• Agranulocytosis,
• Pemphigus vegetans.

Therapy: hydrogen peroxide rinses, metronidazole, antibiotics, improving oral hygiene, painkillers.

25.3.1.2. Streptococcal gingivostomatitis (Coccal gingivitis)

Pathogens: beta-hemolytic streptococci.

Oral lesions: swelling and redness of the gingiva and surrounding mucosa, suppuration, erosions on buccal mucosa, tongue and lips. An immunosuppressed state is a risk factor.

Differential diagnosis:

• Infectious mononucleosis,
• Diphtheria,
• Candida infection,
• Scarlet fever.

Therapy: antibiotics, treatment of the underlying disease.

25.3.2. The most common viral infections

25.3.2.1. Primary herpetic gingivostomatitis

Pathogen: Herpes simplex virus.

Oral lesions: formation of multiple small (1-2 mm diameter) vesicles. As they rupture, rounded ulcerations with an inflammatory halo can be seen covered by a whitish-yellowish pseudomembrane that heal without scarring. A prodromal phase and subsequent systemic symptoms are characteristic. It usually affects children (between the age of 2 and 6)

Differential diagnosis:
• Ulcerative gingivostomatitis.

Therapy: self limiting, local and systemic symptomatic treatment with painkillers, antifebriles. Increased fluid intake is necessary.

25.3.2.2. Recurrent intraoral herpes

Pathogen: Herpes simplex virus.

Oral lesions: vesicles and rounded erosions anywhere in the oral cavity.

Cause of recurrence: reactivation of viruses in trigeminal ganglia following stress or immune suppression.


25.3.3. The most common Candida infections

25.3.3.1. Acute pseudomembranous candidiasis (thrush)

Pathogen: Candida albicans.

Oral lesions: Patchy white plaques that are easy to remove by rubbing. Rubbing reveals an erythematous, ulcerative surface.

Differential diagnosis:
• Lichen planus,
• Leukoplakia.

Therapy: topical antifungal drugs.

25.3.3.2. Chronic atrophic candidiasis (denture stomatitis)

Pathogen: Candida albicans.

Oral lesions: Atrophic, erythematous, sometimes ulcerated mucosa under denture baseplates. Often combined with angular cheilitis.

Differential diagnosis:
• Contact allergy.

Therapy: proper cleaning of denture, topical antifungal drugs.

Figure 3.421. Figure 10. – Chronic atrophic candidosis of the palate and angular cheilitis
26. 3.26. Oral mucosa diseases III. (The most common precancerous lesions and oral manifestations of systemic diseases) – Ivan Mandel

26.1. Premalignancies
Precancerous lesion: is a morphologically altered tissue in which cancer is more likely to occur than in its normal counterpart.

Precancerous condition: is a generalized state associated with a significantly increased risk of cancer. (WHO 1997)

Table 3.28. Table 1. – Precancerous lesions and conditions

<table>
<thead>
<tr>
<th>PRECANCEROUS LESIONS</th>
<th>PRECANCEROUS CONDITIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leukoplakia</td>
<td>Oral lichen planus</td>
</tr>
<tr>
<td>Erythroplakia</td>
<td>Sideropenia</td>
</tr>
<tr>
<td>Chronic actinic cheilitis</td>
<td>Chronic discoid lupus erythematosus</td>
</tr>
<tr>
<td>Abrasive precancerous cheilitis</td>
<td>Syphilitic leukoplakia</td>
</tr>
<tr>
<td>Keratoacanthoma</td>
<td>Xeroderma pigmentosum</td>
</tr>
</tbody>
</table>

Precancerosis may be facultative if the chance for malignant transformation is less than 30% in more than 5 years, or may be obligatory if the chance for malignant transformation is almost 100% in less than 5 years.

26.1.1. Praecancerous lesions

26.1.1.1. Leukoplakia

Leukoplakia is a chronic white patch of the oral mucosa larger than 5 mm that cannot be removed by rubbing, cannot be defined as any other definable lesion and is not a result of any physical or chemical causative factor except for smoking.

Predisposing factors of leukoplakia:

• Smoking,
• Alcohol consumption (spirits),
• Consumption of spicy food,
• Bad oral hygiene,
• Genetic risk factors,
• HPV infection.

Clinical forms of leukoplakia:

• Homogenous (simplex) leukoplakia.
• Non-homogenous leukoplakia
  • Verrucous,
  • Nodular,
  • Erythroleukoplakia,
  • Candidal leukoplakia.

Figure 3.422. Figure 1. – Homogenous and non-homogenous leukoplakia of the tongue
Figure 3.423. Figure 2. – Non-homogenous erythroleukoplakia

Figure 3.424. Figure 3. – Non-homogenous leukoplakia (verrucous)
Predilection sites of oral leukoplakia:

- buccal mucosa,
- tongue,
- retrocommissural area,
- palate,
- lips,
- floor of the mouth.

Differential diagnosis:

- Fordyce’s spot,
- Morsicatio buccarum,
- Linea alba,
- Lichen planus,
- Candidosis,
- Frictional keratosis,
- Leukoedema,
- White sponge naevus.

Diagnosis: presence of etiological factors, clinical picture, biopsy.

Monitoring: brush and punch biopsy.

Rate of malignant transformation of leukoplakia lesions: 3-8%

- homogenous form 1-3%,
- verrucous form 24%,
- erosive form 38%.

Lingual and sublingual leukoplakias have the highest rate of malignant transformation.

### 26.1.1.2. Erythroplakia

An erythematous patch of the mucosa with a velvet-like surface that cannot be attributed to any other pathology. An obligatory precancerous lesion.

Symptoms: mild burning sensation while eating, sensitivity.

Predilection areas:

- soft palate,
- buccal mucosa,
- tongue,
- floor of the mouth.

Differential diagnosis:

- Acute atrophic candidosis,
- Median rhombic glossitis,
- Discoid lupus erythematosus,
- Erosive lichen.

Diagnosis: clinical picture, biopsy.

Therapy: surgical excision, cryo- and laser surgery.

### 26.1.1.3. Actinic (solar) cheilitis

A chronic inflammatory reaction of the lips (especially lower) as a result of chronic exposure to sunlight, wind and chemicals (agriculture). If left untreated, ulceration or hyperkeratosis of the lesion can occur (Cheilitis abrasiva praecancerosa Manganotti).

Clinical picture: elastosis, macrocheilia, crusting, atrophy.

Differential diagnosis:

- EEM,
- Candidosis,
- Radiogen cheilitis,
- Lupus erythematosus,
- Pemphigus vegetans.
26.1.2. Praecancerous conditions

26.1.2.1. Oral lichen planus (OLP)

An oral mucosal manifestation of Lichen ruber planus. It may affect only mucosal surfaces (oral and genital) or may be combined with skin lesions as well.

Predisposing factors:

- autoimmune origin (disturbances of cellular immunity),
- stress,
- systemic diseases,
- viral origin,
- side effects of medication,
- chronic irritating factors.

Clinical forms of OLP:

- reticular,
- annular,
- papular,
- plaque form,
- atrophic,
• ulcerative,
• bullous (rare),
• pigmented (very rare).
• sclerotic and atrophic.

Predilection site: molar region of the buccal mucosa, tongue, lips, gingiva, sublingual area, palate.

**Table 3.29.** Table 2. – Differential diagnosis of different clinical forms of OLPC:

<table>
<thead>
<tr>
<th>RETICULAR FORM</th>
<th>PAPULAR FORM</th>
<th>BULLOUS FORM</th>
<th>PLAQUE FORM</th>
<th>ERYTHEMATOUS (ATROPHIC) FORM</th>
<th>ULCERATIVE FORM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morsicatio buccarum et labiorum (chronic cheek and lip biting)</td>
<td>pseudomembranous candidosis</td>
<td>pemphigus and pemphigoid</td>
<td>leukoplakia</td>
<td>erythroplakia</td>
<td>pemphigus and pemphigoid</td>
</tr>
<tr>
<td>Morsicatio linguae (chronic tongue biting)</td>
<td>Koplik's spot</td>
<td>linear IgA dermatosis</td>
<td>leukoedema</td>
<td>erythematosus candidosis</td>
<td>Oral manifestation of Crohn's disease</td>
</tr>
<tr>
<td>epithelial lysis of the mucosa (toothpaste, mouthrinse)</td>
<td></td>
<td></td>
<td>White nevus of the mucosa</td>
<td>systemic and discoid lupus erythematosus</td>
<td>drug side ulcers</td>
</tr>
<tr>
<td>discoid lupus erythematosus</td>
<td></td>
<td></td>
<td></td>
<td>pemphigus vulgaris</td>
<td></td>
</tr>
<tr>
<td>Hairy leukoplakia</td>
<td></td>
<td></td>
<td>acid burn of the mucosa (aspirin)</td>
<td></td>
<td>pemphigoid</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Erythema exsudativum multiforme</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 3.426.** Figure 5. – Reticular OLP on the bucca
Figure 3.427. Figure 6. – Annular lichen

Figure 3.428. Figure 7. – Reticular and papular lichen
3. Reconstructive dentistry

Figure 3.429. Figure 8. – Skin lesions of lichen ruber planus

Figure 3.430. Figure 9. – Skin involvement in lichen ruber planus
Diagnosis: etiological factors, clinical picture, histological examination.

Therapy:

- white OLP lesions: vitamin-A oil,
- red OLP lesions: vitamin-A oil, topical anaesthetics, retinoids, levamisole, topical steroids, cryo- and laser surgery.

**Table 3.30. Table 3. – Malignant transformation rate of OLP forms**

<table>
<thead>
<tr>
<th>AUTHOR</th>
<th>PUBLICATION</th>
<th>NUMBER OF CASES</th>
<th>PERCENTAGE OF MALIGNANT TRANSFORMATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rajentheran R., McLean Nr.</td>
<td>Eur J Surg Oncol. 1999 Oct; 25 (5)</td>
<td>832</td>
<td>0.80%</td>
</tr>
<tr>
<td>Lo Muzio L., Mignogna Md.</td>
<td>Oral Oncol. 1998 Jul; 34 (4)</td>
<td>263</td>
<td>5.32%</td>
</tr>
<tr>
<td>Mignogna Md., Lo Muzio L.</td>
<td>Oral Oncol. 2001 Apr; 37 (3)</td>
<td>502</td>
<td>3.70%</td>
</tr>
<tr>
<td>Eisen D.</td>
<td>J Am Acad Dermatol. 2002 Feb; 46 (2)</td>
<td>723</td>
<td>0.80%</td>
</tr>
<tr>
<td>Lanfranchi-Tizeira HE</td>
<td>Med Oral. 2003 Jan-Feb; 8 (1)</td>
<td>719</td>
<td>6.51%</td>
</tr>
</tbody>
</table>

26.1.2.2. Sideropenia

A chronic iron deficiency that leads to hypochromic microcytic anemia. Usually affects women.

Predisposing factors:

- chronic blood loss,
• decreased iron intake or impaired absorption.

Symptoms: lip fissures, angular cheilitis, atrophy of the tongue papillae.

Differential diagnosis:
• atrophic candidosis,
• pernicious anemia,
• ariboflavinosis.

Therapy: causal treatment is the competency of an internist, the dentist should only provide symptomatic treatment.

26.1.2.3. Discoid lupus erythematosus

An autoimmune collagenosis with skin and mucosal involvement that usually affects women.

Oral symptoms: A well-demarcated rounded or elliptical painful lesion with central atrophy, ulceration and with white, radiating keratotic striae at the edges. Usually occurs on the buccal mucosa.

Differential diagnosis:
• erosive OLP,
• erythroplakia,
• EEM.

Diagnosis: clinical picture and histological examination.

Therapy: immunological therapy, dentists should only treat symptoms.

26.2. Oral mucosal manifestations of systemic diseases

26.2.1. Neuroendocrine diseases

26.2.1.1. Acromegaly (pituitary gland hyperfunction)

Hyperfunction of eosinophil cells of the frontal lobe of the pituitary gland

Clinical picture: prognathism, macrocheilia, macroglossia, gingival enlargement, enlargement of the ears and nose, fissured tongue, hyperplasia of filiform papillae, sialosis, hirsutism, hyperpigmentation.

Diagnosis: medical history, patient examination, x-ray, CT scan, MRI scan.

Therapy: Surgical treatment of the pituitary gland, irradiation, bromocriptine as an adjuvant.

26.2.1.2. Hypopituitarism

A rare condition with the hypofunction of the pituitary gland.

There are no specific mucosal symptoms. The associated anemia may cause mucosal atrophy and may lead to burning mouth syndrome.

Differential diagnosis:
• Addison’s disease,
• Myxedema,
• Anorexia nervosa.
Diagnosis: medical history, patient examination, laboratory tests, CT, MRI scans.

Therapy: treatment by an endocrinologist (hormone supplementation).

26.2.1.3. Hyperthyroidism

Hyperfunction of the thyroid gland.

Oral manifestations: no specific oral symptoms, early eruption of teeth, burning mouth, osteoporosis that may lead to fractures of the jaw bones.

Differential diagnosis: tumors of the thyroid gland.

Therapy: treatment by an endocrinologist.

26.2.1.4. Hypothyroidism

Hypofunction of the thyroid gland.

• In infants it causes cretism, a severe mental and physical developmental disorder: flat and broad face, large protruding tongue, decreased muscle tone, dry brittle hair, delayed tooth eruption, enamel hypoplasia.

• In childhood and adulthood it results in myxedema: macroglossia, macrocheilia, red and dry mucosa. It is often associated with chronic mucocutaneous candidosis.

Differential diagnosis: amyloidosis, acromegaly.

Therapy: treatment by an endocrinologist.

26.2.1.5. Cushing’s syndrome

Hyperfunction of the adrenal gland (adenoma or carcinoma).

Symptoms: central obesity (‘moon face’, ‘buffalo hump’), muscle weakness, high blood pressure, parotid enlargement, telangiectasias on the face, hyperpigmentation of the oral mucosa, increased susceptibility to bacterial and fungal infections, tendency to bleed.

Therapy: treatment by an endocrinologist.

26.2.1.6. Addison’s disease

Decreased function of the adrenal gland (cortex).

Oral symptoms: diffuse hyperpigmentation and aphytous ulceration of the oral mucosa.

Differential diagnosis:

• famial hyperpigmentation,
• melanoplakia,
• malignant melanoma,
• Peutz-Jeghers syndrome.

Therapy: treatment by an endocrinologist.

26.2.2. Metabolic and nutritional diseases

26.2.2.1. Diabetes mellitus

A complex metabolic disorder that primarily affects insulin mediated glucose metabolism of cells, also characterized by protein and lipid metabolism disorders.
Oral symptoms: xerostomia, glossodynia, impaired taste sensation. Diabetes may also lead to the development of chronic candidosis, angular cheilitis, periodontal abscess, OLP and lichenoid reaction.

Differential diagnosis:

- Sjögren’s syndrome,
- Radiation induced oral symptoms,
- Diseases of the salivary glands,
- OLP, Candidosis, periodontal disease affecting healthy individuals.

Therapy: treatment by a diabetologist. Dental treatment consists of symptomatic treatment (topical antimycotic drugs, artificial saliva, local antiseptics).

**Figure 3.431. Figure 10. – A periodontal abscess of a patient with diabetes mellitus**

**Figure 3.432. Figure 11. – An oral lichen planus of a patient with diabetes mellitus**
26.2.2.2. Diseases related to nutritional deficiencies

Inadequate nutrient intake, impaired digestion and absorption, increased requirements and increased loss of nutrients (vitamins A, B₁₂, B₃, B₅, B₆, B₉, C, folic acid, proteins) may lead to deficient states resulting in different forms of stomatitis, glossitis and cheilitis.

Therapy: nutrient supplementation.

26.2.2.3. Amyloidosis

A disorder of protein metabolism that leads to formation of abnormal, insoluble protein-polysaccharide complexes (amyloids) and to subsequent accumulation of these in various organs causing impaired function. Major forms include: primary, secondary, familial and senile amyloidosis.

Oral symptoms of primary amyloidosis: xerostomia, macroglossia, reddish-yellowish papules on the lateral edge of the tongue, petechiae and purpura on the mucosa.

Differential diagnosis:
- Scleroderma,
- Myxedema,
- Sarcoidosis,
- Kaposi’s sarcoma.

Therapy: only symptomatic.

26.2.2.4. Waldenström’s macroglobulinemia

An abnormally increased chronic immunoglobulin production and deposition in tissues that is often lethal.

Oral symptoms: petechiae, ecchymoses, painful ulcerations, persisting bleeding after tooth extraction.

Differential diagnosis:
3. Reconstructive dentistry

- Amyloidosis,
- Osler’s disease,
- Werlhof’s disease.

Therapy: plasmapheresis.

26.2.2.5. Porphyria cutanea tarda

The most common type of metabolic diseases affecting heme biosynthesis (porphyrias).

Oral symptoms: vesiculo-bullous lesions on the gingiva, angle of the mouth and lips, brownish-blackish erosions, ulcers, atrophy of tongue papillae (a predisposing factor for candidosis).

Differential diagnosis: other vesiculobullous diseases.

Therapy: Causal treatment by an internist/dermatologist. Symptomatic treatment with local antiseptics and antifungal medication.

26.2.3. Oral manifestations of gastrointestinal diseases

26.2.3.1. Crohn’s disease

An immune mediated inflammatory bowel disease that may affect the gastrointestinal tract from the oral cavity to the anus, but most frequently the ileum.

Oral symptoms: pyostomatitis vegetans, erythema and ulceration of the mucosa, granulomatous gingival enlargement.

Differential diagnosis:
- Pemphigus vegetans,
- Candida granuloma,
- Quincke’s edema,
- Hydantoin hyperplasia.

Therapy: causal treatment by an internist.

26.2.3.2. Ulcerative colitis

A type of inflammatory bowel disease with unknown cause that usually affects the colon.

Oral symptoms: pyostomatitis vegetans, non-specific, granulomatous, bleeding ulcerative lesions.

Differential diagnosis:
- Crohn’s disease,
- Candida granuloma.

Therapy: causal treatment by an internist.

26.2.3.3. Coeliac disease

An autoimmune disease of the small intestine characterized by impaired absorption.

Oral symptoms: aphthous ulceration due to iron- and folic acid deficiency, angular cheilitis, atrophic, burning tongue.

Differential diagnosis: other nutritional deficiencies.
Therapy: causal treatment by an internist and local antiseptics.

26.2.4. Oral manifestation of hematological diseases

26.2.4.1. Iron deficiency anemia
Characterized by hypochromic microcytic anemia.
Oral symptoms: atrophic tongue papillae, paleness of the oral mucosa, erosions, ulcerations, fissured lip, angular cheilitis.
Differential diagnosis:
• Pernicious anemia,
• Sjögren’s syndrome,
• Acute atrophic candidosis.
Therapy: treatment by an internist, iron supplementation.

26.2.4.2. Pernicious anemia
Macrocytic, hyperchomic anemia.
Oral symptoms: Hunter-Möller glossitis (smooth, glossy, tender tongue), paleness of the oral mucosa, taste disorder, xerostomia, burning mouth.
Differential diagnosis:
• iron deficiency anemia,
• acute atrophic candidosis.
Therapy: folic acid and vitamin B₁₂ supplementation.

26.2.4.3. Aplastic anemia
A severe disorder characterized by deficiency of all three hematopoietic stem cell lines (pancytopenia).
Oral symptoms: purpura and ulcerations.
Differential diagnosis:
• Agranulocytosis,
• Cyclical neutropenia,
• Leukaemia.
Therapy: treatment by a hematologist.

26.2.4.4. Malignant neutropenia (agranulocytosis)
Almost complete lack of granulocytes in the peripheral blood.
Oral symptoms: necrotic ulcerations without inflammatory halo.
Differential diagnosis:
• Leukemia,
• ANUG,
3. Reconstructive dentistry

- Major aphthous ulceration.

Therapy: treatment by a hematologist, local antiseptics.

26.2.4.5. Leukemias

26.2.4.5.1. Acute myeloid leukemia

Oral symptoms: they may be the first symptoms of the disease. Deep ulcerations of the gingiva and mucosa, loosening of teeth, gingival enlargement.

Diagnosis: clinical picture and blood test.

Differential diagnosis:
- Ulcerative gingivitis,
- Infectious mononucleosis,
- Agranulocytosis,
- Hodgkin’s lymphoma.

Therapy: treatment by a hematologist, local antiseptics, antibiotics, antifungal medication.

26.2.4.5.2. Acute lymphoblastic leukemia

Oral symptoms rarely occur: bleeding and enlarged gingiva, jaw pain.

Differential diagnosis: from other types of leukaemias.

26.2.4.5.3. Acute monocytic leukemia

A rare type of leukemias but it often produces oral symptoms: red gingival enlargements, ecchymoses, gingival bleeding, ulcerations.

Differential diagnosis:
- Ulcerative and herpetic gingivostomatitis,
- Gingival hyperplasia caused by hydanoin.

Therapy: treatment by a hematologist.

26.2.4.5.4. Chronic myeloid leukemia

Oral symptoms are not as characteristic as in the acute form: gingival enlargement, gingival bleeding, deep and slowly healing ulcers, loosening of teeth.

Differential diagnosis:
- Epulis,
- Ulcerative gingivostomatitis,
- Gingival hyperplasia caused by hydanoin,
- Agranulocytosis.

Therapy: treatment by a hematologist.

26.2.4.5.5. Chronic lymphoid leukemia

A leukemia with the slowest progression.
Oral symptoms: ulcerations, bleeding.

Differential diagnosis:

- Other types of leukemias.
- Other ulcerative diseases.

Therapy: treatment by a hematologist.
Chapter 4. 4. Oral surgery

1. 4.1. Clinical Anatomy – Gabor Gelencser

1. Animation

On the video the position of the midface and the mandible compared to each others can be seen, and also exist the differences between the bones’ form and quality. Depending on which part of the skull is damaged by the acting force, how great is the force, of which direction is, and what a bone status of the patient (age, dentition status) has, facial fractures can be greatly various. As much as every human’s splanchnocranium and trauma are also individual, there are no same fractures, injuries and fracture lines. Therefore, the supply of the patient can not be conventional. Every cases is different.

1.1. Mandible

This is a horseshoe shaped, prolated and narrow bone. Compared to its cross section, it is quite long and curved in more planes, and also has two points of support. Consequently, less power is enough for its fracture.

By the mandible, at the fracture categorisation according to the localisation we mention the parasymphyseal and symphyseal-, ramus-, and joint fractures from this point of view according to important jaw parts. As a matter of course, the pars alveolaris above the corpus, and the mentum can be also mentioned as a seperate part. Rarely its fracture can be identified, as well.

Figure 4.1. Figure 1. – The mandible’s special form, it’s position in the face and the thin soft parts shielding all contributes to its affection to get injured

The trauma, which affects the part of the mentum or the corpus mandibulae, efforts the pounding force on to the processus condylaris or the counter side molar region’s lingular part. As a matter of fact a force, which is properly big and comes from a small surface, can cause a direct, on identical place created lumpy fractures.

In case of teeth-attendance, the symphysis, corpus and angulus fractures can be considered as open fractures because the fracture line goes through either of the teeth’s paradontium, in the event of intact gingiva or even a dislocation-free fracture.

1.2. Musculature
The musculature, which is responsible for the opening and closing movements of the mouth, and the direction of the fracture line has a role in whether the fracture is dislocated or not. In the case of a favourable fault line some muscle groups compress the broken ends almost in anatomical situation. However, in a contrary case the muscles abduct the broken ends which cause a huge dislocation.

**Figure 4.2.** Figure 2. – Muscle groups which are responsible for the common fracture dislocation. Muscle groups, which are located under the mandible and open the mouth and the mandible, become responsible for the up- and in moving of the mandible segment, in case of unfavourable fracture line. The masseter, temporalis and the pterygoid muscles dislocate entand and upwards

![Muscle groups](image)

The rear masticatory muscle group, which is mostly responsible for the termination of the mouth, pulls the broken end upwards and entand. /masseter, pterygoid and the temporal muscle/

The lower mouth-opening muscles dislocate the anterior fragment downwards and entand. / m. genioyodeus, m. geniglossus, m. mylohyoideus /

**1.3. Inferior Alveolar Nerve**

It can get injured on the course of corpus mandible fracture, causing provisional and permanent paraesthesia on the area of the lower lip and mentum.

**Figure 4.3.** Figure 3. – Content of the mandible canal. The trigeminal branch which spreads inside the bone, under the pars alveolaris in subapically unconstant depths. Due to its position and the goodly perineurinum, the sensory defects, which are derived from its injuries, are generally provisional

![Content of the mandible canal](image)

Its’ fibrictic sheath protects the nerve even in the case of a 1-1,5 cm dislocation.
There are different grades in the injuries of the nerve: Grade I = neurapraxia, the strain, contusion and haemorrhage of the nerve without the lesion of the fibre. Grade II = axonotmesis, axonrupture is inside the nerve bundle. Grade III = neurotmesis, with intact perineurinum. However, the endonemium discontinues. Neuroaxonal regeneration is questionable here because of the fibrosis. Grade IV = neurotmesis with the saving of the epineurinum, where everything else has interrupted. Grade V = complete transection of the nerve stump.

1.4. Midface

In case of its injury, the maxilla, palate bone, zygomatic bone, nasal bone, - lacrimare and the vomer as well as the ethmoidale bone can be broken. Mostly the fractures can be considered as open fractures because the fracture line almost always reaches the nasal cavity and catches paradontium more infrequently.

These bones have significantly different structures, or constructions, as the lower jaw. Characteristically in the midface, the thin bone and large air-filled, paired outgrowths of the nasal cavity exist. This is the reason why this piece of the fractures are broken more often, and why the situation can not always be reconstructed precisely, or sometimes totally not at all. This does not mean that the functional results are not good, that only means that the total anatomical restoration is not so important here, and sometimes is also not possible.

2. 4.2. Etiology – Gabor Gelencser

There have been huge changes in etiology in the last fifty years. Partially it has been caused by motorisation. Naturally, we can see large differences in the datas of the countryside and towns, in the developed and developing countries. Although, generally we can state that most of the malar bone fractures are caused by fights and traffic accidents. Much less frequently we meet fractures after falls (typical in older patients), sport and household accidents and gunshot wounds with consequent fractures.

The pathological fractures also must be mentioned, but these are so small forces (causing the injury) that would normally produce no fractures in healthy bone. In the background bone system diseases (osteoporosis, Paget's disease, brittle bone disease), cancer with bone involvement, and chronic bone infections occur.

The iatrogenic mandible and malar bone fractures make a segregated group. Usually tooth-removal causes fracture on the mandible and on the malar bone. More frequently the maxillary tuber breaks during the removal of a molar wisdom-tooth. Although, there have been mandible fractures which could have been connected to the removal of an impacted wisdom-tooth.

Males suffer from mandible and malar bone fractures often than females. This type of trauma affects mainly the young aged group and the young middle ageds.

If the jaw-lesion happens due to a fight, mostly the mandibular angle and the joint will get injured. By traffic accidents, corpus fracture is more common.

Characteristically, fights cause malar bone and lateral midface fracture, an injury with bigger energy can generate central bitty midface fracture.

Most of the injuries are double or multiple fractures.

In childhood, the area of the symphysis is the weakest point of the mandible, therefore symphyseal mandible fractures are very common in that age. This area becomes later the most gross and resistant part of the mandible, so we can diagnose fractures at this area very rarely. It is more frequent in the parasymphyseal part.

Apropos of the condylar fractures, a mention must be made about the age differences: around the ages of 20 and 30, corpus/angulus and contralateral condylar fracture caused by fighting, is typical.

The same aged group and the middle ageds are touched mostly in bilateral subcondylar or intracapsular joint fracture with the connected uni- or bilateral corpus fractures, caused by traffic accident/mentum, face hit to the steering wheel or to the dashboard. Falling on mental protuberance, cheek, which is characteristic in childhood and in old age, causes bilateral high joint fractures. In childhood these flops generate symphyseal fractures also.
3. 4.3. Fracture Types – Gabor Gelencser

3.1. Open and closed fractures

We differentiate them by their relation to the external world.

Closed fractures on the midface are only the fractures of the lateral orbit frame and the isolated zygomatic arch fractures without penetrating dermal injury. Fractured of the ascending limb and processes of the jaw, kinking without dislocation and attendance of wisdom tooth or beside of a completely impacted wisdom tooth, and the childhood greenstick fractures are all closed fractures.

Of course these closed fractures are no longer closed if, injured skin or mucosa, and bone surface is exposed, is uncovered. In such cases, the fractures are always open, require serious antibiotic therapy in addition to other surgical treatment.

3.2. Other Fracture Types

Incomplete fracture or rupture and infraction. These fractures in most cases heal without treatment. Sometimes, however, we operate incomplete fractures, but only if no discontinuity at the base can be seen, but forms a step between the teeth themselves. These interdental gap can be 1-2 mm large, that means quite a malocclusion, the reduction and fixation is required

Greenstick fracture is a typical but not too common fracture in childhood and in old age. Smaller energy causes such injuries, bone, skin remains intact, we see dislocation rarely, these are always closed fractures.

Complete fracture is the far most common fracture type, which is generally roaming with dislocation. It is an open fracture which necessary needs fixation.

Fractura impacta or impacted fracture, fractura communitiva and fractura defction, in other words the lumpy and deficiency fractures are all caused by the affect of a large energized injury or rarely by shot lesion. These types of fractures need operative treatment and the reposition usually needs quite serious surgical work.

Fractura pathologica, which has been mentioned in the Etiology chapter, rather affects the lower jaw-bone than the upper one.

Fractura complicata, in this case not only the bone but other structures can also get heavily injured. For example by condylar fracture the meniscus, articular surface, by the lower mandible the inferior alveolar nerver or the facial artery, either other vascular and neural configurations can get injured.

3.3. Fracture with dislocation and without dislocation

Fractures with dislocation, but with axis deviation are almost always greenstick fractures. Bone periosteum allows no deflection but a minimal axis deviation is possible (non-negligible party of joint fractures). The fractures without dislocation and deviation are usually incomplete fractures.

However, those fractures are more common where the broken ends move. These movements can be:

- longitudinal, the fracture ends slide apart or together. (Dislocatio ad longitudinem cum contractionem -A-, cum distractionem -B-),
- axis (dislocatio ad axim -C-),
- sideward (dislocatio ad latus -D-),
- rotating (dislocatio ad periferiam -E-).

Figure 4.4. Figure 4. – Classification of dislocated fractures according to the broken ends’ motion correlated to each other
4.4. Localisation – Gabor Gelencser

4.1. The Le-Fort classification

At the categorisation according to the localisation, several criterias can be taken into account at the midface fractures. Many people use the old division and mention separately the malar bone fractures, and the(special central and centrolateral midface fractures) Le-Fort I, Le-Fort II and Le-Fort III fractures. This classification lets apart from attention many other fracture types. In fact, we meet classical or bilateral Le-Fort fractures extremely rarely because the impact on the patient’s face is never in the middle or even not symmetric.

Furthermore, in the traffic accident cases the impact or the bruise has significantly more energy than those which have been used by Henry Le-Fort in his experiments on skulls, more than hundred years ago. Consequently, the Le-Fort I-II-III fractures are always more complicated then he wrote it down.

4.2. Classification due to the localisation

We often use the lateral-centrolateral and central midface fracture classification, which is definitely better in the sense that all non-classical fracture types can be grouped in, but no one takes this occlusion into account.

4.3. Classification due to the original or changed occlusion

Therefore, nowadays probably the most preffered is that type of midface classification, which affects or does not affect the occlusion.

4.4. Localisation and distribution according to age

Different studies show suprisingly big distincts in the numbers and percents of the differently localisated fracture’s frequences. Usually the lower jaw-bone fractures are a bit more common, than the midface fractures.

The mandible body-, mandible angle- and condylar fractures on the lower jaw-bone have been described over 20%. We classify the fractures of the condylar to intracapsular, high subcondylar and subcondylar fractures. However, symphyseal, parasympyseal and mental region fractures are significantly rear. The number of the ascendant limb- and muscular process fractures are both under 5%.

The number of malar bone fractures in the midface is extremly high. These can be complete malar bone fractures with the little or big displacement of the cheek bone block, even with the impaction backwards to the midline’s direction. These complete zygoma fractures can be further classified to mono- and multifragmental fractures. As a matter of course, less energy impact or shock can also cause incomplete zygomatic fracture when only one or two breaks from the four suspension points of the malar bone. In this case, althought there is step formation, the zygoma does not move as a whole in the atypical medial and posterior direction. The sagittal maxillary body fracture and Le-Fort III fractures are quite rare.

5. 4.5. Diagnostic Methods – Gabor Gelencser

The midface or mandible fractured patients are ordinary in a good general condition, if only few bones got injured. The situation is different in case of a patient, who got injured in a traffic accident, because commonly more organs and organ systems get lesioned at the same time. In such cases the injuries of the face bone cause nearly the smallest complaint for the patient. Naturally, their supply can not be overlooked. The bone reconstruction has to happen in maximum two weeks, if the patients condition is good enough. Optimal if the politraumatised patient, after the stabilization period, gets supplied by more professions during one narcosis.
The operation method of a broken limb does not preclude the simultaneous provision of the malar bone for example.

The supply of an isolated fractured faced patient happens in a department of the oral and maxillofacial surgery. The bone reconstruction can be delayed for a few days but the supply of mandible fractures, which causes serious complaints, has to happen as soon as possible. The extreme haemorrhage and edema, which are always accompanying the midface fractures (these cause much less complaints), are always the largest in the 1-3 days after the accident. The lessening of these after a few days makes a lot easier the surgeon’s work.

The clinical examination of the patient, the X-ray examination happens after through physical inspection. The malar bones, mandible and the surrounding soft part get examined, at first, extraorally, then intraorally. First of all, the examination of the face and of the scalp happens. The harness and mucous membrane injuries, crushed or broken damages’ size, shape, the accurate description together with the trauma are not only part of the medical documentation. In case of traffic accident and scrimmage, it becomes part of the legal documents.

5.1. Extraoral Examination

5.1.1. Examination of the ear

It is necessary to examine the ear and auditory canal, too. If blood or liquor come out of the ear, they are both diagnostic signs. Ear bleeding may be caused by a serious condylar fracture which has crashed through the wall of the auditory canal but in this case, the eardrum is intact and there is no middle scala injury. If fluid is flowing out of the ear, which seems to be serious and it is presumably liquor, the fracture of the scala media is suspected. In such a case, the liquor leaves to the external world through the ruptured eardrum.

Figure 4.5. Figure 5. – There does not have to be necessarily facial fracture behind the bleeding of the external auditory canal. If the trauma has resulted condylar fractures, it is important to think of the injury and the auditory canal or serious damage of the cranial fossa

5.1.2. Examination of the eye region

The examination of the eyes and orbit is essential. In case of periocular injury, it is important to exclude the damage of the bulbus and optic nerve. Nevertheless, after the examination of field of view, it is necessary to make the examination of the eye movement. The herniation of the lower orbital contents can hamper the side and upward movements of the eye which eventuates double vision. In the case of orbit base fracture with huge defect, the bulbus can be placed lower than the other one on the intact side, and its movement is hampered in some directions. In most cases of midface fractures conjunctiva haemorrhage, ecchymosis periorbitalis and edema, which can hinder eye opening completely, can be seen.

Figure 4.6. Figure 6. – After a midface trauma, typical symptoms. At times, an eye-movement uneasiness, which is sometimes transitional but sometimes permanent, can be added to the unfavorable aesthetics
These haematomas are mostly harmless and elapse in a few days. They hardly cause complaints to the patients.

Nevertheless the patient complains about complete loss of vision a few minutes after the accident. In such a case probably a retrobulbaris haematoma has been formed, which has put the optic nerve under compression. Blood has no opportunity to leak next to the intact orbit or next to the perfectly inslating soft part hernia and as the optical fibers of the nervus are sensitive to the variation of pressure, thus blindless develops rapidly. If surgical decompression happens in one or two hours after the accident, blindless can be transitional. Otherwise, the patient’s injured eye remains blind permanently.

In the cases of malar bone and Le-Fort II-III fractures, bone scala formation laterally and below on the orbital frame would be tactile but unfortunately in most cases major haemorrhage and edema covers it.

It is worth to ask for an ophthalmologist concilium, in case of eye and orbit trauma to make the documentation of the preoperative status after the injury.

5.1.3. Examination of the nose

The extraoral examination of midface fractured people also includes the nasal examination. The sinus maxiale mostly gets haemorrhaged in case of midface fractures. (A wall of it gets injured due to the zygomatic bone fractures or the Le-Fort fractures.) Blood leaves through the nasal cavity because the sinus orifice outlet is in the middle nasal meatus. In addition, other reasons of nosebleeding can be nasal bone fracture with deviation, with or without deformity and anterior scala fracture. Although, in this case usually liquor also leaves in the direction of the external world.

5.1.4. Examination of the midface

During the midfacial examination the periorbital edema and the spectacles haematoma can be eye-catcher, just like the prolonged midface and emphysema, air, which got between the tissues. We examine the sensation or the loss of sensation in the infraorbital nerve supply area.

5.1.5. Extraoral examination of the lower jaw

We palpate the outside contour of the lower jaw. In case of dislocated corpus or angulus fracture, the discontinuities if the basis is palpable, despite the haemorrhage and swelling. Examine also the motion of condylars with the little finger placed in the ear canal. (Movement of a condylar, which has luxated out of the valley, can not be detected.) In case of condylar fractures with a little or without dislocation, it is typical to generate pain in the joint, by pusing the mentum of the opened mouth. Articular fractures are associated with deviation during mouth opening but also with a less or greater degree of limited mouth opening.

5.2. Intraoral Examination

In mandibular fractures intraoral examinations give more information than the extraoral. In most cases we meet with striking malocclusion. This malocclusion may occur in the tooth row, or even out of the row of teeth, when angulus fractured, or ramus, or joint.

Most of the mandible fractures cause eye-catching malocclusion. There can be occlusial interferences causing steps ormation in the jaw. Premature molar contact, which has been formed in connection with angulus fracture or condylar fracture. Crossbite and anterior open bite are the most unequivocal fracture signs. However, in these cases the cleavage can not be seen within the jaw. The swelling around the fracture, sublingual haematoma and difficulty in swallowing are typical. Most patients with corpus and angle fractures have a numbness of the lip on the injured side. At the alveolar process fracture of the upper jaw, at the very rare zygomaticomaxillaris fracture intense malocclusion can be seen with or without soft part haemorrhage. In the case of a sagittal maxilla fracture, the soft part rupture along the midpalatal sutur indicates the bone injury above the soft part.
4. Oral surgery

Figure 4.7. Figure 7. – The occlusional discrepancy is the most straightforward by the displaced fractures of the lower mandible, in the jaw. This implies an unequivocal fracture, which can be seen even by a layman. Not only because of the scala formation between the teeth but also because of the commonly torn gingival.

Figure 4.8. Figure 8. – A condylar fracture, which absolutely dislocates the whole pars alveolaris (vid. 8th Figure) or angulus fracture can be confused with a total condylar luxation, but taking into account the patient’s complaints and anamnesis, makes rare real misdiagnoses.

6.4.6. Clinical Symptoms – Eniko Orsi

The narration of the patient facilities making the diagnosis. On several occasions the history and the patient’s superficial inspection is enough to make a correct diagnosis. For example, complaint of a cracked lesion in the mentum region, frontal crossbite and the hit of the mental protuberance foreshadows a condylar fracture.

Mandible fracture produce characteristic symptom of the region, in every localization. (Depending on the size of the dislocation.) It is necessary to discuss the certain and uncertain signs of fractures before we would consider the range of typical symptoms of single type of fractures.

6.1. Signs that refers to fracture

Pain, swelling, which can be edema, haematoma, and paraesthesia, bleeding and soft part injuries are all uncertain sings. Deformation, in case of jawbone’s malocclusion, abnormal mobility and creption (crackling sound caused by the friction of the broken ends to each other) are certain sighs of a fracture./ There can be malocclusion without a fracture if the patient has suffered mandibleluxation./

We can diagnose safely without imagery if we notice any of the certain signs. As a matter of fact, nowadays there is no fracture diagnosis without radiological method, therefore we have to support the diagnosis with a radiogram, at least. The fracture which can be seen on the radiogram is also one of the certain signs of fractures, of course.

6.2. Mandible Fractures

Despite the ruptures, joint and angulus fractures without displacement, or processus muscularis fractures, we are always aware of occlusional deviation. The complaint caused by this, depend on the dental status also, because if the patient is toothless or there are only very few antagonist teeth in the mouth, campitulum motion even with
4. Oral surgery

a large dislocation will not bother the patient, as bad as a millimeter size dislocated condylar bothers someone with intact toothing. A large force of impact with the resulting dislocation of parasympathetic, corpus and angulus fractures or those that are being disimposed by the muscles, because of their unfavorable declaration, causes marked complaint. A less displaced fracture can be more bearable. Nevertheless, mandible fractures have to be supplied in a few days because they it is difficult to talk, eat and often swallowing exists.

6.2.1. Parasympathetic – symphysis Fractures

Bone structure in early childhood of the symphysis mandible is weaker than in other regions of mandible. The fact that in the first years drops, mostly the chin gets bigger hit, laws that fractures in the region of symphysis are very common. It should be noted that in the context of the fall, the force, usually affects as big power on the area of the condylars, as that they get indirectly fractured.

In adulthood, the region of the symphysis of the mandible is the thickest in the labiolingual direction. Furthermore, the cortical layer there is the most leveled. Therefore, only a very small part of adulthood mandible fractures are diagnosed in the mandible.

Figure 4.9. Figure 9. – Bleeding, gingival rupture, horizontal-running and well-mobile fracture line are characteristic. Inability to bite. Sensory loss is not typical

There is step formation between the anterior teeth, usually with medium or small dislocation and with abnormal mobility. The fracture line is vertical or oblique directioned. The gingiva generally bursts, but these fractures should be considered as open fractures, even in case of intact gingiva, because the fracture line goes through the teeth’s paradontium. The patient’s complaint includes pain, but no sensory loss. The presence of condylar fracture always have to be excluded!

6.2.2. Corpus Fracture

If the fracture occurs in this region, it is usually goes along the canin, which is obvious, because the thick canine root significantly weakens the bone, so it extends from the middle to distally. The vertical fracture line along the canine does not always, but the fractures behind usually causes loss of sensation. This is because the fracture line affects the foramen mentale of the mandible canal, therefore the included nerve formulas as well.

In the background of corpus fractures, there are often car-accidents and other high-energised injuries. Consequently, we usually meet fractures with large dislocation, which is hardly reducible. Most opening and mouth closing muscles can also contribute to the dislocation of the broken ends. The lingual bone plate often shows greater movements than that, which is observed on the alveolar process. In this case sublingual haematoma is common. The gingiva can be ruptured and the lingual artery gets injured sometimes also, which can result even bigger haematoma. Contralateral condylar fracture is common as well. Crepitation and abnormal mobility are normally detectable.

Figure 4.10. Figure 10. – At large deformation fractures there is soft part rupture and we can also notice haemorrhage from the sulcus. Bleeding into the external world can be a bit scary in the case of fractures with big soft part discounting. However sometimes the gingiva does not interrupt on a big surface or does not interrupt at all. Nonetheless, deeper vascular structures can tore. Sooner or later the bleeding tamponate itself. Therefore a sublingual haematoma develops which lifts the tongue and causes slurred speech. After a few days the blood effusion starts to sink due to the gravitation and makes blue-yellow-green discoloration on the neck
6.2.3. Angulus Fracture

Most of the cases they are dislocated fractures. Some of them has a favourable fracture line (fracture ends can be compressed by the pterygomassateric muscles to each other, resulting a tiny dislocation or no dislocation at all), but the unfavorable fracture line produces much longer dislocations.

If the fracture line from the alveolar process extending obliqually towards the base, the pterygomasseteric muscle mentle holds the broken ends together and the patient mentions only slight malocclusional complaint. (favorable fracture .) However the fracture line extends forwards and upwards from the bottom, the disintegrative effects of the muscles can be bigger than one centimeter, causing occlusing confusions. In these cases, numbness can be always found and the patient has to be well informed about, that it probably not only temporary. ( Unfavorable fracture) The broken end usually causes gingival rupture, which often occurs on the lingual side.

Wisdom teeth, which have not been grown yet and are mostly vertically impacted, predispose to angulus fracture. In this case the surgeon has to make a decision. From one hand , the existence of wisdom tooth often easiers the fixation after the reposition. On the other hand to keep the wisdom tooth clearly means a second operation. The plate removal and tooth extractions are optimally planned six months after the primary intervention. Sometimes the tooth makes a repositional block. In such a case, the tooth should be extracted.

If the angle fracture is bilateral, it makes an extremely large dislocation and unpleasant opened bite for the patient.

Figure 4.11. Figure11. – If the fracture of the mandible angle dislocates to different directions of space, in different measures. Sometimes the reposition, even of a one or two millimeter size dislocation with the occlusion-correction is a huge job for the surgeon, even with intraoperative mandibulomaxillary fixation. The keeping of a teeth (usually wisdom teeth), which is on the fracture line, depends on its position entireness and the measure of impaction. The extraction of the tooth before the reposition makes the restoration of the bone much harder. One solution should be that we first do the fixation ( tooth is still in the bone ) , then after osteosynthesis we remove the tooth, of course surgically , with the help of a few buccal bone removal, if it is needed. Toothdissection also facilitates the lifting of the tooth. The 11th Figure illustrates a tooth whiteness ( mesioangular slope ) , and behind it exists a fracture with dislocation. Intraoperative photograph, mucoperiosteal flap is done, before to the reduction.
6.2.4. Ramus fracture

It is very rare. It can be a vertical or horizontal fracture. Depending on the displacement, it is connected to malocclusion. Conservative supply does not really make good results. The open reduction and fixation of the fracture is more optimal.

6.2.5. Fracture of the processus muscularis

It is a rare fracture type. It is isolated but sometimes it associates to other fractures. High-energized shock on the zygomatic arch can cause the fracture of the arch and the fracture of the muscular process below. It does not make serious compliment. Conservative supply is good enough.

6.2.6. Fracture of the condylar process

In the case of condylar fracture without dislocation or, fracture with less deviation but without dislocation, the symptoms are significantly smaller, than by patients with the condylar fractures with bigger dislocation. Bilateral abnormal molar contact is characteristic. In case of condylar fracture it can be noticed on both sides and it is connected with frontal open bite.

Figure 4.12. Figure 12. – In untreated cases masticatory deficience and occlusal disfunction can occur and result dyspepsia or indigestion. The treatment is whether conservative or operative, assumes reposition and fixation

Apropos of a fracture, the condylar dislocation can happen in mesially or sometimes laterally. Considerably rarely the condylar perforates the skull and gets into the scala media. Another fracture type is more common. In case of this fracture the condylars’s distal part gets compressed, its volume gets lower and its shape changes, due to the effect of trauma. As a result small complaints could be mentioned. If it couples with haemorrhage in the articular capsule, we have to be afraid of the subsequent ankylosis. In most condylar fracture cases, typical “dislocation ad axim” happens. If this axis deviation is small, the condylar stays inside the socket. We can feel its movements through the auditory canal.

Figure 4.13. Figure 13. – There is a fracture on the picture, which causes significant articular luxation. We put our little fingers into the right and left auditory canal of the
Patient and ask them to open their mouth. We do not feel the movement of the articular condylar.

The size and direction of the fracture-causing force and the fracture line, all play role in the measure and direction of dislocation. In a bit more optimal cases, the broken ends stay in touch, there is bone contact, but in some cases the bone contact absolutely misses. In such cases it is exresential to wait for an optimal form of bone recovery without intervention. Usually the patients mention one frontal or sidewards trauma of the chin of mental protuberance. By children and old ageds, it is mostly falling or bum, by adults it is mainly slamming to the dashboard or fight.

In the region of the joint, pain can be temperate but it intensifies by putting pressure on the mental protuberance while the mouth Is opened. Mouth opening is limited. Mental protubarence deviates to the injured side when the mouth is open. The mandible can be only hardly moved laterally. The molars contact earlier because of the shortening of the ascending limb. We can notice condylar movement in case of fracture in socket, but if the distal broken end left the socket due to the fracture, the articular socket will be empty. Preaurical moderate concavity can be seen but condylar movement can not be felt or seen.

Very rarely bleeding starts from the ear. The fractured and dislocated sharp broken end perforates sometimes the exterior auditory canal and the haemorrhage begins. The ear drum is intact and tranquil. Median scala fracture, which can be associated with joint fracture can lead to liquor discharge beside of a ruptured eardrum.

6.3. Fractures of the midface

6.3.1. Fractures which do not affect the occlusion

6.3.1.1. Nasal fractures

They can be accompanied with nasal deformity, sensibility of the bridge of the nose, pathological movability, edema, oral breathing or with large volume epistaxis. In connection with sport accidents occurs often isolated, but frequently with other midface fractures.

6.3.1.2. Isolated Orbit Fracture, Blow Out Fracture

Mostly it occurs with the outbreaksing of the lower orbit wall, but sometimes the medial or lateral orbit wall can get injured without orbit frame fracture.

The bones of orbit wall are paper-thin. This kind of fracture occurs when the orbit frame does not fend or partcularly fends the hit on the orbit content. The orbit content is able to be umpressed but the thin orbit walls can not bear this pressure, so the walls break out. Commonly, the patients mention special trauma. In case of this fracture the eye should get contact with an object, which has a small surface, does not destructive but has enough force to break out the orbit wall in any direction.

The bone defect’s size is normally a few square millimeter. In extreme cases the defect can be that huge, that the bulbus sinks spectacularly in to the maxillary sinus through the bone defect. In such cases occurs more than one millimeter difference between the vertical position of the pupils.

Usually periorbital haemorrhage and edema also occur.
The fracture can be small or it can has as special position, as the patient can not tell complaints expect the acute complaints. Nevertheless, the leading symptoms are the lack of eye movement, usually while looking upwards, and the consequential double vision. If the bone defect is big, the bulbus can sink. Injury of the intraorbital nerve can also happen.

The optical nerve rarely gets injured, therefore the quality of eyesight does not change, neither on the traumatic eye. If there is no spectacular bulbus sinking, the patient indicates double vision while looking upwards and downwards. The reason of it is one of the ocular muscles’ (usually the lower straight ocular muscle) or fatty tissue herniated into the bone’s effect. As a result the movement of the eye becomes limited.

Double vision can occur even without marked hernia, if a small outbroken bone chip wedges in one of the ocular muscles and offends it. The traction test can be positive with and without explicit hernia, also. In such a case we try to move the eyeball in every direction, by catching the conjunctiva with an anatomical clip. Looking upwards usually, looking laterally sometimes can be blocked.

Isolated orbit fracture can be accompanied with infraorbital nerve paraesthesia and epistaxis.

Figure 4.14. Figure 14. – In case of that fracture which can be seen on the 14th Figure, a significant sized soft part hernia got into the sinus from the orbit. In association with the bone breakout the bone fragments did not lose their connection with the periosteum. There is no real fracture only a door-like tilt, which is foreshadowed by the coronal record. If the tolerance of the patient lets it, these cases are perfectly suitable for lower restrain of the maxillary autrumal balloon, so the bulbus gets its original position back. It is favorable to expose the orbit base independently and put the herniated tissues back to the orbit, under eye control. Otherwise the loss of motion can persist despite the aesthetic restoration. Bridging the defect with foil, titanium mesh or own bone, are also good solutions.

6.3.1.3. Malar Bone Fracture

The most common one is the complete malar bone fracture which causes marked facial asymmetry. It can be monofragmental or multifragmental. The zygoma dislocates into medial and posterior directions. Besides the facial dissymmetry, numbness, it can cause mouth opening backlog and eye movement disorder. The impression fracture of the arch leads to concavity on the skin over the fracture. The incomplete malar bone fracture does not cause facial asymmetry because the zygoma stays in its place. Only step formation can be noticed above the fracture line, but these isolated fractures also can cause functional deviation. Fracture at the lower margin entails with numbness, fracture of the isolated arch entails with mouth opening limitation.

Fighting is very common in the background of zygomatic fractures. If it is associated with central midfacial fracture, then traffic accident etiology is more common.
We never see only one fracture line in case of a typical, complete malar bone fracture. Fracture lines on the lateral orbit frame and on the lower orbit frame are visible and palpable. In such cases, the bone fractures are all along the zygomaticomaxillary suture and the zygomafrontal suture. In fact, any orbit frame fracture can occur some millimeters or centimeters away from the suture also. Fracture along the zygomaticotemporal suture and sphenoidal suture fracture usually associates with the upper suture fractures. Fracture of the lower orbit frame runs towards the front side of the maxilla and often goes through the infraorbital foramen. If bone-outbreak can be noticed there, the fracture is associated with the stacked congestion of the plates, the patient indicates numbness on the injured side of the face, the same side of the nose and anteriors. Fracture partly on the front side of the maxilla does not cause significant complaint despite theese. The alveolar process will not become mobile. The maxillary sinus frequently haemorrhages, which causes epistaxis.

The orbital periphery swells, becomes edematous and spectacles haematoma is produced. Subconjuctival haemorrhage is also typical.

The zygomatic fracture is also orbit fracture at once, because the orbital part of the malar bone forms partially the bottom and side wall of eye socket.

Fracture of the lateral orbit frame causes only pressure tenderness, but the fractures on the arch are associated with multiple concavity, it causes mouth opening limitation, because the temporal muscle is directly under the arch, hanged on the coronoideal process.

Complete zygoma fracture is rarely associated without dislocation. The dislocation is to be expected as a rule in accordance with the direction of the acting force, that means that the malar bone gets placed inwards and backwards. Facial asymmetry is formed and the shape of the orbit changes.

In contrast to theese, incomplete zygomatic fracture causes only some isolated fractures on the lateral frame or lower frame, or on the arch. Sometimes it causes two at once, but the zygoma stays in its place. Therefore, the number of symptoms reduces and they localisate there, where the fracture has happened.

**Figure 4.15.** After the trauma, the explicit swelling and haemorrhage of the injured region and eye periphery covers the symptoms which become obvious later. After trauma the patient usually takes the numbness connected to n infraorbitalis injuries as a matter of course and transient. The dislocation of the bone block can not be seen, even if it is significant, because of the local swelling on the face. After three or five days, when the numbing persists, the patent starts to notice the deformation of the injured side, so the zygomatic fracture gets examined and diagnosed only then. The issue is that, the chance of a successful closed percutan reposition gets smaller due to the soft party, which has been formed in and around of the broken ends. Later intervention needs open exposure and fixation

6.3.2. Fractures which affect the occlusion

6.3.2.1. Dentoalveolar fracture

It is rarely isolated. Associated to Le-Fort fractures. Gingiva rupture, explicit pathological movability and occlusional fermentation characterizes it.

**Figure 4.16.** There is an elderly patient on the picture, who fell to an iron pipe with his face. The right side toothless alveolar process is in uncountable pieces with extreme dislocation beside a destructive soft part injury
6.3.2.2. Fracture of the tuber maxillae

Frequently it is not reasoned by a drop or fight. The patients usually suffers it from a tooth extraction. A deeply undervaulting sinus recessus, a solely wisdom tooth or a second malar tooth and gothic palate are presponding factors. In these cases the beginning of tooth removal is often hard. In addition, when it finally moves, it can be felt that it could be moved with a bigger bone block.

6.3.2.3. Sagittal maxilla fracture

Sometimes the hit of the chin does not cause fracture on the condilars, but it leads the energy towards on to the upper jaw and ruptures it in the midline, along the palate suture. The lack of teeth on the upper frontal area and open bite are both predisposes. The tense gingiva on the hard palate is not that stretchy, therefore this type of fracture is almost always associated with a gingival rupture along the palate raphe. The generated oronasalis connection is quite unpleasant for the patient.

6.3.2.4. Le-Fort I fractures

In a bit simpler way: the pars alveolaris of the maxilla, under the aperture piriformis and above the tooth root sunders from the other parts of the maxilla and from the other parts of the midface. The fracture line runs on both sides from the vomer to outside through the aperture piriformis, into the pterygopalatinal region on the frontal wall of the maxilla. The alveolar process becomes quite mobile but we do not notice pathological movements on the radix nasi and on the infraorbital margin by moving the teeth together with the bone. It does not always associate with great swelling of the face, the soft parts near the eyes neither hemorrhage, but the patient is not able to bite or chew.
Figure 4.18. Figure 18. – The horseshoe-shaped maxilla part, which carries the teeth, gets isolated from the upper part of the maxilla. It was caused by a horizontal hit which was directed a bit downwards and reached the pars alveolaris of the maxilla from the front.

6.3.2.5. Le Fort II Fracture

It is a pyramidal fracture. The fracture line goes through the nasofrontalis sutura, lachrymal bone and ethmoid cells onto the medial-lower wall of the orbit, then forwards on the infraorbital canali, then starts to extend downwards on the frontal wall of maxilla, along the zygomaticomaxillar suture, then rides backwars on to the pterygomaxillary region.

Figure 4.19. Figure 19. – For the first sight they seem to be eye symptoms.
The broken, pyramid shaped area gets placed lower. By moving the frontal teeth, the fracture which is usually palpable on the radix nasi and sometimes on the orbit frame, becomes unequivocal. But this can be hardly examined physically few days after the trauma, because of the periorbital edema and haemorrhage. The occlusion can change moderately or expressly. The patients can complain about premature molar teeth contact. Usually numbness attends, which touches the upper lip and frontal teeth. Eye movement disorder and mouth opening limitation is not too common. We rarely meet symmetric and bilateral Le-fort fractures. The combinataed form is more common: there is Le-Fort II fracture on the one side and Le-Fort III on the other, sometimes only one-side Le-Fort II fracture.

6.3.2.6. Le-Fort III Fracture

The face and neurocranium become separated from each other. The nasofrontal suture, lachrymal bone and ethmoid cells are affected. The fracture line runs out to the lateral orbit frame through the back side of the orbit.
along the zygomaticofrontal and temporal suture and from there out, backwards to the pterygomaxillary field. Step formation on the lower orbit frame and typical facial numbness are missing from the symptoms. We can cause pathological mobility out of the radix nasi by moving the frontal teeth. The midface is elongated and flattened because of its back-placement.

**Figure 4.21. Figure 21. – Leader eye-symptoms without the deterioration of the eye movement function with explicit fracture line on the orbit frame and on the radix nasi, often with damaged nasoethmoid region**

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**6.3.2.7. Zygomaticomaxillary fracture**

It is quite rare. At the frontozygomatic and temperorozygomatic suture, the zygoma sunders from neurocranium. However the maxilla’s alveolar process also breaks down, therefore they move together mesially, causing the marked malocclusion crossbite of the pathological side and facial asymmetry. Of course, it produces the other symptoms of zygomatic fractures also.

**7. 4.7. Radiological Diagnostics – Eniko Orsi**

Although mostly the presence and localization of fracture can be found with physical examination, its exact location, dislocation, single or multiple nature, relationship to the soft part structure becomes unequivocal with different radiological methods. Analysis and evaluation of X-ray and CT-scans from this region are tasks for the maxillofacial surgeon and radiologist as well.

Mostly we can determine with palpation which bones are affected by the fracture or multiple fracture because the certain signs of fractures and symptoms, which characterizes and associated with the fracture, peaches on the fracture.

However only the illustration methods can provide pieces of information about the fracture lines, the one by one position of fracture lines in case of coarse fracture and the direction of dislocation.

As a matter of course, operations can be planned easier with theese extra pieces of information. Radiological findings are part of the accurate documentation they help to make the consequences and terapy of the injury.
understood for the relatives, the postoperative result can be checked with radiograms after the operation and can be used for education, as well.

If we localize the injured bone area with physical examination, we suspect the localization of the fracture. Therefore we can choose the most optimal type of radiogram.

Though nowadays the clinical centers and large hospitals make three-dimensional CT scans in every cases when the patient has suffered malar bone trauma, it is good to exclude the allatory injuries of the skull and the brain, the small treatment centers still use simple radiograms. If there are selected well, we can get greatly useful pieces information of the fractures of the malar bones.

7.1. Radiographies

7.1.1. Orthopantomography

Orthopantomographia is greatly suitable for the certificate of mandible fractures.

Figure 4.22. Figure 22. – On the first radiograph there is an impact lower mandible. The periosteum of the base integrity can be traced, there is no interregnum

Figure 4.23. Figure 23. – On the second picture a left-side fracture without explicit dislocation has been diagnosed. In such a case, the ir almost no occlusional compliment, pain is only expressed under load and there can be numbness. It is not sure that such a fracture becomes visualized on a classical AD scull picture or on an off color OP radiograph

7.1.2. Condylar radiograph

The condylar radiographs illustrate the ascending limb with the processes in case of open and closed mouth. The condylar fractures can be quite precisely localized with this.

Figure 4.24. Figure 24. – We can see on the radiograph the small intermingling displacement of the left side articular condylar. The condylar is in the socket, the case is
suitable for MMF after the enlightenment of the patient. The patient chose the operative therapy from the surgical and conservative therapies

7.1.3. Occipitomental exposure

For the detection of the upper mandible and the malar bone occipitomental expose is used. On this picture, the area of eye sockets and malar bone can be seen well, as well as the maxillary sinuses.

Figure 4.25. Figure 25. – The radiograph shows well the dislocation of the left side zygoma block. The orbit frames are asymmetric, the eye socket narrows on the pathological side, there are 2-3 millimeter sized stepformations on the lower and lateral eye socket frame. Even the step, which seems to be big, can be diagnosed better according to the radiograph, in case of a freshly injured patient because the periorbital edem and haemorrhage makes non-evaluable the physical examination. We notice sooner the zygoma dislocation, which can be seen on the radiograph, but not always palpable at the steps, since the zygomatic tuber rather takes place inboard, than rising out from there
7.1.4. Axial exposure

The fractures of the zygomatic arch can be seen perfectly.

Figure 4.26. Figure 26. – The radiograph shows well the flattening of the left side malar bone arch by the sporting of 3 fracture lines, the impressure is typical. This fracture form is more common in the cases of malar arch isolated injuries, than complete malar bone fractures. In cases like that, usually the broken ends slide simply on each other.

7.1.5. Exposure from lateral direction

The laterally made skull exposure is mostly used for the detection of the orbit base and anterior and posterior walls of the skull.

Figure 4.27. Figure 27. – We can identify the enterior and posterior walls of the sinus and the orbit base beside the mandible. It gives relatively right information, but not always with a good quality. The summarising of the two sides is also disturbing. It does not substitute the CT examination in case of orbit vase fracture suspicion.

If there is no opportunity to make a CT, the a radiogram could be also useful.

This fracture form is more common in the cases of molar arch isolated injuries, than complete malar bone fractures. In cases like that, usually the broken ends slide simply on each others.

In cases of midface fractures, pre and postsurgery occipitomental record, in cases of mandible fractures, just like this, two ortopantomogramms, weather the patient had a CT or not, are part of the pre and postoperative...
4. Oral surgery

documentation in clinics. The success of the operation, the quality of the reposition and fixation can be the best compared to those preoperative radiograms which have been made by one radiologist and on the same advice.

7.2. CT

CT records, in case of ideally thin slices, shows square centimeters of the splanchnonarium by the coronal, axialis and sagittal examinations. Moreover, the 3D splanchnocranium reconstruction, which has been made of the datas, makes sightful and easy to imagine the bones and injuries of the splanchnocranium for the doctor, the patient, and the patient’ relatives.

**Figure 4.28. Figure 28.** – The 3D recod is very sightful. It helps to see in space and full. Usually we show the patient and their relatives the fracture lines on this, the differences of the injured side from the intact one and our plans to during the narcosis. Revealing the mini plate or the screws, which is going to be in the patient for months or for a lifetime, are all part of the enlightenment. We have to calm the patient down that the metals are not allergenic, metallization on their area is minima, the metal detector on the airports will not indicate it and CT or MR can be made of the wearer.

![CT Scan Image]

The anterior and posterior walls of the frontal sinus, nasal septum, posterior and anterior lateral walls of the maxiallary sinus, malar bone with the zygomatic arch, mandible’s condylar, distal end of the ascending limb, alveolar process of the maxilla, palate, lower part of the mandible ascending limb, angulus, pars alveolaris and basis mandible can be seen well on the axial or horizontal section from the calvaria to the mentum.

**Figure 4.29. Figure 29.** – Maybe the horizontal section is the best to diagnose the dislocation of the malar bone block. In the picture, on the right side, a horizontally cut malar bone moved wholly, is dislocated, and terminated the connection with the maxilla and temporal bone, can be seen well. On this radiogram, the sliding of the broken ends on each others can be greatly identified. The adjucnt fractures of the lateral and lower orbit frame are not represented. A fracture on the other side, on the anterior maxilla wall can be seen.
On the frontal and coronal sections we examine the eye socket and orbit base as going anteroposteriorly. Most of the Le-Fort fracture’s fracture lines can be diagnosed here truly. This radiogram helps to diagnose the sagittal maxilla fracture as well.

The segittal sections, completing the frontal ones, clarifies the blow out fractures’ place and size, because due to this slices the fractures of the maxillary sinus’ s anterior and posterior wall are well detectable.

**Figure 4.30. Figure 30.** – We can see the eruption of the left eye base with soft part herniation. If we examine it together with the sagittal picture, we make it easier for ourselves by the fracture exposure because the size of the defect can be greatly estimated.

**Figure 4.31. Figure 31.** – This information is also needed for the opening: how much should we search the fracture mesially or laterally and how low is the lack of bone with hernia.
8. 4.8. Treatment – Eniko Orsi

8.1. Treatments before and nowadays

If the chapter would be only about those treatment alternatives, which are expected today, it would be very short. Nevertheless I consider relevant for the students to get a hysterical overview of the last more than fifty year’s treatment strategy.

During the world wars the injuries of malar bones and cheek bones increased. Its large percentage were gunshot damages, which are very rare nowadays in the war-free zones. The gunshot injuries’s treatment with good function and aesthetics is still hard nowadays. In those times it was mentioned as a bravado.

Formerly there were far less complicated midface fractures, which are connected to traffic accidents and very common nowadays. The simpler bone fracture cases have been treated conservatively. Cases with small dislocation were not even diagnosed. Some luck was needed for good recovery of non-fixed fractures and not to have infection.

The fixation of serious fractures with defect happened with fixatur externe in the last century. After the beginning of antibiotical era, the patients got additional penicillin treatment. At fractures with small dislocation fixations, like the today’s dental splint, were used. When the fracture could not have been repositioned, usage of wirecerclage has happened beside exposure. These fixations gave some reposition for the broken ends, but there were no function stable fixation.

Figure 4.32. Figure 32. – External Fixation Method

Figure 4.33. Figure 33. – Bone S

In the middle of the last century, the traumatology started to use widely the dynamical compressional laminar osteosynthesis, primarily on tubular bones. The available plates and screws which have been used for the fixation
of carpal bones could have been used on the mandible also. It needed bigger exposure, consequently the usage of theese is relegated nowadays.

The upper mandible could have been fixed with splint and wire, because of the inadequacy of the carpal bone plates’s thin bones. The mobile molar bone have been suspended with inner wide ligature to the fix points of the skull. (35th Figure) Metal bone screwed on to the top of the calvaria, could have been used, as well. Most of the midface fractures could be fixed to this.

The inner hangin did not need as big tolerance from the patient as the fixateur externe did, but did not need assure whole immobility.

The reposition of dislocated, wedged midface part without exposure often had difficulties. After the injury it was a quite simple maneuver, happened forcedly with special forceps but the intolerated wedge could have been placed back near to its anatomical position with pulling it for weeks. The occlusion before the trauma could have been restored rarely.

8.2. Reconstruction with miniplates

Nowadays we reconstrate most of the midface and mandible fractures with plates, which are used in handtraumatology, but these are miniatures compared to them. On the upper mandible we also use small mini or micro plates and screws. On the mandible we use a bit stronger ones. On the bones in the area of malar bone and orbit, which has a good quality and there are quite many of them, mini plates can be also used. But in sane cases we use smaller ones on account of the small place superficial plate location.

There have been many experimentations about the tension forces and the plates, which can neutralize theese forces, furthermore with the screw sizes to minimalise the operational loading. The experiments showed, that the usage of big plates and bioartical screws, which need external exposure, strins greater the patient, the aesthetics worsens and does not make such a good result, like the miniplate does on the alveolar process with monocortical screws. On theese smaller plates the tensional forces become neutral and a smaller screw is enough against the contraction of the muscles. Most of the mandible fractures can be treated by inner exposure. ( Despite the condylar fractures many of them can be conservatively treated.) The monocortical teeth do not endanger the roots of the teeth, do not make scar on the face. We do not endanger with the external exposure any facial nerve branches, haemophilia is smaller and the postoperative space of time becomes shorter.

**Figure 4.34.** Figure 34. – The bigger sized lamina, which can be placed only from extraorally to the base and usually needed aggregation dental splin therapy because of the interdental dehisce

**Figure 4.35.** Figure 35. – There is a miniaturized variation, which can be used from intraorally. It has more advantages than disadvantages, therefore it took the autarchy in the last third- quarter of the last century
There is no need to eliminate such a huge tension forces on the upper jaw-bone, malar bone and orbit. During the selection of small, mini and micro plates’s positioning we keep in mind that the measure of the bones in the periphery of the fractured bone, where are suitable for the fractured then reponated part’s fixation. It is often problematic at the reconstruction of the anterior wall of the maxilla because there are no bones where the fracture bone part could be fixed to. (Despite the area next to the aperture piriformis and crista infrazygomatica)

Beside the mini lamina, the drag screws and compressional screws also have to be mentioned. In the case of oblique mandible fracture, the alternative of mini plate and screw usage is good. Three screws give great extensional fixation. In addition, drag screw can be used for high subcondylar or condylar fracture fixation. Fractures in the area of the mentum can be treated with barely screws, as well.

**Figure 4.36. Figure 36.** – Screw fixation (at least 3 screws) happens in case of fracture just like at the planned ortognant interventions when the artificial split ostotoma happens

**Figure 4.37. Figure 37.** – Mentum regional fracture treatment can be seen without plates, functionable

We rarely use reconstructional osteosynthesis with plate. A mention must be made about the absorbable plates and screws which are used at childhood facial traumas.

### 8.3. Immobilisation and bone healing

The immobilization of the broken ends ensure the recovery of the bone. But what is bone recovery exactly? What kind of forms does it have? What happens if there is movement, micromovement between the broken
ends? Namely, why is it so important to prevent the movement between the broken ends with operative or conservative methods?

The recovery of the bone-tissue can be considered as regeneration. We distinguish primer and secunder recovery on the bone, just like the per primary and per secunder recovery types on the skin.

8.3.1. Secondary bone healing

At the secunder bone recovery first we see necrosis. Inflammation, then edema forms with big vasodilation. The haematoma is needed for the recovery cellproliferation’s start. On the area of the fracture, the periostenum makes the epluripotent cells in a great number. Fibroblast proliferation gets started with collagen deposit. The hialinal cartilage formation also begins. The formed cartilage can be found inside and outside of the fracture line. Enchondal bone formation happens and finally the trabecular new bone forms lamelar part.

8.3.2. Primer bone healing

Reposition and immobility, ergo fixation is the precondition of primary ossification.

8.3.2.1. Fissural bone healing

At fissural recovery little gaps and fissures stay between the broken ends. Blood vessels grow inside these gaps from the periostuem, endostuem and havers system, and bring precursor ovums with themselves. If the gap between the bone segment is smaller than 0.3 mm, then immediately a cellar bone forms. The 0.5-1 mm gap charges first with trabecular bone then it changes to lamellar. However, there is no transitional cartilage remodeling in any of them.

8.3.2.2. Contact bone healing

At contact recovery there is no gap between the broken ends. The osteoclasts make tract between the fragments. In theese tracts new bone and bone bridges are formed by the Havers system.

The lack of fixation or not well done fixation cause the chance of secondary bone recovery, even if the fracture was not dislocated. It is simple and not complicated.

Sadly, there is a chance for the formation of inflammation and pseudoarthrosis.

Stable, optimally a function stable osteosynthesis is necessary for a primary bone recovery. Primarily it can be ensuranced by laminar broken end unifaction.

9. 4.9. Conservative Treatment – Eniko Orsi

9.1. Observation

Observation is considered as a conservative treatment because the patient gets medicational treatment sometimes with at least 24 hour long hospitalization after the midface trauma.

Sometimes we do not persuade the patient neither about the classical conservative therapy, nor about surgical treatment after the diagnosis in case of fractures without dislocation, fractures with favorable run, which has been comprimated to each other by the chewing muscles. It is specially true if the patient does not want to have any of the fixation types or it is weak, old or in a poor condition.

If the fracture is opened, the patient gets antibiotical therapy and pain relief in the form of drugs or compress. It is essential to let the patient know that mouth hygiene and keeping the teeth mechanically clean especially incase of fracture of the mesial part of the mandible and the fractures of the corpus and angulus because the fracture line can get easily infected. We propose egg- and milk-free diet with chewing ban. Only pulpy food can be eaten. Rinsing with Betadinesolution or with clorhexidine daily twice is also part of the therapy.

The observed patients have to be called back on control-examinations at least weekly because the fracture, which seemed to be optimal, without or with small dislocation can get inflammed. It can be changed by the impact of a thoughless, stronger chewing movement.
9.2. Conservative treatments

If the fracture does not require surgical treatment but it needs fixation or the patient refuses surgical treatment but after enlightenment undertakes conservative fixation or if the fracture can be hardly fixed surgically (high condylar fracture) but it needs immobility, we make dental splint fixation with wire or gumligature placed on bicortical screws (4,6 interraducal or screws placed over roots) usually for 6 weeks. We can call both of the forms mandibulomaxillar fixation or bimaxillar-intermaxillar fixation.

Figure 4.38. Figure 38. – The less unpleasant but more stable and stronger splint therapy, which gives continuous reposition and bimaxillar or mandibulomaxillar fixation by MMF screws, which can be beared easier. By both of them, we fix the elastically pathological jaw-bone to the intact mandible. Speaking can be carried out hardly but eating is easier.

Figure 4.39. Figure 39. – Though theoretically every type of food can be eaten mixed with a straw (patients with intact teeth also can suck the nutrition behind the last molar teeth and it is even easier for patient with teeth absence), patients can lose 6-15 kg in a few weeks.

Every type of treatment has its advantages and disadvantages. On one hand, fixation with screws spares the paradontium and teeth, there are less foreign substances in the mouth and it can be cleaned well. On the other hand, dental splint fixes the injury on a large surface (it is fixed on 4-5 teeth in every quadrant) and fixes the injured bone zone. The gumligatury versions of fixation let only a few millimeters big mouth opening and the wire form is more rigid. Although, by this type of fixation, very good bone recovery is achievable. The wearing time of MMF for 2, 4 or 6 weeks is very unpleasant for the patient.

MMF fixation is not an alternative of the classical midfacial treatments at the surgical treatments of some corpus-angulus mandible and median lower mandible can be replaced. It is also very useful as the therapy of the not too common sagittal maxilla fracture as the additional therapy of zygomaticomaxillar fractures. It is also used for the therapy of high condylar fractures.
The criterion of MMF usage is the suitability of the patients teeth, namely not to have too many missing teeth. It is also important that the patient has to be able to bear the closure of the teeth. Furthermore, conditions associated with vomiting (concussion of the brain due to the trauma, pregnancy, epilepsy, etc.) excludes the usage of this type of fixation.

A mention must be made about, that the MMF treatment can be a transitional fixation until the definitive surgical supply. Sometimes the open reposition can not be made in a few days but the patient has a mobile fracture with dislocation which causes serious pain and complaints by every speaking attempt. In such a case the broken ends should be in tranquil position, it can happen with screwy method or dental splint.

Sometimes we experience that the fixation of a complicated fracture has not good enough results even with the laminar method. In these cases we use MMF fixation for 4-6 weeks additionally to the laminar osteosynthesis.

MMF fixation of high condylar fractures, where surgery is difficult to be performed happens with wireligature then with a some weeks long elastic fixation. If the occlusion is not perfect, the rigid fixation is not recommended after a few weeks because of the undesirable ankylosis. In case of condylar fracture which is accessible easily surgically and has no or a little dislocation, we offer the two week long MMF therapy for the patient. The fear of operation usually makes the patient to choose the conservative therapy even if we enlight them about the unpleasentness of MMF.

9.3. Different alternative treatments

The Eyelet wire fixation is also a conservative type of fixation but nowadays it is not that used. Its’ only advantage is that it needs only wire, different molded-splints and the Gunning splint. The conservative fixation of edentoulus patient’s mandible can happen with own dentil or with Gunning splint, which is connected to the mandible with concentric sutures.

10. 4.10. Surgical Treatment – Eniko Orsi

10.1. Anasthetic conditions

The supply of the fractures generally needs narcosis, it happens in intratracheal narcosis in the ordinary way. In cases of fractures, which cause malocclusion, the usual control of occlusion during the operation cannot be used. Therefore, the classical tube downconducting through the mouth is not an option. Most of our patients get operated beside nasotracheal intubation.

Figure 4.40. Figure 40. – The tube does not get downconducted through the mouth, it happens from the nasal passage to the pharynx, then gets to the upper respiratory tracks. It needs a bit of proficient and it can cause the accidental brise of the chonchak and epistaxis. But it is an issue only in the case of extreme trismus

10.2. Fractures in childhood
I would like to mention the childhood and old age fractures’s treatment and the differences from the treatment of injured adults, in the beginning of the chapter, separately.

**Childhood Malar Bone Fractures**

Reparative processes are significantly faster in childhood. In the lack of therapy the broken ends get fixed after 6-10 days, therefore we supply childhood fractures in a few days. Splintation beside deciduous set of teeth or mixed dentation is very problematic, even if the child could bear it. In fact, the laminated, screwed osteosynthesis causes difficulties also because of the dental germ.

By children, we usually use conservative methods, sometimes only observative but only in those cases when it can not cause functional deviation or malocclusion which interrupts the fracture. In case of large dislocation, surgical therapy can not be avoided, but the usage of short monocortical screws is very important because of the defence of dental germ. The protection of the anterior wall in the upper jaw-bone has to be thought because of the rudiments. Childhood condylar fracture is very common due to bicycle and motorbike accidents. the expoire of high intracapsular fractures are risky. The conservative therapy has generally good results.

Though, a mention must be made about that quite big percentage of condylar fractures under the age of 12 leads to mandible growing disorders.

If we want to avoid a second plate-removal operation, we can use absorbable plates and screws.

It is a huge mistake to leave the mini titanium lamina in the growing mandible because it can cause serious face asymmetry.

Le-Fort fractures are rare but nasal bone fractures and malar bone fractures occur sometimes. If the fracture causes huge dislocation it must be supplied. Deformed saddle nose and the inability of nasal respiration can be the consequences of nasal bone fracture and septum haemorrhagem if their supply does not happen.

**10.3. Malar Fracture In elderly**

The patients are partly or entirely edentoulus which associates the intense or atrophy of malar bones, the change of blood circulation in the bone and the bones can be osteoporotical, as well. The mandible canal gets close to the surface of the bone. Depending on the quality of the bony substance, we have to make a choice between the classical minilaminated solution and the reconstructive plate placed along on to the base. The placing of the second one can happen through extraoral exposure.

At midface fractures, especially lateral midface fractures we usually choose observation even in the case of fractures with dislocation. In such cases we enlight the patient of the probable facial asymmetry and of the probable permanent facial numbness. By old patients, who are in a bad condition we do not force the burdening surgical intervention. We can not forget the operativ supply if the injury is associated with large functional aviation and the narcosis is not contraindicated due to general condition of the patient.

**10.4. Fracture Line And The Teeth**

I am going to make clear surgical treatment by the operational describing of some predilacional fracture places. But at firstl, some words must be written about teeth in the fracture line. If we start antibiotical supply right after the fracture and surgical intervention happens in a few days, the tooth in the fracture line usually can be kept.

At malar bone fractures, the fracture line gets more rarely vertically between the teeth but we usually can find teeth in the fracture lines on the mandible. This tooth can be healthy but it also can be paradontopathical, vitality-lost, radix or can-not-be restaurated. By angulus fractures, we often meet wisdom tooth, which has not grown out yet, in the fracture line. This plays role usually in the formation of fracture because it can weaken significantly the zone of angulus.

Figure 4.41. Figure 41. – AWe always try to keep the tooth in the fracture line. of course, a tooth, which can not be restaurated, causes complaint, focal or makes harder the reposition, can not stay in the jaw-bone. Even by tooth with uncertain future, we try the keeping of it for the patient’s rquest. But in such a case, the further treatment of the
tooth is needed. Tooth supply after the operation and not giving antibiotics in case of open fractures causes infection of the fracture line or osteomyelitis extremely rarely. There is a non-certanly keepable tooth on the left Figure. (We thought of keeping it because of the easier reposition and fixation)

Figure 4.42. Figure 42. – By the one on the right side, the keeping of the frontal bridge, which is in a bad condition, the teeth need extraction. (The fracture line is along the no.31. tooth. However the no.43. tooth was not in the direct area of the fracture, due to inflammation, which was surrounding the large root, it has been extracted)

The keeping of the tooth in the fracture line is unequivocal if the tooth is healthy or if it is a tooth with intact paradontium and vitality-loss. (Radicular supply is needed as soon as possible.) If its crown is broken, the endodontium is opened but its root is intact or it is an impacted tooth, it does not make repositional obstancle. Keeping the tooth in the fracture line makes anatomical reposition easier and the infactial danger is smaller if we do not leave a big extractional bone wound next to the fracture line.

The tooth is not keepable if it can not be restored or it is mobile-paradontopathical, root-fractured, the root is old or if there is a periapical rarefaction or there is a cyst around its root. We do not keep wisdom teeth if there is a cyst next to or around it or if pericoronitis is happening.

After tooth extraction on the lower jaw we have to pay attention not to cause the dehisce of the basis by fixing the plate interradicularly so high and to avoid the bending of the neighboring crowns in to the extractional gap, causing malocclusion.

10.5. Treatments Of Mandible Fractures

Symphyseal Fractures:

Typical childhood-fracture, often associated with condylar fracture. Sometimes it occurs with adults also but by them, the localization is better parasympyseal.

We can make exact the place and notch of the fracture with an OP-record. We can also get to know the situation and the condition of the rudiments. In case of intratracheal narcissis, the tube has to be driven down through the nose. It can not hinder the checking of occlusion.

If the reposition and fixation happens with a little child, we should not forget that the monocortical screws can not be longer than 4 mm because by injuring or drilling the rudiments under the decidual teeth and we can cause immediatly diversity or prefractional disorders.

Let’s make an incision on the front of the tight and mobile gingival for the exposure of fracture. In case of median fracture we separate the periosteum with the mucous membrane, making the coagulum from the fractural gap with a Volkmann spoon. In case of a few days old gap, we remove the granulational tissues. We
reduce the fracture. We always have to keep in mind that it is a bony reposition and that sometimes we can see occlusional insufficiencies even in the cases of “cell to cell” repositions. If we have to make a compromise, the right occlusion is more important than a broken end connection which seems to be perfect. In fact, it is good to keep trying until the bony reposition is good or excellent and the reposition is the same as the original one. After the manual reposition we choose the optimal-long miniplate and adaptate on to the bone surface. We can use repositional forceps to keep the reduced broken ends. In such cases we drill one-one hole for at least half or one centimeter away from the fractural gap in both two directions, 2-3 mm deep. We put the sharp ends of the forceps into the hole and we keep the reduced position of the broken ends a bit to each others. We put the plate under the forceps subapically and fix it with short screws. By adults, to neutralize the large torsion forces, two plates can be put onto each others. If we use one plate only and put it too deep, onto the bases, we usually experience interdental fracture opening even if the fracture on the basis is disappearing and the broken ends are pushed to each others.

The closure of the wound happens with bunchy stitching. By cooperating patients we better use monofil non-absorptional sewing materials.

After operation we make control radiograph to diagnose the position of plates and the fractural gap. By children, the removal of the plate is proposed after six months. by adults, if the plate interrupts the patient or causes inflammational compliment, the lamina can be removed beside local anesthetization.

**Parasymphyseal Fractures**:

The fracture line is often not vertical but oblique and if it reaches the mental foramen, it can cause numbness. We usually notice triangle-shaped fractures on the basis. The exposures of the paramedian fractures are like the exposures of median fractures. Obviously, the incision line is a bit moved away in the beginning due to the fracture nitch. It is important to idenificate during the lobar transformation the mental nerve and to keep it away and protect it during working. We have to pay attention while we put on the plate, the mental can not push nerve formula. If it is possible, leave the foramen mentale free in a 2-3 mm safety zone.

The symphysealis-parasymphysealis fractures’s surgical solution is the tractional-screw fixation, which has been put onto the fracture line perpendiculary and in two heights. It compregends and compresses the mentum. The removal of these later can be made only hardly, in cases of children, it can not be made. Its insertion needs lobar transformation also. Indeed, the lobe is more enlarged. Pre-drilling is needed before placing the screws beside perfect reposition. The usage of repositional forceps helps.

**Figure 4.43. Figure 43.** – On the preoperative and postoperative OP-record we can compare the situation before and after the operation in case of parasymphyseal fracture. By lobar transformation and by the replacement of plates the protection of the nerve and avoidance of the roots are both important, not only the reposition and occlusion are major

![Figure 4.43](image)

**Figure 4.44. Figure 44.** – On the Figures the fracture did not cause the malocclusion, for that, the angulus fracture was responsible. There was non-negligent dislocation between the broken ends, therefore we can not disregard the osteosynthesis of the frontal fracture

![Figure 4.44](image)
Fractures Of The Corpus Mandibulae:

Typical injury of traffic accidents usually associated with contralateral condylar fractures. If the first examination happens days after the trauma, the patient often mentions that the marked numbness on a big area started to become lighter. In such a case we can be sure that the nerve only got contused or the neural duct haemorrhaged. If the dislocation was big, or if the broken ends press the nerve continuously, or if bonepart hurts the nerve, the numbness does not pass even days after. The numbness can be moderated by exposure and reposition. The dislocation is large in case of bilateral fractures, if the facture line lets the riddles of the mylohyoideal muscle to push backwards and a bit down the anterior bone segment. It is rare that the injury of the nerve, due to the fracture, causes permanent sensory defect. If this happens, all of the neurofibribes of the nerve branch have been torn.

We mention it only as an interesting data that the fixation of corpus fracture happened with fixateur externed in the beginning of the last century. Two bonescrews in front and two bonescrews behind the corpus fracture have been drilled in through the skin. It has been fixed with an external fixational apparate which lets only small movement for the broken ends.

On one hand, there is no need for lobar transformation. On the other hand, there is no eye control during the reposition of the broken ends.

The fixation does not let the patient to sleep comfortable, the external apparatus is disturbing and the bone along the bonescrews can get infected through the skin. Nowadays its usage can be alternative only in case of infected or deficiency fracture. In fact, the reconstructial laminar osteosynthesis seems to be better from every point of view.

Before the minilaminar era, another type of solution was the wire sutural osteosynthesis, which needed extraoral or intraoral exposure. The free bone surface has been drilled in front of and behind the fracture. (1.5-1 cm away from the facture) They threaded wire through the bone and twisted the endings together. It was better to use two wires which has been threaded from different directions. This way further loosenance could have been avoided.

By the Neuer-kind wire suture, they drilled mesially and distally from the fracture. One was over and one was above the canal. The broken ends have been fixed to each others with a wire.

Nowadays the fixation of corpus fractures happen almost only with laminar osteosynthesis.

The small plates, which are used for the time, can be placed onto the alveolar process, under or above the roots can be put only through intraoral exposure. Do not hurt the noble structures of the mandible canal while cleaning the fractural gap.

The usage of repositional forceps can help. The plate has to be fixed with 2-2 screws from the broken end distally and proximally. It means the usage of a plate with at least four or five holes on it. The usage of 3-3 screws, placing two plates onto each others are more optimal.

If the fracture is oblique from buccolingual direction or the sagittal bone surface can be put together on a large surface, we can make stable osteosynthesis with the usage of compressional screws. (minimum 3) If there are roots, we have to pay attention on them because of the longer screws. We also have to pay attention not to hurt the manible canal. The closure of the operation’s area has to happen with knotty stitching. The interruption of the sutural line causes wound separation, hardens the soft part and bone recovery and can lead to bone necrosis.

Fortunately it is rarely needed to supplement the corpusfractures’s laminar osteosynthesis with MMF fixation.
4. Oral surgery

After some weeks of soft food diet, the patient can start to load the fracture which has been strengthened with a plate. Serious diet is not needed anymore.

**Figure 4.45. Figure 45. – Corpus Fractures Before And After Fixation**

![Image](image1)

**Figure 4.46. Figure 46. – The patient’s complete lip and mentum insensibility forms due to the dislocation associated to the trauma and the strain of the nerve. During the operation we noticed the macroscopical intactness of the nerve. Therefore, after the intervention, we told the patient about the probable elapse of the paraesthesia. The lip and mentum insensibility passed completely in 4 weeks**

![Image](image2)

**Fracture Of The Angulus Mandibulae**

It is typically the consequence of fights or sport accidents. By most of the patients we see explicit dislocation with large submandibular and angular swelling, sometimes with haemorrhaged soft parts. In case of formation of an unfavorable fracture line, the masseter muscle, the medial pterygoideal muscle can suddenly pench the broken ends from each others, even for 1 cm away. This can cause the strain or rupture of the nerve and the inferior alveolar artery can tear. In such a case first intensive, then slowly quiet bleeding into the mouth or into the tissues can be noticed.

Wisdom teeth in impaction or which has grown partly out weakens the area of angulus just like canines on the corpus. The young people, who come with angulus fracture usually got injured by heng kicked or hit. We often find third molar tooth under the occlusional plane. The parodontium is generally intersected by the fracture line. Regarding that keeping of the tooth makes easier the reposition and fixation, if there is no pericoronitis we try to keep the tooth. Of course, giving antibiotics is very important and generally we suppose the removal of the plate with the teeth after six months.

The supply of angulus fractures can happen by intraoral exposure but sometimes the patient’s osteal and anatomical particularities require the usage of the transbuccal set. Mucoperiostal flap transformation is needed in any case. This can be envelope flap or triangle flap, the substance is that the bone surface should be accessible. If we can not hook away the soft parts of the cheek and lower bends with a bucca hook, then we prick through the skin and soft parts over the fracture line with a scalpel, then with a trocar. This way we can reach better the injured area with the some millimeter lumende tube without the forcing of the surrounding soft parts. We drill and screw through the trocar. The positioning of the plate happens from the mouth after the adaptation.

Te reposition is often not easy because of the pulling forces of the muscles and the stritching of the broken ends to each others. Always clean the fracture line before. We can not forget the inferior alveolar nerve in some
centimeters deep during the cleaning of a some day old wound with a Volkmann spoon. The hurt of the inferior alveolar nerve is undesirable.

Sometimes the wisdom tooth is a repositional obstacle. (in such a case, removal is indeed.)

The most critical point of this zone’s supply is the adaptation of the plate. In most cases we use external oblique line for the fixation. We put the plate on to this, which has six holes on its back or it is even longer. (Due to the hard placement sometimes we get satisfied with five holes.) The notch of the external oblique line needs the formation of the plate in three planes. After careful transformation happens the input of the screws. In this region we can not use repositional forecaps beside inner exposure, so an assistant is needed to keep the reduced position during the line transformation, the drilling and screwing. There is a special hook for screwing. The occlusion and keeping it in the midline have to be controlled even beside a bone contact which seems to be good.

The MMF fixation, with its bicortical screws and rigid wireligature, makes easier this hard and exhausting work phase by placed on temporarily for the time of the reposition, We leave the intraoperatively drilled MMF screws until suture removal. We remove it with the suture only in case of good occlusion. If the patient complains malocclusion despite the laminar fixation, we can use MMF for some weeks as an additional therapy.

**Figure 4.47.** Figure 47. – Before the intervention, the patient did not dare to open the mouth because of the mobility of the broken ends. Though, the X-ray did not detect serious dislocation

![Figure 4.47](image1.png)

**Figure 4.48.** Figure 48. – One plate placed on in the area of external oblique line is usually enough for suitable stableness. We made the sculption of the wisdom teeth in local anesthetization after six months, together with the line removal

![Figure 4.48](image2.png)

**Condylar Fractures**

One of the most disputed injury type about its supply. Issue of the intracapsular condylar fracture’s supply is rarely caused by hard or risky approach. By prearicula incision we can easily hurt the branches of the facial nerve if we are not enough practiced. Subangular incision makes very hard to approach and look on because of the tightening tissues. Therefore we do not use it in case of intracapsular injuries. This is only one of the reasons. The other is, that as many dental surgeons say that the opening of the articular capsule causes postoperative unpleasantness due to the scaring, that as many dental surgeons propose the exposure of intracapsular fractures. The most accepted manipulation in the articular capsule is the broken ends’s supply assisted with endoscope and laminated with percutan trocar. Most of the clinics agree that the surgical exposure
of the capsule on a big area usually harms more than helps, taking in consideration that patient’s postoperative mouth opening status.

The patient’s complaints depend on the associated injuries. The skin wound in the chin is often squealy. Condylar fracture is commonly associated with contralateral paramedian body or angulus fracture. Bilateral condylar fracture is also common in the middle of the chin, by patients who have been hit from above, beside bilateral condylar fracture, we usually see mandible or midfacial fracture with other localization.

Following the roles of multiple mandible fractures’ supply, we always make the more steril, external interventions first, then the oral cavity operations. Occlusional complaints can be very different due to the variable fracture combinations If the condylar fracture is isolated unilateral, the ipsilateral premature molar tooth – connection dominates as an occlusional disturb.

The patient indicates marked pain on the area of the fracture when we open the mouth and push backwards the chin. Weather we feel articular condylar-motion on the injured side, from the auditory canal or not, it depends on the degree of dislocation and it also depends on weather the broken condylar is luxated outside of the acetabulum or not.

We determine the fracture’s localization with CT, OP or condylarexposure. We decide about the treatment considering the fracture’s localization, the dislocation and the atient’s complaint. If we do not or can not avoid surgical interventions, the patient gets operated from extraoral exposure.

According to our clinic’s point of view, in case of very high or intracapsular condylar fractures, conservative supply should be chosen. After the rehabilitation of our patients in a right way (MMF, then gradual functiotating to avoid ankylozis) the patients do not to have serious occlusional deviation and they can open their mouth well after the therapy.

By the surgically treated patients (high subcondylar fractures with dislocation, lower subcondylar fractures with small dislocation) we use external incision at tha angulus. By the ramus mandible’s posterior and lower one third we separate the masseter fibres. We search for the fracture line under the condylar by going towards the external surface of the ascending limb. We have good chances for the reposision of the condylar, which is usually turned inwards and forwards. If we put a bonehook between the two processes, into the incisures and we move the mandible downwards. The dislocation of the fracture is caused by lateral pterygoideal muscle.

If the broken piece lets it, we can place even two plates onto the condylar. If we put on two plates, we can disregard the “minimum 2-2 screws on to the broken ends’s both side” – rule. At the closure of the wound firstly we stitch the muscle, then the skin. A drain is also gets placed in to avoid large haematoma. We can use intracutan sutural line for the great aesthetics.

Figure 4.49. The laminary fixed articular condylar treats the patient from more weeks of speech and prandial difficulties

Figure 4.50. The MMF indisposes mostly the psychical state as well
Therefore when we can, we choose the operative solution in case of condylar fracture.

**10.6. Midface Fractures**

**10.6.1. Midface Fractures Which Do Not Influence The Occlusion**

*Nasal Bone Fracture*

The supply of the very commonly isolated nasal bone fracture is an otorhinolaryngological task. In our clinic only the midface associated fractures get supplied.

If the nasal bone becomes mobile, gets laminary fixed to the bones next to it, which become stable by the plate, or to non-mobile bones, to the frontale bone or to the maxillae’s part, which is the place of the lower orbit frame.

*Blow Out Fracture*

Isolated orbit base fracture with spared orbit frame is rare. Of course, the orbit base can break out with the margo inferior’s fracture, characteristically associated with malar bone fracture. This form is more common but we do not name it as Blow-out fracture.

Eye movement disorder and double vision are that kind of complaints which worsen the quality of life.

CT, especially the coronal and sagittal segments give good datas about the defect and its position. If we see on the CT that there is a broken piece on the defect’s one or two sides and it is hanged into the sinus by the periostium, then we can make the reconstruction even from the sinus. Nevertheless, if the bones have bursted out and lost their connection with the orbit base, there has to be a bone grafting.

The exposure of the orbit base can happen from more incisions. The transconjunctival exposure would give the most optimal aesthetics but only a small area can be approached by this and the surgeon has a small freedom of movements.

The subciliar section recovers nicely, as well. By this, we make an incision under the lower eyelashes into one of the small eye wrinkles. (2-3 mm at least 2-3 cm long) If the defect is rather medial stated, we make section,
we intersect near to the medial pole. If the defect is situated more laterally, we make the incision more laterally. We find the margo inferior’s periostem under the msculus ocular orbicular muscle’s fibers. We intersect it under the margo or 1 mm lower and start to disconnect it from the bone into the direction of the orbit base. After finding the defect, we pull the herniated soft part out of the sinus. If we can not do or do not want to recover the bone from the sinus, we decide wheather we support the eyeball with bone, titanium mesh or absorbable PDS foil. Defect replacement with own bone is a good solution but it needs another operation to get the bone.

If there are enough bones medially and laterally as well to keep the PDS foil, we choose this. If the bone defect is bigger and not all the sides of the foil can be placed onto the bone, we use the titanium mesh, which keeps its shape and endures the weight of the eyeball. The foil and thetitanium net have to be cut to in to the right shape. The content of the eye socket pushes the PDS onto the edge parts of the orbit base, therefore it does not have to be stabilise. We fix the edges of the titan mesh with microscrews onto the margo inferior.

We check the movements of the eye ball with tractional test, then close the wound in more layers. First we sew the periostium with absorbable sutures, then the muscles, then the skin with monofil autraumatic 5/0 thread.

Making an infraoral section a bit lower into a wrinkle is also a good alternative.

To supply the orbit base from the sinus, we place an inflatable Foley-catheter or atrom balloon into the sinus from the lower turbinate and fill it with physiological saline solution.In case of supercharges, the patient indicates double vision. We can corrige the amount of the fluid.

**Figure 4.52.** Figure 52. – The PDS and titanium mesh, which are used to replace the orbit base defect, are both suitable to keep the bulbus and keep surrounding soft parts in their place beside appropriate indication. In addition, an autologe bonegraft, which can be got by an operation, can be saved. In the picture you can see that we used a titanium mesh for the replacement of a three square centimeters big bone defect. This net is so thin that filigran that it can be hardly seen on the postoperative radiograph. The fracture is not a classical Blow-out fracture, as we had to laminate on the lateral orbit frame, because another fracture is situated also there

![Zygomatic Bone Fracture](image)

Zygomatic bone fracture is the most common form of midface fractures. Lateral midface fracture and other forms are known, as well.

We do not exposure surgically or laminate zygomatic bone fractures without dislocation if it does not cause complaint.

If the zygomatic bone remains in place but there is a distortional fracture on the arch, or if the orifice narrows, we can reponate the fracture closed, according to Gillies. We make a small section in the temporal region and put a repatorium under the muscle’s fascia. While pushing it under the arch we can offset the sunken bone.
The zygoma can remain in place, a little step forms on the margo inferior, causing consequential face numbness. In such a case we expose the nerve area over the infraorbital foramen by using subciliar section. If it is needed, we reduce the bone and restore the anatomical position usually with miniplate.

The monofragmental, few-days-old zygomatic bone fracture is generally suitable for closed, percutaneous reposition with bone hook. By placing the bone hook under the zygomatic body, usually in the width of the lateral eyecanthus we can pull the bone out. In a favorable case, by pulling forward the zygoma, it gets back to its original position and no further fixation is needed. If it stays mobile, we have to expose one of the fracture lines and fix the anatomical situation with a plate. It is the most simple if we do it on the quite superficial lateral orbit frame. We fix it with mini plate and close the wound.

By the multifragmental zygomatic fracture, the percutaneous pulling usually has no results so the zygoma has to be fixed. The supply of those zygoma fractures, which could not have been reduced by pulling out, had happened until the end of the last century’s half by the exposure of the anterior wall of the sinus and by jodoformical tamponage, just like the Luc-Caldwell Operation. Nowadays we do not use this method. These days the to keep the bone sutural in place and fixed is neither used.

At the orbit base injury, associated to zygomatic bone fracture, the original situation is usually get restored. We rarely have to replace defects. If we have exposure the lower orbit frame for fixation, it worth to become considered about that there is no defect, which should be covered by pulling away the periosteum.

Furthermore, in case of supplying zygoma fractures with large dislocation, we have to make a security transaction test on the bulbus conjunctiva.

**Figure 4.53. Figure 53.** – The preoperative and postoperative exposures represent well the dislocation, anatomical situation, which has been restored with reposition and the plates, which have been placed on to the orbit frame and fixes this position

![Figure 4.53](image)

**Figure 4.54. Figure 54.** – Every patient is afraid of periorbital incision but a well-made subciliaris section does not case aesthetical confusion after the operation. Even the young, female patients are satisfied with the results

![Figure 4.54](image)
4. Oral surgery

Figure 4.55. Figure 55. – In this case, an incomplete zygoma fracture operational solution according to Gillies can be seen. (More accurately, an isolated zygomatic arch fracture.)

Figure 4.56. Figure 56. – We put the raspatorium under the temporal muscle’s then lead it forward and downwards, so we can tip the sunk arch out

Figure 4.57. Figure 57. – Section, which has been made under the eyelashes to exposure the margo infraorbitalis’s fracture. After the reduce of the fracture, the fixations happen with a plate which has four holes on it
Figure 4.58. Figure 58. – After the intervention we place in an interrupted suture with 5.0 yarn, then we cover the wound

10.6.2. Occlusion Influential Fractures

*Isolated Fracture Of The Alveolar process*

By itself is rare. It supply happens together with the accompanied Le-Fort level fractures, supplemented by dental splinting.

Figure 4.59. Figure 59. – When we exposure the pathological area of that patient, who has fall to an iron tube, we found awfully damaged gingival and vestibular soft parts. Bone chips of the square millimeter big maxilla’s front and lateral wall have edged into the soft part and lost their connections with the periosteum

Figure 4.60. Figure 60. – The preparing of the soft part for the closure and fixation of the bone pieces with wire are both big issues. The more distal bone fragment was
appropriate for the laminar fixation to the zygoma. We find the anterior bone fragment with biocortical screws to the suitable maxillaries alveolar part

Figure 4.61. Figure 61. – The patient was satisfied with the status after the closure of the wound

Fracture Of The Tuber Maxilla

Dentists can meet this type of fracture, as well. It occurs at the removal of the patient’s upper molar tooth, if the patient has gothic palate with deep recess in the sinus’s back region. Tuber fracture happens with a bigger chance if there is no other tooth in front of or behind the tooth. Luxation initiated in connection with the extraction does not produce only displacement of the tooth but the mobility of the bone plate with the tooth, what we can feel with our left hand, during the soft part protection. In such a case it is worth to remove the tooth with the bone because we can cause large sinus openance and bone-loss with it. We put the tooth in rest for four weeks with acrilate plate and splinting, then beside lobe formation we make a little bone removal. If it is needed, we make the dissection of the tooth to keep the tuber, which has been fractured with by tooth-removal, in its place.

If the tooth has been removed due to acute symptoms and the bone block, which is moving with the tooth is not that huge, after a flap transformation we can finish the removal of the tooth and bone. Sinus closure must be provided with mattress sutures and antibiotic cure for the patient is also necessary.

Epistaxis, facial swelling and pain can be expected after the procedure. However, the patients are complaint-less on the suture-removal, which is 10-14 days after the intervention.

The Maxilla Sagittalis Fracture

It is a rare accompanying fracture of midface fractures. Condylar fracture in one or in both sides, beside maxilla sagittalis fractuent’s complaints are common. The patient ‘s complaints are various because this type of fracture is rarely isolated. This fracture happens just like condylar fractures: the trauma reaches the chin in the middle, from downwards. But in this case, the lower jaw-bone leads the force on to the upper jaw-bone and the maxilla (usually but not always) bursts along the suture line of the mesial or a bit lateral from that. The gingiva, which covers the fracture line, can not follow the sudden changes and cracks. The nasal cavity and oral cavity are opened towards each others. The cusps sulcus-connection of the premolar and molar teeth change. The upper dentil on both sides are situated bit more laterally.

The closure of this typical soft part injury without flap transformation is almost impossible. Similar flaps can be used, like we make at the closure of the palate’s crevices. Approximating of the maxilla, which has split in two pieces, is usually not easy. We place lamina under the apertura piriformis, in submammarial fold after a section in the middle and lobar transformation. The MMF-wearing can be justifiable later also. If the supply does not happen in a few days, the fracture lines can be approximate by Hyrex-device to restore occlusion.
Le-Fort 1 Fracture

It is not a too common fracture type. Rarely clear, symmetrical, bilateral. Rather associated with other side high midface fracture or with sagittal maxilla fractures. The occlusion damages, the whole dental movements pathologically, generally in posterior and inferior directions. It slightly lengthens the midface.

Figure 4.62. Figure 62. – Artificial Le-For I. fracture and fixation. In fact, there is no fracture, but the removal of the alveolar part of the maxilla from the upper jaw-bone happens by using a surgical oscillater saw

Figure 4.63. Figure 63. – The stabilization happens with right and left side, L-shaped miniplates, on the suitable bone areas

We take advantage of the dental mobility, which supervenes in connection with this fracture type at some ortognate operations, when we make an artificial fracture above the upper jaw-bone’s tooth roots. We exploite the obtained mobility to correct the pathological bite of the patient. We make the fixation with laminas and screws, just like in case of accidental fractures. In fact, the bone’s separation by subtle methods does not cause that big damages on the paper-thin anterior maxilla wall, like a blunt force on a face at a traffic accident. A blow that causes midface fracture rarely makes a straight fracture line without defect, which is described in textbooks. In the intraoral, upper deflection, after a deeply lead section (we have to pay attention for the suture line, what has to be on a bone, if it is possible on a bony intact area) which connects the bilateral molar region we separate the periosteum with the gingival and exposure of the fracture line. As it has been mentioned, it is not really a line, but a damaged area of the maxilla’s anterior wall sometimes only with square millimeter bone defects but usually with square centimeter outbrakes. The worthy size and quality bone fragments can be fixed back but in case of a Le-Fort I fracture the really important part is to fix back the alveolar process, which holds the teeth back to the stable maxilla and zygoama parts. The stableness and intactness of the surrounding bones are not enough because in this area we can only find bones, which are suitable for plating around the crista infrazygomat and apertura piriformis. Just like at the ortognate-bite correctional operations in case of the fracture is caused by trauma, we hang the mobile maxilla piece with laminas and screws. Methods of internal suspensive wire-fixations are not used anymore. In case of wire fixation, the recovery is significantly longer because it causes bigger unpleasentness and lets bigger mobility for the bone.

The well carried out reposition of maxilla fracture guarantees lessly proper occlusion, therefore, just like at the angulus fractures of the mandible, intraoperative MMF fastening is needed before the fixation. This way we can ensure the teeth’s pre-traumatic occlusion.

Postoperational swelling and pain is often completely gone until the stretch removal, which is one week after the intervention. From one hand, the patient can eat soft pulpy food. On the other hand it is still better than the situation of those who could not open their mouth for one and a half month due to the splint and wire fixation methods.

Le-Fort II Fracture
It is a bit more common than the Le-Fort I fracture. We meet more often its symmetrical form.

Occlusional deviation is not regular, even if the patient’s toothing is deficient or if the patient is absolutely toothless.

Compared to the Le-Fort I fracture, the damage on the maxilla’s anterior wall is even larger. When we exposure it (like the Le-Fort II fracture) we see explicit bone defect. The fracture affects the whole anterior wall of the maxilla and runs out onto the margo inferior, usually involves the foramen infraorbitale into the fracture line.

In case of large step formation on the lower orbit frame, it is worth to make an external exposure (subciliaris section) and this section can be mesialized the fixation of the nose if it is needed. I would like to note that in case of bilateral upper Le-FortII-III fractures , bilateral section lead on the crown is worth considering. By this incision, the nasal bones and both side of orbit above, bottom and sides can be reached. By those patients, who has at least the average amount of hair, nothing can be seen from the section after the operation. In case of fractures with large dislocation we always check the lower orbit frame and the orbit base. Although many literature datas does not consider justifiable the exposure of orbit base, even if the CT shows positive signs (attendant hernia), but the patient has no eye symptoms.

Since the infra orbital nerve’s location can be seen due to the normally required intraoral exposure, it is worth to look at the nerve exit area because the reposition is often not enough for releasing the nerves. Sometimes bone chips have to be removed from the periphery of the foramen. By intraoral exposure, we search the stable point of the zygoma, where the bone block can be fixed after the reposition. The intraoperative peremptory used MMF’s screws should not be removed in the first one or two weeks because the incidental minimal occlusion correction can happen postoperative with these, as well.

**Figure 4.64. Figure 64. – It is a bilateral, combined midface fracture. It shows the Le-Fort II and III fracture’s typical fracture lines. This is a situation after the reposition and the minilaminar fixation**

*Le-Fort III Fracture*

The patient’s central midface’s is complainant, the nasal bones are mobile, but it has intact lower orbit frames. During the movement of the upper dental, the temporozygomatical sutures can be moved. After the reposition, the supply of the fractures happen on the lateral orbit frame and in the periphery of the nasal bones. Since these fractures are not that simply and clear, always more exposures and more complicated supplies happen. The fracture of the nasoetmoidal region, which is lumpy and causes the flattening and indentation of the nose region and the lateralization of the eye palpebral fissure. The reconstruction of the ligamental canthus and bones are recommended to restore the eye, nose and aesthetics.

The fixation happens with MMF and laminar osteosynthesis, which gives good biting and chewing abilities soon after the operation.

The forces, which damage the fractures and highly affects, often destruct the nasoetmoid and thefrontal bone regions as well. In such case the brain could be injured, as well. The supply of the supraorbital margin region and frontal bone impressed fractures are also tasks for the maxillo-facila surgeon. We have to treat the frontal impression of the bone wall for the favorable aesthetics. We connect it with a titanium mesh if the bone pieces can not be fixed with a plate.
10.7. Complications of the not well treated cases

A failure of supply the different fractures leads to many, typical complications. In the face and mandible region the bone, which “recovers” with malocclusion makes eating and speaking more difficult and worsens aesthetics in extreme cases, as well.

By mandible fractures, which have not been supplied, inflamed resorption, osteomyelitis of the broken ends in a large area is very common. Later it makes therapy significantly more difficult. Extraoral fistula is very frequent in this situation. Usually the classical minilaminar osteosynthesis is not possible intraorally, then we need to make large external exposure to refresh the broken ends, the inflamed tissues and fistulas should be removed and at the end exists the reconstructional laminar osteosynthesis.

The inferior alveolar nerve injury by the reposition usually causes temporary numbness, only. The lack of the fracture supply leads easily to eventual numbness.

By non-supplied patients, or by those, whose rehabilitation was not appropriate sometimes ankylozis occurs with marked mouth-opening limitation.

The lack of zygomatic arch’s supply is likely: the coalescence of the condylar arch and surrounding structures can also cause trismus.

The lack of antibiotic treatment at central midface fractures can lead to the overinfection of the haemorrhaged sinus. In case of absence of the reposition and fixation aesthetic interference and eye-movement disorder can be identified. The numbness of the face will persist easily because of the infraalveolar nerve involvement, if the dislocated fracture is not not replaced. Le-Fort fractures can get recovered, in case of smaller-bigger movements of the denture without surgical therapy, if the patient avoid moving the mouth and denture as well. In fact, the occlusion is not absolutely like it was before.

10.8. Plan of a complicated multiple fracture

Due to the affect force more regions can injured on the face. Particulary, we see injury on the mandible, on the central and lateral midface after falling from high places or at traffic accidents. In such cases we have to keep some rules while making the sequence of the bony fixation.

Of course, every case is unique and special and some conscious rule-breaking can lead to good results.

However, in general:

• Hard bleeding and the face’s opened soft tissues parts must be supplied immediately.

• In cases of bony replacement, we always build from the bottom, to upwards, which means that we start the reconstruction with the mandible’s supply.

• Since it is possible, the external exposure can be made first on account of sterility, first we restore the vertical height of the mandible, taking in advance the condylar fracture. This is followed by the supply of the angulus fracture, it is worth to make the MMF before the flap transformation.

• In the midface’s region we restore anatomy by using the teeth as well (MMF). The setting of the occlusion already assumes a nearly good position. By exposuring the fractural places one by one we fix the midface intraorally.

• If it is necessary, we fix the zygoma to this central frame.

• The naso-orbito-ethmoidalis complex has to be fixed to the reduced midface, just like the fractures of the frontal bone.

10.9. Summary

After the basic cognition of the malar bone and mandible fractures, we can say, that not to recognize a fracture is a mistake, underestimating their supply is a failure, as well. With the tools and methods, which are used nowadays very serious malar bone fractures can be restored. Depending on the trauma’s quality, the aesthetics
and function can be good or even great. In case of satisfied or bad results the patient needs reoperation and reconstruction and of course we almost never get satisfied with tem.

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**11. 4.11. Surgery of the periapical space – Jozsef Szalma**

The definition "periapical surgery" includes several surgical approaches such as the resection of root apices (apicoectomy), removal of periapical inflammations (periapical curette), intraoperative orto- and retrograde root filling techniques, corrections of perforations and false paths, root sections (hemi- and trisections) and transdental fixations of luxation and subluxation injuries of the teeth. Considering the nomenclature, the cystectomy -meaning the total removal of a chronic periapical pathologic lesion- should be mentioned as well.

Because of their relatively high prevalence in everyday dental and oral surgical practice, root apex resection and retrograde root filling are more underlined in this subchapter.

**11.1. The indications and contraindications of root-end resections**

When is resection necessary? There can be anatomical causes (significant apical curvatures of the roots, obstructed or narrowed canals), pathologic processes (chronic periapical infections, such as granulomes, abscesses, cysts or root end resorptions), trauma (horizontal fracture of the apical third of the root), failures of the endodontic treatment (overfilling, overshaped closure of the apical foramen, irretrievable root fillings, false paths or fractured endodontic instruments in the apical third of the root) or in the case of intrapulpal posts, when the root filling is acceptable (adequately obturated) and the post removal is dangerous (risk of vertical root-fracture).

**Figure 4.65. Figure 1.** – In this periapical radiograph the periapical radiolucence around the mesial root and the overfilling of the distal root of the molar tooth can be observed. These are indications for resection separately and together as well
Figure 4.66. Figure 2. – After raising the mucoperiosteal flap, the localization of the root end is possible without bone removal because of the buccal cortex fenestrating overfilled/overobtured guttapercha point
The resection has **relative contraindications**, when the treatment of the patient is safe only after some internal medical or haematological precautions (e.g.: patients with bleeding disorders or those who require prophylaxis), and **absolute contraindications**, when the routine "bloody" treatment of patients are strictly forbidden (in the case of intravenous bisphosphonates or after radiotherapy of the jaws). The most important **local contraindications** are the following: missing or incomplete root filling, parodontally compromised teeth, compromised postoperative root-crown ratio (<1:1), acute periapical inflammation, vertical root fracture and when anatomical structures may be damaged during surgery. Last but not least the most important contraindication of resection: when conventional root filling or re-treatment is possible!

**Figure 4.67. Figure 3. – The success of resection could be significantly decreased by the parodontal communication (arrows)**
11.2. Preoperative diagnostics and differential diagnostics

Preoperative diagnostics should include history taking (anamnensis), physical clinical examination (e.g.: vitality check of the neighboring teeth or measurement and probing of the parodontal depths) and imaging diagnostics (periapical films, panoramic view, occlusal films and cone beam computed tomography /CBCT). In the case of the presence of a fistule when more than one tooth is involved, it could be useful to repeat radiographic examinations with gutta-percha points pushed into the fistule's channel. In sinusitis differential diagnostic problems can occur because of the sensitivity of the teeth to chewing or knocking, similarly present in periodontal infections, but it could be concluded that in sinusitis the teeth are usually vital and knocking sensitivity is characteristic of several teeth or in a full quadrant.

11.3. The surgical steps of resection

a) To perform a resection a full thick, three-layered mucoperiosteal flap raising is needed. The criteria for a correct flap are as follows: it should give a good visibility to the periapical area, the blood supply of the flap is continuous and intact, during the repositioning of the flap the marginal parts should have bony underlying support, they should avoid scar- and cicatrix formation avoiding compromised cosmetic results (cave frenulums, prominences of canines and parasulcular incisions) and finally they should not damage vital anatomical structures (mental-, incisive foramens). The following flap designs are known:

- Partsch
- Pichler
• Reinmöller ↓ (not sulcular!)

• Paragingival flaps (incisions 1-2 mm from the mucogingival border (not in the fixed, keratinised mucosa).

• Wassmund ⌈ (sulcular incision with two releasing incisions).

• L shaped (sulcular! incision with one releasing incision).

In general it should be concluded that sulcular flaps give the best postoperative results with few complications when the teeth have good parodontal status and the oral hygiene is good. If the teeth are parodontally compromised, mainly under crowns or bridges, paragingival flaps should be considered.

b) The localization of the root apex can be performed with the help of measurements of preoperative radiographic images, considering the endodontic working length or with the aiming help of fistules or cortical fenestrations.

c) The preparation of a cortical window can be performed with rotating instruments (e.g.: surgical straight handpiece with carbide round end burs) or piezoelectric devices. Chisels and mallets are avoided nowadays for this intervention. Firstly the periapical area of the apex is localized and next the size and orientation of the window should be corrected according to the size of the pathologic lesion allowing the best admittance.

d) During the periapical curette inflamed tissues should be entirely removed with a sharp spoon or excavator. Sometimes this is difficult or not possible because of the root apex. In these cases the curette is repeated and finished after resection of the root end.

e) During the removal of the apex/apices of the root (= resection) the apical 3-4 mm of the root end should be cut (with carbide fissure bur, piezoelectric tip or, in some, cases, chisel) with a 10-30° external inclination (the cut surface and the obturation of the root filling can be controlled). In this stage it is important to examine carefully the occurrence of rips and fractures.

Figure 4.68. Figure 4. – After the resection of the buccal roots of an upper first molar it is obvious that the examination of the distobuccal resection plan is not too simple from the mirror (arrow)
Figure 4.69. Figure 5. – In an upper front area the control of the resection plan is not problematic. The retrograde filling material (GIC) can be observed.
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Figure 4.70. Figure 6. – After the first unsuccessful resection of the upper lateral incisor, the second operation revealed the cause of the failure: a vertical fracture line can be seen on the buccal surface running apically from the cement-enamel junction

![Image of a tooth with a vertical fracture line](image)

f) When the surgeon indicates the performing of a retrograde root filling a small cavity should be prepared (with a special micro-head handpiece with small round or inverted cone burs or with piezoelectric tips). Then with a micro sized applicator the filling material is placed (amalgam, MTA[mineral trioxide aggregate], glassionomer cement, IRM [intermediate restorative material cement] or, in some cases, composite resins for perforation repairs) into the cavity.

g) The wound care includes the followings: The operating field should be rinsed and disinfected, sharp bone irregularities should be removed and smoothened and the insertion of fibrin sponges (which avoids the retraction of the coagulum) or bone substitutive materials should be considered. The wound is closed with sutures.

h) Control and suture removal occurs on the sixth postoperative day. In the 6th postoperative month a control radiograph is necessary. In many cases intra-, or postoperative radiographs help to control the surgeons’ work and are welcome.

Figure 4.71. Figure 7. – In many cases intra-, and postoperative radiographic examinations improve the accuracy of surgeons’ manipulations. In this picture the earlier represented (4.11.001.) lower first molar can be seen after the resection

![Image of a lower first molar after resection](image)
12. 4.12. The etiology of sialolithiasis, the structure of sialoliths and their surgical treatment – Jozsef Szalma

A sialolith (= salivary calculus) is a usually highly calcified concrete mass with various sizes and shapes, formed in the salivary glands or in their excretory ducts, visible without any magnification. It contains one or more nucleus/nuclei (i.e. core, central structure), the periphery and the external shell.

Figure 4.72. Figure 1. – Submandibular salivary stones with a millimeter scale. The fractured surface of the removed stones represents nicely the central core and the concentric organized periphery
The organic matrix contains glycoproteins, mucopolysacharids, cell fragments and lipids originating from the saliva and cellular membranes. The stones consist of 18% organic and 82% inorganic compounds. Approximately 5-6% (of the stone weight) protein and 1% lipid compose the organic part of the stone. Sialoliths have usually one or more nuclei. Around these nuclei, differently calcified or purely organic lamellar structure is seen. The main inorganic component is hydroxilapatite, but other crystals may be present such as withlockite, brushit, apatite or octacalcium phosphate.

Figure 4.73. Figure 2. – The electronmicroscopic image of a fractured surface of a submandibular stone magnified by 1100. Interesting structures composed of hydroxilapatite crystals can be seen
Scanning electromicroscopic investigations proved mainly irregular and particularly hexagonal, needle-like or sheet formed crystals. These crystals consist mainly of Ca and phosphate ions and in smaller amounts Mg, Na, Cl, Si, Fe, és K ions. Central structures contain these ions in less amount than others, because central parts are more stable and contain a more saturated crystal structure.

Sialolithiasis affects around 1-2% of the population. Mainly adults between ages 30-60 are involved, and it is twice more so frequent in males than in females. In 70-80% of the cases one stone is present, in 20% of the cases two stones and in 5% three or more stones are found. A case is presented where 5 stones were found.

**Figure 4.74. Figure 3.** – The lower occlusal radiograph represents five salivary calculi in the medial and distal portions of the right sided Wharton’s duct

**Figure 4.75. Figure 4.** – Calculi represented in the occlusal radiograph after removal
Stones can be located in the parenchyma of the glands or in the excretory ducts, which determines the therapeutical possibilities.

12.1. The suspected mechanism of stone formation

The mechanism of stone formation is not clear and it is still ambiguous. According to earlier suggestions organic and inorganic minerals are deposited around a central nucleus. Different microorganisms, intracellular microcrystals, foreign bodies, exfoliated epidermal cells or the mucoepidermoid gel formed from increased mucin concentration may serve as an initiating structure in the mechanism. These may form an organic core, where precipitation of phosphates can begin at the current pH and concentration. According to a few authors, foreign bodies and microorganisms can serve as the initiator cores of stone formation, but this nucleus transforms morphologically during the stone development, losing the initial initiator-particles. Research proved that organic structures can be seen in salivary stones (epithelial cell fragments, fine fibrous structures, bacterial fragments), but never in the core. It can be concluded that the organic protein core theory has not been proved yet. Chronic sialoadenitis can play a role in sialolith formation, since the swelling of inflamed areas can compress the glandular structure and cause partial obstruction and salivary stasis which results a highly calcified core genesis in the oversaturated saliva. This core can serve later as the inorganic nucleus of a stone. The first stage develops when sialomicroliths appear and cause obstruction in small intraductal channels and this is followed by an inflammatory response. In the later phase atrophy, fibrosis and inflammation increase. The decreased secretion of the atrophied gland aggravates the chance of bacterial invasion and further inflammation.

12.2. Involvement of the glands

In lithiasis, the submandibular gland is involved in 80-85%, the parotis in 10-13% and the sublingual gland in 7%. Minor salivary gland involvement is rare (<2%) and mainly the upper lip and bucca are affected in contrast with the palate, the tongue, the vestibule and lower lip.

As it was stated, mainly the submandibular gland is affected, more accurately the Wharton's duct of the submandibular gland. This dominance has the following causes: the Wharton's duct is longer and more curved than the Stensen's duct; the submandibular saliva contains more calcium, phosphate and mucin and the pH is more alkalic than others; saliva secretion occurs against gravity; the orifice of the Wharton's duct is narrower and facial mimic muscles help excretion of saliva from the parotid duct. The diagnostics of sialolithiasis includes several approaches, namely physical examination, ultrasonography, radiography, CT, CBCT, MRI imaging, sialography, scintigraphy, sialoendoscopy, sialochemistry and sialometry. If the exact histology of the gland is important, fine needle aspiration biopsy (FNAB) or exploration biopsy can be performed.

12.3. The surgical therapy of sialolithiasis

12.3.1. The submandibular gland

When the sialolith is localized distally from the proximal fourth of the Wharton's duct, the stone can be removed by an intraoral approach. If the stone is located in the hilar region or in the parenchyma of the gland, extraoral gland removal is usually necessary.

The intraoral removal of the stone is usually performed in local anesthesia. The localization of the stone should be exactly determined and then a circum lying suture should be placed distally from the stone to avoid the migration of the stone to gland-proximal direction. Firstly, the mucosa of the floor of the mouth and the
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submucosa are incised by scalpel, then the excretory duct is opened over the calculus and the stone is grasped by forceps, hook or in some cases, with a curette-spoon.

**Figure 4.76. Figure 5.** – After the incision of the mucosa, submucosa and excretory duct, the gland-proximally located calculus becomes visible

![Image](image_url)

**Figure 4.77. Figure 6.** – The elevated salivary stone in the Wharton's duct

![Image](image_url)
Figure 4.78. Figure 7. – The significant dilation of the Wharton's duct is visible after stone removal (arrows)
Figure 4.79. Figure 8. The above mentioned calculi after removal beside the millimeter scale.
After that other adjacent stones or fragments should be checked and removed. Finally, the drain placed into the mucosa and submucosa prevents the formation of any retention cysts (ranula).

**Figure 4.80. Figure 9.** – The wound closure with a rubber drainage (arrow).
12.3.2. Parotid gland

Parotid stones are usually found in the excretory (Stensen's) duct with some exceptions, but the surgical removal has a lot of difficulties, that is why conservative therapy is preferred. If the stone is located near to the intraoral orifice, the exploration of the duct results in stone removal. The papilla should be circumcised, then a probe is driven into the duct and the mucosal incision is elongated distally. Then with a suture of the mucosa, the Stensen's duct could be pulled toward the oral cavity. Up to the site of the palpated stone the duct is bluntly prepared, opened over the calculus and the stone could be removed. A plastic catheter tube is then placed intraductally to prevent any formation of narrowing/obstruction. If the stone is located gland-proximally in the duct, parotidectomy could be necessary. The stone could be removed sometimes only with partial, subtotal or total extirpation. The high risk complication of this maneuver is the injury of the facial nerve (facial palsy).

13. 4.13. Dento-alveolar surgical considerations of the maxillary sinus – Jozsef Szalma

The inflammatory malformations of the maxillary sinus (= Highmore-cavity, =antrum) usually have two etiological origins. The diagnostics and treatment of the rhinogenic causes is the otolaryngologists' task. Dentists and oral surgeons usually discover and eliminate the dental origins.

13.1. Odontogenic sinusitis

13.1.1. Etiology
In **odontogenic sinusitis** the infection and inflammation originates from the teeth, parodontal tissues or from other structures of the oral cavity and spread into the antrum.

**Contact infections** include direct spreading of the infection because of intimate anatomic relations through the narrow barrier (sinus cortex + mucosa, sometimes only the mucosa):

- Periapical infections of a **pulpal origin**
- Parodontitis
- Osteomyelitis, periostal inf.
- Infections of impacted teeth or the cysts of the follicle of these teeth
- Radicular, residual cysts
- Endodontic treatment (mechanical and/or chemical damage)
- **Oroantral communication** (acute perforation after extraction or the chronic antro-oral fistule)
  - Antral foreign body (root remnants, root filling materials)
  - Trauma (fracture of the alveolar process, tuber fracture)
- **Dento-alveolar surgical approaches** (implantation, sinus elevation, resection)
- Odontogenic tumors

**Figure 4.81.** Figure 1. – In the panoramic radiography the spreading of the periapical infection of the upper molar into the maxillary sinus is seen (arrows)
The other form is the lympho-haematogenic spreading (it is rare and develops mainly in childhood). Considering age 16 the final development of the sinuses (entire size and recesses), odontogenic sinusitis is observed usually after this age.

13.1.2. Symptoms

**Major symptoms** (these are characteristic of a concrete disease)

- Pain (head, face) + sensitivity on palpation + dullness of the face: which is sharp and well circumscribed; worst in the mornings; worsens to cough, sneezing or to moving of the head and alcohol consumption or sudden thermal change aggravates it.

- Nasal snuffles.

- Rhinorrhea, nasal discharge.

- Disorders of the smell and taste (anosmia, hyposmia).

- Pus and purulent discharge on anterior rhinoscopy in the nose.

**Minor symptoms** (= characteristic of many diseases)

- Headache (area of the forehead, orbit).

- Fever.

- Fatigue.

- Cough- usually productive, mucus.
• Ear pain.

• Toothache- in the lateral regions on sudden moving of the head, on jumping, on chewing and sometimes spontaneously. The teeth in this region are sensitive on percussion on the affected side, but the teeth are usually vital (except the gangrenous teeth causing inflammation).

• Halitosis (bad breath).

• Nasal sound.

13.1.3. Classification

• Acute: symptoms lasting for a maximum of 3-4 weeks; 1 major+ 2≤ minor, or 1< major.

• Subacute: symptoms lasting for 4-12 weeks.

• Recurrent acute: 4 or more episodes in a year lasting 7-10 days.

• Chronic: more than 12 weeks of purulent discharge from the nose or >2 major or 1 major+ 2≤ minor symptoms.

• Acute exacerbation of chronic infection: sudden impairment, which improves for treatment.

13.1.4. Diagnostics

a) Inspection, palpation:

• Nasal discharge, nasal erythema.

• Erythema or swelling of the skin of the face.

• Purulent discharge in the pharynx.

• Sensitivity of the maxilla on pressure.

• Sensitivity of the vestibule on pressure, may be accompanied by swelling.

• Teeth sensitive on percussion.

• Signs of odontogenic origin (antro-oral communication, avital teeth).

b) Anterior rhinoscopy (with a speculum), posterior rhinoscopy (with an endoscope or fiberscope). A special examination tool is the Hopkins-fiberscope (rigid instrument for examination and biopsy taking)

c) Needle biopsy and antrum lavage through the canine fossa or through the alveolus of an extracted tooth with a trocar needle

• the possibility of analysis of the fluid content,

• sample of mucosa for histology.

d) Lab-tests

• Nasal cytology.

• Gibson-Cook sweat test: the possibility to diagnose cystic fibrosis.

• Testing of the ciliary function: scintigraphic or microscopic analysis of the frequency of the ciliary movements (<10/sec= decreased function).

• Immundeficiency tests.

e) Imaging examinations
• X-ray
  • Water's (occipito-dental central x-ray beam),
  • Velin's (submento-vertebral central x-ray beam),
  • Panoramic depiction (OPG),
  • Occlusal films.

Usually four pathologic malformations can be seen: a nivo in the case of fluids, the covered sinus in the case of increased opacity, swollen mucosa (sinus with "double walls") in the case of chronic inflammation or the polyp in localized chronic cases.

**Figure 4.82.** In the cone beam computed tomography image a polyp-like chronic inflammation can be observed in the antero-caudal portion of the maxillary sinus. The patient had no complaints

It can frequently occur that the normal radiolucency of the sinus superimposes to the apical region of gangrene or root filled teeth resulting in a false positive cyst diagnosis. It is practical to perform a panoramic radiograph, where sinus border and integrity are easy to follow. Finally, the puncture of the radiolucent lesion -as a minimally invasive test- results in the correct diagnosis.

• CT (e.g.: coronal plane slices): give a detailed image, furthermore the bacterial and fungal origin can be separated.

• CBCT (cone beam CT): the dental origin can be exactly examined.

• MRI: cannot be used to examine the bones but the most detailed soft tissue imaging.

f) Trans-illumination: the sinus is lighted through in a dark room, usually only for the diagnosis of acute bacterial sinusitis.
13.2. Therapeutic considerations in sinusitis

One of the most important tasks is to eliminate the aggravating factor (trepanating, extracting, resection of the "bad" tooth). After this approach the inflammation usually improves.

13.2.1. Conservative treatment

- Administration of antibiotics penetrating well into the sinuses (e.g.: macrolids).
- Antipyretic and analgesic therapy + adjuvants (expectorants, mucolytics, nasal drops, inhalations).
- Sinus lavage: It can be applied in acute purulent sinusitis before a surgical procedure (sometimes as monotherapy). After the terminal infiltration anesthesia of the canine fossa, above the roots of the molars, in the vestibular fold we penetrate through the anterior wall of the sinus with a trocar/lumbar puncture needle. Then the antrum is rinsed with disinfectant solutions (physiologic salt, Neomycin or Betadine solutions), applying moderate pressure to avoid unwanted penetration into neighboring tissues (orbit). The opposite nasal entrance should be closed. It should be performed on every third day till the rinsing fluid becomes clear and transparent without bad smell. Otolaryngological method: an entrance is created below the lower concha for the rinsing.

13.2.2. Surgical therapy

- Luc-Caldwell operation: it is performed in local or general anesthesia. After a mucoperiosteal flap is prepared with a horizontal incision (in the vestibule or sulcularly) and with a vertical releasing incision, a bone window is opened anteriorly from the zygomatico-alveolar buttress (i.e. canine fossa where the bone is the most narrow) over the root ends of the lateral teeth. It is important to avoid infraorbital nerve injuries. Then the pathologic components of the sinus mucosa are removed (the healthy mucosa should be kept) and an aperture into the sinus cavity is prepared in the lower concha with a bended, blunt hemostat. Through this opening an iodoformic gauze is placed in the sinus cavity, and the other end of the gauze is located in the nose (the easy and free movement of the gauze through the prepared cavity should be checked). Finally the oral wound is closed and a tamponade is placed into the nose.

- Lotrop operation: (otolaryngological method) the inflammed sinus mucosa is removed through a nasally prepared cavity (under the lower concha) without intraoral wound. The spontaneous regeneration of the sinus mucosa is possible after performing a artificial opening with free ventilation without mucosa removal.

- FESS (functional endoscopic sinus surgery): it is a minimally invasive technique with much shorter hospitalization time. The physiologic opening of the sinus is restored or widened by an endoscope to allow the elimination of inflammation with the help of muco-ciliary function.

13.3. The closure techniques of antro-oral communications

13.3.1. The etiology of sinus perforation

The interdental and interradicular processi of the sinus cavity usually get into contact with the upper, lateral teeth after the age 16 so the sinus mucosa can be injured after an extraction or during root canal treatment. The sinus perforation during root canal treatment needs no further special care, it heals spontaneously. During an extraction perforation develops usually at the upper first molar, more rarely at the upper second premolar and molar and even more rarely at the upper first premolar or the upper third molar (~5.6%). At the canines it is extremely rare. The above mentioned situation could change in the case of extended cysts.

13.3.2. Symptoms and diagnostics of sinus perforation

After an upper lateral tooth extraction (canines too!) a nasal blow test by the patient's open mouth is mandatory. If the test is positive, air could be heard streaming through the alveolus into the mouth. In some cases this test can be false negative, so the anti-probe (blowing the cheeks with closed lips) should be performed to hear air streaming releasing from the nose. The mucosal "trap-door" which is closed at a nasal blow test may open during the anti-probe. Accompanying symptoms:

- Foamy blood discharge from the socket.
• Water/fluids flow out of the patient’s nose on drinking.

• The mirror gets vapor (moisture condensation) near the socket.

• Previous perforations result in fistules.

13.3.3. The surgical care of sinus perforation

After perforation, the closure should be performed as early as possible, but least in the 48th hour. The prognosis is best in 6 hours (postoperative antibiotics are not necessary). Closures between 24-48 hours indicate postoperative antibiotics. A closure after the 48th hour is not possible, the wound is likely to open. In these cases 3–4 weeks should be waited and then the fistule should be removed and closed or a Luc-Caldwell operation could be necessary. Extremely rarely spontaneous closure might be observed even with excellent oral hygiene and healthy sinuses, but its chance is minimal.

Vestibular mucoperisteal (e.g.: Wassmund-, Moczair-, Axhausen-, Czappan-flaps) or palatinal half-thick flaps (Pichler-flap) are prepared for the closure, then the periosteum is cut at the base of the flap for better elongation and the socket is hermetically closed. Horizontal mattress sutures are recommended for better fixation of the flap. The palatinal marginal gingiva should be under-prepared for better possibilities of suture fixation of the vestibular flaps and in certain cases cut some pieces from the palatinal gingiva to put sutures on bony surface. The suture removal -after several controls- is on 10-12th day.
Chapter 5.5. Oral Radiology

1.5.1. Intraoral x-ray anatomy of the lower jaw – Daniel Nemeth

1.1. Anatomy of dental x-ray images

When we are making a radiogram, we are using a physical phenomenon, that the atoms are able to absorb the X-rays. The quantity of the absorption depends on the property of the nuclear, first of all on atomic number. Because the tissues’ components of the human body are different, so we can separate them due to their X-ray absorptive. Because the atomic number of the hard tissues’ (enamel, bone etc.) components are higher than the soft tissues’, the indication of the radiogram is mostly the examination of the bones, teeth and other hard tissues. The different radiogram processes are basic imaging diagnostic methods in dentistry.

In the following chapters we are trying to describe the anatomical structures of the radigrams. We will not mention the different pathological disorders, but we will emphasize the relevant point of views, that are important in the diagnostic.

1.2. Intaroral radiograms

If we are talking about radiograms, first of all we mention the periapical radiographs. The periapical radiograms mean, that the apical part of the tooth and the area around the root are in focus, but the crown should also be represented. By setting angle or parallel techniks are used. Occlusal technic, crown technic or wing technic are also belong to the different intraoral radiograms. But these methods are rare used. In the following chapters the periapical radiographs’ anatomical traits will be represented.

1.3. The radiograms of the teeth

As in the introduction is mentioned, the differences of the tissues are made of the structural and compositional variations. In the fields of the radiology it is difficult to talk about radiodensity or radiolucency. The formulas are always compared to the environment. That’s why on the periapical radiograms the lightest part is the enamel layer, that absorbs the biggest amount of the X-ray. It can be followed by the line of the anatomical crown and the thickness is also could be defined. Because of the crown’s own characteristic there could be differences between the densities. (For example the cusp are projected to each other.) Under the enamel exists a more radiolucence area that’s called the dentin. From radiological aspect the dentin and the cement, that covers the root, could not be separated from each other (Figure 1st). In the axis of the teeth the pulpchamber could be found, that is darker than the dentin. It means the pulpchamber in the crown part and the rootcanal in the root. The characteristic of the pulpchamber is well visible on a radiogram (for example pulphorn, etc). On the anatomic crown the enamel is well marked, so the enamel-cement border could be easily determined. This is important, because the cortical lamina of the interdental septum, that separates the teeth, is situated 1-1,5 mm to the apical direction from the enamel- cement border. The root part is covered by the parodontal fibers. These connect the teeth and the alveolar process (2. and 3. Figure). The three structures are very significant. Due to the triple pattern cement layer of the root is radiodens, the parodontal ligament is radiolucency and the cortical lamina of the alveolar process (lamina dura) is also dens. The triple pattern could be followed all along the roots. The interruption or the change of this pattern refers to a pathological disorder.

Figure 5.1. Figure 1. – Lower jaw radiogram. The premolar and molar teeth are well detected. In the molar teeth exists a filling. No pathological disorders are detected in the spongious bone. 1.: lower jaw, 2.: crown, 3.: root, 4.: enamel, 5.: dentin, 6.: pulpchamber, 7.: rootcanal, 8.: pulphorn
Figure 5.2. Figure 2. – The structure of the parodontium is well marked. The triple pattern is visible till the apex of the molar tooth, and then the lamina dura ends. The healthy parodontal structure is situated between the molar and premolar teeth. 1.: lamina dura, 2.: parodontale ligament, 3.: cement layer

Figure 5.3. Figure 3. – After an extraction the compact bone is better marked
1.4. The lower jaw

The lower jaw’s teeth are situated in the mandible, so the intraoral radiograph usually contains different part of the lower jaw. The mandible connects with joint to the other parts of the skull. During the active movements of the muscles it opens, closes and chews as well. The muscles attachments are located on different anatomical structures.

The mandible’s two main structures are the corpus and the ramus. They join to each other at the angulus mandible. The corpus is divided into the basis of mandible and the alveolar part. The interalveolar septum is separated the alveolars from each other. Similar but smaller interradicular septums are located among the multirooted teeth.

Figure 5.4. Figure 4. – On the radiogram exist the trabecular bone structure, a persistant decidous molar and a permanent molar. On the distal surface of the permanent molar persists a secunder caries next to the filling. 1.: interradicular septa, 2.: interalveolar septa a, 3.: permanent molar teeth, 4.: persistant decidous molar

The symphisis of the mandible is located on the external surface in the midline. The two part of the mandible joins here to each other. The mental protuberance is situated here as well. Laterally exists a tuber, the mental tuberculum. From here passes the external oblique ridge occlusally and distally to the outer surface of the
mandible’s ramus. The external oblique ridge is an attachment point for the different functional muscles. For example the depressor labii inferior muscle, that pulls the lower lip down. The depressor anguli oris muscle pulls the angle of the mouth down, the trumpet muscle ( buccinator muscle ) and a connecting tissue, the raphe pterygomandibularis are also erected from the external obique ridge. Laterally from the protuberance and over the tuberculum occurs the mental fossa. From here erect the mental muscle and partly the muscles around the mouth (orbicularis oris muscle). The mental foramen is situated in the premolar area. Through the mental foramen exists the mandibular canal. The vascular and nerve (alveolar inferior n./a./v.) system , that supply the lower jaw and the bone, are located in the mandibular canal. (Figure) On the internal surface there are resultant areas for the muscles: spina mentalis- 4 protuberances for the tongue and the upper mylohyoideal muscles, under this close to the midline is the stick point of the digastric muscle, the digastric fossa and the linea mylohyoidea- passess through distally and occlusaly. The mylohyoideal muscle erects from the spina mylohyoidea. On the two sides of the internal oblique ridge exist the two impressions of the salivary glands. In the front, occlusaly occurs the sublingual gland’s impression. Underneath and distally exists the submandibular gland.

**Figure 5.5. Figure 5.** – The radiograph represents the main structures of the lower jaw. The mandible is from an elderly patient, because the alveolar process exists just in the front region. 1: basis mandible, 2.: angulus mandible 3.: ramus mandible, 4.: condylus mandible, 5.: processus coronoides, 6.: external oblique ridge, 7.: foramen mentale

**Figure 5.6. Figure 6.** – On the internal surface of the mandible the radiograph demonstrates the lower jaw’s deep impressions, that was made by the submandibular glands under the internal oblique ridge. A canalis mandibulae bemeneti nyílása, a formane mendibulae is jól látható. 1.: foramen mandible, 2.: internal oblique ridge, 3.: fossa submandibular, 4.: spina mentalis
From crosssection a thick cortical lamina covers the mandible. The X-rays, that arrives perpendicular to the mandibular lower ridge, passess through the thick cortical layer and create a radiodense area, that is called the lower ridge of the mandible. (7. Figure)

**Figure 5.7.** Figure 7. – On the radiogram the second lower molar is detected. Next to the tooth the thick cortical layer of the lower jaw is well-marked, that looks like a detached structure that forms the lower ridge of the mandible. The external and internal oblique ridges are also identified on the radiograph. 1.: lower ridge of the mandible, 2.: internal oblique ridge, 3.: external oblique ridge, 4.: mandible canal, 5.: second lower molar

![Radiograph of the mandible](image)

On the radiograms of the lower front teeth’s occurs a triangle-shaped area, that is the project of the protuberantia mentalis. The meaning of the phenomenon is that the lower incisors could be covered by it and so prevents the diagnosis. (8. Figure)

**Figure 5.8.** Figure 8. – The radiograph represents the lower incisors with the pyramid-shaped protuberance. The cortical bone’s shadow could cover the apical region of the lower front teeth. This situation could make the examination difficult. In this case it concerns to the second incisors and canines
The spina mentalis is situated on the lingual surface of the mandible in the midline. It serves the adhesion of the geniglossus muscle and the geniohyoideus muscle. On the close radiograph the foramen linguale is visible. It is a small hole on the bony structure. On the radiograph, that was made by occlusal technic, the spina mentalis is detectable such as the adjacent muscles and the muscles of the floor of the mouth.

On the periapical radiographs the spina mentalis is situated in the neighbourhood of the lower middle incisors’ roots. It is separated from the environment as a radiodense structure. Sometimes the small hole is visible in the middle. (9. Figure)

**Figure 5.9. Figure 9.** – On both figures the radiodense spina mentalis is well detected. Especially on the left radiogram the foramen linguae is very expressive. The surrounding bone is resorped, because of the chorinal inflammation. 1.: spina mentalis, 2.: foramen linguae

As we mentioned before the X-ray examination is good for the detection of the hard tissues. Instead of this theory sometimes the soft tissue is also visible or the soft tissue absorption interferates with the hard tissue absorption and this phenomenon changes it’s liner and density. The situation of the lips in case of the lower front teeth is a good example for it. By the lip covered area is lighter, while the not covered area is much more radiodense. This could be different among patients. The differential diagnostic meaning is that it could make the analysis of the fracture difficult after a traumatic incidence.
Even smaller or bigger nerve and vascular canals are situated in the lower jaw. They are mostly small nutritive canals. Up to their size they become visible, because of the covering bone lamina is thinner. These canals are exist connected to the teeth or independently from them. This canal is responsible for the innervation of the lower teeth from the mandible. The canals could be mixed up with the fracture signs. To make a difference between the fracture and the nutritive canal we should know, that the wall of the canals are made of cortical lamines but the broken ends are not corticated.

**Figure 5.10. Figure 10.** – The radiolucence lines are detected as the nutritive canals. In the nutritive canals different vascular structure are located, that serves the blood supply for the teeth and the surrounding bones. The second lower incisor, the canine and the first premolar are represented on the radiograph. The incisor and the canine are filled. An extended radiolucence area is observed around the incisor’s apex

![Image of teeth and radiograph]

On the lingual surface of the mandible occurs the torus mandible. It’s size is different in every human. Usually it is located on one or two sides and close to the apex of the lower premolars. This is not a pathological disorder. The cortical lamina is thick, so it looks like a radiodense area on the radiographs. It usually covers the canine’s or the premolar’s apexes’ area.

The foramen mentale is situated on the external surface of the mandible, close to the apex of the premolars’. Veins and nerves leaves the canalis mandibulae through the foramen mentale.(mental artery and vein, mental nerve)

**Figure 5.11. Figure 11.** – The radiograph illustrates the first and the second premolar. The mandible canal runs close to the molars’ teeth and could be easily separated from the direct environment. The radiodensity of the thin cortical layer well marks the border of the tooth. The radiolucence structure that is located close to the premolars’ apex, is the mental foramen. All along the premolars’ roots the tripple pattern could be followed. It means, that pathological disorders are not detected here
Figure 5.12. – Around the lower premolar periapical area, especially around the distal apex the translucency is not a normal anatomical phenomenon. The triple pattern disappears around the root. The lamina dura is only detected in the marginal paradontium.

Because of the difficulty of the differential diagnostic this is a very important structure. The foramen mentale is well separated from its environment, well delimited and a regular radiolucence area. The inflammation around the apex is also similar. So to make a difference between this structure and the process is very important. The foramen mentale usually projects to the apex of the tooth. The triple pattern is also exists here and the lamina dura is still continuous. These pieces of information could help us in the diagnostic. In case of an inflammation the lamina dura is not continuous all along the roots. To repeat or to take other radiograms from other directions or to make a mesio or distoexcentrical radiograms could also help us in problematic situations. The radiolucence part of the mental foramen would be visible on the radiographs from a changed position due to the settings.

The next anatomical structure is the mandible canal. The alveolar inferior nerve, that is the branch of the trigeminus nerve, and the alveolar inferior vein and artery are passing here. The canal leaves the foramen
mandible on the ramus mandible and passes through the mental foramen. It ensures the nerve supply of the lower teeth and that’s why it is able to the alveolar nerve block.

The alveolar inferior nerve does not only innervates till the foramen mentale, than it innervates the canines and incisors as well. In many cases occurs the extension of the mandible canal, that is called the anterior loop. (13. Figure)

**Figure 5.13. Figure 13.** – The mandible canal runs under the root of the molar tooth, that passes through the mental foramen by the extracted second premolar’s hole and persists as the anterior loop. 1.: mandible canal, 2.: mental foramen, 3.: anterior loop

The mandible canal is situated under the molar teeth’s roots. It separates well from the direct environment, it is a regular radiolucence area covered by the cortical lamine. (14. Figure)

**Figure 5.14. Figure 14.** – The radiograph represents, that the mandibule canal is determined by the thin cortical layers

The internal oblique ridge is situated lingually on the mandible surface. This bony protuberance serves the attachment of the muscle, that forms a part of the floor of the mouth.
This is an expressive, sharp, radiodense line on the radiograms. This structure is the most visible on radiograms, that are made by by setting technics and it is situated properly to the molars’ and premolars’ apex. Sometimes it has a tight connection to the mandible canal or even they can projected to each other. (15. Figure)

**Figure 5.15. Figure 15.** – On this radiograph about the lower second molar well represents the internal oblique ridge and external oblique ridge. The crown border of the mandible canal and the internal oblique ridge are projected to each other, so on this picture they can not be separated from each other. 1.: lower ridge of the mandible, 2.: internal oblique ridge 3.:external oblique ridge 4.: mandible canal, 5.: second molar teeth

![](image)

The mandibular fossa exists exactly next to the internal oblique ridge. The submandibule gland forms an impression on the lingual surface of the lower jaw. The mandible basis is narrow here, so the fossa looks like a sharp-edged radiolucence area on the radiogram. We should differentiate this structure from extended cysts or tumors. (16. Figure)

**Figure 5.16. Figure 16.** – The submandibular fossa is a radiolucence area under the molars’ apex. This radiogram represents, that the mandible canal probably crosses it, projects on it, but owing to the cortical layer it is distinctive. 1.: canalis mandibulae, 2.submandibular fossa

![](image)

It runs nearly parallel to the internal oblique ridge. The external obliquity ridge is situated on the external surface of the mandible. It passes anteriorly to the direction of the molar teeth’s neck, as the continuation of the anterior edge of the processus coronoideus. This bony protuberance serves the attachment of the buccinator muscle’s fibers. (17. Figure)
Figure 5.17. Figure 17. – The radiodense line of the external oblique ridge and the internal oblique ridge is visible on both radiographs. These structures serve the attachment of the muscles, so they could be also detected after tooth loss. 1.: external obliqua extarna, 2.: internal oblique ridge

The external oblique ridge and the internal oblique ridge look similar structures to each others on the periapical radiograms. The differentiation is not that difficult if we know that the external oblique line is always situated closer to the crown on the radiograms.

2. 5.2. Intraoral X-ray anatomy of the upper jaw – Gyula Marada

2.1. Anatomy of the maxilla

From the frontal aspect of the skull and the upper jaw the bony entrance of the blowhole is easily detected. This is called the nasal fossa (apertura piriformis). It ensures the route for the unimpeded airflow by breathtaking. (18. Figure)

Figure 5.18. Figure 18. – From the frontal aspect of the maxilla the alveolar process of the upper jaw and the apertura piriformis are well marked. The structures of the fossa nasalis could be quite good detected on the radiogram of the upper front teeth, because it is situated close to he apex. 1.: spina nasalis inferior, 2.: apertura piriformis, 3.: nasal septum, 4.: alveolar processus
On the radiograms about the upper incisors the nasal fossa is often represented. (19. Figure) It appears as a radiolucence area and it is well separated from the direct environment. The fossa looks like two routes, a bony disjunctive lamina is situated between them. This structure is the nasal septum. The variable presence of the nasal fossa is owing to septum or to the nasal turbinance inferior. (20. Figure)

**Figure 5.19.** Figure 19. – On the upper middle incisors’ periapical radiograms the radiolucence apertura piriformis is well detected. The nasal septum divides it to two parts. The lower border of the apertura piriformis is the inferior margin of the nasal fossa. 1.: apertura piriformis, 2.: nasal septum, 3.: inferior margin of the nasal fossa

**Figure 5.20.** Figure 20. – On the radiograph the canine is centralised. Next to it occur the lateral incisor and the first premolar. The nasal fossa exists properly to the lateral incisor’s apex and the nasal septum is located on the left side. The nasal turbinance extends to the nasal cavity. In the canine an intrapulpal post could be seen after root canal filling. 1.: septum nasi, 2.: inferior margin of the nasal fossa, 3.: nasal turbinance inferior
The nasal septum, that is the radiodense structure of the cortical bone, proceeds in the inferior margin of the nasal fossa.

The nasal septum and the inferior margin of the nasal fossa cross each other in a bony block. On the radiograms it has a regular liner due to its tissue structure. It reminds us the shape of the diamond. This is the nasal spina inferior. This structure is radiolucence. The nasal spina inferior fixes the cartilage parts of the nose. (Figure 21)

**Figure 5.21.** Figure 21. – The nasal spina inferior is located in the crossover point of the nasal fossa and the nasal septum. This structure is thick and bony. Usually it is a radiodense regular shaped bone shadow, that could project on the apex of the upper middle incisors by an inadequate settings. 1.: inferior margin of the nasal fossa 2.: nasal septum, 3.: nasal spina inferior

**Figure 5.22.** Figure 22. – Fortunately it is not common, but it’s worth to mention, that on the upper incisors’ radiograms different malformations occur sometimes too. The most common is the retention of the canine. The radiogram is a good example for this anomaly. The canine projects on the middle incisor’s apex
The median palatinal suture is a bony suture, that runs through the median-saggital axis of the maxilla. Meanwhile the embryonal development it is formed by the union of the upper jaw’s elements. From the frontal aspect of the upper jaw exists a bony union from the nasal spina anterior, that is the continuation of the median palatinal suture.

On the upper middle incisors’s periapical radiograms the median palatinal suture appears as a radiolucence line, that is encircled by a thin cortical border. This could be followed till the nasal spina anterior. (23. Figure)

**Figure 5.23. Figure 23.** – In the middle of the radiogram, between the two middle incisors the median palatinal suture is visible till the nasal spina inferior. After a trauma it would be important to make a difference between the cleavage and the median palatinal suture. The normal anatomic structure has a thin radiodense cortical layer on the border. The incisors has extended caries and fillings. The hand instruments for the root canal treatment is also represented on the picture. 1.: apertura piriformis, 2.: nasal septum 3.: median palatinal suture

The incisive foramen is situated palatinally from the middle incisors in the midline. Through the palatinal aperture of the incisive canal the nerves and vascular structures leave the canal.
The incisive canal and the incisive foram are often visible on the upper incisors’ radiographs. This anatomical structures are surrounded by the thin cortical layers and do not dislocate the teeth. This is a difference between them and the cysts, that are in the same position, but during their growing the cysts remove the roots from each other. (24. Figure)

Figure 5.24. Figure 24. – On the radiogram exists the incisive foramen, that occurs in the middline, it is darker than the direct environment, and it is a regular-shaped area. Before the radiogram was made, the teeth had been aboutment prepared. This explains the irregular shape of the teeth. The rest of the luting cement’s shadow is also detected on the radiogram.

The soft tissues around the upper incisors also provide shades such as the lower incisors’ soft tissues. The most expressive is the borderline of the nose. It looks like a borderline all along the incisors’ roots. That’s why the teeth are more dense apically, and less lucence to the occlusal direction. This could be also mixed up with the fracture line. (25. Figure).

Figure 5.25. Figure 25. – The borderline of the nose is running in the middle of the crowns on the middle incisors. It should be separated from the fracture line. 1.: the border of the nose, 2.: median palatinal suture, 3.: nasal septum
The cartilaginous part of the nose flap provides shade first of all on the canines’ and lateral incisors’ apical area. The regular and sharp edged radiodense area projects sometimes to the apex of the tooth, that makes the diagnostic more difficult.

The liner of the lips are also visible sometimes on the upper teeth’s radiograms. This phenomenon is similar to the lower jaw’s case and the location is also that much variable.

The nasolabial wrinkle determinates the character of the face and it exists sometimes also on the radiograms. Usually it extends from the lateral incisors to the premolars. It’s presence is very similar to the other soft tissues, because by the thick soft tissue covered part is more radiodense and by the thinner soft tissue covered hard tissue is more radiolucence.

On the upper jaw we distinguish the corpus and the four processes. The sinus (Highmore-cave) filled in the whole maxillary corpus. It has four surfaces and it looks like a tetrahedron. The infraorbital canal is located in the middle of the anterior surface of the corpus, that contains the same named nerves and vessels. The fossa canina is situated under the canal, occlusally from the apex of the canine.

One of the four processes is the alveolar process, that holds the upper teeth. The two processes form a semicircle and create the upper arch (alveolar superior arch), that includes the upper dentil (dental superior arch). The mesial part as an incisive arc terminates in the frontal process. This arc forms the bony blowhole’s (apertura piriformis) lower part. This process is partly the attachment of the trumpet muscle (buccinator muscle). The alveolar process and the sinus are well connected to each other. The apex of the teeth, mostly the molars are situated very close to the sinus. This is well marked on the radiograms. On the radiographs often seems as the apex would be extended to the sinus. Of course it is not exists, but the sinus forms recessus around the apex.

The sinus is well extended on the maxilla so it could be well detected on the upper jaw’s radiograms. The anterior wall of the sinus is located distally from the canine. The radiograms about the canines and the premolars an important structure could be found, that would help us in the detection of area. This are sharp-edged radiodence lines, that are conversed Y shaped. These structures are not unattached anatomical structures. The british calles it „antral Y“. The horizontal arm of the Y is buld up by the basis of the nasal cavity, the vertical arm is the projection of the anterior wall of the sinus. Because the two structures are fromed by the projection, so the presence could change due to the patients and the settings. (26. Figure)

**Figure 5.26. Figure 26. – The antral Y is well marked on the radiogram. On the right radiogram the lying Y is higlihted, that helps orientation on the radiogram. This structure is built up by the anterior wall of the sinus and the palatum durum. 1.: palatum durum, 2.: border of the sinus**
The following anatomical structures are two bony impressions. One is situated between the mesial and the lateral incisors (incisiv fossa), and the other one is between the lateral incisors and the canines (canine fossa). In these areas, where impressions are located between the teeth apexes, the bone is always thinner than the direct environment. These structures are represented as radiolucence areas on the radiograms.

The sinus is an air content cavity, terminated by the cortical bone. It looks like a radiolucence area with radiodense border. (Figure 27)

**Figure 5.27.** The sinus is located close to the upper premolar’s and molar’s apex. The sinus forms recessus, that projects as the teeth would extend to the cavity. 1.: border of the sinus, 2.: separating septum of the sinus’s recessus 3.: cavity of the sinus 4.: Carabelli cusp

After the extraction of an upper molar the bone grows thin and the recessus would spread to the hole of the root. So the sinus’s capacity will enlarged slightly. This is called the pneumanisation. (Figure 28)

**Figure 5.28.** On the radiogram we can observe as the recessus spreads in the hole of the extracted tooth’s root earlier place. 1.: terminate of the sinus, 2.: zygomatic bone
As we mentioned before, the sinus is an irregular cavity. There are many processes separated from each other by the thin cortical walls (septum of the sinus). (29. Figure) It exists as the extended radiodense line of the terminated cortical sinus.

**Figure 5.29. Figure 29.** – The septum of the sinus comes from the apex of the second premolar’s apex, that separates the recessus from each other. It seems if the molar’s roots, especially the palatinal one would located in the sinus cavity. 1.: septum of the sinus, 2.: borderline of the sinus, 3.: cavity of the sinus

On the upper jaw’s lateral surface takes place the other process of the maxille. The tip facing down converse triangle-shaped zygomatic process is a rough protrusion on the contact point of the anterior and posterior part of the corpus and the orbita. The lower ridge of the triangle continued all along the alveolar process as the zygomatic-alveolar crista. It is a basic supporting pillar for the upper dentile. The process and the zygomatic process form a suture together. The zygomatic bone continues as the zygomatic arc.

To take a look at the bony skull from the lateral aspect, the zygomatic bone projects on the upper molars’ apex. This is usually well marked as U or V shaped radiodense boneshades. (30. Figure) The lower jaw and the zygomatic bone join to each other as the bony zygomatic-maxillar suture and the joining parts are lying on each others’ thick cortical surfaces. The identification of the different anatomical structures could be easier, if we know that the lower ridge of the zygomatic arc could join to the U-shaped boneshade’s lower pole and would continue distally.

**Figure 5.30. Figure 30.** – Both radiograms represent the zygomatic arc. The left picture illustrates the typical U shaped projection. The different settings causes another form of
the projection on the right radiogram. In both cases it projects on the molars’ apex and so makes the diagnostic difficult. 1. zygomatic bone (zygomatico-maxillare sture)

Ditally from the last molar in the continuation of the alveolar ridge exists an irregular bone surfaced protuberance. This is the maxillary tuber. (31. Figure)

**Figure 5.31.** Figure 31. – The maxillary tuber and the lateral pterygoideal process are detected on the retromolar’s area’s radiogram from lateral and lower aspect. On the right radiograph the bony suture is visible at the contact point of the zygomatic arc and the bones. 1.: lateral pterygoideal process 2.: maxillar tuber, 3.: zygomatic arcs, 4.: nasal spina inferior

This area often appears in the molar’s radiograph. It’s shape and presence could be variable. Sometimes it has spongious structure, but also the recessus of the sinus could fill this area in. (32. Figure)

**Figure 5.32.** Figure 32. – Distally from the upper wisdom tooth exists the maxillar tuber. In this case the spongious bone fills it in. 1.: zygomaticumbone, 2.: terminate of the sinus 3.: maxillar tuber
We rearly make intraoral radiograms distally from the wisdom tooth. So these two structures are not common, but they still exist. The lateral pterygoideal process is a part of the sphenoidal process and connects to the posterior surface of the upper jaw. (33. Figure)

**Figure 5.33.** Figure 33. – The lateral pterygoideal process connects to the maxillary tuber distally from the wisdom tooth. 1.: borderline of the sinus, 2.: maxillary tuber 3.: lateral pterygoideal process

The other bony structure is the lateral pterygoideal process part of the hamulus pterygoideus. It is illustrated as a thin, pencilformed bony structure in the radiograms.

In case of the upper jaw there are also vessels that run in the sinus. They are the end branches of the internal maxillar artery. On some radiograms the narrow radiolucence lines with thin cortical walls appear. Diagnostically they should be differenciated from the fracture line. (34. Figure)

**Figure 5.34.** Figure 34. – In the upper jaw nutritive canals exists, too. The canals contain mostly vessels that are surrounded by thin cortical layers. This structures are located in the sinus, where the bone is thinner because of the aircontent cavity. 1.: palatum durum 2.: borderline of the sinus 3.: nutritive canals
Another important structure is the basis of the nasal fossa. This is a bony structure, that separates the oral cavity from nasal cavity. This could be the palatum durum. Because the nasal fossa and the oral cavity is terminated it with the cortical layer, two parallel radiodense lines would be expected on the radiogram. Practically it doesn’t look like that. Mostly we see just one line, that seems to be the upper border of the sinus. (35. Figure)

**Figure 5.35.** Figure 35. – The palatum durum is represented as a radiodense line in the picture. It is located close to the sinus so it seems to be the top of it. On the left radiogram there is an enosseal implant in the place of the first premolar. No osseal integration exists. 1.: palatum durum, 2.: borderline of the sinus

In 20% of the patient a thick, bony protuberance could be detected (torus palatinus). It is located in the midline of the palatum durum. It is radiodence and sometimes it has lobar structur.

The inferior nasal turbinance is a protuberance in the bony nasal cavity, that rearly projects on the upper teeth’s radiograms. (20. Figure) Usually it is in the nasal fossa. Sometimes it looks like the torus in the midline, but more anteriorly. But the torus is much more calcified and thicker.

And last but not least the last anatomical structure would be mentioned. It is the part of the lower jaw but it is illustrated on the upper teeth’s radiograms. This is a triangle-shaped protuberance on the mandible. This is the coronoideal process. It serves the attachment of the temporal muscle. From lateral aspect of the skull it is detected, that the coronoideal process is located close to the upper molar teeth when the mouth is opened, and so it could appers on the radiograms as well. (37. Figure)

**Figure 5.36.** Figure 36. – The coronoideal process could be illustrated on the upper molar’s radiographs. It connects to a mobile bone, so not even it’s shape than it’s
position could also be variable. 1.: coronoideal process 2: zygomatic bone, 3.: lower ridge of the zygomatic arc

Figure 5.37. Figure 37. – This picture explains, how the coronoideal process could be projected on the upper teeth’s radiograph, when the mouth is opened. This could represent own personal variations, but this phenomenon exists for sure on the radiograms of the wisdom tooth. 1.: coronoideal process 2. condylus mandibulae, 3.: maxillary tuber

3. 5.3. The anatomy of the panoramic X-ray – Gyula Marada

Nowadays even more dentistry offices use the panoramic X-ray application. The official name is normally the orthopantomograph, but usually it is just mentioned as the panoramic X-ray or OP. In the dentistry it is very popular, because it works with a low exposure dose but serves many pieces of diagnostic information. The intraoral radiographs could not be substitute with the panoramic X-ray. Both methods have their own indicated part.

The panoramic radiographs could illustrate four indication areas. First is the dentoalveolar area. First of all we represent the alveolar process and the included teeth. The next is the maxillary region. This focuses on the upper jaw, maxillary sinus and the orbita. The third one is the mandible. The name refers that we use it to examine the ramus and the basis of the mandible. The last one is the region of the temporomandibular joint, but includes the retro-maxillar and mandible areas as well.

To identify precisely the anatomical structures on the radiograms, we should know the base of the method. During the exposure we make from the 3D object a 2D image. The easiest way to visualise it if we bend a radiogram to the skull, we expose it and then we lay the radiogram again on to the plain.
Figure 5.38. Figure 1. – Compared to the radiographs, that were exposed from different directions, the panoramic radiogram itself serves all the necessary datas.

During the exposure the beam source and the film (nowadays the digital sensor) rotate around the head in the same direction. The radius affects on different part of the sensor and so at the end we get the radiogram. The name of the machine reveals the most important traits of the OP. The central X-ray comes from the orthoradial direction, so it is perpendicular to the arch’s tangential. It is also a tomogram. With a special method we can sharply illustrate only one layer of the upper and lower jaw’s and the other parts are blurred. This could be very helpful in the diagnostic, because due to the settings for us only the most important parts are clearly visible (the area of the teeth). This also means a disadvantage, because the other parts are hardly visible. In the following chapters unfortunately we can not deal more with the principal of the operation. We will make a review about the anatomical structures on the radiograms, the appropriate position of the patient and about the exposure’s mistakes.

3.1. The maxilla

The maxillary sinus is an air contended cavity, that occupies the biggest part of the upper jaw (2. Figure). It is situated cranially from the molar teeth, but also has a close relation with it. The sinus is represented as a radiolucence area, that is terminated by the cortical wall as a radi dense line. On the next image the border of the sinus is well marked. The knowledge of the borders of the sinus and the detectability of the images are very important, because a small deformity also could refer to a malformation. In this case the distal wall is the most significant. Some own variations could happen as well. On the periapical radiograms owing to the pneumatisation the sinus could spread variable. Sometimes it enlarges in to the area of the maxillary tuber or to the space of the extracted teeth (3. Figure).

Figure 5.39. Figure 2. – The borderline of the maxillary sinus is well marked on the panoramic radiogram. In the right sinus a polipous, soft tissue structure exists. 1.: wall of the maxillary sinus.
Figure 5.40. Figure 3. – On the left side the sinus forms a recessus in the place of the extracted wisdom tooth. 1.: maxillary sinus, 2.: pneumatisation

The posterior wall of the maxilla is the part of the sphenoidal bone, the pterygoideal process encloses a drop-shaped area (4. Figure). This is the pterygomaxillar fissure. Anatomically it is an important area because the maxillary artery runs from the infratemporal fossa towards the pterygopalatinal fossa, where the trigeminal nerve leaves the skull.

Figure 5.41. Figure 4. – The conversed drop-shaped structure is the pterygomaxillar fissure in the radiographs. Sometimes the structure is illustrated different than before. 1.: pterygomaxillar fissure
The pterygomaxillary fissure is located on the radiograms distally from the maxillary sinus. It is a drop-shaped radiolucence area. Distally from it the lateral pterygoideal process exists. The lateral pterygoideal process is the part of the sphenoidal bone and the attachment of the lateral pterygoideal muscle and the muscles that goes towards the pharynx. From the lateral aspect the ascendent ramus of the mandible covers the lateral pterygoideal process and only could be visible if we remove the ramus.

The lateral process of the lateral pterygoideal process is the hamulus. It is a skinny, pencil-shaped bony structure. This is also the part of the sphenoidal bone and located distally from the maxillary alveolar process.

The next important structure is the arc of the zygomatic bone, that is built up by other bones. In the front region occurs the maxilla’s zygomatic process, distally from it the temporal process of the zygomatic bone is located, and in the background exists the zygomatic process of the temporal bone. Anatomical structures, that have a connection to the arc of the zygomatic bone, should be separated. The fist one is the zygomatic arc, that is formed by the processes of the temporale and zygomatic bones. The next one is the glenoidal fossa, that is the articular fossa of the temporomandible joint. Anteriorly the articulare tuberculum is located. The condylar of the mandible moves on the incline of the articular eminence during the mouth opening movements. The component processes of the arc join in the zygomaticotemporal suture to each other. Of course the cortical bone parts contact to each other. And the fifth structure is the zygomatic bone. These bony structures should be represented separated from each other, because they look like one continuous structure on the panoramic radiograms.

The zygomatic arc is a homogenous bony structure (*Figure* 5.42). Air contended cavities are found in low percent in the zygomatic bone. Usually these cellular structures are situated bilaterally and could be singular or multiplex.

*Figure 5.42.* *Figure 5.* – On both side the zygomatic arc is well detectable. Different bones form it, but the connected suture between them is not always well marked. If it is visible we should differentiate it from the fracture line. 1.: zygomatic arc, 2.: zygomaticotemporal suture
The zygomaticotemporal suture was mentioned before (5. Figure). It is worth to talk a little bit more about its significance. It is usually mixed up with the fracture line after a traumatic event. In case if the skull is injured, several times the zygomatic arc will be also broken. The best way to differentiate them if we know that the bony suture is always connected by the cortical bones, but in case of the fracture the cortical layer is missing.

Not far away from the zygomatic arc another bony junction is located. In the periapical radiogram’s chapter was already mentioned that the maxilla and the zygomatic bone join to each other with the bony zygomaticomaxillary suture (6. Figure). This is represented as a U or V shaped radiodense shade. It could be easily identify on the panoramic radiograms. It looks like a „J“ letter or almost a fishing hook. It is usually located over the second molars’ apexes. To differentiate it from the posterior wall is very important.

**Figure 5.43. Figure 6.** – The „J“ shaped, radiodense structure between the maxilla and the zygomatic arc is well marked on the radiograph. 1.: zygomaticomaxillary suture

Posteriorly from the zygomatic arc the glenoidal fossa is located. The fossa and the condylar constitute together the temporomandibular joint. In the central relation the condylar is located in the acetabulum. The temporal bone projects on to this part, so this area is not always represented on the panoramic radiographs. But the external acoustic meatus is well detected in most cases, so it serves a good stronghold to the location of the fossa. The bony meatus is radiolucence, round, and bordered by the cortical layer (7. and 8. Figure).
Figure 5.44. Figure 7. – The external acoustic meatus, as a regular radiolucence structure is easily identified on the radiograph. It helps to find the glenoidal fossa, that forms the temporomandibular fossa. 1.: external acoustic meatus, 2.: glenoidal fossa

Figure 5.45. Figure 8. – This specific radiograph was exposed about the former patient. The left image illustrates the temporomandibular joint in an opened mouth position, the right in a closed mouth position. We can observe that the condylar moves down to the anterior direction on the tuberculum’s incline during the opening movement. The articular discus is not visible on the radiograph. 1.: external acoustic meatus, 2.: glenoidal fossa, 3.: articulare tuberculum

The mastoidal process (process of the temporal bone) is located distally from the external acoustic meatus (9. Figure) The structure of the mastoidal process is different from the regular spongious bone. This is an air content cavity, so it is radiolucence and seems to be foamy on the radiographs. Depending on the size and the age of the patient, the process projects totally or just partly to the radiogram.

Figure 5.46. Figure 9. – Large part of the mastoidal process is represented on a young patient’s radiogram. Cranially the medial cranial fossa is located. If we take a look at the various toothings, we can see that the decidual teeth are situated over the permanent teeth. The radiogram also represents the development and the resorption of the decidual tooth. 1.: mastoidal processus 2.: medial cranial fossa
In some cases the bony base of the medial cranial fossa is also detected on the radiograms (9. Figure) it is usually situated over the zygomatic arc, in the upper corners of the radiograms. The diagnostic value of this radiogram is low, but the detection of the anatomical structure could be important.

On every panoramic radiograms the orbita is represented partly or totally. In the orbita takes place the eyeball, the muscles that move the eyes and other soft tissues. It protects the eyes from different traumas. The orbita is represented as a radiolucence area over the maxillary sinus. The orbital ridge is also built up by the cortical bone. (10. Figure)

The lower ridge of the orbita, the infraorbital ridge is illustrated on every panoramic radiograms. It is located close to the maxillary sinus, so they should be differentiate from each other.

Close and caudally from the infraorbital ridge exists the infraorbital foramen. The infraorbital nerve is the continuation of the middle trigeminus nerve and leaves it through the infraorbital foramen with the similar named arteries and veins.

In some special cases the infraorbital canal connects to the infraorbital foramen so it’s liner also could be well marked. These are usually two radiodense parallel lines, running from the foramen through cranially and distally trough the infraorbital ridge.

**Figure 5.47.** Figure 10. – The total or just a part of the orbita could be observed on every panoramic radiograms. The infraorbital ridge and the maxillary sinus should be separated from each other. On some radiogram the infraorbital foramen and infraorbital canal are represented as well. 1.: orbita, 2.: infraorbital ridge, 3.: infraorbital foramen 4.: infraorbital canal
The nasal fossa, is often called the nasal cavity, ensures the breathtaking through the nose. The bony frame is the apertura piriformis, that could be well identified from the frontal aspect of the skull. Laterally it is bordered by the same cortical bone as in case of the anterior maxillary sinus wall.

The nasal septum divides the nasal fossa in two parts. The nasal septum is a thin boneplate. The septum is not symmetric or straight in every cases.

The nasal turbinace is not illustrated in every intraoral images. The panoramic images always represent the nasal turbinance and ensures the easy diagnostic. First of all the inferior turbiance is the most significant. It protruses from the lateral nasal wall to the nasal cavity. The sagittal aspect of the skull represents that the inferior turbinance enlarges all along the nasal fossa. Owning to the special X-rays technics this anatomical structure projects on to the maxillary sinus and could block the examination. (11. Figure)

**Figure 5.48. Figure 11. – The identification of the nasal fossa on a panoramic radiogram is not difficult.** The nasal septum divides it two parts in the middle. The deviation of the septum also could be detected. The inferior turbinance is well marked on the panoramic radiograms contrast to the intaroral radiograms. It’s dorsal process projects to the sinus. 1.: nasal inferior turbinance 2.: the upper edge of the inferior turbinance , 3.: nasal fossa, 4.: nasal septum
The incisive foramen continues in the incisive canal, that passes through the palatum durum and contains arteries and veins and the nasopalatinal nerve. The incisive foramen is almost represented on every panoramic radiograms, but it is not always illustrated as clear as on the periapical radiographs. The radiolucence shade between the two upper incisors’s apexes could be easily mixed up with a cyst or other pathological malformations. The incisive canal could be less observed than the foramen. In case of the total edentulous the thin bone wall of the canal is detectable. (12. Figure)

**Figure 5.49. Figure 12.** – In case of total edentulous the incisive canal is well marked. Usually the soft tissues and the anterior nasal spina could project on it. 1.: incisive canal

The palatum durum is represented clearly on every radiograms. Regular edged, straight running, radiodense line usually illustrated doubled. Owing to the modern technic it happens rarely. The upper line shows the projected shade of the other side palatum durum. (13. Figure)

**Figure 5.50. Figure 13.** – The radiodense line of the palatum durum is located along the upper teeth’s apexes. The other line, that has the same running, but is not illustrated that clearly and located over the palatum’s line, is the projected line of the other side’s
structure. The tuber maxillae exists also on the radiograms, as a bony structure distally from the molar teeth. We have already mentioned it’s structure and the phenomenon of the pneumanisation. 1.: palatum durum, 2.: shade of the other side palatum durum 3.: maxillary tuber

The tuber maxillae is located distally from the last molar, as the continuation of the alveolar crest. It is an irregular surface with a bony protrusion. It is located close to the maxillary sinus that causes variable forms to the tuber maxillae.

At the end we should discuss about the teeth on the panoramic radiograms. In most case of abnormal situated teeth this method ensures the possibility of a right identification. But this is also a reliable technic to identify shape deformations, numeral discrepancies or pathological processes in the bone. But the diagnostic value of the panoramic radiogram in case of caries or paradontological indications is lower than the periapical radiogram’s.

Figure 5.51. Figure 14. – Enlarged cyst exists on the lower jaw. Even the drain for the cyst narrowing is visible

Figure 5.52. Figure 15. – Retinated right upper canine’s radiogram
3.2. The structures of the mandible and the neck

We begin the description with the ascend ramus of the mandible. Transversely the ramus is a flattend, qudrate plate. The mandibular angulus is embraced by the body and the ramus. The angle changes during ages: normally by an adult it is 110-125°, by infants and elder people it is wider (135-150°). On the external and internal surface there are attachment areas for the chewmuscles. These are the massteric and ptrygoideal tubers.

On the upper part of the ramus two processes exist. The mandibule incisure separates them from each other. The coronoid process is located anteriorly, the temporal muscle attaches here. The condylar process is situated distally from the coronoid process. The condylar process is ovoid in shape and its long axis facing forward and inward and part of the TMJ (15. Figure). The madibular collum is located under the mandibular caput. The lateral pterygoideal muscle attaches on the pterygoideal fovea, on the anterior surface of the mandibular collum.

On the internal surface exists the mandibular foramen. A bonelayer is located anteriorly and superiorly from the mandibular foramen: this is the mandibular lingula or the Spix-prickle. The nerve blockade of the lower teeth is implemented in this region. This area overlaps the rotation axis. To find the exact point for the correct inferior alveolar nerve block we ask the patient to open and close his mouth.

Figure 5.53. Figure 16. – The ascendant ramus of the mandible and the processes could be well identified in the panoramic radiograms and could be determinative in case of the pathological progresses. In this radiograms the different parts can be easily separated from each other. If the alveolar nerve block was not successfull, we better take a look at the panoramic radiograms, because the irregular position of the mandibular foramen can cause the failure. 1.: ramus of the mandible, 2.: mandibular incisure, 3.: coronoid process, 4.: condylar processus condylaris 5.: mandibular foramen
The caput mandibulae is situated in the glenoidal fossa and form the temporomadibular articular together. The position and the shape of the condylar is variable in every patient. In case of opened or closed mouth the OP is the basic diagnostic of the articular detection. We have to examine the two articulans at the same time.

The coronoideal process serves the attachment area for the temporal muscle. It is a converse shaped triangle and the tip shows towards the muscle.

The mandibular incisure is an impression that separates the two processes from each other.

On the radiograms the structures in front of or behind each other are projected to each other. In the area of the coronoideal process different bones are located. Despite of their projection we have to differentiate them from each other. So the lateral pterigoideal process and the zygomatic arc summarize with the coronoideal process. The importance of the differentiate diagnostic is, that the lateral pterygoideal process sometimes draws a fracture line shade on to the coronoideal process. The fracture of this process is very rare, but after a traumatic event it can not be excluded. We differentiate the normal anatomical structures from the fracture line, the same way as we mentioned before. The zygomaticotemporal suture also could project on to the coronoideal process, and imitates the fracture line. (17. Figure)

**Figure 5.54. Figure 17.** – The liners of the different anatomical structures are highlighted with different colours. The projections are more well marked on this radiogra. Green: zygomatic arc yellow: lateral pterygoideal process red: coronoideal process
The ramus is situated distally on the mandible. Generally the left and the right ramus are symmetric.

The styloideal process goes from the basis of the mastoideal process towards the angulus. (18. Figure) It serves the attachment of the ligaments and muscles (stylohyoideal ligament, stylohioid muscle, stylogloss muscle, stylopharyngeal muscle). The ligament can contain bony parts, that is represented as the spongy bone surrounded by the cortical layer. We should differentiate it from the distrophic calcification of the ligament, where the earlier mentioned traits are missing.

Figure 5.55. Figure 18. – The thin and pencil shaped styloideal process exists on the radiogram. Somewhere the connected ligaments are also visible. On the right side of the radiogram the vertebrae are numbered. This type of the diagnostic is not valid for the correct identification of the vertebrae. If the vertebrae are projected on the front region, the front teeth will be not well examined. 1.: processus styloideus

On the both edges of the panoramic radiograms the neck vertebrae’s shades are found. This diagnostic is not valid for reliable examination of the vertebrae. Because of the exposure technic the vertebrae are also situated in the middle of the radiogram as well.
As well in case of the intraoral radiograms, as in case in the panoramic radiograms the inferior margin of the mandible is also slightly thick and a regular radiodense structure. It passes through all along the lower jaw. The irregular form can refer to malignant and benign processes.

The external oblique ridge and the internal oblique ridge can be found on the OP as well. The external oblique ridge as the continuation of the ramus goes towards the edentulous ridge, the internal oblique ridge is situated due to the molars’ apexes.

The mandible canal exists also on the OP. It begins with the mandible foramen, where the alveolar mandible nerve, artery and vein enter into the canal. The mandible foramen is located mesiodistally on the narrowest part of the ascendant ramus, in the middle. This point is important in case of the alveolar nerve block. Sometimes this hole could be located in irregular areas. It can exist also on the distal ridge of the ascendant ramus. We should count with it in case of an unsuccessful alveolar nerve block.

The mandible canal is a radiolucence structure that runs under the lower teeth’s apexes and it is surrounded by a thin cortical layer. Usually the two sides are symmetrical. The exact location is variable and sometimes it can not be followed all along the radiograms. This phenomenon could be caused by radiolucence structures, for example the submandibular fossa. An asymmetric shape or a location can also mean pathological disorders.

The other hole of the canal is the mental foramen. It is very important to differentiate it from the inflammatory processes. (19. Figure)

Figure 5.56. Figure 19. – This figure represents the related structures of the mandible. The external oblique ridge and the internal oblique ridge are also as well visible on the panoramic radiographs as on the intraroral radiographs. The borderline of the mandible canal is detectable because of the radiodense shaded cortical layer. The hole is the mental foramen. We have already mentioned its significance. 1.: external obliqua ridge 2.: internal oblique ridge 3.: mandible canal 4.: inferior margin of the mandible 5.: mental foramen

The submandibular fossa is located as an impression on the lingual surface and ensures the location for the submandibular gland. This area is more radiolucence than it’s direct environment. It exists caudally from the molars’ and premolars’ apexes. It is useful to know that other structures can also cause radiolucency.

The mental tuberculum takes place on the mental protuberance. It is illustrated as a radiolucence triangle-shaped shade in the midline on the intraoral radiograms. Individual variancy can happen. This could be enhanced by the different settings.
The spina mentalis is located opposite to the mental protuberance, on the lingual surface. It ensures the attachment for the upper muscle of the tongue and the upper hyoid’s muscle. Sometimes the lingual foramen is also visible, such on the 20. figure.

**Figure 5.57. Figure 20.** – The marked bony radiodense block is the spina mental and in the middle the lingual foramen is visible. 1.: spina mental (foramen linguæ)

Caudally from the basis of the mandible an independent bone exists. This is the hyoid bone. It is situated between the ramuses, it connects to the basis of the tongue and the larynx as well. It fixes the tongue, the larynx and the pharynx. This ensures also attachment for the muscles. Due to the length of the patient’s head or to the bending of the head according to the horizontal plain, the hyoid bone can be seen totally or partly under the mandible. The basis of the mandible also can covers it totally. (20. Figure)

**Figure 5.58. Figure 21.** – The radiographs were taken about two different patients and their hyoids are illustrated different from each other. On the left side it exists under the lower jaw, on the right the hyoid is half covered by the mandible. 1.: hyoid bone

### 3.3. Airways and soft structure
In the head and neck region exist many soft structures, that appear on the radiograms as well. This can make the diagnostic more difficult, because the soft structures can cover important parts, or apper as a fracture or look like tumors.

Because of the mobility the soft tissues ,that mostly are muscles , are represented in variable forms. A typical example is the tongue. In case of the exposing the toung should be constricted to the palate, so it forms a related structure with the soft palate.

In some cases the patient doesn’t constrict it’s tongue to the palate.in this case it appears as well marked radiolucence shaded hole between th etongue and the hard palate. It is called the palatoglossal space.

The palatoglossal space persists distally in to the nasopharyngeal space. The soft palate separates them from each other. Because this is also a soft tissue, that is built up mostly by muscle, it doesn’t have typical shape or location. It is found as the enlargement of the bony radiolucence hard palate.

The neck vertebrales are almost totally covered by the soft tissues of the pharynx distal wall. This structure has a homogenous density. This structure’s diagnostic is necessary.

During inhaling and exhaling the air goes through different airspaces. We have already mentioned two of them but a third is exist too. The nasopharyngeal space, begins with the apertura piriformis, joins the palatoglossal space by the soft palate and they form the glossopharyngeal space together. It goes towards the larynx. These spaces appear as well marked dark or radiolucence shades. In these spaces different structures cross each other by there borderlines and look like fractureline shades. If the cortical layer is continuous and the could be all along followed it should be airspace not a fracture. (22. Figure)

**Figure 5.59. Figure 22.** – The aircontended spaces can be detected on the radiographs. The nasopharyngeal space begins with the apertura piriformis and it can be also detected. The patient didn’t elevate his tongue to the palatum, so the palatoglossal space can be identified. On the border of the two structures the shade of the soft palate exists. The two spaceses connected as the glossopharyngeal space. Red: nasopharyngeal space, yellow: palatoglossal space, green: glossopharyngeal space

![Figure 5.59. Figure 22.](image)

**Figure 5.60. Figure 23.** – On the next CBCT illustration the airspaces are marked with red colour. The way of the air can be well followed through airspaces towards the lung. The illustration represents as well the airspaces as the aircontended cavities

![Figure 5.60. Figure 23.](image)
The ear has also a homogeneous density. It is located under the mastoideal process and it is more radiolucence on the radiograms. If the ear projects on to the ramus, it can make the diagnostic difficult. (24. Figure)

Figure 5.61. Figure 24. – On the radiogram both ears are visible, despite of it is a soft tissue. It makes the diagnostic difficult if the ascendent ramus of the mandible covers and project on other structures. 1.: ear

As on the periapical radiograms the liner of the nose is found close to the upper molars’ apaxes, it exists on the OP as well.

Sometimes the liner of the lips are also detectable, when the patient bites on to the pipe. It goes through the lower and the incisor’s crown parts and should be differentiate from the fractureline.
Sometimes, but we have to mention the nasolabial fold. It separates the thin part of the bucca from a layer, that contains more muscle and fat tissues. A gently vertical shade could be detected, that goes from the nasoflap towards the corners of the lips.

### 3.4. Not anatomical structures on panoramic radiograms

In many cases not anatomical structures can appear also on the panoramic radiograms. These artefacts are sometimes the shade of the machine. This phenomenon can be caused by the positional pipe for example. The patient bite on it with its incisors during the exposure. This is a regular shape all along the front teeth and can cover other anatomical structures.

That’s how can appear the mental pillar as well due to settings failure.

Many machine has lateral positioning pillar, that fixes the temple during the exposure. It can help in the diagnostic as well. The carotis artery is also in a similar position. It is usually not visible on the radiograms, but the atheromal plaques appear. If the plaques are enlarged the wall of the artery can be as homogenous as the positional instrument.

### 3.5. The phantom views

Due to the specific orthopantomograph exposure technic every anatomical structures are projected twice. Because the beam source and the sensor rotate, the x-ray passes through the object, and after 180 turning passes through again. Usually it does not cause any problems, because the first projection will be close to the film and the second will be far from it. Only the tooth that is located close to the film could be clearly illustrated. Due to the specific technic the phantom view is located upper than the original object, is situated on the other side, it is enlarged and the borders are blurred. But the orientation is the same.

The most typical phantom view is the appearance of the mandible angulus. That can covers the mandible’s and maxilla’s structures.

The not anatomical structures, such as the earrings and piercings, can disturb the diagnostic. They are mostly made of alloy and they have expressive radiodense shade. Their phantom shape is also radiodense and can covers anatomical structures, so they become undetectable. (25. Figure)

**Figure 5.62. Figure 25.** – Let’s observe the typical properties of the phantom view: due to the original object it exists on the other side on the radiogram, it is enlarged and the edges are blurred, but they are situated the same way. 1.: earring, 2.: phantom view
When we described the hard palate we mentioned that it is represented as a doubled radiodense line. The one that is located cranially, is the phantom view of the other one, which is located on the opposite side.

The phantom view of the spine can also disturb the detection of the front teeth. Sometimes the vertebrae are also illustrated in the midline as well. It will be more expressive if the patient has spine disorders or if the x-ray is not perpendicular to the spine in the neck. The modern OP machine can easily eliminate this problem. (26. Figure)

**Figure 5.63. Figure 26. – The spine appears in the midline and so the front teeth become undetectable. But the new X-rays can eliminate these problems**

At the end we mention the pseudo-cyst in the mandibular midline. It is not a real cyst, it is just the impression of the mandibular surface. If the patient is located in the sharp contrasted region, the cyst would look like as a radiolucent shade.

### 4. 5.4. The successful panoramic x-ray exposure – Gyula Marada

The panoramic x-ray exposure is an irreplaceable part of the diagnostic in dentistry. The first expose was taken in 1934 and since the rules of the method has not changed a lot. Nowadays different type of machines could be purchased, even the basic concept is the same.

The part will represent the necessary pieces of information about the correct way of exposing. But we always have to follow the rules of the producers.

First of all we would like to introduce the failures that can happen during the exposing. This is important to know, because sometimes also the person, who is responsible for the exposing, does not know the origin and the reason of the failure. Every factor is detected as a failure, that harms the diagnostic. So first of all the most important task is to eliminate these failures.

#### 4.1. The panoramic specificity

In the anatomic part of the panoramic radiograms we mentioned that this is tomodiagnosis. The tomodiagnosis means that only defined shaped and defined thick layer can be clearly illustrated. The structures, that are located out of the sharp contrasted region, will be shadowy and less visible. The further they are located the more obscure they are. This way try the producers to highlight significant areas. In most of the cases it means the arc of the teeth. The location of the sharp layer and size could be defined just avarage by the producers. So the irregular sized or shaped upper and lower jaws’ important structures can not be located in the well contrasted
area so the identification become more difficult. The shape and the size of the sharp areas are implemented by different methods of the producers. Mostly they change the location of the focus point or the rotational speed.

4.2. The correct position of the patient

We also have to follow the producer’s rules here as well. Due to some machines the patient have to sit down. Others would let the patient stand or even lay down.

First of all we have to ask the patient to remove every prosthesis or other objects that can disturb the identification of the exposure from it’s oral cavity. It could be the nowadays very fancy tongue and lippiercings. Than the patient has to take of it’s glasses and every metal hair-slides. The jewels such as earings and necklaces should be removed to.

Before we expose we have to inform the patient about it’s roles during the process. We have to show the patient how to bite on the positional pipe and inform him about it’s importance. The tongue should be constricted to the soft palate all along the process as well. We use lead overcoat to protect the patient. This overcoat is different from the intraoral used coats, because it covers the chest and the back as well. (27. Figure)

**Figure 5.64. Figure 27. – The right way to use the lead overcoat and take the right position.** The patient always has to pay attention that it should keep it’s tongue in the right position and the positional has also an important meaning. The straight body pose eliminates the possibility, that the X-ray during the rotation touches the patient's shoulder, and the radiogram could become unusable. On the left picture the head is in the prescribed position

![Image of patient with lead overcoat]

We have to choose the right expositional rates to reach the correct sharpness and contrasting. In most of the cases the device offers average settings, but in specific cases we have to transgress the rules and set it by our own. Usually we can not change the expositional time just the amperage and the voltage. In case of an overweight patient or if the patient’s mandible is thicker than the average we can use higher kV or mA rates. In case if someone has a very thin bone, or is totally endentulous we have to work with lower rates to reach the successful expose.

1. Failure

**Figure 5.65. Figure 28. – The front teeth are seem to be very thin, and because they are not in the sharp contrasted area, the borders are not clearly visible.** The spine’s projection disturbs the detection on both sides
The description of the failure: the frontal teeth’s area is shadowy, the teeth are thin, the spin projects on to the ramus and an overlap exists in the premolar region.

The reason of the failure: the patient was not well positioned. The patient was located too forward, so the frontal area got out of the sharp area.

How can we correct it: it is important that the teeth should be situated in the correct part of the pipe. We usually recognise this failure if the patient does not bite on to the right part or if the frontal teeth are missing. In this case we should put gauze between the positional pipe and the endentulous ridge.

2. Failure

Figure 5.66. Figure 29. – The front teeth do not have sharp edges, they are wide and cannot be well examined because the spine covers them. The ascendent ramus of the mandible is enlarged so the condylars are not on the radiogram.

The description of the failure: the frontal teeth’s area is obscure, the teeth are thick and the spine projects on to the frontal teeth’s region.
The reason of the failure: The patient was not well positioned. He stood too much in the back, so the frontal area bypassed from the sharp region.

How can we correct it: the teeth should be located in the right part of the pipe during the exposure. This failure will be very common if the patient’s frontal teeth exist to much in the anterior region. Because the bending of the teeth the apex and the crown can not be found together in the sharp region. In this case we can make the best exposure if we set the patient further than before. The apex exists then in the sharp region. In this situation the crown part is not that important radiologically, because we can examine that clinically.

3. Failure

**Figure 5.67. Figure 30.** – The patient bended it’s head too much in the front. The lower jaw became V shaped then. The lower teeth are not situated in the sharp contrasted region, so their exposure is obscure. In the premolar region the approximal parts cover each other.

The description of the failure: the apexes of the lower incisors are out of the sharp region and obscured. The hyoid bone projects on to the basis of the mandible. The condylar of the mandibular joint is also often missing from the radiograms. The premolars overlap each other.

The reason of the failure: The patient wasn’t well positioned. The patient’s head was too much bended anteriorly.

How can we correct it: it is important to follow the producer’s rules. This contains the reference points and the description of the plains. There is a device that uses the tragus-nasal flap line and others use the Frankfurt-horizontal plane as a reference line. If the reference plains are not well marked, we should set the occlusal plane. The occlusal plane should enclose 5 with the horizontal plane.

4. Failure

**Figure 5.68. Figure 31.** – The settings failure is well detectable on this radiogram. The lower incisors look like a fan, because they are out of the sharp contrasted region. The upper incisors’ follow the lower incisors’ arc. The radiodense shade of the hard palate could covers the apexes.
The description of the failure: the upper incisors are out of the sharp region. The apexes of the maxilla’s teeth are covered by the hard palate. The condylars are also missing from the radiogram.

The reason of the failure: The patient was not well positioned. The head was bended backwards.

How can we correct it: it is important to follow the producer’s rules. This contains the reference points and the description of the plains. There is a device that uses the tragus-nasal flap line and others use the Frankfurt-horizontal plane as a reference line. If the reference plains are not well marked, we should set the occlusal plane. The occlusal plane should enclose 5 with the horizontal plane.

5. Failure

Figure 5.69. Figure 32. – The median sagittal plain was not well setted. The lower and upper jaw are not symmetric with each other, the right angulus projects lower than the left.

The description of the failure: It seems that the patient bended his head laterally. In this situation the angulus and the teeth are bigger on one side than on the other.
A hiba oka: A páciens feje oldalra dőlt a felvétel során.

The reason of the failure: The patient bended his head laterally.

How can we correct it: The incorrect settings of the median-saggital plain is not common. Many devices have temper pillars, that can help the correct position. If this situation occurs, than it has anorganical reasons, such as orthopedic disorders.

6. Failure

Figure 5.70. Figure 33. – The patient did not bend his head laterally than he turned it. As it’s result some of the teeth are out of the well contrasted region and they become enlarged and the ridges will be blurred

![Image](image_url)

The description of the failure: on one side the teeth are wider are overlaps on each other, and on the other side they are rather thinner. Due to the thickness the ascendant ramuses and the condylars show some differences.

The reason of the failure: The patient’s head turned laterally.

The symmetry of the face is sometimes different from the symmetry of the teeth or jaws. During panoramic radiographs we usually set the symmetry of face. How can we correct it: The incorrect setting of the median-saggital plain can cause the failure. The soft structures and the hard tissues simmetry is also differnt. So we set the reference plain due to the face’s simmetry.

7. Failure

Figure 5.71. Figure 34. – In all cases the patient should pay attention that its’ tongue should be all time long on the palatum. If it fails the palatoglossal space would be free and its’ radiolucence property will make the detection of the upper incisors’ more difficult
The description of the failure: On the radiogram a thick radiolucence area is represented under the hard palate, and this area can cover the upper teeth’s apexes.

The reason of the failure: The patient did not elevate it’s tongue to the hard palate.

How can we correct: We always have to ask the patient to constrict the tongue to the palate. If it can be observed only just on one part of the film, than it means that the patient did not constricted it’s tongue to the palate all along the exposure. It helps the patient to understand the right position if we ask the patient to swallow and tell to hold the tongue in the same position.

8. Failure

Figure 5.72. Figure 35. – The not straight bodypose is also a reason of the failure expose. Its’ result is well marked on the radiograph as a light triangle-shaped form on the front mandible. In this area the teeth are not well recodetermined. This failure is can not be avoided beacause of the patient’s physique or other malformations.

The description of the failure: In the middle of the radiogram the spin looks like a triangle shaped radiodense area.
The reason of the failure: The patient did not hold it’s body straight during the exposure.

How can we correct: We always have to ask the patient to keep straight it’s body. The spin always should be straight. Sometimes we can not eliminate this failure if the patient has shorter neck or suffers from arthritis.

Other failures can occur as well, we just tried to mention the most common types. There is also another case when the patient moves during the exposure. We can see there step formation. Or technical problems can block the exposure as well. For example the whole radiogram can not be done, because the exposure is not perfect during the rotation.

**Figure 5.73. Figure 36. – The underage child moved during the exposure. Still he was standing calm, the radiogram was sharp (on the right side) and then because of the movements the radiogram become totally blurred**

Nowadays most of the devices are digital. This is represented not only in the beam reduction, than it also eliminates several failures of processing.

### 5. 5.5. The anatomy of the CBCT images – Gyula Marada

During the last decades the technical development, the possibility of the correct diagnosis and the necessity of the well planned treatment allow the radiological image technics applications in the dentistry as well. Nowadays the expension of the CT is getting even more popular. First of all it was used by the maxillo-facial surgery, but later when the enosseal implants’s application became more common, the dentoalveolar surgery also began to use the CT. In the 90’s a totally new CT was represented, that was developed especially for the head and the neck region, so it was well designed for the dentistry other departments as well. The cone beam CT (CBCT) works with a lower radiation dosage, than the conventional one. The size of the CT machine is also adapted to the dental clinic’s size, so it is even much smaller, than the conventional one. The application of the software formed it very user-friendly. A new slice dimension was used, that contains more pieces of information than the commercial 3D slices had.

The computer tomography doesn’t have an old historical background. The first CT was represented in 1967 by Sir Godfrey Hounsfield. Since that it went through many technical innovations, especially owing to the development of the computing technology. Nowadays the 5th generation is applied, but the innovations are still on progress. The basic theory is built on the X-ray. The different geometrical formed ray beams passes over the body and blocked in to the detector. The beam source circles the whole surface of the body. The recorded images contain all the pieces information to reconstruct the 3D image about the examined structure. The exactness of
the image depends on the number of the images’ series and the intensity of the rays and so many other different structure.

The CBCT is designed due to the application borders of the commercial CT. The most important difference is that the CBCT works with cone shaped ray beams. The flat panel detector (FPD) or a charge coupled device (CCD) detects after amplifying. That is why it works with lower ray density than the commercial CTs. After the images are taken then it is able to produce and store pieces of information in the DICOM (Digital Imaging and Communication in Medicine, DICOM).

The most significant differences between the CBCT and the commercial CT are the following:

- The beam density of the CBCT is much smaller because the amperage is also smaller. The voltage (90-120 kv) is similar, but the amperage changes btw. 1 and 8 mA. The commercial CT works around 80 mA but could increase to 200 mA.

- The detector system is different. (FPD or CCD).

- The CBCT’s resolution of the elementary units is better, the size of the voxels are smaller.

- The number of the different artefacts, that were made by alloys are smaller, but the lower density causes higher noise and less information is available about the soft tissues.

- The CBCT is significantly cheaper and smaller.

Nowadays several company produce CBCT. These machines could be compared to each other in many ways, but the most useful if compare the size of the projected areas. There are CBCTs, that make low field of view (FOD, <8cm). These represent usually a segment of the upper or lower jaw. The higher FOV (8-15 cm FOV) CBCTs could illustrate the whole upper or lower jaw. The even more higher FOV (15-21 cm FOV) CBCTs could examine them together and could applied by orthodontic treatment. 21 cm or even higher FOV CBCTs are also exist and used for the whole skull or head-neck region diagnostication.

### 5.1. Consumption of the CBCT

First of all the CBCT is used for diagnostic. It helps to detect the wisdom teeth’s position and their relation with the direct environment. For example it could be very important, when we want to define the status of the wisdom tooth due to the canal. The widest application field could be the planning of the implantation method. But it ensures the examination of the temporomandibular joint, the planning of the orthodontic and other operation treatments, the detection of the pathological processes in the bone, examination of the airways and adjunct cavities. The undeformed and unenlarged images are well serves the orthodontic treatments. Recording a CBCT image doesn’t even mean a plus beam overload for the body due to the other summarised technics (OP, Cephalogram or intraoral).

The commercial technics don’t represent as exact images in case of the periapical and morphological examinations (number of roots, running, position) as the CBCT does. The paradontology could utilize also more details by the 3D examinations about the tooth surrounded by the hard tissues. The diagnostic opportunities are almost infinite. Later owing to the innovative technics the indicated examinations could widening.

The CBCT could be used direct clinically and not only for diagnostic. We get real 3D datas about the structure, so we can use it for virtual or concrete spatial modelling. To represent the computer created 3D objects we have appropriate technology and apparatus. Summary these is called rapid prototyping. The rapid prototyping summarises many technical methods, when from the computer data a spatial 3D object is created. The most famous of them is the CAD/CAM technic (Computer Aided Design/Computer Aided Manufacturing), when a fraser exactly carves the object from a stable block. Other methods are also belong here. The 3D printing also forms 3D models, that looks like a common printer. But in this case the on each other printed images together are create a real 3D object. This technic is applied in the medical fields and dentistry as well. The most popular part is the maxillo-facial surgery. The computer plans the skull from the datas of the CBCT’s images and than a rapid prototyping create the real sized skull. With this opportunity the necessary operations and the plated osteosynthesises could be designed. The expected results could be augured, and the operation could become easier.
The Ct and the CBCT usually represent segments in 3 plains. These are the axial or transversal, frontal and sagittal plains. By comparison the CBCT is able to lay perpendicular segments on the marked plain and than to represent it. This is a very practical method and it can serve important pieces of information for the dentist.

5.2. Axial slices

The first axial slice was made in the height of the frontal sinus. The bony ridge of the orbita and the ethmoidal sinus are well detected on the images.

**Figure 5.74.** Figure 1., 2. – 1.: orbita, 2.: sphenoidal bone, 3.: temporal bone, 4.: zygomatic bone 5.: frontal sinus, 6.: ethmoidal sinus

The next slice goes through the orbita, concerns the ethmoidal and the sphenoidal sinus.

**Figure 5.75.** Figure 3., 4. – 1.: zygomatic bone, 2.: vomer, 3.: nasal septum, 4.: sphenoidal sinus
The next slice was made under the orbita. The biggest air contented cavity is the maxillar sinus. The maxillar sinus, the mandible caput of the temporomandible join and the slice of the articulare tuber are clearly represented on the image. The aircontended mastoideus process’s cavity could be examined as well.

**Figure 5.76.** Figure 5., 6. – 1.: mastoideal process, 2.: caput of themandible, 3.: sinus 4.: zygomatic process of the upper jaw

The next slice illustrates the upper jaw’s teeth position to the sinus. The 3 roots of the first upper molar and distally the second molar could be found. This image also demonstrates as the molars apexes are covered by the thick cortical bone layer and the root surface doesn’t extend in to the sinus. In the front region the apexes of the maxillary front teeth appear and dorsally from the lateral incisors the incisive canal is detected.

**Figure 5.77.** Figure 7., 8. – 1.: ramus of the mandible, 2.: maxilla, 3.: sinus , 4.: atlas (C1), 5.: dens axis (C2), 6.: first molar’s root molaris
The next slice was made at the beginning of the mandible canal in the plain of the mandible foramen. The entering hole of the canal is well marked. The roots of the upper jaw’s teeth are also detected on the image. The 2 roots of the first premolar are also properly visible. The sinus forms a recessus between the second premolar and the first molar.

Figure 5.78. Figure 9., 10. – 1.: mandible foramen, 2.: root of the first molar 3.: sinus recessus

The next slice passes through the crown parts of the mandible’s teeth. In the front region exist the molars in normal occlusion. Number three indicates the second class filling in the first molar on the radiogram. The right lower wisdom tooth is represented from root segment.

Figure 5.79. Figure 11., 12. – 1.: mandible canal, 2.: left lower wisdom tooth 3.: filling in the left first molar
The last axial segment goes through the basis of the lower jaw in the front region. The bone follows the roots all along the vestibular side, especially in case of the canines, and forms holes on it. On the right side (marked with arrow) the mesial and distal roots of the first premolar could be examined separated from each other and the interradicular septum is also found here.

**Figure 5.80. Figure 13., 14. – 1.: left lower canine (root) 2.: right lower first premolar’s mesila root**

![Figure 5.80](image)

**5.3. Coronal segments**

The coronal slices contain important datas about the horisontal bone, that couldn’t be given by a 2D image (panoramic radiogram). In this case the plain doesn’t follow the arc of the upper and lower jaw, so it is better used in the molar areas. To avoid this disadvantage the transaxial plain was introduced, that will mention in the followinfg chapter.

On the first image the lower and upper incisors are found on the coronal segment. The teeth shouldn’t be located in th esame plain. This is different from the panoramic method. The section passes through the vestibular dentin in the lower incisors and in case of the upper incisors it goes through the root canal. The front sinus is also situated on the image.

**Figure 5.81. Figure 15., 16. – 1.: frontal sinus, 2.: mandible 3.: left upper middle incisor**

![Figure 5.81](image)
The next segment goes through the orbita, the sinus, and the lower and upper jaw. In the nasal fossa the inferior nasal turbinate is located, on the palatum durum the median palatinal suture exists.

**Figure 5.82.** Figure 17., 18. – 1.: front sinus 2.: orbita, 3.: inferior nasal turbinate, 4.: nasal septum, 5.: sinus s, 6.: median palatinal suture 7.: maxilla, 8.: right upper first premolar, 9.: mandible

The next slice was made distally from the maxillary sinus. The area, that is located distally from the maxillary tuber and the ramus of the mandible could be observed on the image. The mandible canal is circumscribed by the cortical ridge.

**Figure 5.83.** Figure 19., 20. – 1.: temporal bone 2.: sphenoidale bone, 3.: canal of the optic nerve, 4.: zygomatic arc, 5.: sphenoidal sinus, 6.: medial pterygoideal process, 7.: lateral pterygoideal process, 8.: mandible
The mandibular condylar and thinnig neck part is illustrated on coronal slice. Because the condylar was not parallel to the median-saggital plain, so the two condylars are not exist together on one image.

**Figure 5.84.** Figure 21., 22. – 1.: os sphenoidem, 2.: condylus mandibulae, 3.: ramus mandibulae margo posterior

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On the last coronal slice we can follow all along the right acoustic meatus. The aircontened cavites of the mastoideal process surround the acoustic meatus.

**Figure 5.85.** Figure 23., 24. – 1.: acoustic meatus, 2.: mastoideal process 3.: cochlea, 4.: dens axis, 5.: C3
5.4. Saggital slices

The saggital slices are usually analysed together with the other plains. The first slice was made in the plain of the mandibular condylar. Due to this slice the temporomandibular joint’s structure, the component bones and the relation to other formulas are could be well analysed. The separator part between the external acustic meatus and the mandibular condylar could be detected. This part allows to examine the movements of the condylar from the external acustic meatus.

Figure 5.86. Figure 25., 26. – 1.: condyl of the mandible, 2.: meatus 3.: articular tuberculum 4.: mastoidal process 5.: zygomatic bone

The next image represents the ascendent part of the mandible and the mandible canal. The protuberance of the maxillary sinus exists also in the segment.

Figure 5.87. Figure 27., 28. – 1.: condyl of mandibulae, 2.: angulus of the mandible, 3.: mandible canal, 4.: occipital bone, 5.:maxillary sinus
On the next image the wisdom tooth exists in the section plain. Because the lower jaw is wider than the upper jaw only the mandible’s wisdom tooth is visible. The relation between the wisdom tooth and the mandible canal should be analysed, that has a determinative role in the dentoalveolar surgery.

**Figure 5.88.** Figure 29., 30. – 1.: maxillary sinus, 2.: mandible, 3.: lower upper wisdom tooth

In the next picture the second and first molars and the antagonists are well marked. We can observe the upper teeth’s relation to sinus and the recessuses between the teeth.

**Figure 5.89.** Figure 31., 32. – 1.: maxillary sinus, 2.: first lower molar, 3.: second lower molar
Comparing to the former image the recessus is wider between the two premolar. In this image the continuous cortical layer, that covers the roots, is much more well marked than before.

**Figure 5.90. Figure 33., 34. – 1.: frontal sinus, 2.: maxillar sinus, 3.: ethmoidal sinus, 4.: optic nerve canal, 5.: atlas**

The last saggital segment was made in the median-saggital plain. The sella turcica is also represented on the picture. It ensures the position for the hypophysis, that is terminated mesially by the etiberculum sellae, distally the dorsum sellae.

**Figure 5.91. Figure 35., 36. – 1.: frontal sinus, 2.: sphenoidal sinus, 3.: sella turcica, 4.: clivus, 5.: incisive canal**
5.5. Transaxial slices

As it was mentioned before, some parts of the dentistry focus on the teeth and the surrounding bones. So the mentioned 3 plains are not always helpful in the diagnostic of the anatomical structures. For example one reason could be the arc of the upper and lower jaw. In case of the CBCT we can specify a plain. The CBCT is able to make perpendicular slices on to the choosen plain and to illustrate it. The mandibular plain is the most popular to choose. Probably it is incorrect to call it as a plain, because this is a curved shaped structure. We can use also the upper teeth’s plain, the lower teeth’s plain and the plain of the mandible canal. Other different plains could be used for example in case of the jaw’s joints examination. But to detect the choosen plain is simple.

The Figure explain, that the plain was marked by the upper teeth arch. To make the examination easier we can lay it in to a plain, that looks like a panoramic X-ray. Than the machine represents the perpendicular slices to the choosen plain.

Figure 5.92. Figure 37., 38. – The chosen plain is exist on the right image and marked with a green zone. The thickness of the zone could be changed (in this case it is 5 mm). After the layout of the plain we get a similar image to the panoramic radiogram on the right side. Theese teeth could be precise detected and the eaxamination of sought area becomes easier. The upper and the lower jaw’s arch form and size are different from each other and from different perspectives the variant teeth of the jaws’s are visible.

The first slice was made on a plain, that was layed on the second molars. The left red line’s plain is red framed on the right. But we can detect the plains together before and after the chosen section. Both the upper and lower pulp chambers are well illustrated. The sinus is located close to the upper teeth.
Figure 5.93. Figure 39., 40.

The following 41-42. figures highlight the mandible canal with red colour in the different segments. The lower first molar’s mesial root canal could be observed all along tooth.

Figure 5.94. Figure 41., 42.

The 43-44. figures represent as the end of the mandible canal, the mental foramen leaves the vestibular cortical layer between the two lower premolars.

Figure 5.95. Figure 43., 44.
The last transaxial slices were made on the middle incisors area. The incisive canal is the most important structure on the image, and the running of the canal could be followed all along the incisors.

**Figure 5.96. Figure 45., 46.**

At the end of the chapter let’s mention some other opportunities about the CBCT’s usage. We would like to introduce other potential possibilities that we just slightly apply.

### 5.6. Airways

The significance of the airways were also highlighted in the chapter about the panoramic X-rays chapter. In case of the CBCT the aircontended cavities and the different airways don’t hamper the diagnostic. With this method these spaces could be expressively illustrated. The orthognath surgery and the E.N.T. would gently utilise these advantages of the CBCT. But in case of a dental originated sinusitis the CBCT also could be important for a dentist as well.

**Figure 5.97. Figure 47–52. – The variant aircontended cavities and the airways could be illustrated and easily examined**
The examination of the different sinuses could cause also difficulties, but this new method would be very helpful clinically in the diagnostic. It could be also used when we want to determinate a space-occupying process. Due to the CBCT the exact localisation, the expansivity, the relation to the direct environment are could be easily detected. The augmentation also could be well implemented, if the exact localisation of the augmentation and the amount of the missing bone is known. The operation would become easier and we can count with some obstacles as well.

**Figure 5.98. Figure 53–56.** – In the introduction was mentioned, that the common CT image is better for the soft tissue illustration, but this technic serves useful pieces of information about the sinuses. Due to the different intersection plains the connection of the roots and the maxillary sinus could be illustrated in 3D.
Figure 5.99. Figure 57–62. – The 3D skull image could be examined from the most appropriate aspect or we can turn it to every direction of the space.
Last but not least one of the most popular usage field of the CBCT is orthodontics. The disadvantage of the cephalogram that it disfigurates and enlarges. The projection of the two sides causes diagnostic difficulties. Compare it to the CBCT, the CBCT is always exact, never deforms and the sizes are always the same. On these images every necessary measurement and other diagnostic detection could be performed. The right and the left side of the skull could be represented separately from each other, so the projection could be avoided.

Figure 5.100. Figure 63. – The cephalograph was made by CBCT. The soft tissues could be detected next to the precise bone illustration as well. This is also an important method in the orthodontic diagnostic.
6. 5.6. Test

1. What is the most radiodense structure on the x-ray taken from a tooth?
   - Enamel
   - Dentin
   - Cementum
   - Periodontal ligament

2. Which is not part of the“ three stripe” along the root?
   - Periodontal ligament
   - Lamina dura
   - Cementum
   - Dentin

3. Where is the lingual foramen on the intraoral x-ray?
   - Around the apex of upper central incisors
   - Around the apex of upper premolars
   - Close to the apex of lower incisors
   - Close to the apex of lower premolars

4. Which anatomical structure takes part in the formation of antral Y with the hard palate?
   - The distal wall of the maxillary sinus
   - The anterior wall of the maxillary sinus
5. Oral Radiology

- The base of the maxillary sinus
- The roof of the maxillary sinus

5. Sometimes the coronoid process could be seen on intraoral x-rays taken from which tooth?
- Incisors
- Canine
- Premolars
- Molars

6. What is the explanation of the double radiodense line of the hard palate on panoramic x-rays?
- Because of the thickness of the hard palate the upper and lower cortical could be seen
- The upper is the ghost of the contralateral side
- The lower is an unremoved denture
- The upper is the lower border of the nasal turbinate

7. What is specific on the panoramic x-ray taken from the TMJ?
- The TMJ could be seen with open and closed position on both side
- The TMJ could not be seen on panoramic x-ray properly because of the ghost of the other side
- The x-ray always has to be made with fully open mouth
- The panoramic technique is not proper for TMJ at all

8. What is the anatomical structure on panoramic x-ray which may overlap the zygomatic arch and the lateral pterygoid plate?
- Condylar process
- Coronoid process
- Mastoid process
- Styloid process

9. The following statement is true for one panoramic x-ray setting mistake. What is the mistake? The roots of the lower teeth are blurred and not sharp. The hyoid bone overlaps the mandible and the premolars are also overlapping each other. Most of the cases the condyles could not been seen.
- The head is tilted backward
- The head is tilted forward
- The head is moved backward
- Not straight spine

10. Why do we have to ask the patient during panoramic x-ray exposure to lift up the tongue?
- Otherwise he is not able to bite the positioning pipe
- Otherwise the crown of the lower teeth will be blurred
- With the lifting up the tongue the hyoid bone moves downwards and will not overlap the mandible
• Otherwise the roots of the upper teeth will be blurred

11. What does the “FOV” mean during CBCT?
   • Maximum kVP
   • The maximum resolution of the machine
   • Maximum dose
   • Field of view

12. In addition to the axial, coronal and sagittal planes what is the fourth plane which could be achieved from a CBCT?
   • transaxial
   • transcoronal
   • transsagittal
   • transfrontal

13. What is the advantage of the CBCT in orthodontic cases?
   • Because it has lower dose than a normal caphalograph
   • Because there is no magnification and distortion
   • Because is able to show the caries lesions
   • Because it could be made in laying position

14. What is the transaxial plane on the CBCT images?
   • The plane which is crossing through the CEJ
   • The plane which connects the mental and mandibular foramen
   • The plane which goes across the axis
   • The plane which is perpendicular to a chosen plane

15. What is the form of the images which could be shared among different medical machines?
   • MOV
   • MP3
   • DICOM
   • TXT
Chapter 6.6. Prosthodontics

1.6.1. Treatment plan, anamnesis – Beata Benke

For fabrication of a complete denture a treatment plan is needed, which contains general anamnesis and a dental anamnesis.

Anamnesis:

During the anamnesis, the patient is asked about their health status (illnesses), such as cardiovascular diseases (cardiac failure, arrhythmia, high blood pressure), bleeding disorders, digestive, respiratory system disorders (e.g: asthma), infectious diseases (HIV, HBV, HCV, TBC), tumours, glaucoma, allergies, and epilepsy.

After the registration of the illnesses, next step is the documentation of the regularly taken medications, which can influence the treatment in many aspects. (e.g.: Syncumar therapy – by surgical intervention causing bleeding, Ca channel blockers – gingival hyperplasia, antihypertensive medications - dryness of mouth).

It’s important to note the possible drug hypersensitivities/allergies.

Previous surgeries can be also relevant: “Did the patient receive any transfusion?” As well as post-treatment status in tumour therapy (Bisphosphonate, chemotherapies, and irradiation of the head-neck region), because in these cases an indicated vestibuloplastic surgery or a denture caused decubitus can be of greater importance.

In female patients due to pregnancy or breastfeeding more caution should be taken with taking X-rays, and use of medications.

The patient is asked about smoking, drinking and creational drug habits, as well as previous dental treatments. “When did the patient last visit a dentist? “Does/did the patient have a fix or removable denture?” Does the patient have any joint complaint?

The overview and evaluation of previous medical history and documentation is also of importance, in addition to the consultation with other specialists in some cases. Henceforth comes the dental anamnesis, information can be obtained about why and when the patient lost their teeth. Did the patient get an immediate denture after losing their teeth? Does the patient wear a denture now? How many dentures did the patient have? What are those properties that the patient likes/dislikes in this denture and what would they like to change?

1.1. Clinical examination

The clinical examination consists of two parts, an extraoral and an intraoral process.

During the extraoral, the face, the mandible, the maxilla and the head-neck region undergoes general examination, especially the symmetry and functional integrity. After inspection follows the palpation, all the regional lymph nodes will be felt from the suboccipital to the supracalvicular region.

Examine the temporomandibular joints, if also there are any tenderness, pain, voice phenomenon, or any deviation during movements.

After summarising the results of the extraoral examination short and precise, the intraoral examination will proceed. During the inspection and palpation of several types of mucosa, evaluate the colour, characteristics (smoothness of the surface, pathological deformities), movement and mobility.

1.1.1. The examination process

Intra- and extra oral examination: need good illumination for inspection and digital palpation, a defined sequence should be followed:

• lips
• margin and base of the tongue
• floor of the mouth
• lymph nodes
• soft palate
• oropharynx
• neck
• thyroid gland
• TMJ: clicking, crepitating, pain.

1.2. The causes of tooth loss

There can be several causes of tooth loss. Genetic, congenital problems (aplasia, as like anodontia partialis and totalis, development disorders of the face, maxilla and mandible, e.g: cheilo-gnatho-palatoschisis) and acquired diseases. Acquired diseases are the caries, which nowadays are the main cause of tooth loss in Hungary, periodontal disorders, and mainly the traumas, as causing factors.

1.3. Aftermath of tooth loss

Losing the tooth occurs immediate and late damage:

• Chewing and speaking ability decreases.
• Decreased muscle tone of the face and the lips (Picture 1).
• Alveolar bone resorption.
• Changes in the intraoral structures.
• Decreased work ability.
• Psycho-social problems.

The absorption of the nutrition from the gastrointestinal tract can be disturbed (vitamin absorption), lumpy, hard digestible foods can lead to stomach disorders.

For this reason, it’s necessary after the extractions to start the fabrication of a denture as soon as possible. (Picture 2.)

Figure 6.1. Figure 1. – Decreased muscle tone of the face and the lips

![Figure 1](image1.png)

Figure 6.2. Figure 2. – Positive changes after the reconstruction

![Figure 2](image2.png)
2. 6.2. The anatomy of edentulous upper jaw – Beata Benke

The retention and stability of the upper denture mainly depends on the form and size of the arch.

a. The posterior border of denture is defined by the following structures: the bilateral tuber alveolare maxillae, the vibrating line and the pterygomandibular raphe.
   - Tuber alveolare maxillae (Hamular notches).
   - Vibrating line: the border between the soft and hard palate, the two foveolae palatinae help to define its position. Ask the patient to blow their nose, whilst kept shut with the fingers, so can determine and mark the exact location of the denture’s posterior border.
   - Pterygomandibular raphe: a distally mucosal wrinkle behind the tuber alveolare maxillae (hamular notches), it will be bowed by opening the mouth, therefore in case of an overextended denture base it will destabilise the denture.

b. Ridge form

c. Frena

d. The depth of the vestibulum

e. Bony areas

The edentulous upper jaw can be divided into two parts: hard palate (palatum durum) and soft palate (palatum molle). The bony base is created by two bones: lamina horizontalis ossis palatine and the processus palatinalis maxillae.

The parts of the hard palate:
   - palatum alveolare – the oral incline of the edentulous ridge,
   - palatum prorium – the oral part of the palate, palatum alveolare bounded area.

2.1. Structures on the hard palate

a. papilla incisive:
   - located posteriollly to the upper central incisors,
   - roll or pear shaped mucosal structure,
   - it is above the incisive canal,
   - during the bone resorption it can be moved from the oral incline of the processus alveolaris to the top of the ridge, or the vestibular surface (Picture. 3),
• relief is suggested.

b. palatal raphe:
• a fusiform crest in the midline,
• starts from the papilla incisive and goes distally,
• relief is suggested.

c. rugae palatinae:
• high individual variations exist, mucosal wrinkles,
• plays a roll in the phonation,
• if the patient has pronounced rugae, worth to provide space on the denture base, otherwise if the patient has a denture with smooth denture base, expressed rugea on the new denture can disturb the patient,
• the number is between 3 and 7.

d. palatal torus:
• on the area of palatum proprium in the midline,
• relatively increase with age,
• covered by thin mucosa without submucosa,
• relief is needed,
• in the case of denture fabrication, it’s a disadvantaged anatomical area, because of the bone resorption it can destabilise the denture, causing swaying and fracture.

e. foveola palatina:
• little mucosal indentations on the border of the hard and soft palate,
• the vibrating line is 1,5-2 mm distally.

2.2. Soft palate (palatum molle)

It’s the continuation of the hard palate between the the palatal bone and oropharynx. Following muscles build up the soft palate: m. tensor veli palatine, m. levator veli palatine, m. uvulae, m. palatopharyngeus, m. palatoglossus. The posterior border of the denture - the vibrating line - is on the soft palate, it’s able to push the prosthesis edge into the mucosa. The vibrating line is not as the same as “A”-line.

2.3. Maxillary frena

• Maxillary labial frenum: fold of mucous membrane at the median line.

• Buccal frenum: located opposite the premolar region, it contains muscle fibers, its movements are in connection with the buccinator muscle.

• Pterygomandibular raphe: extends from the tuber alveolare maxillae to the trigonum retromolare. The prosthesis base shouldn’t cover or touch this structure.

2.4. The maxillary tuberosity (Tuber alveolare maxillae (1))

• Located on the dorsal end of the upper ridge (on the site of the wisdom teeth).

• It contains bone and dense fibrous connective tissue.
• Always covered by the denture base.
• It can form an unit with the pterygomandibular raphe (it moves by opening the mouth).
• In some cases relief is needed (in case of large undercuts it should be blocked out, otherwise won’t be able to insert the denture).

2.5. Buccal vestibule

• It’s between the vestibular surface of the maxillary tuberosity and the buccal mucosa.
• 3-10 mm wide.
• Plays an important role in vacuum effect.

2.6. Ridge forms

The ridge can be divided to frontal and lateral region. The front region is extended till the mesial border of zygomatico-alveolar crista. The lateral region contains the zygomatico-alveolar crista and the maxillary tuberosity.

There are three different arch forms:
• triangle (V-form),
• rounded rectangular (this type is the most ideal for denture support),
• and U-shaped (egg-shaped).

By height there are four different forms:
• high ridge (Picture 4.),
• low, only vestibular high ridge (flatter palate),
• flat ridge,
• hyperplastic mucosa.

The type of the vestibular mucosa:
• fix mucosa,
• moving mucosa,
• movable mucosa.

Figure 6.3. Figure 3. – Papilla incisive on the vestibular side
3. 6.3. The anatomy of edentulous lower jaw – Beata Benke

The form of the mandibular arch is even more critical than the maxilla, since there is less surface area for retention, and the moveable structures of the tongue and floor of the mouth can cause denture displacement, due to overextension of the denture - inform patients of any potential retentive problems.

Edentulous ridge/arch forms on the lower jaw:

• whole held, high ridge,
• in the front region held, lateral flat ridge (Picture 5.),
• flat ridge,
• negative ridge,
• deep negative ridge.

The ideal ridge:

• a good unlimited tuberculum alveolare mandibulae,
• not sharp linea mylohyoidea,
• deep retromylohyoideal gap,
• low attached frena,
• plenty of firm keratinized mucosa,
• appropriate ridge height.

The bony base of the edentulous lower jaw is the mandibule. The important anatomic structures are the following.

• mental foramen,
• external oblique ridge,
• mylohyoid ridge.

3.1. Retromolar region

Mandibular alveolar tuberculum (tuberculum alveolare mandibular): behind the location of the third molar, contains dense fibrous connective tissue, rigid, and is incompressible.
If:

- it’s less separated from its environment, hardly elevated,
- tuberculum – masseter fissure doesn’t narrow, during opening of the mouth,
- the lingual packet can’t be exploited,
- there isn’t significant difference in level between the occlusal surface of the ridge and the mandibular alveolar tuberculum the denture base should be extended till the middle of the tuberculum.

If:

- it’s separated well form its environment, outstanding, connected to the bony base,
- the tuberculum – masseter fissure narrows,
- there is significant difference in level between the flat or negative ridge’s occlusal surface and the mandibular alveolar tuberculum, it’s not suggested to extend the denture base on it.

3.2. Tuberculum masseter fissure

A triangular space, formed by the opening of the mouth, between the buccal surface of mandibular alveolar tuberculum and the buccal mucosa. It can be exploited, if doesn’t tighten during mouth opening, and the buccal is not in contact with the mandibular alveolar tuberculum.

3.3. Lingual pocket

This area is located between the lingual surface of mandibular ascendant ramus and the tongue.

In case of extending the denture behind and below the mylohyoid ridge it is referred as retromylohyoid extension.

Forms of extension:

- only on the mandibular ascendant ramus,
- into the retroalveolar fovea and as well as retromylohyoid.

The usage of this area has great importance for future stability of the prosthesis (hold of the prosthesis, stabilizing effect of tongue, the removal effect of tongue can be eliminated).

The lingual pocket can be exploited if it is firmly covered, the bone fused mucosa on the lateral surfaces and mucosa do not change their position and form by the movement of the tongue.

The usability of the lingual pocket is examined by palpation. By positioning the index fingers into each lingual pocket and asking the patient to elevate their tongue, followed by swallowing movement, observe weather the tongue stabilizes or pushes out the fingers.

3.4. Mandibular accessories recessus (recessus mandibulae accessorius)

It’s only presented in cases of flat, negative and deep negative ridge between the margo anterior of the coronoid process temporal crista.

Borders:

- lateral: oblique ridge,
- medial: alveolar ridge,
- dorsal: tuberculum – masseter fissure,
• frontal: the mesial surface of the second molar.

In the presence it should be used, because it reduces the lateral movements.

3.5. Buccinator pocket

Located between the place of the first premolar, the mandibular alveolar tuberculum and the frontal border of the tuberculum – masseter fissure. It’s created by muscles (musculus depressor anguli oris, musculus buccinator, musculus masseter).

Borders:
• frontal: dorsal fibers of m. depressor anguli oris,
• lingual: vestibular slope of the edentulous alveolar ridge,
• base: vestibular fornix,
• distal: the mesial border of mandibular alveolar tuberculum,
• lateral: buccal mucosa,
• upper: the horizontal line between the vestibular surface of the teeth and bucca.

It’s important to establish the denture’s polished surface individually in favour of better stability.

By evaluation of the buccinators pocket’s lateral wall, need to take notice how it moves by opening of the mouth compared to the median plain (Picture 6).

Figure 6.5. Figure 5. – In the front region held, lateral flat ridge

Figure 6.6. Figure 6. – Position of the buccinator pocket’s lateral wall

3.6. Torus mandibularis
Bony prominence found lingually and bilaterally near the premolars midway between the soft tissues of the floor of the mouth and the crest of the alveolar process. It’s covered by an extremely thin layer of mucous membrane, relief is always required. In some cases it’s necessary to be removed or diminished.

3.7. The floor of the mouth

It’s located between the oral fornix and the tongue. It can be divided into two parts (sublingual and paralingual area).

3.7.1. Sublingual area

This is the anterior part, between the oral fornix and the sublingual raphe, in the middle is the lingual frenum. It can be thick and hard, which will destabilize the denture or soft in favourable cases.

3.7.2. Paralingual area

It’s situated between mesial border of the mandibular alveolar tuberculum and the site of the first premolar.

3.7.3. The types of the floor of the mouth

There are two types, non-extending and extending form, the second form can be soft or thick touched.

Non-extending form:
- smooth surface,
- by elevation of the tongue rises only a bit,
- even by flat or negative ridge it’s not higher, than the alveolar bone.

Extending form:

a. Soft touched:
- it is reponable with slight pressure,
- by tilting the head back it almost disappears,
- it follows hardly the movements of the tongue,
- unfavourable.

b. Thick touched:
- the shape is less impressionable,
- during function the shape doesn’t change,
- it is hard to move.

3.8. Edentulous ridge/arch forms on the lower jaw

- whole held, high ridge:
  favourable
  supportable
  smaller interalveolar distance, which influences the stability advantageous
- in the front region held, lateral flat ridge (5. picture):
the patient lost the premolars and molars sooner, than the front teeth

- flat ridge:
  the level of the residual ridge’s occlusal surface is as high as the mental spinae, oblique ridge and mylohyoid ridge

- negative ridge:
  the mucosal level of the floor of the mouth is higher, than the edentulous ridge

- deep negative ridge:
  till the base resorpted alveolar process
  thin crest of residual ridge

3.9. Frena

- Thin labial frenum: in the midline between the alveolar bone and the inferior lip,
- buccal frenum: around the place of the premolars.

3.10. Types of the mucosa

On the vestibular surface there is fix mucosa in the premolar and molar region, with movable mucosa in the frontal region.

On the oral surface in the frontal region fix mucosa exists, the moving mucosa is in the premolar and molar region.

4. 6.4. Information of the patient, treatment plan – Beata Benke

Limitation of dentures: The complete dentures are much different, than natural teeth. In cases of better alveolar arch format, the chance for success is bigger: as neutral as well as chewing stability is achievable. For the patients with negative or deep negative alveolar arch usually a denture with neutral stability can be fabricated. In these unfavourable cases it is recommended to inform the patient about the possibility of implantation – if the general status allows.

4.1. Adaptation

It is verified by many studies, that it may take 6-8 weeks for the patient to adapt to their new denture. It is important to make the patient realize this. The reduced adaptability’s physiological explanation is the following: in patients; wearing complete denture proprioceptors of the periodontal ligament have been lost and large areas of mucosal proprioceptors are covered by the acrylic base.

Adaptation for a new denture is affected by:

- time of wearing prosthesis (since when has the patient complete denture),
- amount of remaining ridge (height and thickness),
- degree of modifications in new denture compared to the “old”,
- individual variation (sensitivity and load capacity of the mucosa).

4.1.1. Limitations of denture

- A denture is less effective, than the natural teeth (degree of chewing force is lower).
Some of the patients are not able to eat every kind of food, but it is not so common.

Generally the better the alveolar arch form and higher the ridge, the less problems are encountered. In those cases, where the ridge is low, especially on the lower jaw (flat, negative or deep negative ridge) have to advise the patient that the denture won’t be stable, it will move, causing bad chewing ability.

Patient with minimal ridges will have more complaint, because of sore spots.

It is recommended to inform the patient about these limitations before starting the work, rather than an excuse.

**4.1.2. Adaptation to chewing depends on**

- CO/CR relationship,
- changes of the position of the teeth (comparing the previous denture),
- changes in the vertical dimensions.

The patient may experience a decreased chewing force, including lip and cheek biting. The adaptation can be easier, if the patient eats soft food at the beginning, slowly moving to hard food and also by suggesting to cut the food into small pieces. Patient should try to chew by placing food pieces towards the corners of the mouth. During this period, production of saliva can increase temporarily. We have to explain to the patient, that they should be patient throughout this time, till their neuromusculature adapts to the new prostheses.

**4.2. Speaking skills**

The changes and difficulties in the speech are maybe caused by the modified tooth position, the decreased tongue space and the palatal contours. The initial speaking difficulties are usually transitory, because the tongue is highly adaptable.

Appearance may be changed in some individuals, the most common causes of these changes are the following:

- increasing the length of the incisors (worn),
- changes in the vertical dimension,
- more significant lip support.

In most of the cases the changes are beneficial and not a cause of concern. The patients sometimes get scared, when the changes in appearance are radical, it is important to reassure them, till they get used to their new appearance.

**4.3. Hygiene**

Complete denture wearing patients have a special oral milieu. The mucosal integrity mainly depends on the effective plaque removal methods.

Denture cleaning rules:

- separate toothbrush is needed,
- all surfaces of the denture must be clean with a nonabrasive cleanser (pl. liquid soap),
- to use abrasive paste is forbidden,
- denture must be cleaned after every meal and before going to bed,
- it is suggested to cover the washbasin with a towel while cleaning and removing (to prevent fracture if dropped),
- additional cleaning possibilities: using denture cleaning tablets regularly (every second day), and ultrasonic machines,
6. Prosthodontics

- cleaning of the mucosa is even more important, using a proper brush,
- it is recommended to remove the denture during night, not to disturb the mucosal regeneration.

### 4.4. Continuing care

- Yearly control,
- it is easier to solve smaller problems (wear, fractures, resorption - rebasing, relining),
- complete denture life is about 5-7 years in case of proper hygiene and care.

### 5. 6.5. Steps of complete denture fabrication – Zsofia Muzsek

The steps made by the dentist are in close co-operation with the dental laboratory, which is documented on laboratory work paper.

**Table 6.1. Table 1. – Steps of complete denture fabrication**

<table>
<thead>
<tr>
<th>Occasion</th>
<th>Dental Office</th>
<th>Laboratorial</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>anamnesis, oral examination, upper and lower alginate impression, stone cast fabrication, drawing of custom tray borders*</td>
<td>fabrication of light curing acrylate custom tray</td>
</tr>
<tr>
<td>2.</td>
<td>remaking the custom tray to functional tray, border moulding, functional impression*</td>
<td>beading, fabrication of functional cast and upper and lower occlusal rims</td>
</tr>
<tr>
<td>3.</td>
<td>control of the occlusal rims (ridge line), defining VDO and CR, spatula probe, fixing, tooth colour matching</td>
<td>articulation, tooth set up</td>
</tr>
<tr>
<td>4.</td>
<td>trial denture*, spatula probe</td>
<td>flasking, reocclusion</td>
</tr>
<tr>
<td>5.</td>
<td>delivery, spatula probe</td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>remontage</td>
<td></td>
</tr>
</tbody>
</table>

* possibility for relief

### 6. 6.6. Impressions – Zsofia Muzsek

#### 6.1. Preliminary Edentulous Impressions

For fabrication of a complete denture a custom tray(1) is always needed, which fits perfectly to the patient’s dental arches in the mouth. For making a custom tray, a study cast is required, for this first an alginate impression is taken.

Choosing the material: after shaking the alginate, - which is kept in a tight box – put the desired amount of the powder with the measuring cup (included in the pack) into the rubber bowl. Add the appropriate amount of cold water (measuring cup is also included) and mix the material with a spatula. During mixing the aim is to have a homogeneous material in time (45sec), afterwards place the mixed alginate material into the stock tray that was chosen. It is also possible to use an alginate mixing machine. (Picture 7.)

**Video 1.**

**Figure 6.7. Figure 7. – Alginate mixing machine**
6.2. Selection of stocktray

The selected stock tray should cover all the areas, which are responsible for the retention. The irreversible hydrocolloid impression materials need 5 mm gap to be stable. (Picture 8.) If the tray is not long enough can be enlarged with composition impression material. The retention between the tray and the impression material can be achieved by using perforated impression trays or alginate adhesives.

Figure 6.8. Figure 8. – Alginate impression

6.3. Patient preparation

- Practice placing and removing the tray with help of an assistant, this way dentist and patient are better prepared.
- Select the right position of the patient: straight supported head, the chair must be set to the dentist’s height.
- The patient should be reassured.
- Locate and mark the vibrating line.
- Evaluating the ridge form, have to put some extra impression materials on the big undergoing areas and the gothic palate.

6.4. Impression taking

Put the mixed (by hand or machine) impression material into the stock tray and insert it into the patient’s mouth by moving the tray quickly (vibrating movements) and pushing first at the posterior parts then moving forward. Have to take care on the position of the tray – the shaft must be in the midline. During the procedure the lips can be held by two dental mirrors to ensure visual control. Taking impression from the lower jaw, the tongue must be in an elevated position to provide enough place for the tray and material. The impression must be removed by
one definite movement to prevent the irreversible deformations. If the removal is difficult, ask the patient to blow up the face and insert your index fingers in the vestibule between the mucosa and impression’s border and remove the impression by slight rotational movements.

6.5. Disinfection

The removed impression must be washed with tap water first (to eliminate the saliva, blood etc.), than shake out the water. Thereafter disinfect the impression’s all surfaces with the help of a disinfection spray. Wait for the disinfection time, and wash the material with tap water again.

6.6. Evaluation of the alginate impression

- Evaluation of the surface: smoothness, bubble- and particle-free, homogenous surfaces (Picture 9).
- Good retention between the impression material and stock tray.
- The thickness of the impression material (5 mm).
- Position control (all the anatomic areas are inside the border of the tray, midline).

Figure 6.9, Figure 9. – Material shortage in the alginate impression

6.7. Diagnostic cast

Pouring:

After evaluation of the impression should pour it in the second 15 minutes after the impression was taken. The first step is the measuring of the gypsum powder and coldwater; Mix it in a rubber bowl with the help of spatula, or use a vacuum mix (less time, stronger cast). Modulate speed of pouring by tilting back and forth or pressing the tray more firmly onto the vibrator. Have to ensure the model is moist during trimming soak in slurry water, or soak the base of cast into water. Thinnest part of the cast should be a minimum of 12mm. Separate the alginate impression from the stone cast after 45 minutes.

Trimming:

It is important to trim the base on the model trimmer parallel to the residual ridges, leave the mucous membrane reflection intact for custom tray construction. Remove extensive areas of the cheek or lip reflections that create undercuts and those which will make it difficult to make or remove the custom tray. All anatomical surfaces should be included with minimum voids.

6.8. Tissue conditioning

Definition: all those non-surgical treatments, which enable the mucosa for denture wearing, including the use of tissue rest, occlusal correction, temporary soft liners and/or improvement of hygiene.

The mucosa can be temporary inappropriate for impression, because of:
• pathologic conditions: the result of systemic disease - nutrition, hormone imbalance, autoimmune diseases (Lupus),

• local factors: inaccurate denture base, ill-fitting old denture, occlusal disharmony, bad oral hygiene.

**Figure 6.10. Figure 10. – Papillary hyperplasia on the lower jaw**

The tissue regeneration is the requisite of the work, the red, inflamed, swollen, oedematous mucosa, ulcerations, Candida infection should be eliminated. If there is granuloma fissuratum or papillary hyperplasia in the mouth, the conservative therapy will be supplemented with surgery (Picture 10.). It is important to inform the patient, how the correct cleaning of the denture and mouth can help the regeneration of inflamed mucosa.

**6.9. Custom tray**

The custom tray is used for making functional impression. The custom tray is made of light curing acrylic (individolux, Super-Tac) on the study cast.

**Figure 6.11. Figure 11. – Study casts**

**Figure 6.12. Figure 12. – Borders of custom tray**
Mark the border of the fix and movable/moving mucosa and the vibrating line on the study cast in the presence of the patient (Picture 11). On the upper jaw mark one more line around 2mm away from the previous line on the fix mucosa, take care to ensure space for the frena is provided (Picture 12). On the lower jaw have to do the same on the lingual side. The next step is blocking out the undercut areas, and application of placeholder material. Thereafter the light curing acryl will be adapted to the study cast and will be cut accurately with a scalpel, then insert the handles. Position of handles: the height and thickness of the handles are the same as the teeth’s at the mentioned region, apply 3 handles in the following way: one in the front region, and one on both sides on the position of the second premolar and the first molar on the ridge line. Now perform the photopolymerisation based on manufacturer's instructions (Picture 13.). Make the borders of the completed custom tray smooth and rounded with hard metal Frazers and rubber polishers.

**Figure 6.13. Figure 13. – Polymerisation of the light curing acryl custom tray**

Selective custom tray: It is possible to create a special type of custom tray, in this case mark those (sensitive) areas, where relief is needed. On the most sensitive areas apply 2-3 layer of placeholder (for example: wax), while on the other surfaces only one layer. So don’t have to be afraid of the patient won’t be able to do the functional movements correctly because of sensitivity or pain.

**6.10. Border moulding**

At the first step have to check the custom tray in the patient’s mouth and evaluate if there is enough place for the border moulding and impression material on all surfaces. If it is necessary make the modifications. Handles should be verified, whether they are on the correct place, they shouldn’t disturb the functional movements.

For border moulding can use compositions impression materials, border moulding wax and silicon impression materials.

Border moulding with compositions impression material:

- dry the custom tray,
- prepare 40-50°C water and wax tray,
- pull the material through the flam 3-4 times, till it gets warm (it will be even more shinier) Take care, not to overheat, over 70°C irreversible structural changes occur. If the rod became bubbly (like boiling), cut that part and don’t use it. When it is soft, it should be applied to the borders (2-3cm long) of the custom tray immediately, and pull it across the flame once more, to ensure the retention,
- cool down the material, insert it to the patient’s mouth, and ask to do the functional movements,
- the material becomes hard in 37°C, check the surface (smooth and shiny),
• if one part is accepted, can go on with the next section/part,
• if the form of an area is not appropriate, warm it again, and repeat the procedure,
• can have the vacuum effect with the completed functional tray (Pictures 14, and 15.).

Figure 6.14. Figure 14. – Mucosal view of a lower custom tray after border moulding

Figure 6.15. Figure 15. – Vestibular view of a lower custom tray after border moulding

Video 2.

6.11. Functional impression

Materials to use theoretically:

• irreversible hydrocolloid (irreversible, elastic impression material): hydrophilic, but viscous,
• zinc oxideeugenol (irreversible, rigid impression material): rigid, bad taste,
• polysulfide (irreversible, elastic impression material): bad taste, bad dimension stability, bad retracts capacity,
• silicone (irreversible, elastic impression material): acceptable taste, good dimension stability, good retracts capacity, hydrophilic, thixotropic,
• polyether (irreversible, elastic impression material): bad taste, good, dimension stability, good retracts capacity, expensive.

6.11.1. Impressions with zinc oxide eugenol impression material

The border moulding material should be 1mm reduced before the impression, to ensure space for the ZnOE to flow on the vestibular surface of the tray. Take care on the surface’s smoothness, remove all the edges. It isn’t necessary to use adhesives, but the tray must be dry.

6.11.2. Impression with silicone impression material
The same procedure should be done as the ZnOE, but adhesive is needed. Before using the adhesive dry the tray, after applying the tray adhesive allow to dry (based on manufacturer's instructions, about 5min.). It is possible to mix the material with hand or use silicone mixing machine (Picture 16).

**Figure 6.16. Figure 16. – Silicone mixing machine**

![Silicone mixing machine](image)

Ask the patient not to use the old denture 24 hours before the impression taking; the tissues can return to the original form. Show the functional movements to the patient, and practice them. Take care on the amount of the impression material during the preparation the border moulding must be covered. Quantity for the upper and lower jaw impression is double and one and half of the tray’s width respectively. Apply the homogeneous material into the tray, and insert to the patient’s mouth, while holding the lips with dental mirrors, start the functional movements.

**Video 3.**

**Video 4.**

### 6.12. The functional movements (1), (2), (3), (4)

#### 6.12.1. Functional movement of the upper jaw

Opening and closing, inward and outward movement of the lip corner, asymmetrical movements of the angle (moving the mandible from side to side), instruct the patient to pucker and smile, breathe deeply and blow the air through the nose while closed and swallowing.

#### 6.12.2. Functional movement of the lower jaw

Opening and closing, inward and outward movement of the lip corner, asymmetrical movements of the angle (moving the mandible from side to side), swallowing, moving the tongue from side to side and forward

(It is easier to explain the patient the functional movements, by asking them to spell specific letters like s, m, o, u, e).

The time needed for the functional movements are determined by the impression material. In case of using Zincoxide Eugenol (ZnOE) material (e.g. SS White), setting time of the material is 5-6 minutes, which means that the manipulation time of the material is approximately 4 minutes. After complete setting of the impression material the custom tray will be removed from the mouth and controlled. The optimal thickness of ZnOE should not be more than 1 mm. If it is more, the positioning of the custom tray was not correct. The surface of the impression should be smooth, not allowed to have irregularities or broken parts, if present, correction is needed. (Fig. 17, Fig. 18., Fig. 19.)

**Figure 6.17. Figure 17. – Lower functional impression with ZnOE material from mucosal view**
The functional impression requires a final check: by inserting back into the patient’s mouth, ask the patients to perform the functional movements again while controlling the stability of the tray. Pharyngeal seal will be re-
marked again. After washing, disinfecting and drying of the impression marking of the beading will follow. The master cast is poured with type 3 gypsum. (Fig. 20. Fig.21.)

**Figure 6.20. Figure 20. – Master cast with the marked ridge line**

**Figure 6.21. Figure 21. – Master cast with the marked ridge line and relief**

### 6.13. Beading and Boxing

After the functional impression on the vestibular side of the tray a wax plate has to be applied 5 mm away from the margin. Thickness of the wax should be 3-4 mm. This procedure enables to ensure the accurate size of the functional land area. This form will be important later by flasking. In case of lower tray, wax is placed on the lingual and vestibular side (Fig. 22.), and only on the vestibular side for the upper tray (Fig. 23.). The margin of the posterior border of the upper tray should be 1 mm above the investment material.

**Figure 6.22. Figure 22. – Position of the wax on the lower tray**

**Figure 6.23. Figure 23. – Position of the wax on the upper tray**
6.14. Relief (1), (2), (3), (4)

The aim of the relief is optimal loading, and to secure the path of insertion. During the procedure a 0.1 - 0.3 mm thick lead foil or wax is applied, without changing the anatomical forms.

Places which have to be relieved:

• On the lower jaw: mylohyoid ridge, mandibular tori, genial tubercles, sharp edentulous ridge, and the upper contour of mental foramen (Fig. 24., Fig. 25.).

• On the upper jaw: maxillary tuber, to reduce the difference between the most prominent and the undercut areas, incisive papilla, palatinal raphae, palatinal torus and the palatinal rugae.

Figure 6.24. Figure 24. – Marking the mylohyoid ridge and mandibular tori for relief

Figure 6.25. Figure 25. – Marking the mylohyoid ridge for relief

7. 6.7. Theoretical background of Gnathology – Zsofia Muzsek
In order to understand Gnathology, need to define the relation of the upper and lower jaw to each other, and the occlusion theories determined by natural teeth contacts. Afterwards describe the most important jaw movements.

In the last decades Gnathological terms were described in different ways. Now the latest, accepted definitions are reviewed

7.1. The resting position of the mandible
The mandibular position assumed when the head is in an upright position and the involved muscles, particularly the elevator and depressor groups, are in equilibrium in tonic contraction, and the condyles are in a neutral, unstrained position. During this position the distance between upper and lower teeth is 2-5 mm. In this resting position the distance between upper and lower jaw is called VDR (vertical dimension of rest).

7.2. Intercuspid position (ICP)
The position of the mandible to the maxilla when the relationship of opposing occlusal surfaces provides for maximum planned contact and/or intercuspation. Because this is a tooth determined position, sufficient teeth must be present for the mandible to be placed in this position. The complete intercuspation of the opposing teeth is independent of condylar position. The incisal or occlusal surfaces of the teeth guide the mandible to ICP, and according to this, canine, incisal or group guidance are defined.

7.3. Retral contact position (RCP)
That guided occlusal relationship occurring at the most retruded position of the condyles in the joint cavities. According to Gnathological studies 90-95 % of the population are able to retract the mandible with 0.5-1 mm from ICP. If in this position a contact occurs between upper and the lower teeth, this position is called RCP. In 5-10% of the cases it means maximal ICP. The first contacts are on the slopes of the cusps. These slopes drive the mandible into ICP, and the RCP is an unstable position. In RCP the condyles are situated in the most retruded position of the centric relation range.

7.4. Centric relation (CR)
Centric relation is not an occlusion at all. CR has nothing to do with teeth because it is the only ‘centric’ that is reproducible with or without teeth present. Centric Relation is a jaw relationship: it describes a conceptual relationship between the maxilla and mandible. Centric Relation can be described as the position of the mandible to the maxilla, with the intra-articular disc in place, when the head of the condyle is against the most superior part of the distal facing incline of the glenoid fossa. It is clinically determined position of the mandible placing both condyles into their anterior uppermost position.

The mandibular position is assumed when the head is in an upright position and the involved muscles are in active contraction, condyles are in an upper and anterior position symmetrically. In CR the mandible is restricted to a purely rotary movement around the transverse horizontal axis.

7.5. Central occlusion (CO)
If the opposing teeth are contacting each other while the condyles are in CR, can be defined as CO position. CO is different from ICP, because ICP is not describing the position of the condyles. The maximal intercuspidation in CR is the most stable position.

7.6. Movements of the mandible
The opening and closing movements of the lower jaw was first described by Posselt. During opening first the condyles have a rotation then they move forward and downward along on the slope of the articular eminence. The Posselt diagrams demonstrate these movements. The maximal values of the mandibular movements are the following: in lateral direction 10 mm, in vertical direction (opening) 50-60 mm, propulsion 9 mm, retraction 1 mm. The inclination of the condyle is 35° in average; this angle is formed by the Frankfurt horizontal plane and the condyle pathway in sagittal plane. Bennett angle is the angle formed by the sagittal plane and the path of the
advancing condyle during lateral mandibular movement, as viewed in the horizontal plane, its average value is 15°.

Fischer's angle was defined as the difference between the sagittal condylar inclinations during protrusive and lateral excursions on the non-working side, by keeping the corresponding horizontal distance from the intercuspal position (ICP) equivalent at the incisal point.

During laterotrusion the side where the bolus is grinded is called working side, while the opposite side is called balancing side.

8. 6.8. Recording the VDO (vertical dimension of occlusion)/Determination of occlusal height – Zsofia Muzsek

Is based on the master cast, the dental technician prepares the occlusal rims (Fig. 26, Fig. 27) by using that:

- Determine the vertical and horizontal dimensions of the occlusal height.
- Set values in the articulator.
- Execute the tooth alignment.

Figure 6.26. Figure 26. – Upper occlusal rim

Figure 6.27. Figure 27. – Lower occlusal rim

The occlusal rim consists of two parts (base plate and wax rim):
The base plate can be made in a traditional way (e.g. shellac or light-curing acrylate material), or be polymerized immediately with the (heat and pressure polymerizing) acrylic material of the ready denture; in which case use the acrylate base plate method.

In the case of the acrylate base plate method, the final base plate is used as bite registration plate, and later will be used as the base plate of the tooth set-up and that of the real prosthesis. Therefore during this phase it can be decided, whether the base plate does have sufficient vacuum effect and stability; can the work proceed, is it even worth, or is any adjustment required.

On the base plate along the ridge (leading) there is a 2-3 mm high and 1 mm wide „ridge”; which has the following functions:

- Strengthening/stiffening the base.
- Facilitate easy placement of the wax rim.
- Shows the dental technician the form of the ridge line.
- Provides a secure base for the tooth alignment.

The benefits of the method:

- A gothic arch can be used.
- Control of the tooth alignment is more accurate.
- Repairable: if the base is not stable enough, can make immediate corrections to it, or can be relined.

The disadvantages of the method:

- It can happen that the edge of the double-polymerization becomes visible, which can lead to irritation/problems during talking or smiling in esthetically sensitive zones.
- The master cast breaks during the first polymerization; during the articulation a gypsum key will be needed first.

The wax rim is made of red wax; during application we need to pay attention that the width is similar to that of natural teeth; it must be placed exactly on the ridge but be higher than natural teeth. Measured from the upper bite plate’s frontal vestibular edge, the wax ridge’s height is approximately 23 mm in average. Correct formation of the wax rim provides the basis of the tooth arrangement in progress.

Control of the bite plate has two steps: stability, smoothness of edges, form and size are controlled first on the sample, than in the mouth. Evaluation of the template is done on the master cast; if any adjustments are required it can be done immediately. After disinfection (e.g. Descosept spray) and washing, comes the control in the mouth. As the bite plate provides the basis of the denture in use, the retention therefore must be examined thoroughly. Any instability is experienced, must evaluate to see the base plate is not extended over or the retention area used is sufficient enough. Too much relief can deteriorate the exact matching of the base plate. In case only slight movement is experienced, stability can be adjusted using denture adhesive. If the bite plate does not require any adjustments, can proceed with the determination of the vertical dimension of the occlusial height.

8.1. Determination of the vertical dimension

Requirements: pencil, felt pen, adhesive tape, ruler or caliper, Bunsen-burner, wax knife, putty knife, and parallelism-measuring tool – a bite fork. The physiological occlusal height must be measured five times, then calculate the average of the measured values. To achieve an accurate result must take note of the following:

- The Appropriate position of the patient: ask the patient sitting in the dental chair not to lean their back and head towards the chair, the spine must be in a natural stance, shoulders rested loosely, with the eyes focused to a distant object.
• Distance of the two jawbones can be determined using specified points. Marking of those must be unequivocal along the middle line of the face (e.g. nasal tip, nasal base and Mentum).

• After that, the patient should wet their lips, and then perform the movements shown earlier: soft contact of the lips (as if you want to blow a feather) or saying the letter „S” long. During this period the distance can be measured.

After calculating the average, the physiological occlusal height is determined, by deducting 2mm, the physical occlusal height is obtained. Now the wax rim can be carved. The aim is, besides the rims contacting along the surfaces, reproduce the physical occlusal height’s value between the measurement points of the face. Two bite plates secure the interalveolar distance in the mouth; the ideal division with regards to the upper rim: lower rim ratio is 2:1 or maximum 1:1. Always start to carve the upper bite plate, because can use different anthropologic measurement points due to the maxilla’s binding to the facial skull.

During carving of the upper occlusal rim have to note the following factors:

• The wax rim on the area of the frontal tooth must be slightly ahead of the ridge / spine, aligning to the natural leaning of the teeth.

• During smiling 1-2 mm should be visible of the rim.

• The edge of the rim must reach the lower lip when the patient says „F” or „V”. Ask the patient to count loud from fifty to sixty, to control the contacts / connections.

• The incisal edge of the rim on the frontal area must be parallel to the bipupillar line.

• The occlusal surface of the rim on the molar area must be parallel to the Camper plain (tragus-nasal flap line).

During carving of the lower occlusal rim take notice of the following factors:

• The rim must be placed exactly symmetric over the spine / ridge line, otherwise increasing the unfavorable conditions.

• Height of the wax rim in the molar region must not be higher than two thirds or half of the retromolar pad’s height (Fig. 28).

• Height of the wax rim in the frontal region ought to be as high as to reach the line between the corners of the lips in relaxed state (of the lips).

• On the frontal area the upper wax rim must have a 1-2 mm undershot relative to the lower rim.

• The base plates must not contact each other, the wax rims must always be in contact.

Figure 6.28. Figure 28. – Incorrect rims
Adjustments to the wax rims are always performed outside of the mouth: Must place the bite plate on the gypsum model, to avoid deformity of the shellac base plate. Watering the gypsum cast reduces contamination with wax. During adjustments to the upper wax rim’s height, parallelism to the reference plains must be maintained (Fig. 29). To achieve this, use two rulers/spatula/palette knives: one is placed on the reference plain; the other is placed on the occlusal surface of the rim, now evaluate the parallelism of these. Or use a (ho-ve-sa) plain measurement / leveling tool, which aligns with the bite surface of the upper wax rim to stabilize it; use the ruler in our other hand aligned with the reference surface and evaluate the ratio of the splains. When the ideal state is reached, proceed by carving the lower rim. After reaching the physical occlusal height, need to check if the interocclusal distance (2 mm) is secured.

Too elevated occlusal height of the ready / final denture can cause the following problems:

- Muscle pain.
- Pain on the surface of the stress-bearing mucosa.
- Fast absorption of bones.
- The denture makes clicking sound during speech.

Too low occlusal height of the final denture can cause the following problems:

- The face appears hollow.
- The patient gets tired when chewing.
- Muscle- and joint pain can develop.

Now indicate the directional lines on the carved rims, which will help the dental technician during orientation of tooth alignment:

- The mid lines (upper and lower): determine the position of the upper and lower large incisors.
- The nasal branch line or the position of the corners of the lips during smiling: determine the position of the tip of the upper canines or the distal surface.
- Mark the position of the tooth neck on the upper rim, the smile line; on the lower rim show the incisal edge of the upper canines.

Using the marked lines, clearly indicate on the dental worksheet which lines have been chosen.
8.2. Determining the horizontal dimension of the occlusal height, spatule probe(1), (2), (3), fixation

Measure the connection between the two rims using a metal spatula / applicator / palette knife, in order to avoid the Christensen phenomena.

If the contact is stable, the spatula cannot be turned, the result of the test is negative, and the work can proceed by bonding the rims together.

If the contact is instable, the spatula can be turned between the closed rims, the shellac base plate does not align with the mucosa bone base, so the result of the test is positive. By not adjusting this, the tooth alignment will not be evaluable. The gap between the rims must be eliminated, the rims must be adjusted, and then the test must be repeated.

For fixation purpose can choose from various materials: bite fixing silicone, ZnOE, wax (Fig. 30).

**Figure 6.30. Figure 30. – Fixed bite plates**

Various techniques will presented for fixing the wax rims, but have the possibility to use facebow record of the maxillary position which can be performed prior to bonding.

Fundamentally, have to present two main types of the methods based on the fact that the patient can co-operate or no. If the patient cannot cooperate with the dentist (e.g. in the case of facial paralysis or cerebrovascular dementia), then the dentist places the rims with active hand lead into the ideal position. If the patient can cooperate actively in the work, a drawing machine or maneuvers based on myodynamics can be used. The basis of the latter is the linguo-mandibular homotropia, the definition of which the following: based on the effect of the tongue’s movement the jaw is moving similarly. Therefore, by sticking out and retracting of the tongue, the position of the jaw can be fixed. The same happens during swallowing and lifting of the tongue. We can try the Dawson-method as well: the patient is in a slightly leaned position; the dentist fixes the rims with both index fingers, with both thumbs being underneath the mentum. Slightly move the mandible, which has to move freely, without hindrance. After that let the patient close the mouth but the dentist must not put pressure on the occlusal rims. In order to achieve a perfect binding during the process, use soft fixing material (e.g. silicone).

The bonded rims have to be removed from the mouth together, using a 90 degree turn. After washing and disinfection, place the occlusal rims on the gypsum model. Then determine the tooth color and send the work to the dental laboratory.

8.3. Application of the Gothic arch

To carefully determine the horizontal dimension of the occlusal height, use a so called intraoral drawing machine. The distance of the drawing pin and drawing plate is in the correct vertical dimension, the acrylate rims made for the acrylate base are 1-2 mm smaller than the measurements determined earlier in order to secure a good view (Fig. 31). Should ask the patient repeatedly to touch the rims together several times, and the area marked most densely by the drawing pin determines the adduction field, with the adduction point sitting in the middle (Fig. 32, Fig. 33).

**Figure 6.31. Figure 31. – Gothic arch**
After determining the ideal position of the mandible, have the possibility to measure the extension of the eccentric movements, in which case the drawing pin makes a so called arrowhead design on the drawing plate (Fig. 34). As the last step, guide the edge of the drawing pin in the adduction point marked on the plate and fix the rims together using a pink wax (Fig. 35, Fig. 36).

Figure 6.34. Figure 34. – Arrowhead design
8.4. Preparation of samples for tooth alignment

8.4.1. Marking of lower gypsum cast

The form of the ridge line provide help for the tooth alignment, draw a parallel line on the side of the lower gypsum model, marking the lowest point of the ridge / spine and the arch of the ridge line.

To control the height of the tooth to be aligned, mark the half point height of the retromolar pad. Strengthening the ridge line in the molar area, is necessary to provide help for the palatinal cusps position of the upper teeth in the case of normal bite, securing the stability of the denture and compensating the lifting forces. Strengthening the ridge line in the frontal region and projecting both sides of the gypsum cast is necessary, so can align its position during the setup of the frontal teeth, lowering the possibility of torque development.

8.4.2. Marking the upper master cast

Project a line connecting the incisive papilla and the median palatine suture on the upper gypsum model, evaluate if the mid line drawn on the wax surface matches with that. A perpendicular line on the mid line of the
gypsum cast through the middle of the incisive papilla can be used to control the cusps position of the canines as well as the symmetry of the carved rims (Fig. 37). On average, the large upper incisors are positioned 8-10 mm in front of this line.

**Figure 6.37. Figure 37. – Marked upper and lower master cast**

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9. 6.9. Tooth arrangement – Zsofia Muzsek

Next step after mounting the master cast is the tooth setup. On the already prepared upper master cast the distance between the two canines are measured and technician chooses the proper artificial tooth garniture. This requires combination of science and artistic ability based on observation. For example, if the bizygomatic width is measured by a facebow and the distance is divided by 16 the width of the central incisor is obtained. Whereas dividing the bizygomatic width by 3.3 the width of the six anterior front teeth are acquired. If documentations of the patient are available e.g. previous casts or photographs, they provide information about the ideal tooth form, occlusial type, overjet and overbite and the possibility of diasthema. The tooth shape is influenced by the face character, female type teeth are more rounded, and on the other hand masculine form teeth are more rectangular. The tooth shade selecting is influenced by the color of the skin, hair and sclera, and by the age: teeth get darker with age. Patients usually pick the whitest shade from the shade tab, offer your opinion but do not be persuasive.

The material of the artificial teeth could be acrylic or porcelain. On one hand the porcelain teeth wear less and they are more translucent, on the other hand their bonding ability to acrylic base is less and if the patient drops the denture while cleaning, has a higher risk of damage.

Acrylic teeth on one hand wear well and usually last for 5-7 years, on the other hand due to their chemical bonding to the denture base they are easy to repair. In most of the cases fracture of the denture base occurs; using high impact acrylic material is emphasized. While selecting the posterior teeth form, can use anatomic, semi-anatomic or non-anatomic tooth selections. Difference is only the cusp angulations, anatomic teeth has an inclination of 30 or 40 degree, semi-anatomic teeth has an inclination of 20 or 10 degree, non-anatomic teeth has no inclination.

How to choose them?

Anatomic and semi-anatomic teeth will be used:

- if esthetic is important even in the premolar region,
- if there is no contraindication: severe alveolar ridge desorption, uncoordinated jaw movements,
- if like to achieve bilateral balanced occlusion, need to select the tooth cusp angle similar to the condylar inclination,
• if the opposing teeth are natural teeth.

Non-anatomic teeth will be used:

• if there are jaw size discrepancies, e.g. cross-bite situations or Angles Class III malformation,

• if there is an extreme alveolar bone resorption and like to reduce the horizontal component of the forces,

• in case of uncoordinated jaw movements,

• if esthetic is not so important,

• if we setup the tooth without overbite in monoplane occlusion.

9.1. Choosing posterior teeth

• Need to accord to the size of the already chosen front teeth, their cervico-incisal height influences the size of the molar teeth.

• The distance between the distal margin of the canine and the anterior ramus of the mandible (29, 30 mm) will guide the tooth setup.

• The bucco-lingual size of teeth affect the neutral zone, if there is not enough space for the tongue movements, need to use smaller teeth.

9.2. Rules of tooth arrangement

During the anterior teeth setup, need to take care about symmetry.

The incisal edge of the central incisor and the canine should be placed on the occlusal plane, while the lateral incisor should be placed 0.5 mm above the level of the occlusal plane. From frontal view the axis of the central incisor is parallel with the midline, the long axis of the lateral incisor and the canine shows a slight distal inclination to the perpendicular.

From sagittal view the centrals and laterals should have their incisal edges placed more anteriorly than their necks to support the lips in a slightly prominent and natural position. The canine is perpendicular to the occlusal plane (Fig.38).

The incisal edge of the lower anterior teeth should be all placed on the occlusal plane; the inclination of them is similar to the upper teeth from frontal view. From profile view the incisal edge of the central incisor is more anteriorly than its neck, the lateral incisor is perpendicular to the occlusal plane, and the neck of the canine is more anteriorly than its incisal edge. So the neck of the canine is more prominent. Under ideal circumstances there should be a 1-2 mm horizontal overlap (overjet) and a 1-2 mm vertical overlap (overbite) between the upper and lower front teeth. In case of monoplane occlusion there is no overbite hence needs to determine the type of the arrangement in advance.

From the lateral aspect there should be one half tooth offset between the maxillary and mandibular teeth to ensure the posterior teeth can have a normal cusp to fossa relationship. After the front arrangement have the opportunity to have a primary control. Check the lip support, the position of the corner of the mouth, and the esthetic of the front teeth: the symmetry, overjet, overbite, size and color of them. It is easier to make changes now rather restart the whole arrangement again.

Figure 6.38. Figure 38. – Tooth setup from profile view
9.3. Concepts of tooth setup

There are many philosophies of arranging the denture occlusion, which are based on scientific experience, to make it easier to determine which type of occlusal scheme to use, will describe three occlusal schemes (there are others): the monoplane occlusion, the lingualized occlusal scheme, and the concept of bilateral balanced occlusion.

9.3.1. Lingualized occlusal scheme

Lingualized occlusion is a type of bilateral balanced occlusal concept. The philosophy of a balanced occlusal scheme is to improve denture stability while the patients feel less discomfort as dentures are more stable. There is indirect evidence that balanced occlusion may reduce ridge resorption and denture stability will be present even in case of increased forces. Lingualized occlusion differs from classical bilateral occlusion only, that there are contacts just between upper palatinal cusps and mandibular teeth.

Its features:

• Palatinal cusps of upper premolars and molars shall contact central fissure of the lower premolars and molars in ICP, but no frontal teeth contact is allowed.

• The mesial cusps of the upper first molars are positioned on the occlusal plane, but the distal cusps of the upper first molars are slightly above it, while the cusps of the upper second molars are elevated more, this is how Curve of Spee is formed.

• On the maxilla it is recommended to use artificial teeth with anatomic cusps, but on the mandible semi-anatomical or non-anatomical teeth are preferred.

• In each eccentric movement there is contacts on both working and balancing sides.

• The method has the advantage that the more dominant upper teeth are anatomic teeth, so the appearance is esthetic. The forces are transferred in the direction of the long axis of the lower molars. The tooth alignment is technically challenging, it is contraindicated if recording of jaw relations is complicated, if the patient has in- coordination problems, or severe ridge resorption.

9.3.2. The monoplane occlusion (1), (2)

The artificial teeth without cusps form a completely flat occlusal plane, there is no cusps-fissure contact. The maximum value of the overjet is 2 mm between the front teeth to avoid the biting of the lips. In ICP there is no contact among upper and lower front teeth, the value of the overbite is 0 mm. During eccentric motions usually contact could not be found on the balance side, but contact may occur on the front region. The philosophy of this method is to avoid the destabilizing effect of the cusps. The advantage of the method is the easy technical workout; the disadvantage is the non-favorable esthetic, thanks to the non-anatomical artificial teeth. So the monoplane occlusion is contraindicated if the esthetic appearance for the patient is the primary goal, the very steep condylar guidance makes the denture unstable. At the molar region the cusps follow the occlusal plane but have to keep in mind not to setup molars to the upward anterior mandibular ridge. If the usable space is limited it is better to leave the second premolar and set only the two molars. During the arrangement (after the front teeth setting) should start with the setup of the upper molars. Then continue with the lowers. In this case have to
be careful not to extend the occlusal surface of the lower molars over the height of the half point of the retromolar pad. In many cases due to the unfavorable positions of the edentulous ridges or because of the skeletal malocclusion have to set the teeth in crossbite position. This is easily achieved in monoplane occlusion.

The tooth setup could be determined by the so called interalveolar angle. The interalveolar angle is formed between the line connecting the upper and lower ridgeline and the occlusal plane (α). If this angle is an acute angle have to setup the molars in crossbite position. In case of a right angle it is better to choose edge-to-edge contact. And whenever the angle is obtuse angle normal occlusal scheme should be selected (Figure 39., Fig. 40.).

**Figure 6.39. Figure 39. –**

![Figure 39](image1)

**Figure 6.40. Figure 40. –**

![Figure 40](image2)

**9.3.3. Bilateral balanced occlusion**

In natural dentition when the mandible makes eccentric motions (protrusion and lateral pulsion) the condyle moves downwards along the slope of the articular eminence. Due to this motion a gap occurs between upper and lower dentition. This separation is called Christensen phenomenon (1). During protrusion the sagittal, and during laterotrusion transverse Christensen phenomenon could be defined. So in this case there is no balanced position achieved by the teeth. This is not required with natural teeth. In completely edentulous situations the Christensen phenomenon has to be compensated during the tooth arrangement. According to Gysi the mandibular movements depend on condylar and incisal inclination (1910). He was the first to emphasize the importance of bilateral balanced occlusion in completely edentulous cases. According to his hypothesis to ensure a proper stability at least three point contacts are needed between upper and lower denture, and this kind of contact has to be achieved during protrusion and lateral movements. So in completely edentulous situations during mandibular movement there is no gap which may cause instability of the dentures. This means that there is no Christensen phenomenon. During protrusion the contacts are found among front and molar teeth (balance in protrusion). During lateral movement the buccal cusps of the uppers and the buccal cusps of the lowers get into contact. As well as upper and lower lingual cusps meet (cross arch balance). For balanced occlusion anatomical teeth should be used. In the absence of anatomical teeth can choose non-anatomical with the help of balancing slopes and compensating curves.

If the condylar inclination is too expressed should use non-anatomic (without cusps) or anatomic teeth with flat cusp slopes. The rules of artificial tooth selection were set by Hanau (Hanau’s quint).
• condylar angulation,
• incisal inclination,
• steepness of the cusps, depth of the fissures,
• Curve of Spee and Monson,
• position of the occlusal plane.

The correlation of these five factors and their effect on the balanced occlusion is described by the formula of Theilman:

\[ C = \frac{\text{condylar inclination} \times \text{incisal guidance}}{\text{occlusal plane} \times \text{cusp inclination} \times \text{compensating curve}} \]

The condylar inclination is given. The proper place of the occlusal plane is determined by biomechanical concepts. The place and shape of the front teeth is influenced by esthetic demands. For example, increasing the length of the incisors, better esthetic appearance is achieved but gap may occur on the molar region which has to be compensated. From the Theilmans formula can conclude, which factors should be modified to compensate the changes.

As the condyle moves downwards a gap may occurs among the upper and lower molars. In order to get contact should modify the angulations of the occlusal plane to be similar to the condylar inclinations. Or choose artificial teeth with steeper cusp slopes. Because the long axis of the front teeth determines the incisal guidance the dentist has to choose the proper angulations in order to avoid Christensen phenomenon among molars.

Our main goal in balanced occlusion is to achieve stable contact during every eccentric movement while we trying to setup the teeth to the ridgeline (except upper front teeth).

Rules:
• with articulating foil, mark the contacts in CO, and modify until have same contacts on both sides,
• avoid having contacts on the slope,
• evaluate the eccentric motions,
• use different color foils for each movement and have to ensure that the marked CO contacts are to be visible through the whole procedure,
• the proper set working side and balancing side contact is not a single point but a line,
• the balancing side contact cannot be stronger than the working side,
• during eccentric movements the motion of the teeth on each other are in harmony and unobstructed sliding movement has no hidden jump,
• if the antagonist is natural dentition should use anatomic form teeth to obtain stability.

10. 6.10. Try-in – Daniel Nemeth

Purpose of the try-in is to examine the denture in work from every aspect:
• outlook,
• phonetics,
• occlusal conditions,
• comfort of the patient.
There is the possibility to perform multiple try-ins; by making adjustments to the test denture, another try-in is required. Must take note of the minor problems as cannot make any adjustments after processing. Using multiple try-ins we can save time and money with regards to the later corrections / adjustments.

10.1. Primary try-in

In complicated cases it is recommended to ask firstly for alignment of the front teeth, in order to evaluate the aesthetic appearance, which has importance to the patients. If corrections are necessary, it is easier to relocate these few teeth and the dentist has to make these adjustments as the dental technician does not see the face and mouth of the patient. Of course, the supervision of the occlusal height is necessary as well.

10.1.1. Vertical dimension

The interocclusal space can be 2-4 mm which is measureable and perceptible for the patient. Finding the vertical dimension is incorrect; have to modify this value immediately as it directly effects the teeth’s contacts and the aesthetics of the face. (When the mouth opens the vertical dimension increases, the incisal edge of the lower incisors moves down- and inwards. So the vertical dimension increases, may observe greater overjet, therefore reaching a Class II skeletal malformation). If the occlusal height is too large or have to modify one or both jawbones, it is recommended to get that done by the dental technician as re-alignment of all teeth will be required (both the frontal and the rear teeth’s height will have to be adjusted).

10.1.2. Horizontal dimension

Control the alignment of the denture to the mucosal bone base using a metal spatula; if the test is negative, precede with the observation of the connection points using articulation paper or foil. After that, ask the patient to bite on a silicone based bite registration material, then place back the disinfected test denture on the gypsum model, and place into the articulator to control the spatial accuracy. (Examine if, can observe the same state with and without bite registration silicone on the articulator.)

10.1.3. Position of teeth

• Position of the canines.

• Overjet: correctly chosen overjet will help to avoid biting of the lips and the bucca, in some cases the 1-2 mm relocation of the tooth bodies will be required (Fig. 41).

Figure 6.41. Figure 41. – Ideal position of the frontal teeth

• The formation of the correctly determined tongue space: the teeth must not be positioned too lingual, ask the patient to evaluate comfort of the prosthesis and ease of speech.

10.1.4. Excentric connections

Evaluation of the work side and balance side connections are done using naked eyes and articulation paper/foil, comparing to the contacts observed in the articulator.

10.1.5. Aesthetics
Control the position of the front teeth relative to the lips, the harmony of the upper teeth relative to the line of smile, accuracy of the middle line and the occlusal plain / sheet. Control the exact soft tissue profile, contours, lip support, aesthetics of the vermilion border, the nasolabial angle. Ask the patient to express their own opinion without, influencing them. It might be of help if a family member or friend accompanies them to the treatment, be able to have an independent/unbiased opinion. Completely try changing everything that the patient does not like, and not trying to persuad the patient to accept the form designed by us, rather ask the dental technician to help execute the required modifications.

10.1.6. Phonetics

Phonetics can be controlled with the try-in as the patient can comfortably talk in this state. Observe the spoken words are lisping, if true it can be a sign of incorrectly chosen overjet between the front teeth, diastema between the teeth, or incorrectly designed palatal contours. If the patient did not have denture for a longer period of time or had made dramatic adjustments to the new denture (position of teeth, vertical dimension), shall ask the patient to read a newspaper loud at least for 5 minutes to get used to these changes and the altered state. During the speech of alveolus tones (s, z) the edges of the upper and lower incisors touch each other, so ask the patient to say e.g. Mississippi and check the position of the teeth. In the case of labial-alveolus tones (f, v) the upper incisors just slightly touch the rear one-third of the lower lip.

10.1.7. Base plate contours

The base plate contours effect phonetics, sense of comfort and retention. It can be stated in general that the base plate must not be Convex, should be rather slightly concave. Unnecessary wax should be removed from the lingual areas to ensure sufficient space for the tongue. Make sure that the denture does not have any areas that are unnecessarily thin or thick.

10.1.8. Opinion of the patient

Let the patient ask any questions they like to be answered. Do not let the patient put pressure on you so you rush this phase to save time. Do not ask closed questions from the patient, rather open questions where the patient needs to elaborate. Feel that the patient is unsatisfied; ask as long as you need to find out about their problem.

10.2. Last try-in

In this part of the treatment, check if the required changes are appropriate. Feel the need for any adjustments; ask for those changes to be executed. Whether the vertical or horizontal dimension needs to be changed or even the position of the teeth, then these changes have to be verified again. During the last try-in, concentrate on the aesthetic and phonetic aspects and the comfort of the patient. Send the disinfected, washed and accepted try-in to the laboratory, to prepare for finalization. If we have asked the reocclusion from the laboratory, raise their attention to use the correct embedding technique accordingly. Have the possibility to choose the color of the acrylate base plate material as well, in this case go by the mucosal color of the patient. Request a so called clear palate too: some patient’s comfort is increased when the upper denture’s palatal part is transparent, if their earlier denture was like that as well (Fig. 42).

Figure 6.42. Figure 42. – Prosthesis with clear acrylic palatal plate
11. 6.11. Processing – Daniel Nemeth

Prior to arrival of the patient, place the final denture into clean water for 24 hours in order to dissolve the surplus monomer. During control of the prosthesis attention must be paid to eventual sharp edges and burrs – those must be smoothened if any present.

Placement of the denture into the mouth must be started with the upper prosthesis. Ask the patient if they feel the prosthesis is comfortable, and describe or rather show the areas of discomfort. If any instability is experienced and the denture does not have retention, first explore the extension of the rear closure. Mark the pharyngeal seal on the mucosal using an ink pencil; after placements of the prosthesis in to the mouth compare the relativity of these to each other. Control this function using a fit checker; evenly disperse a small amount of paste (prepared on the mixing plate) on the unpolished surface of the dried denture using a paintbrush; then place the denture in the mouth and remove it after setting of the material. Later evaluate the result:

- On the areas where the paste does not cover the acrylate denture base at all: disencumber the mucosal surface for relief, remove material from the given surface using a fraser.
- If the original brush strokes are visible on the surface of the paste, it means there was no contact to the mucosa on that area, showing that the modification of other overloaded areas is necessary to enable contact on this area too.
- If the paste covers the unpolished surface of the denture base evenly, then the contact is appropriate.

Repeat the process until stability of the denture is deemed acceptable: lack of contact only on minimal surface; and there are no larger areas not covered by the paste. In the next step evaluate the overextensions on the edges. Apply a min. 5mm wide indicator material on the vestibular edge of the prosthesis, then place the denture in the mouth and ask the patient to cautiously perform the functional movements. Carefully reduce the denture plate where the paste does not cover the marginal parts of the denture. Extra attention must be paid to the area of the frenulum, as that is the predilection location of the irritation.

If all necessary corrections are made, remove the residual paste from the denture and remove the fat with alcohol.

Then proceed by checking the lower prosthesis in similar steps. The modified areas of both the upper and lower prosthesis must be polished using a wet polishing brush and polishing paste.

Now the occlusion can be checked. Both prosthesis are placed in the mouth, place cotton wool between the molars on both sides, then ask the patient to press with high force. This pressure will cause the feeling on the mucosal as if the patient was wearing the denture for a longer time. Release the dentures to maximal intercuspid position and check the connections using articulation paper or foil; then perform the necessary modifications. Return of the patient is necessary after 24, 48 and 168 hours; and after that as many times as needed until maximum comfort feeling is achieved. Information of the patient about the appropriate hygienic rules is absolutely necessary.

12. 6.12. Remounting – Daniel Nemeth

During this procedure the new dentures are remounted. It is a time consuming procedure because less appointment is needed. It is comfortable for the dentist to correct the occlusal surfaces, outside of the mouth; there is no need to apply the dentures to the mouth repeatedly. It is easier to detect occlusial interferences which may cause painful mechanical irritations and denture instability. During the conventional flasking the master cast has been destroyed that is why a new cast is needed for mounting. If the lab was previously asked to reoccluding the dentures (primery remounting) the existing casts could also be used. If new casts are needed, follow these instructions:

The unpolished surface of the denture has to be isolated with petroleum jelly, block out the undercut areas (e.g. with wet cotton rolls). Then mix a fast setting gypsum and apply in order to ensure proper stability. Take care to avoid covering the vestibular surface with too much material otherwise it will difficult to remove the denture from the cast (Fig.43.).

Figure 6.43. Figure 43. – Upper denture with block-out
Steps of remounting:

• Cleaning the dentures, insert them into the patient’s mouth.

• Facebow recording: the position of the maxilla is recorded with facebow. For the fixation of the denture, compound material (e.g. Bite Tabs) has to be placed in three sides of the bitefork (Fig. 44.). The material has to be warmed up to 50°C in water bath, then has to be applied to the occlusal surface of the denture, take care of the midline (Fig. 45.). Next step is the adaption of the compatible facebow on the patient’s face. First have to adjust the facebow to the outer meatus as far as the patient will feel slight pain, then the third fixation point is the glabella, and have to stretch the facebow until reaching proper stability (Fig. 46.). Because the support of the facebow was on soft tissue, the patient should be in laying position to minimize the effect of gravity. Then the bite fork has to be attached to the fixed facebow with the attachment. Then the fixation will be loosened and removed from the patient’s head while taking care of the tightened assembly (Fig. 47.).

**Video 6.**

• Recording the occlusion: for this procedure can use registration silicone material or aluminum containing wax as well. Recommend the wax, because controlling the position of the mandible is easier. The lower denture has to be immersed to the previously used water bath (50°C) with the occlusal surface already covered with wax (Aluminawax) (Fig. 48.). Have to control the proper plasticity of the wax and double check the adaptation to the denture. The dentures should be inserted into the mouth and ask the patient to close slowly until the first contacts. If having premature contacts need to reduce the wax with a sharp scapula until we see bilateral contacts. Extra orally check the proper stability of the dentures to each other. The dentures should be washed and disinfected.

• Mounting: Start mounting of the upper denture. In order to keep the position of the facebow a cast support is used (Fig. 50.). Fast setting gypsum is recommended for this procedure. After the setting of the gypsum, continue with mounting of the lower denture which is still covered with the Aluminawax. Adjust the articulator first, the incisal guidance should be increased from zero point with the thickness of the wax (approximately 1mm) (Fig. 51.).

• After setting of the gypsum remove the wax plate, and set the incisal guidance to zero point. With the articulating foil or silk, are able to detect the premature contacts, and the occlusal surfaces should be corrected. After arranging the bilateral contacts in centric occlusion, continue with analyzing of the eccentric positions.

• Finally we use polishing paste to smoothen the modified surfaces.

**Figure 6.44. Figure 44. – Bite tabs on the bite fork**
Figure 6.45. Figure 45. – Position of the upper denture

Figure 6.46. Figure 46. – Facebow recording

Figure 6.47. Figure 47. – Assembly already separated from the facebow

Figure 6.48. Figure 48. – Adapted Aluminawax on the lower denture
Figure 6.49. Figure 49. – Cast support

Figure 6.50. Figure 50. – Mounting the upper denture

Figure 6.51. Figure 51. – Mounting the lower denture
13.6.13. Problems after the delivering – Daniel Nemeth

Mechanical irritations mostly occurred because of occlusional problems, modifying the denture base is only allowed if these corrections are already made.

Remounting of the denture is always useful. If irritated surfaces are detectable, need to mark them with e.g. indelible marker, and then the previously dried denture should be inserted into the mouth; that is the way of correct localization of the problem. If the irritation is not yet visible, palpate the sensitive area. Keep in mind, that the patients sometimes cannot exactly define these areas; need to give them time for proper localization.

Ask patient to describe in detail:

- Where are the painful areas? - Dentist needs to locate with fit checker or with a blunt instrument.
- When does the pain occur? - By chewing or in rest position.
- How long does it last?
- Does anything make it better or worse?
- Have patient demonstrate the problem if you have difficulty diagnosing the cause.

Other problem could be the lack of retention, stability, not well defined vertical dimensions, allergies, infections and not correct tooth arrangement.

Most common areas requiring adjustments:

Maxillary

- Hamular notches – ulceration can occur if over-extended.
- Labial frenulum – requires adequate relief otherwise feels too bulky to the patient.
- Palatinal raphe.

Mandibular

- Lingual frenum – impingement can cause displacement of the denture or ulceration.
- Retromylohyoid area- overextensions can cause sore throat; or instability of denture when swallowing.
- Buccal shelf- overextension can cause instability of denture when speaking.

13.1. Single complete dentures

If there is only a single denture made for the patient, it is usually the upper one, because the patients loose first the maxillary teeth. Despite of having tooth present in the maxilla, and a complete denture needed to be done on the mandible, there is almost no chance for success. Because of the difference in loading, this fact may explain the reason should try to keep root(s) for overdenture (Fig.52.). The lost dental support could be substituted with enosseal implants.

Otherwise fast and obvious alveolar ridge resorption might be observed.

Figure 6.52. Figure 52. – Tooth kept for overdenture support
The upper natural teeth have a negative effect on the lower complete denture, and can cause crack or fracture. Previously formed malocclusion (e.g. elongation or rotation) causes the denture to be less stable even there will be difficulty to balance the occlusion. In such cases before starting the procedure it is useful to analyze occlusal plane first.

On the study cast an occlusal rim is made, and the vertical and horizontal dimension of occlusion will be recorded. Evaluate how to reach positive outcome from the unfavorable starting situation. The over expressed Curve of Spee could be reduced, elongated teeth could be nivellated, occlusal surface of rotated teeth could be restored (even with composite filling material).

If there are only 6-8 remaining teeth in the frontal region on one jaw, the prosthetic rehabilitation has to be proceeded simultaneously. While trying the framework of the removable partial denture on one jaw, on the opposite jaw the functional impression should be done. It will be possible to record the jaws relation at the same time. That is the method for optimized tooth arrangement. If a fixed denture (crown or bridge) is planned on the opposing jaw a mock-up is recommended for the same reasons.

While setting up the upper front teeth, positioning them is sometimes difficult, focusing only on esthetics can have a negative effect on denture stability. Like to avoid excessive overbite set up in a higher position and the result will be also a negative effect, because the upper lip will overlap them.

Setting up the molars: instead of reducing the occlusal surfaces it is recommended to decrease the base of the artificial teeth.

If natural teeth are opposing artificial teeth, the wear of the acrylic teeth will be increased, patient’s regular checkup is essential more often. It is unfavorable if the opposing teeth are porcelain teeth, because it may leads to abrasion of natural teeth and later to sensitivity.

The already mentioned stress area; problems may cause fracture of artificial teeth or even damage of the denture base. If there is multiple tooth fracture in the case history, occlusal examination and remounting is required. If denture base fracture occurs more frequently, the solution could be reinforcement.

For this procedure, use metal or fiber reinforcement.

Using metal plate, wire or net only macro- mechanical connection could be found, so we should take care on proper bonding (Fig.53.). Enough perforations will maintain space for the acrylic that is why metal net is used in upper dentures. The disadvantage is the grayish color; even the gold colored nets are visible.

**Figure 6.53. Figure 53. – Metal plate reinforcement**
Nowadays fiber reinforcement is more frequently used in dentistry. Can use aramid, carbon, ultrahigh molecular weight polyethylene or glass fibers. Clinical studies have proved an effective strengthening effect whilst using glass fibers (Fig. 54.).

The advantage of the glass fibers are the possible chemical contact to the acrylic base, and the reinforcement could be used during the processing of a new denture and even during the repair of an older denture. The glass fiber reinforcement could be used for both the upper and lower dentures. The disadvantage is only their technological sensitivity and their price.

Figure 6.54. Figure 54. – Fiber reinforcement

Also to increase the strength of the denture base can use the modern high impact acrylic resins for denture fabrication (for example Luciton 199 Dentsply International, USA).


Fully removable acrylic dentures often need to be repaired, to modify the denture base, the acrylic teeth or both. The correction could be either material reduction or addition.

Methods for material reduction:

• remounting,
• correction / grinding of the denture base.

Methods for material addition:

• repair of the fractured base,
• repair of the damaged (missing) artificial tooth,
• relining/ rebasing.
Artificial tooth fracture may happen due to incorrect technology. If the connection between the artificial tooth and the denture base is not appropriate, the tooth will fall out during use. In this case a piece of the acrylate denture base is missing at the base of the artificial tooth. In those cases can count on further breaking of the teeth. If the denture falls to the ground or other physical impact occurs and the polymerization of the artificial tooth into the denture base was done in correctly; the tooth often breaks together with the acrylate around it. If the broken tooth is available and can be placed back then nothing special needs to be done. The laboratory stabilizes the broken piece correctly and polymerizes the artificial tooth again using indirect method. If the broken artificial tooth cannot be placed back accurately or is not available, an antagonist mould must be made. It is submissive to fix the occlusion prior to moulding. If the acrylate surrounding the artificial tooth breaks as well but does not affect the unpolished surface or the edges of the denture; a situational cast does not need to be made with the denture. In any other cases – breaking of the acrylate does effect the unpolished surface and / or the edges of the denture – a situational impression has to be made using alginate impression material. The denture remains in the impression, and so send it to the laboratory immediately; or pour it in the clinic according to the rules of alginate moulding.

If the denture base breaks, first need to find out about the possible causes. It may happen that the chewing pressure caused by the masseter molars causes breaking of the denture base. In this case it is expedient to strengthen the denture base, even preventively. The denture may break due to falling or other physical impact, frequently they fall into the basin or onto the floor of the bathroom during cleaning. The denture breaks or cracks on these hard surfaces, but breaks may also occur due to wear. Anatomical structures found in the mouth, such as palatal torus may cause mucosal bone atrophy around the tooth root left in the mouth under an overdenture prosthesis; the denture waggles on the non-atrophic surface, and that may lead to fracture of the denture. Typical breaking / fracture forms may occur: in the case of palatal torus, the denture will typically break close to and along the median-sagittal line.

It must be determined first if the ruptures can be joined at all. If not, the denture is irreparable, new denture has to be made. If the ruptures can be fixed, get it repaired in the dental technician laboratory using indirect method / technique (picture 55). If the rupture is caused by the atrophy of some parts of the jawbone, resulting in the waggle that has caused this, relining / rebasing may be necessary besides reparation of the fracture. Intend to improve the contact of the denture using relining / rebasing. This may affect a part of the unpolished surface or the whole area. Relining / rebasing are necessary if retention of the denture is insufficient; the patient reports that a gap is formed and therefore food gets underneath the denture. Criterion of relining / rebasing is that occlusion of the denture, the vertical dimension and chewing surface of the artificial teeth is appropriate. Using relining / rebasing may correct only retention problems caused by inappropriate contact. Relining / rebasing are contraindicated in other cases, such as insufficient stability caused by unfavorable spine form / ridge line. Relining / rebasing can be performed using direct or indirect method. For relining / rebasing use acrylate or silicone based material. Direct relining / rebasing is done using a cast. During molding have to pay attention that the thickness of the material is appropriate and that it is applied only in the necessary locations. If material is applied in locations where contact is appropriate, it may lead to changes in the vertical dimension and change of the occlusion. To avoid that, always have to check the occlusal contact / catching and the vertical dimension. After checking the occlusion and eventual correction, modify the denture base by removing the undercuts, enabling removal of the denture from the cast later. The denture has to be cleaned thoroughly (e.g. in an ultrasonic cleaning tub). Using the cleaned denture check the functional edges; if any deficiency is experienced have to correct those using the method explained earlier at the functional impression. Afterwards have to remove 1 mm thick material from the unpolished surface of the denture base if the interocclusal space is appropriate. Orientation grooves may be used to enable / help removal of an even layer. For molding, use
silicone or zink-oxide eugenol impression material. In the case of silicone the appropriate adhesive material must be applied. The moulding material should be applied in an even layer thickness on the prepared denture base. Then the denture is placed in the mouth, first in the front then proceeding towards the posterior areas. During binding of the moulding material the patient shall close the mouth with light pressure so can check the occlusion besides controlling the vertical dimension. Finally, check the mould and send it to the laboratory.

15. 6.15. Denture Pain and Looseness – Gyula Marada

Clinicians can save time and minimize repeat visits for patients with complete denture problems by employing five strategies for eliminating etiological factors:

a. establishing a differential diagnosis,

b. identifying variations from normal,

c. allow patients demonstrate their problems,

d. always using an indicating medium when making adjustments to prostheses,

e. measuring patient’s satisfaction after corrections.

a: establishing a differential diagnosis:

In order to find out the possible factors causing problems for denture user’s, a good clinical history and a thorough clinical examination is needed. Listing the potential problems, ranking them and eliminating them one by one is suggested. If the cause of the problem is removed, pain will be reduced and ulceration will disappear within 10-14 days. Every wound on the mucosa which is not healed within 2 weeks, especially if denture irritation is ruled out, a biopsy is recommended.

b: identifying variations from normal:

Many denture wearing problems could be eliminated by carefully controlling the prosthesis: not enough extension, contour, flange and tooth position could influence the stability. Correlation of anatomic areas to irritated surfaces should be analyzed by intraoral examination.

c: allow patients demonstrate their problems:

It is easier to find out the cause of the problem, if the patient is demonstrating it. For example if the problem occurs by chewing, cut a piece of cotton roll, wet it, and give it to the patient in order to orient the location. If speaking, singing, or simply wide opening of the mouth is limited, ask the patient to demonstrate it, so can carefully watch, what is exactly happening during these activities.

d: always using an indicating medium when making adjustments to prostheses:

Even if it is only a small wound on the mucosa, do always use an indicating medium for localization of the problem. This medium can be fit checker (light bodied C silicone) or any medium bodied silicone or marker. For adjusting the occlusal surfaces use of articulating paper, foil or silk is recommended.

e: measuring patient’s satisfaction after corrections:

After finishing the adjustment, ask the patient whether feels a difference. Do not only ask the patient whether it is better, but ask to describe how much it is: e.g. 20% or 80% improvement. It is never possible to eliminate the sensitivity of an irritation immediately with100%.

15.1. Diagnosing Denture Problems

Many denture problems are based on either pain or instability.

1. Causes of denture pain may occur because of: occlusal disharmony, non-satisfactory fitting and contouring of denture base, vertical dimension problems, infections, systemic diseases or conditions and rarely allergy.
2. Causes of instability (denture looseness) may occur because of: occlusal disharmony, non-satisfactory fitting and contouring of denture base, problems of tooth arrangement, or poor anatomy of the patient.

It is not proven, but many adjustments are needed because of occlusal disharmony and non-satisfactory fitting and contouring of denture base. That is why patient’s case history is so important: characteristic of pain: starting time, length, does anything make it better or worse, followed by a thorough clinical examination.

16. 6.16. Copy denture – Gyula Marada

Making a new complete denture for an older patient needs many treatment time and great experience from the dentist. It is recommended the use of cost-effective materials and methods if the patient has several systemic diseases.

It is a difficult task to satisfy elderly denture wearers who walk into dental office for new dentures. In such a situation it is really important that all aspects of their previous dentures be reproduced in the new denture. Elderly patients who have been wearing the same dentures for years may find it difficult to adjust to a new prosthesis. The original dentures provide valuable information that can be used to increase the success of the patient’s adaptation to the new dentures. First need information from the patient: why they would like to have a new prosthesis, is there anything on the old denture they wants to change or any reason for a new denture fabrication. If it is possible to make correction on the older denture: e.g. relining or rebasing, than with a cost effective treatment are able to satisfy the patient’s need. Or this denture will be a base for a copy denture method.

When is this procedure recommended?
• If there is an elderly patient who got used to their denture.
• If the patient has systemic disease which is influencing the success of the new prosthesis e.g. Parkinson’s disease, stroke, internal disturbances.
• For implant planning and implant template.
• For interim denture if the patient has multiple denture base fracture.
• If the patient had stomatitis prothetica and a new denture is needed because of possible re-infection.

What is the goal of copying method?

One treatment option is to provide the patient with same esthetics of the previous denture. The attempt to reproduce those aspects of the denture that the patient is satisfied with, for example, form or fullness of denture in regard to esthetics and arrangement of the teeth, while selectively improving those aspects of those the patient and clinician have become dissatisfied.

There are several techniques developed and described, which are only different from each other in the used materials. Describing here is a technique in detail, where the previous denture was copied to the new prosthesis.

• First step, make a bite registration and an antagonist (situational) impression, clean the denture in a calculus/stain remover solution for 15 minutes and then brush with soap and water.

• The denture is flasked, and putty silicon material is used as the investment material. The polished surface of the denture is placed into the material to the level of the denture borders. This creates a flat border for material around the denture and the investment material is allowed to polymerize completely. (Fig.56.)

• A new amount of putty silicon is mixed and placed over the unpolished surface of the denture. The reverse side of the cuvette is immediately placed on top of the mixed putty material. This ensured a seal and prevents leakage of the resin (cold cure) material during the duplication procedure. The second mix of the material is allowed to polymerize and the two halves of the cuvette are separated after 10 minutes. The denture is retrieved from the impression, cleaned and returned to the patient. (Fig.57.)

• The monomer and polymer of the resins are thoroughly mixed, then poured into the cuvette. When polymerized, the replica denture is retrieved from the mould and rough areas are trimmed and the denture is polished.
• The replica now is mounted to the articulator, for this step can also use facebow recording or the already existing bite register.

• Because the copied denture is completely pink, the teeth are needed to be removed and replaced with new denture teeth of approximately same mold. First the anterior then the new posterior teeth are placed. The denture base is festooned in preparation for the try-in. The trial denture is evaluated for stability (spatule probe), speech and esthetics and minor adjustments are done.

• New polymerization process is done, and the denture is inserted into the patient’s mouth. The denture instructions are given to the patient followed by a recall appointment.

The procedure is simple and requires less time and cost and the advantage of this technique is the patient acceptance of complete dentures.

Another technique is used if the goal is to create an implant template. In this method the steps of the first clinical appointment and laboratory work is the same as mentioned before, there is only a difference in the material of the duplicate: need to use clear acrylic. It provides the dentist and for the surgeon to choose the optimal place and position, and are able to manage the needed place for the control of the abutments.

**Figure 6.56. Figure 56. – Cuvette and the silicone used for copying method**

**Figure 6.57. Figure 57. – The unpolished surface of the denture is placed to silicone**

**17. 6.17. Neutral zone – Gyula Marada**

The eruption of the teeth in the oral cavity is influenced by the forces exerted by tongue, cheeks and lips. These muscular forces collectively determine the final dental arch form and position of the tooth in the oral cavity. This muscular environment continues throughout life, even after teeth have been lost and greatly influences this potential space. It is one of the major determining factors for any prosthesis that will be placed in the oral cavity to replace these missing teeth.

So the denture teeth should be arranged in the neutral zone, where during function the forces of the tongue pressing outward are neutralized by the forces of cheek and lips pressing inward. This potential space is known as neutral zone, which is bounded by the tongue medially, and the lips and cheeks laterally.
The success of any prosthesis depends on the proper position of the artificial teeth, and on creating a correct flange form and contour of the denture base within the neutral zone. This technique needs only some extra time during fabricating the denture, one possible method is using the acrylic base technique.

On this acrylic base, occlusion rims will help to establish the vertical dimension and centric relation. Two casts are needed for mounting the bases to articulator, and after the stone is set need to block the position of the occlusal rims with putty silicon material, and wax rim will be removed and acrylic rim will be replaced instead (with same height and width).

Once the patient is trained regarding the functional movements PVS light body is placed on the labial as well as lingual surfaces of the new rims and denture surfaces, they are placed in the mouth and patient is asked to perform the functional movements. This procedure is carried out for both the maxillary and mandibular arches. This records the polished surfaces of the denture according to the neutral zone. Need to remove from areas which are not covered with silicon, and to repeat the application of silicon until it will cover the whole surface.

Now block the rims again in the articulator, then have to remove the acrylic rims and the space between the matrices representing the neutral zone and indicating where the teeth should be positioned. After selection of the proper size, occlusal morphology and material of the teeth to be used, continue the positioning or arrangement of teeth.

The control of the trial denture is the same as the control by the regular method.

We recommend using the technique not only as a treatment of choice in atrophic mandible but also in patients with partial glossectomy, mandibular resections or motor nerve damage to the tongue which have led to either atypical movement or an unfavorable denture bearing area.

Since the forces are developed through muscles contraction during the various functions of chewing, swallowing and speaking, they vary in magnitude and direction in different individuals and in different periods of life, the neutral zone philosophy is also helpful by already existing older dentures as well.
Chapter 7. 7. Dental anatomy

1. 7.1. Bones of the chewing organ – Balazs Gaszner, Tibor Hollosy, Pal Toth

The osseous structures of mastication comprise three bones. The two maxillae that form the upper jaw, and the mandible (lower jaw). These bones accomodate the upper and lower teeth. The maxilla also contributes to the formation of the anterior, larger portion of the hard palate. The mandible, together with the temporal bone, forms the only large joint of the skull (the bilateral temporomandibular joint). Both bones contain all the vessels (arteries, veins and lymph vessels) and nerves that supply the teeth.

1.1. Maxilla

The maxilla is the largest paired bone of the viscerocranium. Specific junctions (plane sutures, a type of syndesmosis) connect the maxilla to most of the other bones of the viscerocranium. These are: the nasal, the frontal and the zygomatic bones on the outer aspect of the face, and the lacrimal and the ethmoidal bones, the inferior nasal concha, the palatine and the sphenoid bones internally. The maxilla also contributes to the wall of the orbit, the nasal and the oral cavities.

The major part of the maxilla is the body, from which the four processes originate: the frontal, zygomatic, alveolar and palatine. The largest paranasal sinus, the maxillary sinus, occupies the entire body.

Figure 7.1. Figure 1. – Left maxilla with the zygomatic bone and part of the sphenoid bone (anterior view). 1: anterior surface, 2: orbital surface, 3: frontal process, 4: alveolar process, 5: zygomatic process, a: anterior lacrimal crest, b: infraorbital foramen, c: nasal notch, d: infrazygomatic crest, e: canine fossa, f: alveolar yokes, M: medial, S: superior

The body of the maxilla is shaped like a three-sided pyramid that has four roughly triangular surfaces. On the anterior surface longitudinal ridges (alveolar yokes) are produced by the roots of the upper teeth. The root of the canine tooth produces the strongest and longest elevation, which is bordered by the canine fossa laterally. High on the anterior surface is the infra-orbital foramen, the anterior opening of the infra-orbital canal, transmitting

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infraorbital vessels and nerve. Delicate alveolar canals, arising from the infra-orbital canal, descend to the anterior upper teeth. These contain the superior anterior alveolar arteries, veins and nerves.

The orbital surface of the body produces the largest anterior part of the floor of the orbit. On it the infra-orbital groove extends anteriorly, from where alveolar canals pass to the middle upper teeth carrying the superior middle alveolar arteries, veins and nerves.

The infratemporal surface faces posteriorly. This surface is separated from the anterior surface by the strong infrazygomatic crest (also called zygomatico-alveolar or jugalcrest; these are not official terms, but widely used by dentists!). A round elevation on this surface is called the maxillary tuber, from where alveolar canals start by alveolar foramina. Through these canals the posterior superior alveolar arteries, veins and nerves reach the posterior upper teeth.

Figure 7.2. Figure 2. – Left maxilla with the zygomatic bone and part of the sphenoid bone (posterolateral view). 1: greater wing of sphenoid bone, 2: zygomatic bone, 3: maxillary tuber, 4: alveolar process of maxilla, 5: lateral plate of the pterygoid process (of sphenoid bone), A: anterior, S: superior

Part of the lateral wall of the nasal cavity is formed by the nasal surface of the body. This surface of the isolated maxilla is occupied mainly by the large maxillary hiatus, the wide gate of the maxillary sinus. This opening is narrowed considerably into the small, definite aperture by four neighbouring bones (see Chapter 9).
Figure 7.3. Figure 3. – Left maxilla with a part of the sphenoid bone (medial view). 1: frontal process, 2: palatine process, 3: alveolar process, 4: medial plate of the pterygoid process (of sphenoid bone), a: nasal notch, b: maxillary sinus, c: incisive canal, A: anterior, S: superior

The **frontal process** projects upwards, and unites with the frontal (*frontomaxillary suture*), the nasal (*nasomaxillary suture*), the lacrimal (*lacrimomaxillary suture*) and the ethmoidal (*ethmoidomaxillary suture*) bones. It forms the anterior boundary of the fossa for the lacrimal sac (*anterior lacrimal crest*).

The **zygomatic process** extends laterally to connect the maxilla with the zygomatic bone (*zygomaticomaxillary suture*).

The **alveolar process**, together with the same process of the contralateral maxilla (united in the *intermaxillary suture*), forms the horseshoe-shaped upper *alveolar arch*, which accommodates the upper teeth. The teeth occupy their socket (eight *dental alveoli*) separated by the *interalveolar septa*. In more posterior alveoli, individual spaces are formed by *intraradicular septa* for the multiple roots of the molar teeth.

Figure 7.4. Figure 4. – Left maxilla with the zygomatic bone and part of the sphenoid bone (inferior view). 1: zygomatic bone, 2: zygomatic process, 3: alveolar process, 4: palatine process, 5: pterygoid process (of the sphenoid bone), a: infrrazygomatic crest, b: incisive foramen, A: anterior, M: medial
Figure 7.5. Upper dental arch and the hard palate (inferior view). 1: palatine process of the maxilla, 2: alveolar process of the maxillae, 3: horizontal plate of the palatine bone, 4: pterygoid fossa (pterygoid process of the sphenoid bone), a: incisive foramen, b: median palatine suture, c: transverse palatine suture, A: anterior
The palatine process extends medially to form the anterior major part of the hard palate. The palatine processes of the two maxillae meet at the median palatine suture, while posteriorly they unite with the horizontal plates of the palatine bones, forming the transverse palatine suture. In the anterior part of the hard palate, the incisive canal starts at the incisive foramen which, by splitting, leads into both halves of the nasal cavity.

The two maxillae, together with the nasal bones, surround the piriform aperture, the anterior opening of the bony nasal cavity. The lateral borders of the opening are formed by the laterally convex anterior margins (nasal notches) of the bodies of maxillae. Inferiorly the aperture is bordered by the united alveolar processes, which form a sharp protrusion in the midline, the anterior nasal spine. The spine together with its posterior continuation, the nasal crest give support to the lower part of the cartilaginous nasal septum. The two nasal bones only contribute superiorly to the rim of the piriform aperture.

1.2. Mandible

The mandible is the biggest and toughest bone of the viscerocranium. It consists of a body and two rami, which meet at the two angles. The mandible is formed by the midline fusion of its two halves, which are joined at the mandibular (or mental) symphysis during fetal life.

Figure 7.6. Figure 6. – Mandible (anterolateral view). 1: body of the mandible, 2: ramus of the mandible, 3: angle of the mandible, a: mental foramen, b: oblique line, P: posterior, S: superior
The body of mandible is horseshoe-shaped, curved in the horizontal plane. The upper part carries the lower teeth (alveolar part) that occupy the 16 dental alveoli. The alveoli are separated by the interalveolar septa, and the roots of the teeth by the interradicular septa.

The bulky lower part of the body below the alveolar part is the base of the mandible. In the middle of its outer surface is the triangular mental protuberance that ends laterally at the two mental tubercles. Further lateral, approximately below the second premolar tooth, are the mental foramina, through which the mental artery, vein and nerve leave the mandibular canal. The oblique line is a stronger elevation that starts at the mental tubercle and rises backwards to continue into the anterior margin of the ramus of mandible. It is worth to mention that one of the most compact area of the mandible lies deep to that part of the oblique line that passes on the outer aspect of the alveolus accommodating the lower wisdom tooth. Therefore, the forced lateral bending of this tooth during extraction frequently results in the fracture of one or more of its roots. Behind the last dental alveolus, medial to the oblique line, a small, flat area is called the retromolar triangle, which is an important, palpable bony landmark used for the proper orientation of the injection anesthetizing the lower teeth.

**Figure 7.7.** Figure 7. – Mandible (anteror view). 1: alveolar process of the mandible, 2: base of the mandible, a: mental protuberance, b: mental tubercle, c: mental foramen, d: oblique line, S: superior
On the lower surface of the base, next to the midline, the two *digastric fossae* provide for the origin of the anterior bellies of the digastric muscles.

On the inner aspect of the base, close to the midline, sharp processes project posteriorly, the *mental* (or *genial*) spines. The genioglossus muscles originate from the two superior, and the geniohyoid muscles from the two inferior mental spines. Starting next to the spines, the two *mylohyoid lines* extend backwards and superiorly on both halves of the base. The main muscles (mylohyoid muscles) of the floor of the oral cavity arise from these lines. Therefore, structures lying above the mylohyoid lines are related to the oral cavity, but structures below these lines are related to the neck. The mylohyoid line separates two depressions. The smaller and shallow anterior one (the *sublingual fossa* for the sublingual gland) lies above the line, the wider and deeper posterior one (the *submandibular fossa* for the submandibular gland) is located inferior to it.

**Figure 7.8.** Figure 8. – Mandible (posteror view). 1: alveolar process of the mandible, 2: angle of the mandible, a: mental spine, b: sublingual fossa, c: digastric fossa, d: mylohyoid line, e: mylohyoide groove, f: submandibular fossa, g: retromolar trigangle, S: superior
The ramus joins the body of the mandible at the angle of mandible. The angle amounts to 125° in general, but is considerably larger before the teeth erupt, and also increases in elderly persons due to the loss of the teeth. The roughness on the outer aspect of the angle (masseteric tuberosity) serves as attachment for the masseter muscle. Similar roughness is seen on the inner aspect of the angle (pterygoid tuberosity), to which the medial pterygoid muscle and the stylomandibular ligament insert.

Figure 7.9. Figure 9. – Mandible (lateral view). 1: alveolar process of the mandible, 2: base of the mandible, 3: ramus of the mandible, a: mental tubercle, b: mental foramen, c: oblique line, d: coronoid process, e: mandibular notch, f: condylar process, g: masseteric tuberosity, A: anterior, S: superior

In the centre of the inner surface of the ramus of mandible the mandibular foramen is located, through which the inferior alveolar vessels and nerve enter the mandibular canal. From the inferior border of the mandibular foramen a fine groove, the mylohyoid groove starts. It passes anteriory and downwards below the mylohyoid line, within it runs the mylohyoid nerve. A fine, flat process extends upwards from the anteroinferior margin of
the foramen, the *lingula of the mandible*, which serves as insertion for the sphenomandibular ligament. Since the lingula indicates the position of the mandibular foramen, it is important to locate it precisely when anaesthetizing the lower teeth. The method of Szokolóczy helps to find the lingula: the lingula lies at the crossing point of the lines dividing both the length and the width of the ramus into two equal halves.

**Figure 7.10. Figure 10.** – Left half of the mandible (medial view). 1: ramus of the mandible, 2: alveolar process of the mandible, 3: base of the mandible, a: head of the mandible, b: mandibular notch, c: coronoid process, d: lingula of the mandible, e: mandibular foramen, f: retromolar triangle, g: pterygoid tuberosity, h: mylohyoid groove, i: mylohyoid line, A: anterior, S: superior

The *mandibular canal* descends close to vertical within the ramus. Reaching the body, it continues almost horizontally beyond the mental foramen till the midline, and establishes tiny connections with each of the inferior dental alveoli. Exceptionally, the mandibular canals of the two sides may communicate.

**Figure 7.11. Figure 11.** – Mandible (lateral view). 1: alveolar process of the mandible, 2: base of the mandible, 3: ramus of the mandible, a: dental alveolus, b: retromolar triangle, c: coronoid process, d: mandibular notch, e: pterygoid fovea, f: mental tubercle, g: mental foramen, h: interalveolar septum, i: interradicular septum, j: masseteric tuberosity, A: anterior, S: superior
Superiorly, the ramus divides into the anterior coronoid and the posterior condylar processes, which are separated by the mandibular notch. The flat, sharp and triangular coronoid process is the site of the insertion of the temporalis muscle. The condylar process starts with a neck, which supports the head of the mandible (condyle). Most of the head is covered by fibrous cartilage; it articulates with the temporal bone in the temporomandibular joint. A slight depression is seen on the anterior surface of the neck, the pterygoid fovea, into which the lateral pterygoid muscle inserts.

2.7.2. Temporomandibular joint – Balazs Gaszner, Tibor Hollosy, Pal Toth

The temporomandibular joint (TMJ) allows the mandible to move, relative to the rest of the skull*, during several important actions, such as food intake (sucking, biting, grinding, chewing), yawning, talking, deep breathing, etc. It is a synovial joint between the mandibular fossa above and the head of the mandible below. The joint surfaces are covered with avascular, fibrous tissue containing variable amount of chondrocytes (fibrous cartilage; bearing record to the desmocranial origin, as well as to the intramembranous ossification of both the temporal bone and the mandible). The joint cavity is completely divided into two compartments by an articular disc. The upper part is frequently referred to as discotemporal joint, and the lower one as discomandibular joint. Any movement of the mandible requires either homologous or alternate movements in both the left and the right TMJs, simultaneously.

Relations: The TMJ is covered by skin laterally, anteriorly it is directly related to the lateral pterygoid muscle, medi ally to the spine of the sphenoid bone and the foramen spinosum transmitting the middle meningeal artery, posteriorly to the parotid gland, the superficial temporal vessels, and the auriculotemporal nerve. Further to the back is the anterior wall of external acoustic meatus. Superiorly only a very thin bony plate separates the joint from the middle cranial fossa.

Figure 7.12. Figure 1. – Left temporomandibular joint (3D reconstruction, lateral view). Courtesy of the Diagnostic Center of Pécs. 1: zygomatic arch, 2: mandibular fossa, 3: head of the mandible, a: anterior, s: superior
2.1. Components of TMJ

2.1.1. Articular surfaces

2.1.1.1. Mandibular fossa

Figure 7.13. Figure 2. – Left temporal bone (lateral view). 1: zygomatic arch, 2: mandibular fossa, 3: articular tubercle, a: anterior, s: superior
The **mandibular fossa** is an oval depression in the inferior surface of the squamous part of the temporal bone at the root of its zygomatic process. It is somewhat narrower medially than laterally. The long axis of the fossa is almost transversely oriented and corresponds to that of the head of the mandible (see below). Anterior to the mandibular fossa lies the downward convex, transversely elongated **articular tubercle**. The fibrous cartilage covering the tubercle is continuous with the same tissue lining the mandibular fossa. The posterior boundary of the fossa is an almost vertical bony plate which, in high percentage of individuals, enlarges laterally as the **postglenoid tubercle**. Behind the tubercle, the **squamotympanic fissure** separates the squamous and tympanic parts of the temporal bone. Due to the presence of the **tegmental crest**, a thin anterior edge of the tegmen tympany belonging to the petrosal part of the temporal bone, medially the fissure splits into two. The anterior continuation is the **petrosquamous fissure**, immediately behind is the **petrotympanic fissure**. The petrosquamous fissure is a gap between the back side of the mandibular fossa and the middle cranial fossa, and is completely closed by dense fibrous tissue. Through the petrotympanic fissure the chorda tympani (branch of the facial nerve) exits from the tympanic cavity into the infratemporal fossa. Also in this fissure, the anterior ligament of the malleus of the middle ear inserts and gets continuous with the sphenomandibular ligament (see below) that extends downwards and anteriorly. This continuity is a good indication of the common developmental origin of the malleus and the mandible, both deriving from the 1st branchial (pharyngeal) arch.

**Figure 7.14.** Figure 3. – Left temporal bone (inferior view). 1: squamotympanic fissure, 2: petrosquamous fissure, 3: petrotympanic fissure, arrow: tegmental crest, *: mandibular fossa, a: anterior, m: medial
2.1.1.2. Head of the mandible

Figure 7.15. Figure 4. – Mandible (superior view). 1: head of the mandible, 2: neck of the mandible, 3: pterygoid fovea, p: posterior
Figure 7.16. Figure 5. – Left half of the mandible (inner view). 1: head of the mandible, 2: neck of the mandible, 3: pterygoid fovea, p: posterior, s: superior
The head of the mandible fits into the socket formed by the mandibular fossa, but it does not exactly conform to the shape of the latter. Viewed from above, it can be simplified as being oval in outline. The head has a shorter sagittal dimension (about 10 mm) and a bigger transverse dimension (about 20 mm). Even though the lateral aspect of the head is the main load-bearing area, it is slightly narrower than the medial. The long axis of the head medially tends to the back, thus the extended long axes of the two heads cross each other at an obtuse angle of 145-150° just in front of the foramen magnum. The convex superior and anterior surfaces of the head articulate with the mandibular fossa, therefore only these are covered with fibrous cartilage. A thin neck connects the head to the ramus. On the anterior aspect of the neck, a small depression, the pterygoid fovea lies, to which the inferior head of the lateral pterygoid muscle attaches.

Figure 7.17. Figure 6. – Horizontal MR image at the level of the heads of the mandible. Courtesy of the Diagnostic Center of Pécs. 1: maxillary sinus, 2: head of the mandible, 3: external acoustic meatus, a: anterior
2.1.2. Articular disc

The articular disc is an S-shaped fibrocartilaginous plate located between the articular surfaces, the posterior two thirds of it being convex upwards. It has weak blood supply and innervation in most of the peripheral areas, except its lateral aspect, where the load-bearing is maximal. The disc is of variable thickness, being thickest in its posterior part (approximately 3 mm). It tapers centrally, where it may possess a hole (then we call it a meniscus). With the mouth closed, the articular surface of the head of the mandible lies against the thinnest central part of the disc, and faces to the posterior surface of the articular tubercle.

The articular disc has three bands: anterior, intermediate and posterior. In the thinnest intermediate zone, the collagen fibre bundles are oriented mostly sagittally, while in the anterior and posterior bands they run both sagittally and transversely.

Figure 7.18. Figure 7. – Sagittal section through the left temporomandibular joint (lateral view). 1: articular tubercle, 2: mandibular fossa, 3: head of the mandible, 4: joint cartilage, 5a: fibrocartilagenous part of the articular disc, 5b: bilaminar zone of the articular disc, 6: joint capsule, 7: external acoustic meatus, 8: styloid process, a: anterior, s: superior
Peripherally, the disc merges with the joint capsule through which it has fibrous attachments. Anteriorly, the insertion of these fibrous bands corresponds to that of joint capsule (anterior margin of the articular tubercle above and the anterior margin of the articular surface of the head of the mandible below). Medially and laterally, strengthened triangular zones of the joint capsule anchor the disc to the sides of the head just below its articular surface. Posteriorly, the disc is attached to the capsule by the bilaminar zone at which the disc splits into two laminae, superior and inferior. The superior lamina is rich in elastic fibres and, through the capsule, it anchors the disc to the posterior margin of the mandibular fossa. The inferior lamina contains mostly collagen fibres and, again through the capsule, it inserts to the posterior margin of the articular surface of the head of the mandible. The triangular space between the two laminae and the joint capsule contains the retrodiscal pad, a highly vascularized tissue made of collagen and elastic fibres, fat, vessels and nerves. The bilaminar zone is the most distortable part of the disc, in it a relatively large venous plexus fills with blood when the mouth is opened.

The specific shape, structure and attachments of the disc may act as a self-centering automatism, which maintains its correct relation to the articular surface of the head of the mandible during movements in the TMJ.

2.1.3. Joint capsule

The fibrous capsule is thin and loose allowing high freedom of movement of the TMJ, but giving weak support for it. Embedded into this layer there are various receptors, including nociceptors. The capsule completely encloses both articular surfaces of the joint. Superiorly, it comes from the common rim of the articular tubercle and the mandibular fossa, including the tegmental crest and the squamotympanic fissure behind, but leaving the petrotympanic fissure free (!). It attaches inferiorly to the margin of the articular surface of the head of the mandible. Anteriorly, the superior head of the lateral pterygoid muscle inserts into the capsule (some muscle fibres may pass through it to attach directly to the anterior border of the disc). The lateral (temporomandibular)
ligament strengthens the capsule on its outer surface. (fibres both on the lateral and the medial aspects of the capsule, originating from the outer and the inner margins of the articular disc, and attaching to both sides of the head of the mandible are sometimes referred to as lateral and medial collateral ligaments, respectively.) The posterior part of the capsule is associated with the bilaminar zone of the articular disc.

The synovial membrane lines the inner surface of the fibrous capsule and the peripheral part of the disc. This layer secretes the synovial fluid, which lubricates the joint and provides the important metabolites to the articular cartilages.

2.1.4. Ligaments

Figure 7.19. Figure 8. – Left temporomandibular joint (lateral view). 1: articular tubercle, 2: lateral (temporomandibular) ligament, 3: joint capsule, 4: external acoustic meatus, 5: mastoid process, 6: stylomandibular ligament, 7: mandibular foramen, 8: sphenomandibular ligament, a: anterior, s: superior

2.1.4.1. Lateral (temporomandibular) ligament
Along the outer aspect of the TMJ the joint capsule is toughened by the lateral (temporomandibular) ligament, the only proper ligament of the joint. It is composed of two separate bands:

The outer oblique part originates from the lateral surface of the articular tubercle, descends posteriorly and attaches to the lateral side of the neck of the mandible thus limiting the opening of the mouth.

The inner horizontal part is weaker, comes together with the outer oblique part and inserts to the lateral sides of both the disc and the head of the mandible thus limiting posterior movement of both.

2.1.4.2. Accessory ligaments

The accessory ligaments of the TMJ are the sphenomandibular and the stylomandibular ligaments, out of which only the sphenomandibular ligament is likely to have any biomechanical significance.

The sphenomandibular ligament is the remnant of the perichondrium of the Meckel’s cartilage, the core of the 1st pharyngeal arch. It originates from the spine of the greater wing of the sphenoid bone and the rim of the petrotympanic fissure, and attaches to the lingula of the mandible. The ligament becomes tense when the mouth is opened and, by keeping a fairly constant distance between the oval foramen of sphen bone and the mandibular foramen, prevents stretching of the inferior alveolar nerve.

The stylomandibular ligament is just a thickened band of the deep cervical fascia, which extends from the lower third of the styloid process of the temporal bone to the posterior margin of the angle of the mandible. It limits protrusion in the TMJ.

Though not true ligaments, some connective tissue elements have also been described as ligaments. One of them is the pterygomandibular raphe. It serves as origin for the buccinator muscle anteriorly and the buccopharyngeal part of the superior constrictor muscle of pharynx posteriorly. Being extended between the hamulus of the medial plate of the pterygoid process of the sphenoid bone and the posterior end of the mylohyoid line of the mandible, it helps the function of the sphenomandibular ligament. The other, the retinacular ligament, is a connective tissue band connecting the articular tubercle with a strengthened part of the masseteric fascia near the angle of the mandible. A commitant vein is associated with this ligament, which is likely to have connection with the veins in the retrodiscal pad. In spite of barely having any mechanical function, it may facilitate venous drainage from the TMJ during mastication.

2.2. Movements at TMJ

Although morphologically hinge, functionally the TMJ is a ginglymoarthrodial (combination of hinge and sliding) joint, which acts in unison with the contralateral joint. The movements of the mandible are controlled more by muscles than by either the shape of the articular surfaces or the ligaments. Movements in the TMJ are combinations of the following chief components.

2.2.1. Rotational movement (depression and elevation, opening and closing the mouth)

Figure 7.20. Figure 9. – Opening the mouth (left lateral view)
During the initial phase of the jaw opening (depression), the head of the mandible rotates forward around its own axis. This phase lasts till the distance between the upper and lower incisor teeth does not exceed 20 mm (till 15 degrees of opening). Closing the mouth (elevation of the mandible or occlusion) requires backward rotation of the head of the mandible. During elevation and depression, the TMJ acts as a hinge joint. These rotational movements occur in the lower compartment of the TMJ, i.e. in the discomandibular joint.

2.2.2. Translational movement of TMJ

Figure 7.21. Figure 10. – Protrusion and retrusion of the mandible (superior view)

The strict forward (protrusion) and backward (retrusion) movements, during which the base of the mandible moves in a horizontal plane, are limited only to 1-2 mm. During further protrusion, the head of the mandible, together with the articular disc, glides downward and forward on the posterior slope of the articular tubercle (forward translation). This movement is needed for opening the mouth wide (beyond 15 degrees; the distance between the upper and lower incisor teeth exceeding 20 mm). Closing the mouth requires backward sliding of the head of the mandible and the disk into the mandibular fossa (backward translation), the sliding of the disc is mostly helped by elastic fibres in the superior lamina of the bilaminar zone. The axis of the translational movements can be determined as a transverse line connecting the left and right mandibular foramina. This
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allows for a low-tension environment for the vessels and nerves entering the mandibular canal. Translational movements occur in the upper compartment of the TMJ, i.e. in the discotemporal joint. During these movements, the TMJ acts as a sliding joint.

2.2.3. Grinding or lateral movements (side to side excursion)

Figure 7.22. Figure 11. – Grinding movement of the temporomandibular joint (superior view)

During grinding (chewing), the mandible moves around a vertical axis, which can be located anywhere along a line connecting the two heads of the mandible. The side toward which the mental protuberance moves is referred to as either the rotating or working (= resting) side, while the other side is referred to as either the orbiting or balancing (= swinging) side. The first terms precisely define the sides by the movements of the respective head of the mandible.

When the mandible is moved into a lateral excursion, the head on the rotating side only performs rotation, while the head on the orbiting side performs forward, downward and inward translation. The rotating and orbiting sides dynamically change. In a quite rare phase of grinding, in which the vertical axis is positioned at the middle of the line connecting the heads of the mandible, protraction in one TMJ equals in extent to the retraction in the other joint, and the other way around. The angle of lateral excursion of the mandible is called the angle of Bennett, which gives the lateral movement of the mental trigone measured in degrees. The TMJs also perform rotational movement during functional grinding, when biting and smashing of the food is also done by the teeth.

2.3. Vascularization and innervation

2.3.1. Blood supply
The initial part of the superficial temporal artery, one of the terminal branches of the external carotid artery, is located behind the neck of the mandible, where it gives small branches to the TMJ. From medial and behind, tiny branches of the deep auricular and the anterior tympanic arteries, both originating from the maxillary artery, supply the joint. The veins from the TMJ are collected by the superficial temporal and the maxillary veins, that join to form the retromandibular vein. The latter, through the common facial vein, drains to the internal jugular vein.

### 2.3.2. Sensory innervation

Since the TMJ develops from the first pharyngeal arch, branches of the first branchial (pharyngeal) nerve, the trigeminal nerve supply the joint. The auriculotemporal nerve, coming from the sensory division of the mandibular nerve, after embracing the middle meningeal artery, turns posteriorly between the neck of the mandible and the sphenomandibular ligament, and then laterally behind the neck of the mandible, innervates most of the capsule. Small sensory twigs emerging from the masseteric and the deep temporal nerves, branches of the motor division of the mandibular nerve, aid the innervation of the anterior and lateral parts of the capsule.

### 2.4. Clinical correlates

Circa one quarter of the population – mostly women - suffers from TMJ dysfunction of variable severity. The most common causes are degenerative joint diseases, trauma, infections, displacement of the articular disc, and bruxism.

#### 2.4.1. Arthritis and ankylosis

Degenerative joint disease refers to both arthritis and arthrosis. Osteoarthritis is a mechanical abnormality due to degradation of articular cartilage and underlying bone. The causes may be hereditary, developmental, metabolic, and mechanical (trauma, occlusal dysharmony), but no preceding inflammation. Autoimmune disease (rheumatoid arthritis) can also affect the TMJs. The arthrosis is usually a consequence of a mild inflammation. Degenerative joint diseases may lead to ankylosis, a disorder characterized by deformities of joint surfaces, limitation of mandibular movements, and pain.

#### 2.4.2. Trauma

The most common traumatic injury is the fracture of the mandible, that can be a result of blunt force applied to the chin (a punch), motor vehicle collision, and objects flying with high velocity (golf balls and bullets). It is also a rare complication of lower third molar extraction. Other, more severe injuries can be either caused by or coupled with the fracture of the mandible. Fractures, such as a midline fracture or bilateral fracture, may impair the function of genioglossus muscle, and the relapsed root of the tongue may block the airway. Far displaced pieces of the broken mandible may cause severe bleeding from the internal jugular vein or from one of the carotid arteries.

The force that breaks the jaw can be great enough to cause intra-cranial injury. If the head of the mandible is forced to move up, it can easily rupture the thin bony plate separating the mandibular fossa from the middle cranial fossa, and branches of the middle meningeal artery can be injured resulting in epidural bleeding. If the head of the mandible is forced to move backward, it can destroy the thin anterior wall of the external acoustic meatus or the tympanic cavity, that may result in the impairment of hearing.

#### 2.4.3. TMJ dislocation

The TMJ is the only joint that can be dislocated without the action of an external force. Dislocation of the mandible is almost always bilateral, and it happens when the mouth is open wide (yawning). The displacement is anterior, the head of the mandible is located on the anterior slope of the articular tubercle. Replacement is accomplished by depressing the back of the jaw with two thumbs positioned at the retromolar trigones and elevating the chin simultaneously.

#### 2.4.4. Bruxism

Bruxism is an unconscious activity of TMJs that involves excessive clenching and grinding of the teeth. It can occur during sleep or whilst awake. The relationship of bruxism with TMJ dysfunction is disputable.
3.7.3. Muscles of facial expression and muscles of mastication – Balazs Gaszner, Tibor Hollosy, Pal Toth

The muscles of facial expression are located around the openings of the face (the orbit, nose, ear and mouth) and most of them insert to the skin. These muscles derive from the second branchial (pharyngeal) arch, and therefore are innervated by the branches of the facial nerve (cranial nerve VII, the 2nd branchial nerve).

3.1. Muscles of facial expression

3.1.1. Muscles around the orbit (orbital group)

The largest and the most important in this group is the orbicularis oculi muscle, which protects the eye. Its orbital part closes the eye forcibly, the palpebral part gently closes the eyelids during blinking, and the lacrimal part assists the drainage of tears. The frontal belly of the epicranius (occipitofrontalis) muscle lies beneath the skin of the forehead, it elevates the eyebrow and produces transverse wrinkles. Deep to these two muscles are the corrugator supercilii and the depressor supercilii muscles. The former draws the eyebrow medially, the latter pulls it down.

Figure 7.23. 1. ábra

3.1.2. Muscles around the nose (nasal group)

The nasalis muscle is a thin muscular lamina, the muscles of the two sides cover the back of the nose like a tent. Some of its fibres dilate, others contract the nostril (forced nasal breathing). The levator labii superrioris alaeque nasi muscle also dilates the nostril, thereby producing one of the visible signs of hypoxia. High on the nose, close to the midline is the procerus muscle, which pulls the skin on the bridge of the nose into wrinkles (paying strong attention). The depressor septi nasi muscle inserts to the nasal septum from below and pulls the nose inferiorly.

Figure 7.24. 2. ábra
3.1.3. Muscles around the ear (auricular group)

The auricular muscles are responsible for the precise orientation and shaping of the ear in many animals, but are rudimentary and non-significant in humans. These muscles have no medical importance, and only few individuals can contract them voluntarily. According to their position, the anterior, superior and posterior auricularis muscles can pull the whole auricle in different directions.

3.1.4. Muscles around the mouth (oral group)

The orbicularis oris muscle is a ring-like muscle that occupies the central part of the lips, and has no bony attachment. The muscle determines the shape of the lip, the size and shape of rima oris, therefore plays an important role in food intake and speech. The radially arranged other muscles of the group mostly insert to the orbicularis oris muscle.

Two muscles belong to the upper lip: The levator labii superioris muscle originates from the maxilla, lateral to it the zygomaticus minor descends from the zygomatic bone. Both muscles elevate the lip.

Five muscles insert to the angle of the mouth: (1) The levator anguli oris muscle is completely covered by the former two muscles, and it raises the angle of the mouth. Three muscles are superficial: (2) The zygomaticus major muscle originates from the zygomatic arch, it elevates the angle of the mouth and pulls it laterally. (3) The risorius muscle arrives from lateral. When smiling, this muscle produces a small depression on the skin next to the oral opening. Also superficial is the triangular (4) depressor anguli oris muscle, which lowers the angle of the mouth. The main muscle of the cheek is (5) the buccinator muscle, which originates from the pterygomandibular raphe anteriorly. Most of its central fibres decussate before inserting into the angle of the mouth, some fibres proceed forward and intermingle with the orbicularis oris muscle in the lips. In spite of belonging to the muscles of facial expression, the main function of the buccinator muscle, working together with the tongue, is to keep the bolus between the masticatory surfaces of the teeth during mastication. It also helps to forcibly blow air, and to create vacuum for sucking. The duct of the parotid gland pierces the buccinator muscle before opening to the oral vestibule (see Chapter 8).

Two muscles belong to the lower lip: The depressor labii inferioris muscle is partly covered by the depressor anguli oris muscle, it ascends medially and pulls the lip down. The deepest is the mentalis muscle, which protrudes the lip.

Figure 7.25. 3. ábra
Figure 7.26. 4. ábra

Figure 7.27. 5. ábra
3.2. Muscles of mastication

The very synchronous and precise action of all these muscles moving the mandible results in the fine frittering and grinding of the bolus. All develop from the first branchial (pharyngeal) arch, and therefore are innervated by muscular branches of the anterior (motor) division (masticatory nerve) of the mandibular nerve (cranial nerve V/3, the 1st branchial nerve). The arteries supplying these muscles arise from the maxillary artery.

3.2.1. Masseter muscle

The masseter muscle is quadrangular, and consists of two overlapping heads. The superficial head originates from the inferior margin of the body of the zygomatic bone and the anterior two-thirds of the lower border of the zygomatic arch. Its fibres pass inferiorly and backwards to insert into the lateral surface of the angle of the mandible (masseteric tuberosity) and into the inferior-posterior part of the lateral surface of the ramus of the
mandible. The much smaller deep head arises from the posterior third of the lower border of the zygomatic arch and from the inner surface of it. The fibres of the deep head pass more vertically down, and insert to the upper half of the lateral surface of the ramus of the mandible and to the outer aspect of the coronoid process. Inside, the muscle has numerous septa that increase the surface for muscle fiber origin. This multipennate arrangement provides for greater power of contraction. The masseter muscle elevates the mandible, thereby closes the mouth. It is particularly active when grinding tough food. Due to the oblique fiber direction of the different parts of the muscle, it can – in a lesser extent – also contribute to the protraction, retraction and the lateral movements of the mandible.

The masseteric branch of the masticatory nerve innervates the muscle, which arrives through the mandibular notch together with the masseteric artery.

**Figure 7.29. 7. ábra**

![Image of temporalis and masseter muscles](image)

### 3.2.2. Temporalis muscle

The fan-like temporalis muscle is the largest muscle of mastication. It widely originates from the entire temporal fossa along the inferior temporal line and from the overlying temporal fascia. The fibres of the temporalis muscle converge considerably towards their inserting tendon, thus the muscle is considered as a bipennate muscle. The anterior fibres descend vertically, the posterior fibres pass horizontally, all of them running beneath the zygomatic arch. The strong common tendon inserts to the tip, the anterior and posterior margins and the inner surface of the coronoid process of the mandible, extending down on the anterior margin of the ramus of the mandible almost till the 3rd molar tooth. The anterior part of the muscle elevates the mandible (closes the mouth), the posterior part pulls the protruded mandible backwards.

The temporalis muscle receives its innervation from the deep temporal branches of the masticatory nerve, which ascend on the deep surface of the muscle together with the deep temporal arteries.

**Figure 7.30. 8. ábra**
3.2.3. Medial pterygoid muscle

The shape of the medial pterygoid muscle is quadrangular, and it consist of two heads. The smaller *superficial head* originates from the tuber of the maxilla and from the adjacent part of the palatine bone (pyramidal process). The bulkier *deep head* arises from the inner surface of the lateral plate of the pterygoid process of the sphenoid bone. Many tendineous septa provide the muscle a multipennate arrangement of fibres (see masseter muscle above). The fibres of both heads descend backwards and laterally (like the fibres of the masseteric muscle), and insert into the rough area on the inferior and posterior part of the inner surface of the angle of the mandible (pterygoid tuberosity). The main function of the muscle is to elevate the mandible but it also aids protrusion and grinding movement. The medial pterygoid muscle, together with the masseter muscle, forms a muscular stirrup that suspends the mandible.

The medial pterygoid muscle is innervated by a muscular branch of the masticatory nerve, and receives blood supply through direct muscular branches of the maxillary artery.

Figure 7.31. 9. ábra
3.2.4. Lateral pterygoid muscle

The fibres of the lateral pterygoid muscle are aligned horizontally. This muscle consists of two heads. The smaller *superior head* takes origin from the infratemporal surface of the greater wing of the sphenoid bone, including the infratemporal crest, and most of its fibres attach to the capsule and to the articular disc of the temporomandibular joint. The *inferior head* arises from the outer surface of the lateral plate of the pterygoid process, and insert into the pterygoid fovea on the anterior surface of the neck of the mandible.

The lateral pterygoid muscle pulls the neck of the mandible anteriorly. If the muscles of both sides simultaneously contract, the mandible is protruded. Protrusion bigger than 1-2 mm is needed for the opening the mouth, this latter movement is assisted by the geniohyoid and the digastric muscles. The unilateral contraction of the muscle moves the mandible towards the opposite side (lateral excursion). Alternate contraction of the lateral pterygoid muscle with the medial pterygoid muscle of the opposite side plays an important role in grinding movement.

The lateral pterygoid nerve of the masticatory nerve reaches the muscle on its medial side. The direct muscular branches arise from the maxillary artery, which passes immediately below the muscle, occasionally between its two heads.

Figure 7.32. 10. ábra

3.3. Other muscles functionally associated with mastication

Additional muscles play an essential role in frittering of the food. In spite of being a muscle of facial expression, the primary function of the *buccinator muscle* is to give tension to the cheek against the food, thereby keeping the bolus between the upper and lower rows of the teeth during mastication. This action is greatly assisted by the *muscles of the tongue*. The muscles of oral diaphragm (*mylohyoid muscles*) and the suprahyoid muscles (*geniohyoid and digastric muscles*) are essential for opening the mouth; the effective contraction of these muscles is supported by the infrahyoid muscles (*sternohyoid, thyrohyoid, strenothyroid and omohyoid muscles*), which pull the hyoid bone down.

4. 7.4. Vascular and nerve supply to the head and neck – Balazs Gaszner, Tibor Hollosy, Pal Toth

4.1. Arterial supply to the head and neck

Two main systems are responsible for the arterial supply to the head and neck. The head is mostly supplied by the branches of the *common carotid artery*, which on the left side arises from the brachiocephalic trunk, and on the right side is a direct branch of the aortic arch. The common carotid artery bifurcates at the level of the 4th cervical vertebra (upper margin of the lamina of the thyroid cartilage). One of its branches, the internal carotid artery, enters the neurocranium, and supplies mostly intracranial organs (not to be discussed here in detail). It is relevant to mention the first intracranial branch of the internal carotid artery, the *ophthalmic artery*, which gives rise to small branches supplying part of the walls of the nasal cavity and some paranasal sinuses (*anterior and
posterior ethmoidal arteries), as well as the frontal region (supraorbital and supratrochlear arteries). The face and its cavities, as well as most of the cervical organs receive blood from the branches of the external carotid artery. These branches are discussed below.

The other important artery supplying the head and neck is the subclavian artery.

**Figure 7.33. 1. ábra**

4.1.1. Branches of the external carotid artery

The external carotid artery arises from the common carotid artery at the upper margin of thyroid cartilage. It leaves anteriorly the carotid sheath covering the main vessels of the neck, ascends till the base of the mandible where it enters the parotid gland, and split into endbranches within the gland. The most important endbranch for dental students and professionals is the maxillary artery, the detailed description of which is given below.

4.1.1.1. Superior thyroid artery

The superior thyroid artery arises anteriorly from the initial part of the external carotid artery, and sharply turns down to reach the upper pole of the ipsilateral lobe of the thyroid gland. A branch of it (sometimes directly coming from the external carotid artery), the superior laryngeal artery, contributes to the blood supply to the larynx, and other small branches reach the sternocleidomastoid muscle.

4.1.1.2. Lingual artery

The lingual artery is the second anteriorly directed branch of the external carotid artery arising at the level of the hyoid bone. It may originate together with the facial artery. Passing on the medial side of the hyoglossus muscle, it gives a branch into the sublingual region (sublingual artery) to bring supply to the sublingual gland. After turning medially and superiorly, the lingual artery pierces into the muscles of the tongue, where it splits into two main branches, the dorsal lingual artery and the deep lingual artery. Small branches of the dorsal lingual artery turn backwards, and contribute to the blood supply to the palatine tonsil. Knowledge of the presence of anastomoses between the left and right lingual arteries is important when staunching bleeding of the tongue.

**Figure 7.34. 2. ábra**
4.1.1.3. Facial artery

The facial artery originates anteriorly, slightly above the lingual artery, and crosses deep to the posterior belly of digastric and the stylohyoid muscles. In the submandibular trigangle, it gives the ascending palatine artery to supply the soft palate, and the submental artery to the floor of the oral cavity. It passes on the medial and superior surfaces of the submandibular gland to which it gives glandular branches, and enters the face just in front of the insertion of the masseter muscle. At this point, the facial artery is palpable, and can be compressed against the base of the mandible in case of severe bleeding. While proceeding towards the angle of the mouth, deep to the muscles of the lower lip, it gives rise to the inferior labial artery. After making a turn at obtuse angle, it gives supply to the upper lip (superior labial artery) and continues towards the medial angle of the eye. Small branches are turning to the back of the nose, and the artery terminates as angular artery, which anatomose with the branches of the ophthalmic artery (from the internal carotid artery). The course of the facial artery on the face is rather tortuous, allowing wide opening of the mouth.

Figure 7.35. 3. ábra
4.1.1.4. Ascending pharyngeal artery

This artery is the smallest branch of the external carotid artery, arising posteriorly and medially near the bifurcation of the common carotid artery. It passes superiorly between the lateral wall of the pharynx and the carotid sheath, and gives fine branches to the palatine tonsil, the auditory tube and the tympanic cavity. Its endbranch continues through the foramen magnum to supply the dura mater lining the posterior cranial fossa (*posterior meningeal artery*).

4.1.1.5. Occipital artery

The occipital artery originates posteriorly from the external carotid artery within the carotid triangle. It ascends along the medial-inferior margin of the posterior belly of the digastric muscle, and turns to the occipital region to reach the scalp. It contributes to the blood supply to the dura mater, the skull and the occipital muscles. Via fine anastomoses, it communicates with the branches of the superficial temporal artery.

4.1.1.6. Posterior auricular artery

The posterior auricular artery is a small branch that passes below the parotid gland and turns up behind the ear. It gives small twigs to supply the parotid gland, the muscles originating from the mastoid and styloid processes, the external ear and the tympanic cavity.

4.1.1.7. Superficial temporal artery

This artery is one of the two terminal branches of the external carotid artery. It begins behind the neck of the mandible within the upper portion of the parotid gland, and ascends in front of the ear. At this point the artery is palpable, and can be compressed against the root of the zygomatic arch to staunch bleeding. The *transverse facial artery* emerges anteriorly from it, passes horizontally below the zygomatic arch, and anastomoses with the infraorbital artery (from maxillary artery). The frontal branch turns towards the frontal region, and anastomoses with the supraorbital artery (a branch of ophthalmic artery originating from the internal carotid artery). The parietal branch turns backwards, and establishes anastomotic connections with the occipital and the posterior auricular arteries.

4.1.1.8. Maxillary artery
The maxillary artery is the other terminal branch of the external carotid artery, and is of great significance for dental students and professionals. For practical reasons, this artery is subdivided into three portions.

**Figure 7.36. 4. ábra**

![Diagram of the maxillary artery](image)

**a.) Retromandibular(or mandibular) part**

It begins posterior to the neck of the mandible, and turns anteriorly between the ramus of the mandible and the sphenomandibular ligament. The superiorly originating branches are the *deep auricular artery* contributing to the supply of the temporomandibular joint, the *anterior tympanic artery* accompanying chorda tympanica back to the middle ear, and the *middle meningeal artery* entering the middle cranial fossa through foramen spinosum. Bleeding from the latter may result in epidural haematoma. The inferiorly originating most important branch is the *inferior alveolar artery*. Before entering the mandibular foramen, it gives the *mylohyoid branch (or artery)* which turns onto the inferior surface of the mylohyoid muscle. While passing along the mandibular canal, it gives rise to branches supplying either the lower teeth (*dental branches*), or the alveolar bone, the periodontium and the gingiva (*peridental branches*). The preterminal branch, the mental artery, emerges from the mental foramen, and contributes to the supply of the lower lip and the chin. Inferior to the chin, this branch anastomoses with the submental artery (from the facial artery).

**b.) Intermuscular (or pterygoid) part**

Most vessels giving supply to the muscles of mastication and to the cheek originate from this segment. The *masseteric artery* reaches the deep surface of the masseter muscle through the mandibular notch. Short branches are directed towards the lateral and medial pterygoid muscles (*pterygoid branches*). The *anterior and posterior deep temporal arteries* pass between the skull and the temporalis muscle, and give supply to the entire muscle. The initial part of them anastomoses with the middle meningeal artery. The *buccal artery* turns obliquely anteriorly and inferiorly between the inserting part of the temporalis muscle and the medial pterygoid muscle. It reaches the outer surface of the buccinator muscle, which it supplies, together with the lining mucous membrane of the cheek. This artery has excessive anastomoses with the branches of the facial artery.

**c.) Pterygopalatine part**

The vessels originating from the maxillary artery above the tuber of maxilla and within the pterygopalatine fossa give supply to the upper teeth and the cavities of the viscerocranium. The *superior posterior alveolar arteries* descend to enter the maxilla through the alveolar foramina on the tuber of maxilla. These vessels supply the upper molar teeth, and give fine branches to the alveolar bone, the periodontium and the gingiva.
The *infraorbital artery* enters the orbit through the inferior orbital fissure, passes within the infraorbital sulcus, and through the infraorbital canal reaches the infraorbital foramen, where it becomes superficial. Branches to the upper teeth arise either from the part lying in the sulcus (*superior middle alveolar arteries, to the premolars*) or the part passing within the canal (*superior anterior alveolar arteries, to the canine and the incisive teeth*). These arteries reach the teeth through delicate canaliculi, and also supply the alveolar bone, the periodontium and the gingiva.

The *descending palatine artery*, after giving branches to the pharynx, splits into two. The *greater palatine artery* enters the oral cavity through the greater palatine canal. While turning anteriorly, it supplies the hard palate till the incisive teeth, and anastomoses with the terminal branch of the sphenopalatine artery coming from the nasal cavity through the incisive foramen. The *lesser palatine arteries* descend to the oral cavity through the lesser palatine canals, and give supply to the soft palate and the palatine tonsil.

The *sphenopalatine artery* enters the nasal cavity through the sphenopalatine foramen, and splits into a number of *posterior nasal arteries*. These branches provide blood to the mucous lining of the posterior-inferior part of the nasal cavity, both on the septum and on the lateral wall, and also contribute to the supply of the sphenoidal and ethmoidal sinuses. The longest of them, the *nasopalatine artery* continues into the oral cavity through the incisive foramen (see above).

The *artery of the pterygoid canal* help to supply the auditory tube and the epipharynx.

### 4.1.2. Branches of the subclavian artery

The right subclavian artery arises from the brachiocephalic trunk, the left is the third direct branch of the aortic arch. It is subdivided into three segments, all of which contribute to the arterial supply to the head and neck.

#### 4.1.2.1. Thoracic part

This segment lasts till the medial border of the anterior scalenus muscle. Its first branch is the *vertebral artery*, which enters the transverse foramen of the 6th cervical vertebra. After ascending through the transverse foramina of vertebrae C6-C1, the artery turns posteriorly on top of the posterior arch of the atlas, pierces the posterior atlantooccipital membrane, and enters the posterior cranial fossa through the foramen magnum. It mostly provides blood to the brain and the spinal cord.

The short *thyrocervical trunk* has several branches. The *inferior thyroid artery* ascends to the thyroid gland, but also contributes to the supply to the larynx (inferior laryngeal artery), the pharynx and the cervical part of the esophagus. Its crossing with the inferior laryngeal nerve is of clinical importance, since the common ligation then transection of the artery and the nerve will impair the function of the laryngeal muscles. The *ascending cervical artery* provides blood to the deep muscles of the neck and the spinal cord. The *suprascapular artery* contributes to the supply to the shoulder region, and anastomoses with the circumflex scapular artery, providing the possibility of collateral circulation in case of constriction or obliteration either of the distal part of the subclavian artery or of the proximal part of the axillary artery. The *superficial cervical artery* commonly arises from the thyrocervical trunk, and it supplies the anterior scalenus muscle and the trunks of the brachial plexus. The *transverse cervical artery* has variable origin (see below). It commonly comes from the third segment of the subclavian artery, but in about the third of the cases it originates from the thyrocervical trunk.

The *internal thoracic artery* turns into the thorax behind the sternum.

It contributes to the supply to the anterior thoracic wall (anterior intercostal arteries), the diaphragm (musculophrenic artery), the pericardium (pericardiacophrenic artery), the mediastinum (mediastinal branches) and the anterior abdominal wall (superior epigastric artery. It is frequently used as graft in coronary bypass operations.

#### 4.1.2.2. Muscular part

This is the portion of the subclavian artery which passes between the anterior and middle scalenus muscles, immediately on top of the clavicle (scalenus hiatus). Its only branch is the *costocervical trunk*, which splits into two. The *deep cervical artery* supplies the deep muscles of the neck, the *highest thoracic artery* turns into the first intercostal space (gives no branch to the neck).

#### 4.1.2.3. Cervical part
This segment is located between the lateral border of the middle scalenus muscle till the clavicle. It gives rise to the dorsal scapular artery supplying the muscles of the back. This artery most frequently arises together with the superficial cervical artery as transverse cervical artery, which may be the branch of the thyrocervical trunk (see above).

After crossing the clavicle, the subclavian artery continues as axillary artery.

### 4.2. Venous drainage of the head and neck

Two main veins drain the blood from the head and neck, which are highly variable. Most of the blood collected by the internal jugular vein arrives from the dural sinuses of the neurocranium. The description of these sinuses is omitted here for the sake of simplicity, only the extracranial roots are mentioned. The external jugular vein drains the blood from most of the structures of the neck and the occipital region. (The inferior thyroid veins directly drain into the left brachiocephalic vein.)

#### 4.2.1. Internal jugular vein

Begins at the base of the skull with a moderate dilation, descends within the carotid sheath, and unites with the subclavian vein. The junction is called the venous angle (angulus venosus), from where the brachiocephalic vein starts. Into the left venous angle the thoracic duct opens, the right receives the right lymphatic trunk. At these angles the lymphatic circulation meets the blood circulation. Several small veins join the internal jugular vein near the base of the skull (meningeal veins). While descending, it collects blood from the pharynx (pharyngeal veins), the tongue (lingual vein), and the thyroid gland (middle thyroid vein). Two larger veins join the internal jugular vein, the facial and the retromandibular veins.

##### 4.2.1.1. Facial vein

The facial vein begins as angular vein at the medial angle of the eye, and obliquely descends till the anterior margin of the insertion of the masseter muscle. After having crossed the base of the mandible, it passes superficial to the submandibular gland, and unites with the retromandibular vein. The two veins together (common facial vein) open into the internal jugular vein. The facial vein receives blood from the frontal region (supratrochlear vein), the eyelids (superior and inferior palpebral veins), the nose (dorsal nasal veins), the parotidomasseteric region (deep facial vein, parotid veins), the soft palate and palatine tonsil (external palatine or pterygoid veins), the submandibular region (submental vein), the thyroid gland (superior thyroid vein), and the upper part of the larynx (superior laryngeal vein).

It is worth noting that the facial vein has no valves to prevent backflow of the blood. It connects with the cavernous sinus by two ways: through the angular vein and the superior ophthalmic vein or its supraorbital root, and through the deep facial vein and pterygoid plexus. Infections may thus freely spread from the face to the dural sinuses.

##### 4.2.1.2. Retromandibular vein

The retromandibular vein is formed by the fusion of several veins within the parotid gland. Its main tributaries are the superficial temporal, the transverse facial and the maxillary veins. The maxillary vein drains the pterygoid plexus, which collects blood from the areas supplied by the branches of maxillary artery.

Important to mention the connection between plexuses around the openings of the base of the skull (e.g. venous plexus of foramen ovale, internal carotid venous plexus) and the pterygoid plexus. The latter has excessive communication with the veins collecting blood both from the face and from the orbit (inferior ophthalmic vein), thus establishing route for inflammation to spread into the neurocranium.

#### 4.2.2. External jugular vein

The external jugular vein passes down between the platysma and the superficial lamina of cervical fascia. It receives blood from the occipital region (occipital vein), from the area behind the ear (retroauricular vein), and from the shoulder (suprascapular and transverse cervical veins). Commonly it enters the subclavian vein. The anterior jugular vein collects superficial veins from the chin and the anterior part of the neck. It anastomoses with the corresponding contralateral vein, and either directly or via the external jugular vein, joins the subclavian vein.
4.3. Nerve supply to the head and neck

4.3.1. Innervation of the face

The sensory nerves of the face are mostly branches coming from the three main divisions of the trigeminal nerve (V/1-2-3). The muscles of facial expression are all innervated by the facial nerve (VII), and the muscles of mastication by the mandibular nerve, the third main branch of the trigeminal nerve (V/3).

4.3.1.1. Sensory innervation of the face

The orbit and the skin of the forehead is innervated by branches of the ophthalmic nerve (V/1). The cutaneous branches arrive from the orbit, and pass superiorly beneath the skin. These are the medial and lateral supraorbital, and the supratrochlear nerves. The infratrochlear nerve turns to the back of the nose. The terminal branch of the anterior ethmoidal nerve arrives from the nasal cavity below the nasal bone, and innervates the tip of the nose and the philtrum.

The branches of the maxillary nerve (V/2) give sensory supply to the skin between the angles of the eye and the angle of the mouth. The infraorbital nerve emerges through the infraorbital foramen, and supplies the skin of the ala of the nose, the inferior eyelid and the part of the upper lip lateral to the philtrum. The zygomatico facial and zygomaticotemporal nerves, branches of the zygomatic nerve, come through tiny foramina of the zygomatic bone, and innervate the skin overlying the bone and above the zygomatic arch.

The skin on the posterior surface of the head, up almost till the level of the coronal suture, receives sensory innervation from the lesser (branch of the cervical plexus), the greater (dorsal ramus of the 2nd cervical spinal nerve) and the tertiary (dorsal ramus of the 3rd cervical spinal nerve) occipital nerves.

4.3.1.2. Nerve supply to the muscles of facial expression

All the muscles of facial expression are innervated by the facial nerve (VII), which emerges from the skull through the stylomastoid foramen. Here it gives branches to the stylohyoid muscle and the posterior belly of digastric muscle (not belonging to the muscles of facial expression). It splits into two branches (temporofacial and cervicofacial trunks), and enters the parotid gland, where the two branches unite and form the parotid plexus. The endbranches arise from the plexus, and leave the gland in five different directions: towards the muscles in the temporal region and above the eye the temporal branches turn, the zygomatic branch supplies the muscles below the eye and those originating from the zygomatic bone. Muscles around the mouth and below the mouth are innervated by the buccal and marginal mandibular branches, respectively. The cervical branch descends to the platysma.

While passing within the facial canal in the temporal bone, the facial nerve gives the greater petrosal nerve (to the lacrimal gland, and to the small glands in the mucous membrane of the nasal cavity, the paranasal sinuses and the soft palate), the nerve to stapedius muscle (to a muscle in the middle ear) and the chorda tympany (giving taste sensory innervation to the anterior two thirds of the tongue, and secretomotor innervation to the submandibular and sublingual glands).

4.3.1.3. Nerve supply to the muscles of mastication

The muscles of mastication are innervated by direct muscular branches originating from the smaller anterior (motor) division of the mandibular nerve (V/3). Find the detailed description of these nerves in the chapter addressed to the muscles of mastication (Chapter VII/3).

4.3.2. Innervation of the neck

4.3.2.1. Nerve supply to the muscles of the oral diaphragm and the suprahypoid muscles
Chapter VII/5 deals in details with the oral cavity. It is worth briefly to mention here the mylohyoid muscle, the muscle of the oral diaphragm, which receives innervation from the mylohyoid nerve (inferior alveolar nerve, V/3). The same nerve supplies the anterior belly of digastric muscle, a muscle belonging to the suprahypoid group. The geniohyoid muscle, also a member of this group, is innervated by fibres arriving from the cervical plexus via the hypoglossal nerve (XII). The other two suprahypoid muscles, the stylohyoid and the posterior belly of digastric muscle were mentioned earlier (see the facial nerve above).

4.3.2.2. Nerve supply to the infrahyoid muscles

The infrahyoid muscles (sternohyoid, sternothyroid, thyrohyoid and omohyoid muscles) play important role in the opening the mouth, phonation and swallowing. All are innervated by branches arising from the ansa cervicalis (see below).

4.3.3. Cervical plexus

The cervical plexus is formed by the union of the ventral rami of the first 4 cervical spinal nerves. It gives four sensory, and two mainly motor branches. The sensory branches pierce through the superficial lamina of the cervical fascia at the middle third of the posterior margin of the sternocleidomastoid muscle (punctum nervosum of Erb), and innervate the skin behind the ear (lesser occipital nerve), below the ear (great auricular nerve), on the anterior surface of the neck (transverse cervical nerve), and on the shoulder (suprACLavicular nerves). These branches can be anesthetized by an injection applied at the punctum nervosum. One of the motor branches, the ansa cervicalis, was mentioned in relation to the infrahyoid muscles. It is formed by the union of two roots. The superior root provides fibres from C1 spinal segment carried by the hypoglossal nerve, the inferior root comes from the cervical plexus. The other important motor branch originating mostly from C4 spinal segment is the phrenic nerve, which innervates the diaphragm, but also carries sensory fibres from the pericardium, pleura, peritoneum, gall bladder and the capsule of the liver. This explains why pain originating from these organs is frequently referred to the skin of the shoulder. The cervical plexus, together with the brachial plexus, gives direct muscular branches to innervate the anterior, middle and posterior scalenus muscles, which play role in movements of the head, the neck and also help inspiration.

4.3.4. Nerve supply to the viscera of the head and neck

For the sake of entirety, and to give a brief summary, a short synopsis follows regarding the innervation of internal organs of the head and neck. Nerves to the nasal cavity and paranasal sinuses come from the trigeminal nerve (ophthalmic and maxillary nerves), which also innervates the oral cavity (maxillary and mandibular nerves) (see in detail in Chapter VII.5). The glossopharyngeal nerve (IX) is responsible for sensory innervation of the root of the tongue, and for sensory and motor innervation of the upper half of the pharynx. The lower half of the pharynx (pharyngeal branches) and the larynx (superior and recurrent laryngeal nerves) receive both sensory and motor fibres from the vagus nerve (X). The hypoglossal nerve (XII) supplies the muscles of the tongue.

5. 7.5. The oral cavity – Balazs Gaszner, Tibor Hollosy, Pal Toth

The oral cavity is the initial part of the alimentary canal. It starts with the oral fissure (oral opening or rima oris) anteriorly, and continues into the oropharynx (mesopharynx) through the fauces (oropharyngeal isthmus). The two main parts of it are the oral vestibule and the oral cavity proper.

5.1. Oral fissure

The oral orifice is bordered by the upper and lower lips, which meet at the angle of the mouth forming the labial commissure. The middle portion of the upper lip is the philtrum that protrudes down in the midline as the tubercle. Opposite to the tubercle, the lower lip has a shallow depression. The upper lip is separated from the cheek by the nasolabial grooves laterally, and the mentalabial groove separates the lower lip from the chin.

Figure 7.37. Figure 1. – Mouth. 1: superior lip, 2: inferior lip, 3: philtrum, 4: mentolabial groove, 5: nasolabial groove, a: tubercle of the superior lip, b: angle of the mouth
The core of both lips are formed by the orbicularis oris muscle and connective tissue, which is covered by skin externally (cutaneous part) and by mucous membrane internally (mucous part). The red zone of the lip (vermilion or rubor labii) is sharply separated from the skin of the lip by the vermilion border. The red zone is characteristic of humans only, and the rapid change of its colour may indicate pathological conditions (cyanosis, anaemia). In the skin of the cutaneous part are sweat and sebaceous glands, while the mucous membrane of the mucous part contains many mixed salivary glands (labial glands). The vermilion has no glands.

The lips receive sensory innervation from the infraorbital (branch of maxillary nerve), from the buccal (branch of mandibular nerve), and from the mental (branch of inferior alveolar nerve) nerves. The torpidity of the lower lip indicates well the sufficient anesthesia of the lower teeth.

The rich arterious supply to the lips is ensured by the superior and inferior labial arteries, the branches of the facial artery. The veins are collected by the facial vein, the lymph vessels drain into the submental and submandibular lymph nodes.

### 5.2. Oral vestibule

The oral vestibule is a narrow, horseshoe-shaped space bordered by the lips and cheeks externally, and by the teeth and gums (gingiva) internally. Even if the mouth is closed and the dental arches are complete, the vestibule communicates with the oral cavity proper through a small space behind the last molar teeth. This allows fluid intake when the teeth are occluded.

The main component of the cheek is the buccinator muscle supported externally by the buccal fat pad of Bichat. It is lined by mucous membrane medially, and its lateral surface is covered by the skin of the face. A small, elongated (7–17 mm in length, 1–2 mm in diameter) epithelial cord is situated lateral to the buccinator muscle, the juxtaoral organ (of Chievitz). The organ is surrounded by a highly organized, perineurium-like connective tissue, and is very rich in nerves and receptors innervated by the buccal nerve. This structure presumably acts as a mechanical sensor.

The oral vestibule is lined by the stratified squamous non-keratinized epithelium of the mucous membrane. There are many small mucous salivary glands scattered in tunica submucosa (buccal glands). In the tunica mucosa of the chin, ectopic sebaceous glands with no associated hair follicles appear as pale yellowish spots. These are the
Fordyce spots, which can be seen in most of individuals, and their number is thought to increase with age. Opposite the second upper molar tooth, the parotid duct (Stensen’s duct) open into the vestibule on the top of a small mucosal elevation called the papilla of parotid gland.

The mucous membrane lining the lips and the cheeks reflects on to gums, forming a groove. In dentistry, this groove is frequently called mucogingival (mucolabial and mucobuccal sulci) or vestibular fornix. Deep in the sulcus, sickle-shaped mucosal folds connect the labia to the gums in the midline both superiorly and inferiorly. These are the frenulum of upper lip and the frenulum of lower lip, and are of variable length. Less prominent folds may traverse the sulcus close to the canine teeth.

The gingiva is firmly attached to the alveolar periosteum. It continues beyond the bone, and fills the spaces between adjacent teeth as interdental papillae.

5.3. Oral cavity proper

The roof of the oral cavity proper is formed by the hard palate anteriorly, and by the soft palate posteriorly. It extends anteriorly and laterally till the inner surface of the teeth. The floor of it is the oral diaphragm.

The mucous membrane lining the oral cavity everywhere has stratified squamous non-keratinized epithelium, but can still be divided into three areas according to its surface characteristics and the special features of the underlying tunica submucosa:

- **Specialized mucous membrane**: it is found on the dorsum of the tongue, occasionally becomes keratinized and contains taste buds.
- **Mucous membrane of masticatory areas**: it covers the gingiva and the hard palate, and strongly attaches to the underlying periosteum.
- **Lining or smooth mucous membrane**: it lines all other areas of the oral cavity, and mainly mucous glands are always found deep to it (in tunica submucosa).

5.3.1. Oral diaphragm

The oral diaphragm is formed by the two mylohyoid muscles. These are flat, almost triangular muscles, which arise from the mylohyoid lines of the mandible. Most of their fibres attach to the mylohyoid raphe that extends from the mental spines to the center of the body of the hyoid bone, to which the most posterior fibers also insert. Both surfaces of the muscles are covered by fascia ensuring sealing of the floor of the oral cavity, as far back as the last molar tooth. Structures arriving from back and posterior can only enter the oral cavity through a slit-like space named the sulcus lateralis lingue. This sulcus starts behind the posterior margin of the mylohyoid muscle, and extends anteriorly and medially above it. The medial wall of the sulcus is formed by the hyoglossus muscle (see below). The mylohyoid muscle aids in opening the mouth. When the muscles of mastication keep the mandible fixed, it helps to elevate the hyoid bone and the larynx, and also increases the pressure in the oral cavity (very important for babies when sucking milk). The muscle develops more strength in elderly individuals having lost their teeth, and thus participate in chewing. The mylohyoid nerve (branch of the inferior alveolar nerve) innervates the muscle.

Figure 7.38. Figure 2. – Floor of the mouth 1 (inferior view). 1: base of the mandible, 2: left mylohyoid muscle, 3: anterior belly of the left digastric muscle, 4: angle of the mandible, a: mylohyoid raphe, A: anterior
Figure 7.39. Figure 3. – Floor of the mouth 2 (inferior view). 1: base of the mandible, 2: right mylohyoid muscle, 3: anterior belly of the left digastric muscle (reflected), 4: angle of the mandible, a: mylohyoid raphe, A: anterior
Figure 7.40. Floor of the mouth 3 (inferior view). 1: base of the mandible, 2: right geniohyoid muscle, 3: mylohyoid muscles (reflected), 4: angle of the mandible, A: anterior
Figure 7.41. Figure 5. – Floor of the mouth 4 (inferior view). 1: base of the mandible, 2: right genioglossus muscle, 3: left geniohyoid muscle, 4: right geniohyoid muscle (reflected), 5: mylohyoid muscles (reflected), 6: angle of the mandible, A: anterior
The *geniohyoid muscles* enstrenghen the floor of the oral cavity. These slender muscles lie above the mylohyoid muscles just next to the midline. They originate from the inferior mental spines of the mandible, and attach to the body of the hyoid bone posteriorly. Fibres deriving from the *first two cervical segments* of the spinal cord, but carried by the hypoglossal nerve, innervate these muscles.

### 5.3.2. Hard palate

The hard palate is the anterior two thirds of the palate, which is formed by the *palatine processes of maxillae* and the *horizontal plates of the palatine bones*. These meet in the *median* and in the *transverse palatine sutures*. In the midline, a sharp process, the *posterior nasal spine*, projects backwards. There are openings on the hard palate, each of them are covered by the mucous membrane.

In the midline of the palate anteriorly, the *incisive papilla* overlies the *incisive foramen* (sometimes foramina, left and right) that transmits the *incisive nerve* (branch of the posterior nasal nerves), *the incisive artery* (from the sphenopalatine artery) and the corresponding *vein*. In the nasal cavity, these structures are also called *nasopalatine artery, vein and nerve* (the nerve of Scarpa).

In the posterolateral region of the hard palate, the *greater* and *lesser palatine foramina*, the openings of the corresponding canals, are located. Through them pass the *greater* and *lesser palatine nerves* (branches of the maxillary nerve), and the *descending palatine artery* (from the maxillary artery).

Deep to the tough masticatory mucous membrane of the hard palate are the mucous *palatine glands*. The *palatine raphe* extends backwards in the midline from the incisive papilla. The mucous membrane firmly attaches here to the underlying periosteum with no intervening tunica submucosa. Three to seven transverse folds of the mucous membrane extend laterally from the incisive papilla and the anterior part of the palatine
raphe, the *palatine rugae*. The arrangement of these folds is unique to a person, like fingerprints, therefore can be used to identify individuals in forensic medicine. Occasionally, bony prominence, the *palatine torus*, may develop in the midline of the palate, which may require surgical removal for the proper fitting of an upper denture.

The hard palate continues into the soft palate posteriorly, the border between them is readily seen and palpable. Related to that of the hard palate, the colour of the soft palate is more vivid red, and has a tint of yellow. This border is frequently referred to as 'vibrating line' (movement of the soft palate can be observed here when the patient says 'ah' with the mouth open).

**Figure 7.42.** Figure 6. – Palate. a: uvula, b: palatopharyngeal arch, arrow: vestibule of the oral cavity, dotted line: border between hard and soft palates, continuous line: terminal sulcus of the tongue

The *nasopalatine nerve* innervates the area of the hard palate behind the incisive teeth, i.e. the intermaxillary segment, and the palatine surface of the gums. The *greater palatine nerve* supplies the bigger posterior area both by sensory and secretomotor fibres, the latters originating from the pterygopalatine ganglion. Similarly arranged branches of the *nasopalatine* and the *descending palatine arteries* give blood supply to the hard palate.

### 5.3.3. Soft palate

The soft palate is a mobile, fibromuscular flap attached to the posterior border of the hard palate, which slopes down and back between the posterior parts of the nasal and the oral cavities. The upper surface of it is covered by the nasal, and the lower by the oral mucosa. The 'skeleton' of the soft palate is formed by a thin, fibrous plate, the *palatine aponeurosis*, which is primarily composed of the expanded tendons of the tensor veli palatini muscles, but all the other muscles of the soft palate also attach to it. The anterior part of the soft palate contains little or no muscle. Near the junction of the hard and soft palates, there are often two small depressions on both
sides of the midline. These palatine foveae may extend into sagittally elongated pits receiving common openings of the converging ducts of the mucous palatine glands. The posterior part of the soft palate that forms the superior-lateral boundary of the fauces (oropharyngeal isthmus) is mostly muscular. A median cone-shaped process, the uvula, hangs down from its free posterior border.

There are several muscles originating from or attaching to the soft palate, making it fairly movable. For the complete separation of the nasopharynx (the part lying behind the nasal cavity) from the rest of the pharynx during deglutition, simultaneous contraction both of levator and of tensor muscles of the palate is essential.

The muscles of the soft palate:

Figure 7.43. Figure 7. – Right muscles of the soft palate (posterior view). 1: levator veli palatini muscle, 2: tensor veli palatini muscle, a: stylopharyngeus muscle, b: glossopharyngeal nerve, c: stylohyoid muscle, d: posterior belly of the digastric muscle, S: superior

a.) Levator veli palatini muscle: originates from the cartilaginous portion of the auditory tube, passes above the superior constrictor muscle of the pharynx, and inserts to the upper surface of the palatine aponeurosis. It elevates the soft palate.

b.) Tensor veli palatini muscle: arises from the scaphoid fossa and the lateral plate of the pterygoid process, the lateral lamina of the cartilage of the auditory tube and the sphenoid spine. From here its fibres converge into a delicate tendon that turns into horizontal around the pterygoid hamulus to be inserted to the palatine aponeurosis. A small bursa reduces friction between the tendon and the pterygoid hamulus. Alone, the muscle pulls the soft palate to its side, contracting on both sides the muscles stretch, therefore tauten it. Together with
the levator, the tensor veli palatini muscle opens the auditory tube thus allowing the ventilation of the tympanic cavity.

c.) Palatoglossus muscle: takes origin from the palatine aponeurosis, and radiates antero-inferiorly and laterally to pass deep into the side of the tongue, where it intermingles with the fibres of the transversus linguae muscle. Covered by mucous membrane, the muscle forms the palatoglossal arch. It elevates the root of the tongue and narrows the fauces for deglutition.

d.) Palatopharyngeus muscle: comes from the posterior part of the hard palate and from the palatine aponeurosis, and passes laterally and downwards behind the palatine tonsil within the fold of the mucous membrane (palatopharyngeal arch). Some of its fibres attach to the posterior border of the thyroid cartilage of the larynx, others blend with the fibrous tissue of the pharynx thus forming an incomplete inner muscular layer in the wall of it. The palatopharyngeus muscles elevate the pharynx and larynx, pull the pharynx over the bolus during swallowing.

e.) Musculus uvulae: originates from the posterior nasal spine and the palatine aponeurosis, and inserts into the mucous membrane covering the uvula. The unilateral paralysis of the muscle results in an oblique position of the uvula (it bends to the side of the unaffected muscle).

Except for tensor veli palatini muscle, which receives motor branch from the mandibular nerve (V/3), all the muscles of the palate are innervated by nerve fibres leaving the brain through the cranial roots of the accessory nerve (XI) and reach the pharyngeal plexus via the vagus nerve (X).

The lesser palatine nerves (branches of the maxillary nerve) supply sensory and secretomotor fibres to the soft palate, the latters arising from the pterygopalatine ganglion. The arteries arise from the descending palatine artery (branch of the maxillary artery).

5.3.4. Oropharyngeal isthmus (isthmus faucium or fauces)

Figure 7.44. Figure 8. – Oropharyngeal isthmus. 1: cheek, 2: root of the tongue, a: uvula, b: palatoglossal arch, c: palatopharyngeal arch, d: palatine tonsil

The oral cavity communicates with the middle level of the pharynx at the oropharyngeal isthmus. It is superiorly bordered by the soft palate, from which the uvula hangs down in the midline. On the sides, the soft palate continues inferiorly into two arches, each containing a muscle corresponding to the arch. The anterior is the palatoglossal, the posterior is the palatopharyngeal arch. They diverge inferiorly, the depression between them is the tonsillar fossa, into the mucous membrane of which the palatine tonsil is embedded. The floor of the fauces is formed by the root of the tongue.

5.4. Tongue
The tongue is a muscular organ with its posteriorly located root attached to the floor of the oral cavity. The tip (apex) of the tongue is the most movable part, the main portion is its body. The upper and lower surfaces meet at the margin of tongue.

*Figure 7.45. Figure 9. – Tongue. 1: root of the tongue, 2: body of the tongue, 3: apex of the tongue, 4: margin of the tongue, a: median sulcus of tongue, b: fungiform papillae, continuous line : terminal sulcus of the tongue*

The upper surface (*dorsum of tongue*) is divided into two asymmetrical parts by the anteriorly opened V-shaped *terminal sulcus of tongue*. The anterior 2/3 is the *papillary (presulcal) part*, which is characterized by the presence of tiny mucosal protrusions, the lingual papillae. Behind the terminal sulcus lies the *follicular (postsulcal) part* with many lymphatic follicles embedded in its mucous membrane. Posterior to the angle of the V of sulcus terminalis, a small depression (*foramen caecum*) is seen, which indicates the site of development of the thyroid gland. The *median sulcus* divides the tongue into left and right halves. The median and the terminal sulci correspond to the lines of fusion of different primordia of the tongue.

The inferior surface is covered by a thin mucous membrane. A mucosal fold, the *frenulum of tongue*, connects the midline of this surface with the floor of the oral cavity. If the frenulum long extends sagittally (ankyloglossia), it may restrict movements of the tongue. Next to the lower attachment of the frenulum are the *sublingual caruncles*, on which the submandibular and the (major) sublingual ducts open. The caruncles are situated on the anterior ends of the forward converging mucosal elevations, the sublingual folds, which are produced by the sublingual glands. Two folds, the *fimbriated folds* converge on the inferior surface towards the tip of tongue. Through the thin tunica mucosa of these folds, the deep lingual veins are readily seen.

Three folds, the *median* and the two *lateral glossoepiglottic folds*, connect the root of the tongue to the epiglottis (the uppermost cartilage of the larynx), between them are the two *epiglottic valleculae*.
**Papillary (presulcal) part**: The papillae of the tongue are minor protrusions of lamina propria of the mucous membrane. The most numerous are the filiform papillae that appear as whitish, cylindrical elevations with pointed tips. They are 2 to 3 mm long, the epithelium covering their apical portion may be keratinized. These papillae are responsible for the roughness of the tongue, and have an important role in shaping and moving the bolus. Scattered between the filiform papillae are the larger, reddish fungiform papillae, which are mostly found at the tip and near the margins. The foliate papillae are seen as a series of parallel ridges on the sides of the tongue, close to the attachment of the palatoglossal folds. Eight to fifteen circumvallate papillae, the largest of all papillae, are arranged in a single row lying immediately anterior to the sulcus terminalis. Each of them is surrounded by a deep, circular trench, into which the ducts of the underlying serous glands of von Ebner open. Similar ducts may open into the grooves of the foliate papillae. Several small, mostly mucous glands are found in the entire extent of the tongue (lingual glands), the somewhat larger anteror lingual glands of Nuhn-Blandin are located in the tip. Except for the filiform papillae, the sides of the papillae contain numerous taste buds.

The papillary part receives general sensory innervation from the lingual nerve (branch of the mandibular nerve). The taste sensory and the secretomotor fibres carried by the chorda tympani (branch of the facial nerve) join the lingual nerve to arrive to the tongue.

**Figure 7.46. Figure 10.** – Circumvallate papillae of the tongue. 1: palatoglossal arch, 2: body of the tongue, a: uvula, *: circumvallate papilla

**Follicular (postsulcal) part**: Aggregated lymphatic follicles produce round elevations of the mucous membrane forming the lingual tonsil. This part is also characterized by the abundance of mostly mucous glands. The continuous stream of saliva removes particles from the depressions around the follicles.

The general sensory, taste sensory and the secretomotor fibres to the follicular part of the tongue are supplied by small lingual branches of the glossopharyngeal and the vagus nerves.

The muscles of the tongue either change the shape of the tongue (intrinsic muscles), or move the tongue within the oral cavity, or even outside of it (extrinsic muscles). Many muscle fibres attach to the lingual septum, the thick median fibrous sheet separating the tongue into two halves.

The intrinsic muscles of the tongue are the superior and inferior longitudinal, the transverse and the vertical muscles. The superior longitudinal muscle elevates the tip, thereby produces a sagittal concavity on its dorsum. The inferior longitudinal muscle lowers the apex, and increases the convexity of the dorsum. Contracting together, they shorten the tongue. The transverse muscle makes the tongue narrower and taller, the vertical muscle flattens and widens it. The precise combination of synchronous uni- or bilateral contraction of all these muscles is required for the fine movements of the tongue, especially during speach.

The extrinsic muscles are:

a.) The genioglossus muscle originates from the superior mental spine of the mandible. Most of its fibres fan out to be inserted in the body, the most superior fibres ascend to the tip of the tongue, and the lowest fibres insert to the body of the hyoid bone. By entering the tongue, the left and right muscles run on either side of the lingual septum. The main function of the genioglossus is to pull the tongue forward and down. Contracting on one side only, the muscle curls the tongue towards the opposite side (in case of paralysis, the tongue turns to the side of damage).
b.) The hyoglossus muscle takes origin from the upper margin of both the body and the horns of the hyoid bone and ascends into the lateral part of the tongue where its fibres merge with those of the styloglossus muscle. It depresses the tongue, and forms the medial wall of the sulcus lateralis linguae. On its lateral side are the lingual nerve, the submandibular duct and the hypoglossal nerve, medial to it the lingual artery proceeds forward.

c.) The styloglossus muscle arises from the tip of the styloid process, the lowermost fibres may come from the stylohyoid ligament. The muscle descends anteriorly to the side of the tongue where its fibres intermingle with the fibres of hyoglossus muscle before they continue towards the tip of the tongue. The styloglossus muscle retracts and elevates the tongue.

d.) The palatoglossus muscle equally can be listed amongst the palatine muscles and the muscles of the tongue (see its description above).

All the muscles of the tongue derive from the occipital myotomes, therefore all are innervated by the hypoglossal nerve.

Figure 7.47. Figure 11. – Right extrinsic muscles of the tongue (lateral view). 1: styloglossus muscle, 2: hyoglossus muscle, 3: genioglossus muscle, a: stylohyoid muscle, b: posterior belly of the digastric muscle, c: mylohyoid muscle, d: anterior belly of the digastric muscle, S: superior

Figure 7.48. Figure 12. – Right extrinsic muscles of the tongue (lateral view), 1: styloglossus muscle, 2: hyoglossus muscle, 3: genioglossus muscle, a: posterior belly of the digastric muscle, b: stylohyoid muscle, c: anterior belly of the digastric muscle, d: geniohyoid muscle, e: mylohyoid muscle, S: superior
The main artery supplying the tongue is the lingual artery (branch of the external carotid artery). It first runs in the carotid triangle of the neck, then continues forwards medial to the hyoglossus muscle. In case of serious bleeding from the tongue, the artery can be ligated in the triangle of Pirogov, which is bordered by the hypoglossal nerve superiorly, the posterior border of the mylohyoid muscle anteriorly, and the intermediate tendon of the digastric muscle postero-inferiorly. In the triangle, the artery passes anteriorly on the inner aspect of the hyoglossus muscle, slightly above the greater horn of the hyoid bone. The lingual artery finally enters the tongue near the frenulum as deep lingual artery. Some of its branches (dorsal lingual branches) supply the dorsum of the tongue at the region of the root. These branches also provide blood supply to the structures of the oropharyngeal isthmus (the palatoglossal arch, the palatine tonsil, the epiglottis). Another branch (sublingual artery) distributes blood to the floor of the oral cavity, and anastomoses with the submental artery (branch of the facial artery). It is of medical importance that the branches of the left and right lingual arteries, mainly the sublingual arteries, establish excessive anastomoses within the tongue (bleeding).

Beneath the mucous membrane covering the inferior surface of the tongue and the floor of the oral cavity is a rich venous plexus, from where the absorption of some drugs acting on the heart is fast. The lingual vein drains into the internal jugular vein (occasionally through the facial vein).

The lymph vessels draining the tip of the tongue lead to the submental lymph nodes, those from the body to the submandibular lymph nodes. From the root of the tongue the lymph is collected by the deep cervical lymph nodes.

### 6. 7.6. Anatomy of the teeth – Gyula Marada

#### 6.1. Teeth of the upper-jaw

##### 6.1.1. Upper central incisor

From the vestibular or labial view it is trapezoid-shaped and mesiodistally the widest of all the other anterior teeth. Out of the two parallel walls of the trapezoid the longer is the incisal side, the shorter is in gingival one. Generally the incisogingival length is slightly larger than the mesiodistal.

The vestibular surface is convex mesiodistally and incisogingivally. The incisogingival convexity is the greatest in the gingival third and flattens towards the incisal third.
The mesial outline of the tooth is slightly convex, but sometimes it can also be quite flat. The crest of the curvature is the contact area which is situated close to the incisal edge. The mesioincisal angle is usually acute angle.

The convexity of the distal outline contour is far more prominent. The distoincisal corner is usually rounded and obtuse angle. The peak of the curvature is also the contact area, which is located more gingivally than on the mesialis surface.

On the incisal edge irregularities, mamelons can be found. The mamelons are mostly expressed after eruption. Later due to the wear of the teeth, the incisal edge straightens and will be perpendicular to the longitudinal axis of the tooth.

The cervical line (cemento-enamel junction, CEJ) curves evenly towards the root.

On the vestibular surface two shallow developmental depressions can be found. These depressions extend from the incisal edge towards the gingival direction where they fade. They divide the surface into three lobes.

Close to the CEJ thin imbrication lines are usually present. They are parallel to the gingival outline.

The height of the incisocervical contour on the vestibular surface is in the gingival third.

The lingual surface is also trapezoidal in shape but shorter mesiodistally than the vestibular surface. The reason of this is that the approximal surfaces converge slightly towards the lingual direction. Incisal two-thirds of the surface is concave (lingual fossa), while the gingival third is convex (cingulum). The concave surface is bordered mesially and distally by marginal ridges. Generally the surface is smooth but occasionally “W” shaped ridges may extend in the gingival direction.

In most of the cases a groove separates the fossa from the cingulum (linguogingival groove) and in the middle of the groove a pit (lingual pit) can be found.

From approximal view the crown is triangular-shaped. The apex of the triangle is the incisal edge and is situated over the long axis of the tooth. The sides of the triangle are also not flat. They follow the curvature of the vestibular and lingual side. The distal surface is shorter incisogingivally and the contact area is closer to the gingival third than on the mesial side.

From incisal view the crown is rather triangular. The vestibular surface is slightly convex and the height of contour is located mesially to the midline. The mesiodistal dimension of the lingual surface is narrower than the vestibular. The lingual surface is more convex due to the cingulum.

### 6.1.2. Upper lateral incisor

Its shape is really similar to the central incisor, but smaller. From the vestibular view the mesial side of the tooth is more convex, than the central incisor and its mesiooncisial edge is more rounded. Even the contact surface is located closer to the gingiviva. The distal surface is rather rounded and the distoincisial angle is the most rounded. The whole vestibular surface is much more convex than of the central incisors. Depressions can be found.

The cingulum and the marginal ridges are more significant on the palatal surface. The foramen coecum and the groove, that separates the fossa from the cingulum, are also deeper. An adjunct groove is tending towards the gingiviva and softly laterally.

Because of the prominent protrusions of the palatal and the vestibular surfaces the tooth is diamond-shaped from the incisial view.

**Figure 7.49. Figure 1., 2., 3.**
6.1.3. The upper canine

The incisivo-gingival length of the crown is similar to the central incisor, but mesiodistally shorter and orovestibularly much wider.

Its shape resembles a pentagon from the vestibular view. The surface is convex in every direction, mainly mesiodistally. The mesial side is rounded and the widest part is at the contactpoint, that is situated at the border of the incisal and the middle third. The distal surface is shorter than the mesial. Usually it is straight or slightly concave. The contact surface exists in the middle third. The mesial and distal surfaces pass through the rounded corners to the incisal edge.

The incisial edge consists of a mesial and a distal slope. These are erected from the peak of the cusp, proceeding distally and mesially. Generally the mesial slope is shorter and less steep.

The most significant protrusion on the buccal surface is the central protrusion, that extends in the axis of the tooth from the cusps towards the gingiva. Laterally from this, there are two depressions, but they are shallower than on the incisors. The widest part of the surface is situated at the cervical third.

The cingulum and the marginal ridges are also more prominent on the palatal surface, than on the incisors. The incisial third is usually smooth. The fossa is devided in two parts by the palatal ridge. Grooves or pits are really rarely found on the border of the cingulum.

From the mesial view it’s triangle-shaped. The orovestibular dimension is wider and more rounded than on the incisors. The distal surface is smaller, than the mesial. The contactsurfaces are closer to the gingiva.

It is rounded and irregular diamond-shaped from the incisialview.

Figure 7.50. Figure 4., 5., 6.
6.1.4. Upper first premolar

From buccal view it is pentagon shaped. Its’ cusp is not that prominent as the canines’, but more than the lateral incisors’. It is slightly bigger than the second premolar. The mesials margin and the mesio-occlusal inclane terminate in an obtuse angle. The mesial surface is gently concave. The widest part is the contact surface at the border of the occlusal and the middle third. The distal surface is similar to the mesial but a little bit shorter and the the angle is just barely expressed. The mesial slope of the vestibular cusp is longer and straighter than the distal. On the buccal surface from the cusp toward the gingiva, an enamel ridge passes through till the half of the tooth, like on the canine. A depression exists on the two sides.

From the palatal view it is visible that the palatal surface, such as the palatal cusp is smaller than the buccal. The surfaces are also similarly shaped, but a bit more rounded. The mesial concavity could be more pointed. The mesial slope is shorter than the other slopes of the palatal cusp. Even the cusp is rounded.

From the mesial view the tooth is trapezoid shaped. The buccal wall is convex, the widest point sets on the cervical third. From the palatal sight the equator occurs in the middle third.

The occlusal ridge is irregular, concave. The mesial marginal ridge gives the biggest part of the occlusal ridge. From this point of view the difference between the buccal and palatal cusps height is well-marked. The palatal cusp is usually 1 mm shorter. The mesial concavity is specific to the first premolar. It is situated in the middle of the gingival third.

The shape of the distal surface is similar to the mesial. The occluso-gingival height is a bit shorter. The surface is convex and not concave, the contact surface is bigger.

From occlusal view it is hexagonal-shaped, from orovestibular view its wider than mesiodistally. Because of the enamel ridge the buccal surface is more convex. The mesial and the distal marginal ridges are straight and converge palatally. A groove can appear on the mesial ridge. The buccal cusp is more chopping, sharper and straighter. Four enamel ridges connect to the buccal cusp. The palatal cusp is triangle-shaped goes towards the marginal ridges. The mesial and the distal are connecting to the buccal end of the marginal ridges on the same sights. The enamel ridge from the palatal cusp meets the enamel ridge from the buccal cusp at the central groove and produce the crista transversa. This divides the central groove to a mesial and distal groove. These are triangle-shaped. On their deepest points some pits exist.

Figure 7.51. Figure 7., 8., 9.
6.1.5. Upper second premolar

The second premolar is smaller than the first one. Its sights are more convex than that of the first premolars, the height of their cusps are nearly the same size.

From buccal view mesial slope of the occlusal surface is slightly shorter. From the mesial sight the convex surface is missing. The two cusps have nearly the same height.

From the occlusal view the tooth is not that angular. The central groove is shorter, the adjunct grooves are frequent.

6.1.6. Upper first molar

Its crown is the biggest in the arch. It is oro-vestibulary wider than mesio-distally. Occlusio-gingivally it is a bit shorter, than the premolars. All the axial walls are trapezoid-shaped.

From the buccal view the mesial wall is straight. The widest point is the contactsurface that exists at the border of the occlusal and the mesial third. On the occlusal surface it passes by a rounded angle.

The distal surface is rather convex. The equator passes in the middle third.

The two cusps (mesio-buccal and disto-buccal) divide the occlusal line to two parts. The mesial cusp is wider, the distal is sharper. The two cusps are nearly of same height. The mesiopalatal cusp persists between them. Between the two cusps a shallow groove extends till the middle of the buccal surface. It could terminate in a pit.

From the palatal view it is trapezoid-shaped, too. The distal part of the tooth is more rounded because the distopalatal cusp is smaller than the disto-buccal. A groove divides the occlusal marginal ridge to two equal parts. It terminates in the middle of the palatal surface in the foramen coecum. The mesio-palatal cusp is longer and bigger, than the distal.

The Carabelli’s cusp exists palatally from the mesio-palatal cusp. A groove separates the Carabelli’s cusp from the mesiopalatal cusp.

From mesial view the tooth is trapezoid-shaped. The cervical third is the widest. The cervical third is significantly convex and the equator is also passing here. Towards the occlusal direction it is rather flat. The all palatal surface is convex and the Carabelli cusp can make it irregular. The mesial marginal ridge interconnects the two mesial cusps.

From the the distal view the tooth is similar to the mesial view. The distal cusp and the two mesial cusps are also visible. The distal marginal ridge is situated a bit lower than the mesial.

The whole occlusal surface slopes to distal.
From the occlusal view the tooth is diamond shaped. The mesio-buccal and disto-palatal angles are acute angle. Grooves pass through the mesial and distal marginal ridges and divide the surfaces equally. One groove divides the buccal surface into two pieces and the mesial would be longer. The most prominent groove, is the one that reaches the palatal surface. From this side mesially, that part is longer and more convex.

The mesio-buccal cusp is the second biggest. Four enamelridges arise from its peak. The one that goes to the palatal direction is the most important. It unites with the mesiopalatal cusp and form the crista transversa. The biggest cusp is the mesiopalatal one. Its enamelridge and the distobuccal cusp’s ridge create an oblique ridge.

The deepest point of the surface occurs in the middle of the central groove. From here arise the buccal bended groove. The central groove proceeds from the deepest point towards the distal direction. Theese terminate in mesial and distal pits. From the distal pit erects the palatal bended groove.

**Figure 7.52. Figure 10., 11., 12.**

![Figure 10.](image1)
![Figure 11.](image2)
![Figure 12.](image3)

### 6.1.7. Upper second molar

The shape is similar to the upper first molar, but smaller especially at the mesio-distal part.

The groove on the buccal surface is located more distally, thus the mesiobuccal cusp is bigger.

From palatal view the distopalatal cusp seems to be smaller in every cases, than that of the first molar. The Carabelli’s cusp is missing. The palatal groove is shorter.

The distal surface is shorter occluso-gingivally, than the mesial. The distal slope of the occlusal surface is more prominent.

From the occlusal view the crown is similar to a diamond or rearily to a heart. In the case of heart similarity the distopalatal cusp is missing.

**Figure 7.53. Figure 13., 14., 15.**
6.2. Teeth of the lower-jaw

The teeth of the mandible are very similar to the same toothgroups of the maxilla. In the following text we compare them to the maxilla’s teeth and just the most important differences are mentioned.

6.2.1. Lower central incisor

This is the smallest tooth. Its crown is very similar to the crown of the first upper incisor. From the vestibular view the crown is trapezoid-shaped, the axial walls are nearly straight. The approximal surfaces and the incisial edge intersect in acute angles. The contact surfaces are located very close to the incisal edge. The surface is just a little bit convex, in some cases it is flat.

From the lingual view it is really similar to the vestibular view. Compared to the upper incisors it is flatter and just a shallow fossa and a prominent cingulum are located here. The marginal ridge and the foramen coecum are also missing.

From approximal view the vestibular surface is slightly convex, the lingual is barely concave.

The incisival edge is sharp. The equator occurs in the cervical third.

From incisal view the tooth is diamond-shaped, the distal and the mesial parts are symmetric and the incisal edge is straight.

Figure 7.54. Figure 16., 17., 18.
6.2.2. Second lower incisor

It is really similar to the middle incisor. It is a little bit bigger, the ridge slopes distally, and the distal marginal angle is rounded.

6.2.3. Lower canine

Its shape looks like the canine of the upper jaw, but the inciso-gingival dimension is smaller.

From the vestibular aspect it is pentagon-shaped, the enamel ridge is less prominent, therefore, the vestibular surface is more convex. The mesial surface is straight. The ridge and the mesial surface intersect in an obtuse angle. The contact surface is situated in the incisial third. The distal surface is convex and with the ridge the formed angle is rounded. The contact surface is located more gingivally, than on the mesial surface, and rather closer to the gingival third. The cusp is even not that prominent as on the upper canine. The distal slope is a bit longer.

The lingual surface is flatter, than the upper canine. The cingulum does not extend far incisally and the marginal ridges are barely visible. The shallow fossae form a concave surface.

From the approximal aspect the tooth is more triangular, than the upper canine. The vestibular and the lingual surfaces are hardly concave or even convex. The oro-vestibular surface is significantly narrower and the widest points are situated in the gingival third.

Figure 7.55. Figure 19., 20., 21.
6.2.4. Lower first premolar

It is a little bit smaller than the second premolar. The mesio-buccal and the orovestibular heights are the same. Usually the lower premolars have more than two cusps, compared to the upper premolars the lingual cusp is smaller. The first premolar is looks morelike acanine, than the second premolar.

From buccal view it is pentagon-shaped. It is convex to both direction. The mesial side is convex from the contact surface towards the cervical third third. The equator and the contactsurface is present in the middle third. The distal surface is similar to the mesial, but a bit shorter. The cusp is not in the midline, it is located more mesially, therefore, the mesial slope of the cusp is shorter. An enamelridge proceeds from the cusp to the buccal surface. It forms two fossae. The mesial and distal contactsurfaces are of the same hight.

From the lingual aspect the buccal cusp is also visible. The lingual cusp is smaller and the whole occlusal surface slopes to the distal direction. The approximal surfaces are also converged to the lingual direction. The lingual cusp is smaller than the buccal one and situated slightly mesially. Usually a groove is present, that bends to the mesio-lingual surface, mesially from the lingual cusp.

The difference between the two cusps is visible from the aproximal view. Because the buccal and the lingual surfaces slope to the lingual direction, it seems, that the whole crown is tilted towards median-sagittal plain. The marginal ridges and the occlusal plain meets in a 45 degrees angle.

From occlusal view the tooth is diamond-shaped and the only the mesio-lingual groove is situated here. Because the cusps are located more mesially, the distal part of the tooth is bigger. The tooth slopes to the lingual direction, so a bigger part of the buccal surface is visible, but the lingual is covered. The buccal cusp tip stands in the longitudinal axis. 4 marginal ridges are erected from the cusp tip. The height of the lingual cuspsdoes not even reach the half of the buccal cup. A crista transversa is formed by the enamelridges, that are coming from the buccal and the lingual cusps. Pits are found mesially and distally from the crista transversa. The central groove interconnects them.

Figure 7.56. Figure 22., 23., 24.
6.2.5. Lower second premolar

Compared to the first premolar it is a bit bigger, the buccal cusp is lower and occlusio-gingivally shorter. The buccal cusp is situated approximately in the middle, the mesial and the distal slopes are nearly of the same length.

From the lingual aspect it is conspicuous, that the lingual cusp is more developed, and sometimes two cusps are present instead of one. Because the cusp is higher, the crown hardly tilted to the lingual direction and the occlusal surface is barely visible. If two cusps occur, a groove will pass between them.

The greatest difference between the first and second premolar can be observed from the interproximal view.

From the occlusal aspect it is rather square-shaped, especially if it has two lingual cusps. The lingual convergence of the approximal surfaces is not that pronounced. Its groove is H or Y shaped. In case of three cusps the mesial and distal pit is not present, a pit is situated in the middle of the tooth, which is the deepest point of the fissuresystem.

6.2.6. Lower first molar

From the buccal aspect it is very obvious that its buccal surface is the biggest among the teeth. It is also trapezoid-shaped. The mesial contact surface is located at the border of the occlusal and mesial third. From this point gingivally the surface is concave, occlusally convex. The distal surface is rather convex. Two grooves are located mesially and distally. The mesiobuccal and the distobuccal cusps are slightly higher than the distal one.

From the lingual view it is also trapezoid-shaped. The mesial and distal surface converge distally. Lingually a groove occurs, between the mesio-lingual and disto-lingual cusps. The mesio-lingual cusp is a bit shorter, than the disto-lingual one. The lengths are the same. The lingual groove often terminates in a foramen coecum.

From the approximal aspect the tooth is diamond-shaped. It is wider gingivally than occlusally. This is the only lower molar, with a crown tilted lingually.

From occlusal view the tooth is pentagon-shaped. The distal part of the buccal wall deviates lingually and forms the fifth wall. The mesiodistal part of the tooth is the widest and the contactsurface is located buccaly. Usually 5 cusps occur and the distal one is the smallest. Instead of its name the distal cusp, the mesio-buccal cusp and the disto-buccal cusp create together the three cognated cusps. The mesio-buccal cusp is the biggest. The occlusal slopes of the cusps do not form a crista transversa. The central groove is bounded by the mesial and distal marginal ridges. The deepest point of the related groove system is the pit, in the imaginary middle of the tooth. Two grooves extend from this point. They are bending to the buccal and lingual surface.

Figure 7.57. Figure 25., 26., 27.
6.2.7. Lower second molar

Compared to the first molar it is smaller and its surfaces are more regular. It often has four cusps, the distal cusp is missing.

From the buccal aspect a groove is present, that divides the buccal surface to two nearly equal parts. It usually terminates in a pit. From the lingual aspect a groove passes close to the midline.

The four cusps are located regularly on the four corner of the tooth. The biggest cusp is the mesiobuccal, the smallest is the distolingual. The central groove is nearly symmetrical, bordered by the mesial and the distal marginal ridges. The deepest point is situated in the middle of the tooth. The buccal and the lingual groove extends from this point.

7. 7.7. Anatomy of the periodontium – Balazs Sandor

The periodontium:

The periodontium surrounds and supports the teeth. There is a resilient anchorage between the roots of the teeth and the periodontium. The main components of the periodontium are (Figure 1.):

- gingiva,
- periodontal ligaments,
- cementum,
- alveolar bone (alveolar process).

Figure 7.58. Figure 1. – Structure of the periodontium
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7.1. Gingiva

The oral mucosa consists of three parts:

• Gingiva and the covering of the hard palate, termed as the masticatory mucosa
• The dorsum of the tongue, with specialized mucosa
• The oral mucous membrane (sublingual, vestibular and buccal mucosa).

Gingiva is part of the mucosa, and the most superficial part of the periodontium. Basically can be separated into two parts (Figure 2.):

• Attached gingiva (gingiva propria)
• Marginal gingiva (gingival marginalis, free gingiva or unattached gingiva)

Figure 7.59. Figure 2. – Parts of the gingiva: attached gingiva and marginal gingiva, separated by the gingival groove
7.1.1. Attached gingiva

Attached gingiva lies between the free gingival and the oral mucous membrane, covers and firmly attaches to the alveolar process. The border between the oral mucous membrane and the attached gingival is the mucogingival junction. This is a transition between the keratinized epithelium (attached gingiva) and the non-keratinized epithelium (oral mucosa). This borderline is absent on the palatal side. The coronal border of the attached gingiva is the epithelial junction or attachment (see later). Attached gingiva is wider in permanent dentition ranging between 1-10 mm (widest in the area of the upper lateral incisors, and narrowest in the area of the lower canines and premolars.

7.1.2. Marginal gingiva

Marginal gingiva or unattached gingiva is the terminal border of the gingiva surrounding the teeth. In about 50% of the cases it is separated from the attached gingival with a linear depression called the gingival groove (Figure 3.) It is the outer wall of the sulcus. The apical border is the epithelial attachment. The gingival groove is the line of the epithelial attachment.

7.1.3. Clinical morphology of the gingiva

The color of the healthy gingiva is usually pale pink, but may contain melanin pigmentation to a different extent. The texture of the healthy gingiva is similar to the orange peel. It surrounds the teeth like a collar. Can be separated into buccal and oral gingiva and interdental gingiva.

The interdental gingiva is found in the interdental space (between the teeth) under the contact points of the teeth. This area is called gingival embrasure. The interdental gingiva may have a pyramidal tip called the papilla, and a shallow valley that connects the lingual and the buccal papilla, called col area (Figure 3.) There may be some variations depending on the contact point between the teeth. If diastema (gap) between the teeth is present, the interdental gingiva is rounded, without interdental papillae and col area (Figure 4.).

Figure 7.60. Figure 3. – The interdental gingiva after the avulsion of the tooth. Interdental papillae (buccal and lingual) shown with blue arrows. Col area (shallow depression between the buccal and lingual papilla) is shown with the red arrow. Note the border between the attached gingiva and the free gingiva (green arrow)
Figure 7.61. Figure 4. – The absence of papilla between the two central incisors, due to diastema (gap) between the teeth

7.1.4. Histology of the gingiva

A central core of connective tissue (lamina propria) is covered by the layer of stratified squamous epithelium (lamina epithelialis), separated by the basement membrane. There is no submucosa in the gingiva.

7.1.4.1. Gingival epithelium (lamina epithelialis)

Gingiva is covered by keratinized stratified squamous epithelium. The border of the epithelium and the connective tissue is irregular. Protuberances and depressions in the papillary layer (outer layer) of the lamina propria covered by the epithelium, producing the "orange peel" texture.

The epithelium consist of four layers:

- Stratum basale (basal layer, or stratum germinativum) is a layer of continuously proliferating keratinocytes producing the cells of the next layer
- Stratum spinosum (spinous layer), named after the shape of the cells caused by the desmosomal connections between the cells
- Stratum granulosum (granular layer) containing keratohyalin granules
- Stratum corneum, the outmost layer of dead cells, without nuclei and organelles.

The main function of the gingiva is to protect the underlying layers. This is achieved by the proliferation and the migration of keratinocytes. The keratinocytes proliferate by mitosis in the basal layer of the epithelial cells, and migrate to the spinous layer. The differentiation of the cells involves the process of keratinization during the migration. There is a flattening in the cells with increased number of tonofilaments. During differentiation the cells loose their mitotic activity. Heading towards the outer layers the cytoplasm comprises more keratohyalin granules. Finally the cells lose their organelles and nuclei through biochemical reactions, and turn into corneocytes (stratum corneum, “horned layer”). This process is called the orthokeratosis. If the cells retain their
organelles and nuclei, it is called parakeratosis. 70-80% of the gingival mucosa is parakeratinized, 20-30 orthokeratinized.

The total turnover of cells in the gingiva is around 25-30 days.

Other cells found in the epithelial lamina (next to keratinocytes) are:

- Langerhans cells in the spinous layer which are involved in the cellular immune defense of the gingiva, by activating T-helper cells
- Merkel cells tactile receptors in the basal layer of the epithelial lamina
- Melanocytes are found in the stratum basale.

Lamina propria

The gingival connective tissue (lamina propria) consists of:

- Papillary layer
- Reticular layer

Papillary layer consist of fine loosely arranged fibers. Papillary projection can be found between the rete pegs of the epithelial layer (epithelial extensions that project into the underlying connective tissue).

Reticular layer consists of dense irregular connective tissue.

The connective tissue consists of cellular (mainly fibroblasts, mast cells, white blood cells), and extracellular compartment (ground substance).

The main types of fibers found in the gingival are the type I, III and V collagen fibers. They radiate in the cementum, the periosteum of the alveolar bone, and directly in the alveolar bone. Their main functions are to connect the gingival to the tooth, to provide rigidity against mastication. According to their orientation they can be classified as:

A. Dentogingival fibers: running from the supraalveolar root cementum coronally (coronal), horizontally (horizontal) or towards the apex (apical).

B. Alveologingival fibers: running almost vertically from the alveolar border to the interdental and vestibule-oral gingiva.

C. Interpapillary fibers: connecting the interdental buccal and oral interdental papilla. They cross in the col area.

D. Transgingival fibers: fibers connecting the gingival fibers of the adjacent teeth.

E. Circular, semicircular fibers: supraalveolar position. Circular fibers are running in the marginal gingiva, they encircle the teeth. Semicircular fibers are found on the buccal side of the teeth.

F. Dentoperiostal fibers: running between the root cementum and the periosteum of the alveolar bone.

G. Transseptal fibers: also called interdental fibers running above the interdental alveolar septum and connecting the cementum of the adjacent teeth.

H. Periostogingival fibers: radiate from the buccal and lingual periosteum of the alveolus to the attached gingiva/running on the buccal and lingual side everywhere in the attached gingiva connecting the periosteum and the gingiva.

I. Intercircular fibers: connecting the intergingival fibers to the fibers running between the teeth.

J. Intergingival fibers: continuously running fibers converging towards the molar teeth.

7.1.4.2. Basement membrane
The epithelium and the underlying connective tissue is joined by the basal lamina. Consists of two layers: the lamina lucida (mainly laminin) and the lamina densa (type IV collagen fibers).

7.1.4.3. The gingival sulcus

The gingival sulcus is a virtual gap surrounding the neck of the teeth. The coronal border is the crest of the gingival margin and the apical limit is the junctional epithelium. The depth of the healthy sulcus ranges between 0.5-2 mm.

7.1.4.3.1. Sulcular epithelium

The gingival wall of the sulcus is lined by non keratinized stratified squamous epithelium; the opposite wall is the enamel of the tooth or the cementum in the elderly (Figure 5). There are no rete pegs in the sulcular epithelium. The sulcular epithelium has crucial role in the development of periodontal diseases.

Figure 7.62. Figure 5. – In the case of advanced gingival recession the opposing side of the sulcus is the root cementum

7.1.4.3.2. The junctional epithelium (JE)

Ten to thirty layer thick stratified squamous nonkeratinizing epithelium. The attachment between the tooth and the gingiva is exceptional in the body. It is formed by the oral epithelium and the reduced enamel epithelium during tooth eruption. The junctional cells are attached to the tooth surface (epithelial attachment) by an internal basal lamina produced by the reduced ameloblasts. The internal basal lamina is continuous with the external basal lamina that has the same structure as other basal laminae in the body. Consist of two groups of cells (Figure 5.):

- Basal (cuboid proliferating cells)
- Suprabasal.

The junctional epithelium attaches to the tooth surface through the the glycoproteins and hemidesmosomes of the internal and external basal lamina. Although polymorphonuclear leukocytes (PMN) are always present in the junctional epithelium. Junctional epithelial cells have low defense power against microbial attacks.
7.1.5. Blood and nerve supply of the gingiva

1. Blood supply

   - Supraperiosteal arterioles: on the buccal and the oral surfaces of the bone. Capillaries extend to the sulcus epithelium and to the rete pegs of the external gingival surfaces.
   - Vessels in the periodontal ligament reach the gingiva
   - Arterioles from the crest of the interdental septa.

2. Gingival innervations derive from the nerves of the periodontal ligaments, the buccal, labial and palatal nerves.

3. Lymphatic drainage: from the lamina propria to the regional lymph nodes.

7.2. Periodontal ligament (PDL)

Periodontal ligament is connective tissue rich in cells, ensuring resilient connection (gomphosis) between the alveolar bone and the cementum through type I collagen fibers (principle fibers), and type III reticular fibers. Other fibers of the periodontal ligament include immature forms of elastin fibers (oxytalan) running parallel to the surface, their exact functions yet unknown. The tasks of the periodontal ligaments are distribution of masticatory forces and the blood supply of the neighboring tissues. The fibers are synthesized by fibroblasts, osteoblasts, and other cells.

The fibers connecting the calcified tissues (bone, cementum) are called Sharpy’s fibers. The fibers are arranged in bundles of six type according to their orientation (Figure 6.):

   - Transseptal group: interproximally between the cementum of the adjacent teeth.
   - Alveolar crest group connects the cementum of root with the alveolar crest, running obliquely.
   - Horizontal group: are positioned perpendicularly to the surface of the root and the alveolar bone.
   - Oblique group: running from the cementum obliquely in a coronal direction. This is the largest group of fibers.
   - Apical group: they are found in the apical region of the teeth, their radiation is rather irregular.
   - Interradicular fibers: only found in multi rooted teeth in the furcation area.
Figure 7.64. Figure 7. – PDL fibers. TS: transseptal group, IR: interradicular group, AC: alveolar crest group, T: horizontal group, O: oblique group, A: apical group, IDS: interdental septum of the alveolar process, IRD: interradicular septum of the alveolar process

The cells of the periodontium are responsible for coordinating the physiological remodeling of the cementum and the bone.

- Fibroblasts: responsible for the control of collagen turnover. Intercellular desmosomes provide the bases for the 3D structure of the tissue.

- Cementoblasts only appear during the cellular cementum formation.

- Osteoblasts and osteoclasts are responsible for the remodeling of the alveolar bone.

- Epithelial rests of Malassez are cluster or latticework of cells remained after the disintegration of the Hertwig’s epithelial root sheath, some of the may be in connection with the JE. They decrease in number with age.

- Other cells found in the periodontal ligament, such as neutrophils, lymphocytes, macrophages, mast cells and eosinophil cells are responsible for immune defense of the tissue.
The functions of the periodontal ligament:

- Protection of nerves and vessels.
- Transmission of occlusal forces to the bone.
- Attaching the teeth to the bone.
- Maintaining the gingiva-dental connections.
- Shock absorption.
- It is assumed that the innervations of the PDL has proprioceptive functions.

7.3. Cementum

Found on the surface of the root. Mainly consist of a calcified avascular matrix and collagen fibers. The width of the cementum layer ranges between 20-250 µm with the widest layer of cementum usually located around the apex, to compensate attrition. It is considered to be part of the periodontium because periodontal ligaments anchor or originate from the cementum.

Cementum can be classified as primary or acellular cementum and secondary or cellular cementum. Cementum is formed by cementoblasts which differentiate from mesenchymal cells of the connective tissue of the dental follicle. They produce an organic intercellular matrix called the cementoid, which later is mineralized. In the acellular cementum the the cementoblasts stay on the surface, while in the case of cellular cementum they incorporate into the matrix (lacunae). After mineralization they are called cementocytes.

- Acellular cementum covers the coronal 1/3-1/2 of the root. Sharpey’s fibers are the main component of the acellular cementum. Sharpey’s fibers are calcified terminal endings of the PDL fibers.
- Cellular cementum is formed after the tooth has erupted in the apical region.

The cementum is arranged in lamellae parallel to the tooth surface. Incremental lines are visible between the lamellae representing the periodicity of cementum formation.

7.3.1. The composition of cementum

- Inorganic component- hydroxyapatite 45-50%.
- Organic components and water 50-50%.

7.3.2. Cementoenamel junction

- Cementum overlaps enamel 60-65% (Figure 7.).
- Edge to edge connection 30% (Figure 8.).
- The cementum and the enamel is separated 10% (Figure 9.).

Figure 7.65. Figure 8. – Cementoenamel junction: cementum overlaps enamel
Figure 7.66. Figure 9. – Cementoenamel junction: edge to edge connection

Figure 7.67. Figure 10. – Cementoenamel junction: cementum and the enamel is separated
7.4. Alveolar process (processus alveolaris)

- Part of the mandible and the maxilla, supporting the tooth by the tooth socket.
- Consists of the following layers (Figure 11; 12):
  - Cortical compact bone covered by periosteum on the lingual and the buccal surfaces (external plate).
  - Alveolar bone proper: the inner socket wall in connection with the PDL. Thinner than the external plate. It is perforated (cribriform plate). Also called lamina dura (radiographic).
  - Trabecular bone is a cancellous bone located between the alveolar bone proper and the cortical bone.

Basal bone is part of the alveolar process, apically from the teeth, but not in connection with them.

**Figure 7.68. Figure 11. – CBCT image, frontal slice: red arrow-cortical compact bone, green arrow-, trabecular bone, blue arrow- alveolar bone proper**
Figure 7.69. Figure 12. – CBCT image, frontal slice: red arrow-cortical compact bone, green arrow-, trabecular bone, blue arrow- alveolar bone proper, yellow arrow- periodontal ligaments
The part of the alveolar bone between the teeth is called the interdental septum. The most coronal part of the interdental septum is the alveolar crest or limbus alveolaris. The septum between the roots of multirooted teeth is called interradicular septum (Figure 6.).

7.5. Blood and nerve supply of the periodontium

7.5.1. Blood supply

- Mandibular blood supply: inferior alveolar artery, mental branch of inferior alveolar artery, lingual and buccal artery.

- Maxillary: superior alveolar arteries, infraorbital artery, greater palatine artery, incisive artery.

7.5.2. Nerve supply: second and third branch of trigeminal nerve

- Mandibular nerve supply:
  - Bone and PDL: inferior alveolar nerve.
  - Buccal gingiva distally from the premolars: buccal nerve.
  - Buccal gingiva mesially from the premolars: mental nerve.
  - Lingual gingiva: lingual nerve.

- Maxillary nerve supply
  - On the buccal side branches of the superior alveolar nerve.
7. Dental anatomy

- On the buccal side, in the front region the infraorbital nerve and the superior labial nerve.
- On the lingual side between the canines the incisive nerve, behind the canines the greater palatal nerve.

7.5.3. Lymphatic drainage

- Mandible:
  - Labial and sublingual region submental lymph nodes.
  - Molar and premolar region submandibular lymph nodes.
- Maxilla:
  - Palatal side: deep cervical lymph nodes.
  - Maxillary buccal side: submandibular lymph nodes.

7.6. Questions

1. What are the parts of the periodontium?
   A. A) enamel, dentin, cementum, periodontal ligaments
   B. B) alveolar process, cementum, gingiva, dentin
   C. C) alveolar process, cementum, gingiva, periodontal ligaments
   D. D) dentin, cementum, gingiva, periodontal ligaments

2. Characteristics of the gingival sulcus:
   A. A.) apical border is the epithelial attachment
   B. B.) the depth of the healthy sulcus ranges between 4-5 mm
   C. C.) valley that connects the lingual and the buccal papilla
   D. D.) None of the above

Right answers:
1. C
2. A

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8. 7.8. Morphology and histology of the salivary glands – Balazs Gaszner, Tibor Hollosy, Pal Toth

The salivary glands produce a thin film of fluid, which – under normal physiological circumstances – always covers the oral mucosa. Many of the important functions of the saliva, such as keeping the oral mucosa wet, defense against infections, beginning of digestion, preventing some tooth diseases, producing slippery surface to facilitate swallowing, etc. are discussed in physiological textbooks. This chapter only gives details of the macroscopy and histology of the salivary glands. The glands producing saliva are conventionally divided into groups of big and small salivary glands. To the former group the parotid, the submandibular and the sublingual glands belong.

8.1. Big salivary glands

8.1.1. Parotid gland

It is the largest of all the major salivary glands, located in front and below the external acoustic meatus. The gland sits in its own bed, called the parotid nest, which can be demonstrated either on a horizontal section of the head cut through the angle of the mouth, or on a preparation, after the removal of the gland. The latero-medial sequence of the anterior border of the nest is the masseter muscle, the ramus of the mandible, and the medial pterygoid muscle. The nest extends medially till the muscles originating from the styloid process, directly till the stylohyoid muscle, and may reach the carotid sheath. The posterior border of the nest is formed by the mastoid process superiorly and the anterior surfaces of the sternocleidomastoid and the posterior belly of digastric muscles. A strong fascia, the parotidomasseteric fascia forms the lateral wall of it, which extends anteriorly to the outer surface of the masseter muscle. The gland extends upward to reach the external acoustic meatus and the zygomatic arch. Inferiorly, the nest has no strict border, here the parotidomasseteric fascia from the angle of the mandible projects posteriorly to fuse with the superficial lamina of the cervical fascia enclosing the sternocleidomastoid muscle (angular tract of Eissler). Due to the absence of a strong lower border, the inflamed gland (parotitis, mumps) can extend down to the neck.

There is a number of delicate structures passing through the parotid gland, which can be injured during operation. The facial nerve enters at the posterior-medial margin of the gland and splits into two branches, the temporofacial and the cervicofacial trunks. The trunks unite to forms a plexus (parotid plexus) that splits the parotid gland into a superficial lobe and a deep lobe. The lobes are connected by the isthmus. From the parotid plexus, the terminal branches arise anteriorly, which innervate the muscles of facial expression. Hence, any damage to these fine nerves results in the paralysis of the corresponding muscle group. The auriculotemporal nerve, a sensory branch of mandibular nerve, pierces the parotid gland in medio-lateral direction. It innervates the temporomandibular joint, the external acoustic meatus, part of the auricle, and the skin of the temporal region, but also carries parasympathetic motor fibres, which control the secretory activity of the gland. These postganglionic fibres derive from the otic ganglion, to where the preganglionic fibres arrive through the lesser petrosal nerve (continuation of tympanic nerve from glossopharyngeal nerve). The terminal portion of the external carotid artery, and the beginning of its two endbranches, the maxillary and the superficial temporal arteries are also embedded in the gland, together with their accompanying veins and the retromandibular vein.

The parotid duct (Stensen’s duct) crosses horizontally the outer surface of the masseter muscle. The duct is frequently palpable here, if the muscle is contracted. After having pierced the buccal fat pad (of Bichat) and the buccinator muscle, it opens into the vestibule opposite the second upper molar tooth, on the parotid papilla. During oral interventions, a tampon inserted in the vestibule can prevent the continuous pouring of the saliva.

Histology of the parotid gland

The parotid gland is a lobulated organ. The lobules are visible with naked eyes, the connective tissue septa between them often contain nerves (see above). It is a compound tubuloalveolar gland, which utilizes merocrine secretion mechanism, and consists exclusively of serous acini. The cytoplasm of the acinar cells is basophilic, due to the abundant rough endoplasmic reticulum, the nucleus is round and more or less centrally located. Outside the basal lamina of the secretory cells, myoepithelial cells embrace the acini, the contraction of which play an important role in the release of the saliva.

Figure 7.70. Figure 1. – Low power magnification image showing the structure of the parotid gland. The gland is divided into lobules by numerous connective tissue septa (s).
The basophilic characteristics seen in the low power magnification image is determined by the histological features of the serous glands. Large secretory ducts (d) in the connective tissue septa are recognizable even in the low power magnification image.

Many adipocytes are scattered throughout the gland, and ducts of different sizes are abundant. The smallest ones, the *intercalated ducts* are lined with cuboidal epithelium. The bigger *striated* (or salivary) ducts have simple columnar epithelial lining. The cytoplasm of the columnar cells is eosinophilic. At the base of the cells, basal striation is seen, caused by the numerous infoldings of the basal cell membrane, and the accumulation of mitochondria in the cytoplasm between the foldings. The enlarged surface facilitates, the mitochondria serve energy for the intensive active ionic transport through the cells (Na+/K+ exchange). The big *excretory* (or *interlobular*) ducts are in the interlobular connective tissue septa, and are lined by pseudostratified, the biggest ones by stratified columnar epithelium.

The serous acini produce a thin digestive saliva, which is rich in α–amylase (ptyalin) that catalyzes the hydrolysis of starch. Antibacterial molecules (IgA and lysozyme) are also secreted. Mostly in newborn, it also contains lipase and growth factors (e.g. EGF, epithelial growth factor). The concentration of ions and salt in the saliva is controlled by the striated ducts. Some drugs, hormones and toxins may be excreted in the saliva. The parotid gland secretes approximately 20-30% of the total saliva.

**Figure 7.71.** Figure 2. – High power magnification of the parotid gland. Among serous secretory portions small secretory ducts (intercalated duct, i; and salivary ducts, s) can be observed.
8.1.2. Submandibular gland

The submandibular gland is situated in the submandibular triangle of the neck, which is bordered superiorly by the base of the mandible, anteriorly by the anterior belly of digastric muscle, and posteriorly by the stylohyoid muscle together with the posterior belly of digastric muscle. The gland lies superficial to the mylohyoid and hyoglossus muscles, and is covered by the superficial lamina of the cervical fascia and the platysma. The facial artery crosses the medial and superior surfaces of the gland in a deep groove, while the facial vein descends superficial to it. The deep part of the gland lies in the oral cavity between the hyoglossus muscle and the mandible, and reaches the posterior surface of the sublingual gland. The submandibular duct wraps around the posterior margin of the mylohyoid muscle, and passes anteriorly and medially within the sulcus lateralis linguae, i.e. between the mylohyoid and the hyoglossus muscles. It opens into the oral cavity at a small elevation next to the frenulum of the tongue called the sublingual caruncle. If the sublingual gland has a common duct, it frequently opens together with the submandibular duct. The postganglionic parasympathetic secretomotor fibres directly reach the gland from the submandibular ganglion that lies medial to the gland. The preganglionic fibres to the ganglion arrive through the lingual nerve, to which the chorda tympani carries them from the facial nerve.

Figure 7.72. Figure 3. – Low power magnification image showing the structure of the submandibular gland. The gland is divided into lobules by numerous connective tissue septa. The basophilic characteristics seen in the low power magnification image is determined by the histological features of the serous glands. Besides these, in lower proportion mucous (m) secretory portions can be observed, showing pale appearance.
Figure 7.73. Figure 4. – Submandibular gland, mucous (m) and serous (s) secretory portions
Histology of the submandibular gland

Like the parotid gland, the submandibular gland is also lobulated. The majority (70-80 %) of its acini is of serous type, the structure of which corresponds to those of the parotid gland. The cytoplasm of cells of the fewer mucous acini remain unstained or stain very pale eosinophylic with the conventional hematoxylin-eosin method. These cells produce mucin, which is very rich in carbohydrates. The use of specific carbohydrate stains therefore (e.g. PAS) results in the intense staining of cytoplasm of the mucous cells. The mucin is a glycoprotein with a great amount of carbohydrate that is added to the protein component in the Golgi apparatus. The well developed Golgi cisterns tend to compress the nucleus into the basal part of the cell. The lumen of the tubuloalveolar mucous acini is small, but somewhat wider then those of the serous ones. Small, sickle-shaped accumulation of serous cells may be found at the bottom of the mucous acini. These are frequently referred to as serous demilunes of Gianuzzi (a histotechnical artefact). The thick mucous or protective saliva acts as lubricant during mastication, swallowing and speech, and also defends the mucous membrane against minor mechanical injuries. The submandibular gland secretes approximately 65-75% of the total saliva.

**Figure 7.74.** Figure 5. – Serous demilunes of Gianuzzi at the mucous secretory portions of the submandibular gland (yellow arrows)

**Figure 7.75.** Figure 6. – Secretory duct profiles lined by simple columnar epithelium in the submandibular gland
8.1.3. The sublingual gland

The sublingual gland is the smallest of the three major salivary glands. It is located between the mucous membrane of the floor of the oral cavity and the mylohyoid muscle. The gland is bigger anteriorly and gradually narrows backwards. The anterior and posterior parts are frequently separated. A common duct, the sublingual duct (of Bartholin) that drains the anterior portion of the gland and opens into the oral cavity at the sublingual caruncle, may be present. The rest of the gland is drained by numerous small ducts opening on the fold of the mucous membrane above the gland (sublingual fold). The postganglionic parasympathetic fibres, as for the submandibular gland, originate from the submandibular ganglion, and reach the gland via the lingual nerve.

Histology of the sublingual gland

The histological structure of the sublingual gland is basically similar to that of the submandibular gland. It is also considered to be a mixed gland, but with the predominance of mucous acini. (It is worth mentioning that the so-called serous cells in the sublingual gland are immature mucous cells.) The duct system is poorly developed, the striated ducts are almost completely absent. As a consequence, the saliva produced by the sublingual gland is rich in sodium. The sublingual gland secretes approximately 5-10% of the total saliva.

Figure 7.76. Figure 7. – Low power magnification image showing the histological structure of the sublingual gland. The high proportion of mucous secretory portions is characteristic in the lobules bordered by connective tissue septa
Figure 7.77. – High power magnification image showing the mucous secretory portions of the sublingual gland
8.2. Minor salivary gland

Numerous minor salivary glands are scattered all along the oral mucosa, their estimated number varies between 400 and 700. The labial glands are located in tunica submucosa of the lips, and are readily palpable with the tongue as small, irregular nodes of the mucosa. Similar glands are beneath the oral mucosa of the cheek (buccal glands), the hard and the soft palate (palatal glands) and the tongue (lingual glands). The glands of von Ebner drain into the circular groove around the circumvallate papillae of the tongue, the saliva produced by them ensures the continuous wash-out of molecules from the gustatory buds and serves new taste sensations. A relatively voluminous gland is embedded in the anterior, apical part of the tongue (anterior lingual gland of Nuhn-Blandin), its duct opens on the fimbriated fold located on the inferior surface of the tongue.

The minor salivary glands are primarily mucous, except for the serous glands of von Ebner.

Figure 7.78. Figure 9. – Mucous secretory portions appear in a purple colour due to the their carbohydrate content in a PAS-strained preparation

Figure 7.79. Figure 10. – Low-power magnification photomicrograph showing the vallate papilla of the tongue. Yellow arrows point at the Ebner’s gland draining into to grooves next to the palillae
Figure 7.80. Figure 11. – For the Ebner’s glands are the serous secretory portions characteristic
Figure 7.81. Figure 12. – Numerous small salivary glands embedded into striated muscle tissue of the tongue can be observed, which may have either mucous (yellow arrow) or serous (green arrow) histological structure.

9. 7.9. Anatomy of the maxillary sinus – Balazs Gaszner, Tibor Hollosy, Pal Toth

The paranasal sinuses are air-filled spaces communicating with the nasal cavity. All are paired and vary in size and shape in different individuals. The sinuses are lined by the same mucous membrane (pseudostratified ciliated columnar epithelium with goblet cells and with numerous glands in the lamina propria) as that of the respiratory region of the nasal cavity but being thinner, less vascular and less adherent to bone in the sinuses. This similarity of the mucous membranes favours the spread of inflammations from the nasal cavity to the sinuses. The mucus film is swept into the nose through the apertures of the sinuses by kinocilia. The functions of the paranasal sinuses are complex: in addition to warm the inhaled air, they lighten the skull, add resonance to the voice and increase the surface area of the respiratory mucosa. The sinuses are rudimentary or absent at birth, and only enlarge considerably during the eruption of the permanent teeth and after puberty, appreciably altering the size and the shape of the face. The paranasal sinuses are the frontal, the ethmoidal (consists of 3 to 15 ethmoidal air cells, the ethmoidal labyrinth), the sphenoidal and the maxillary sinuses.

Figure 7.82. Figure 1. – Anteroposterior X-ray picture of the head. 1: orbit, 2: maxillary sinus, 3: frontal sinus, a: bony nasal septum, b: bony palate, S: superior.
9.1. Description of the maxillary sinus

The maxillary sinus (the cavity of Highmore*), occupying most of the body of the maxilla and having the greatest importance in dentistry, is the largest of paranasal sinuses. The left and right sinuses are frequently asymmetrical, their size is determined by the size of the nasal cavity as well as the overall width and height of the skull. In adults, the approximate anteroposterior length of the sinus is 34 mm, the vertical is 33 mm, the transverse is 23 mm, the average volume of it amounting to 15-20 ml.

Figure 7.83. Figure 2. – Frontal MR slice of the head. 1: orbit with the eyebulb and the extraocular eye muscles, 2: maxillary sinus, 3: tongue, a: ethmoidal air cells, b: inferior nasal concha, c: base of the mandible, S: superior
Figure 7.84. Figure 3. – Sagittal MR slice of the head. 1: orbit, 2: maxillary sinus, a: root of the tooth, S: superior
Figure 7.85. Figure 4. – Horizontal MR slice of the head. 1: maxillary sinus, 2: external acoustic meatus, 3: cerebellar hemispherium, a: nasal septum, b: inferior nasal concha, c: lateral pterygoid muscle, d: neck of the mandible, A: anterior
The maxillary sinus is pyramidal in shape, its base is formed by the lateral wall of the nasal cavity, whereas its apex extends into the zygomatic process of the maxilla. The roof is part of the floor of the orbit, frequently ridged by the overlying infraorbital canal. The fine branches of the infraorbital vessels and nerve passing within this canal reach the upper anterior teeth through delicate canals in the anterior wall of the sinus. Frequently, the wall of these canals are partially formed by bone, and the vessels and nerves are separated from the cavity of the maxillary sinus by its mucous membrane only. Canals descending to the upper middle and upper posterior teeth within the lateral and the posterior walls of the sinus, respectively, are similarly related to the cavity of the maxillary sinus. The anterior wall of the sinus corresponds to the anterior surface, the posterior wall to the infratemporal surface of the body of the maxilla. The latter protrudes backward as maxillary tuber, and its medial narrow part forms the anterior wall of the pterygopalatine fossa containing the final branching of the maxillary artery, a rich venous plexus and the pterygopalatine ganglion. The floor of the sinus is formed by the alveolar process of the maxilla, and lies usually 1-1.5 cm lower than the floor of the nasal cavity. Cone-like elevations produced by the roots of the first and second molar teeth (sometimes by the roots of the premolars and the wisdom teeth, occasionally by the root of the canine tooth) project into the floor, which they may perforate. When extracting fractured roots of these teeth, care must be taken to avoid making a fistula, a passage lined by epithelium connecting the oral cavity with the maxillary sinus (sinus apertus).
Figure 7. Lateral wall of the right half of the nasal cavity with reflected middle nasal concha. 1: frontal sinus, 2: anterior cranial fossa, 3: sphenoidal sinus, 4: aperture of the maxillary sinus, 5: middle nasal meatus, 6: inferior nasal concha, a: semilunar hiatus, b: incisive canal, c: palatine process of the maxilla, d: horizontal plate of the palatine bone, e: pharyngeal opening of the auditory tube, f: soft palate, A: anterior
On the medial wall facing the nasal cavity (nasal surface of maxilla) is the **maxillary hiatus**, which almost entirely occupies this surface. This opening is narrowed considerably by the neighbouring bones: the *ethmoidal bone* anterosuperiorly, the *inferior nasal concha* anteroinferiorly and the *perpendicular plate of the palatine bone* posteriorly. The definite opening of the maxillary sinus is its narrow *aperture*, which is located near the roof of the sinus. It opens into the lowest posterior part of a curved opening on the ethmoidal bone, the *semilunar hiatus*, which is bordered by the *uncinate process* anterior-inferiorly and by the *ethmoidal bulla* posterior-superiorly. Frequently, a second opening is also present in or just below the hiatus.

The maxillary sinus has several outpocketings (*recesses*), the size and shape of which vary considerably amongst individuals. These are**:

- **the anterior recess**; at the junction between the anterior and the medial walls of the sinus,
- **the ethmoidal recess**; high on the medial wall of the sinus,
- **the posterior recess**; at the maxillary tuber,
- **the zygomatic recess**; a funnel-shaped space extending into the zygomatic process,
- **the alveolar recess**; protrudes between the roots of the distal premolar and the mesial molar teeth. Larger recess may extend between the second and third molars, and occasionally between the distal incisive and the canine roots. The recess may invade the space between the palatine and buccal roots of molars (*interradicular sinus*) or may send processes low between the dental alveoli (*interdentalsinus*).

### 9.2. Development of the maxillary sinus

The maxillary sinus is the first paranasal sinus to develop. It appears at the beginning of the fourth intraembryonic month as an outpocketing of the nasal mucosa. It is small and contains fluid at birth. After birth, the maxillary sinus enlarges and pneumatizes in two phases. During the *first phase of pneumatization* (years 0-3), it grows backwards, whereas during the *second phase* (years 6-12), together with the vertical elongation of the face, the growth proceeds downwards. The sinus is fully developed after the eruption of the permanent teeth. At this time, its base lies lower than that of the nasal cavity, and is closely related to the roots of the fourth to
seventh teeth (see above). The cavity of the sinus may expand into the dental alveoli of the lost or extracted distal molar teeth.

9.3. Clinical considerations

Out of the paranasal sinuses, the maxillary sinus is of greatest significance for dentists. Because the dental alveoli of the molar teeth are only separated from the sinus by a thin bony plate (occasionally by the paradontium of these teeth and the mucous lining of the sinus), inflammation can easily spread from the sinus to the roots of teeth, or the suppuration of the roots can invade into the sinus causing sinusitis. During extraction of the molar teeth, the broken root of them may accidentally be pushed into the sinus (see above).

Inflammation of the mucous membrane of the paranasal sinuses (sinusitis) occurs frequently, typically caused by a cold and not related to tooth diseases. Most common and most severe is the sinusitis of the maxillary sinus. This can develop secondarily due to the flow of pus from the frontal and ethmoidal sinuses via the semilunar hiatus into the maxillary sinus, which becomes a reservoir for pus. The sinus is most often chronically infected, resulting in the loss of kinocilia of the lining epithelium and an impaired stream of the mucus film. Because the aperture of the sinus is high on its medial wall, the natural drainage of it is hindered. The chronic sinusitis may be treated by irrigation through a puncture (antrostomy) in the lateral wall of the nasal cavity below the inferior nasal meatus. The antrostomy is usually left open to allow drainage of the maxillary sinus into the inferior nasal meatus.

The nerves that supply the upper teeth are closely related to the maxillary sinus, therefore the pain arising from the sinus may irradiate into these teeth.

Fractures of the maxilla and damages to its sinus are classified according to Le Fort. The fracture Le Fort I is restricted to the alveolar process. In case of fracture Le Fort II, the body and the frontal process of the maxilla are damaged, together with the nasal bone and the medial part of the floor of the orbit. The serious trauma resulting in an almost complete separation of the viscerocranium from the base of the neurocranium is classified as fracture Le Fort III. In each of the above cases, the maxillary sinus is also involved.

A sudden impact of power delivered to the eye bulb enormously increases the intraorbital pressure that breaks the thinnest wall of the orbit, i.e. the roof of the maxillary sinus (blow-out fracture). In this case, orbital content (adipous tissue, muscle) may be pressed into the maxillary sinus causing orbital herniation.

In addition to the imaging methods, the maxillary sinus can be investigated by endoscopic techniques.

*Nathaniel Highmore, 1613-1685, British surgeon and anatomist

** The names in this paragraph are widely used by dentistry personnel but not included in Terminologia Anatomica

10. 7.10. Test questions (multiple choice)

1. Parts of the maxilla among the listed structures is/are:
   a. body of maxilla
   b. zygomatic process
   c. alveolar process
   d. pterygoid hamulus

2. The following statements is/are characteristic for the mandible:
   a. Intramembranous ossification.
   b. In its canal, the trunk of the mandibular nerve can be found.
   c. The mandibular notch is located between the processes of the ramus.
   d. Only muscles classified as muscles of mastication are inserted into it.
3. Articular surface(s) of the temporomandibular joint is/are:
   a. mandibular condyle
   b. neck of the mandible
   c. mandibular fossa
   d. mental tubercle

4. Movement(s) of the temporomandibular joint is/are:
   a. elevation
   b. rotation
   c. depression
   d. protrusion

5. It/they belong(s) to the muscles of facial expression:
   a. levator anguli oris muscle
   b. buccinator muscle
   c. mentalis muscle
   d. masseteric muscle

6. The following statements is/are characteristic for the muscles of mastication:
   a. They insert into the mandible.
   b. They are derivatives of the first pharyngeal/branchial arch.
   c. Their innervation is given by the branches of the mandibular nerve.
   d. Their main common function is the depression of the mandible.

7. It/they belong(s) to the branches of the maxillary artery:
   a. Inferior alveolar artery
   b. Buccal artery
   c. Posterior superior alveolar arteries
   d. Middle meningeal artery

8. It/they belong(s) to the branches of the inferior alveolar nerve:
   a. mylohyoid nerve
   b. dentales rami
   c. mental nerve
   d. nasopalatine nerve

9. Structure(s) belonging to the oral diaphragm:
   a. genioglossus muscle
b. mylohyoid muscle
c. verticalis muscle
d. geniohyoid muscle

10. It/they participate(s) in the sensory innervation of the tongue:
   a. lingual nerve
   b. hypoglossus nerve
   c. glossopharyngeus nerve
   d. maxillary nerve

11. Its/Their duct(s) drain(s) to the oral vestible:
   a. submandibularis gland
   b. buccal glands
   c. anterior lingual (Nuhn-Blandin) gland
   d. parotid gland

12. The extraction of the following tooth/teeth is/are often complicated by sinus apertus:
   a. 12
   b. 26
   c. 33
   d. 15

Correct answers:
1. A
2. B
3. B
4. E
5. A
6. A
7. E
8. A
9. C
10. B
11. C
12. C
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working length