HANDBOOK OF DENTAL HYGIENIST

Handbook of BSc level Dental Hygiene Program
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Preface

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Chapter 1. ANATOMY, PHYSIOLOGY, MICROBIOLOGY

1. Head and Neck anatomy (András Mihály, MD)

**BONES AND JOINTS**

The head (caput) and the neck (collum, cervix) have their bones: the skull (cranium) and the vertebrae of the cervical vertebral column. The bones are connected by joints: the skull bones are united by the sutures and a pair of synovial articulations, the temporomandibular joints. The cervical vertebrae join to each other in synovial joints, intervertebral connective tissue discs (intervertebral discs) and several ligaments. The first cervical vertebra (atlas) articulates with the skull (atlantooccipital joint). The second cervical vertebra (axis) articulates with the atlas in synovial joints (atlantoaxial joints), which, together with the atlantooccipital joint, participate in the movements of the head. There is one bone on the anterior side of the neck which does not articulate with other bones (hyoid bone). The hyoid bone is fixed by muscles and ligaments to the mandible and to the larynx.

1. **Skull (cranium)**

The skull has two main parts: the facial skull (viscerocranium) and the upper skull, which protects the brain (neurocranium). The viscerocranium forms several cavities, which protect organs: the orbit (eyeball and lacrimal gland are inside), the nasal cavity and the oral cavity.

A. **Viscerocranium**, the important bones except for the mandible, ethmoid and vomer, are paired.

- Zygomatic bone
- Nasal bone
- Vomer and inferior nasal concha (inside the nasal cavity)
- Maxilla (upper jaw)
- Mandible (lower jaw)
- Palatine bone
- Ethmoid bone

*The bones are connected by sutures, which are thin, connective tissue connections and unite the bones tightly. Therefore, the facial skull often reacts to injuries with bone fractures. Some of the bones of the facial cranium contain air-filled cavities (the paranasal sinuses) which are fragile structures, too. The maxilla contains the maxillary sinus, and the ethmoid bone has the ethmoid air cells inside. There is one movable, synovial articulation: the temporomandibular joint (between the mandible and the temporal bone).*

B. **Neurocranium**: two parts are distinguished, the roof (calvaria) and the basis (basis cranii: cranial base). The base has two important and functionally different surfaces: the internal base (basis cranii interna) and the external base (basis cranii externa). While the internal base is for the protection and holding of the brain, the external base is for the attachments of important neck structures: the pharynx and muscles. The cranial base is the site of entry and exit of several important structures into and from the the skull cavity: cranial nerves, internal carotid artery, and internal jugular vein. Bones of the neurocranium are as follows:

- Frontal bone (unpaired)
- Parietal bone (paired)
- Occipital bone (unpaired)
- Temporal bone (paired)
• Sphenoid bone (unpaired)

The calvaria is made by the frontal, occipital and parietal bones. The basis is made by every bone, except for the parietal. The bones are connected by sutures; sutures do not permit movements, therefore, the neurocranium is a fragile structure. The lower part of the frontal bone participates in the formation of the facial skull: it forms the orbit and contributes to the nasal cavity. The frontal bone contains a large paranasal sinus: the frontal sinus. The temporal bone contains a complicated cavity system: these are the cavities of the external ear and middle ear. The temporal bone also contains the labyrinth, which is the inner ear, with the delicate receptors of hearing and equilibrium inside.

2. The temporomandibular joint.

The articulation is between the head of the mandible and the articular fossa of the temporal bone. The surfaces are covered by fibrocartilage, and the articular cavity contains a fibrocartilage disc. The articular capsule is strong, the joint has four ligaments. Movements: opening and closure of the mouth (hinge movement, elevation and depression of the mandible), protrusion–retrusion of the mandible (gliding movement), and right and left lateral excursions. Combination of these in the two joint sides results in the complex movement patterns of mastication.


The seven cervical vertebrae joint to each other through movable synovial joints, ligaments and intervertebral fibrocartilage discs. The cervical vertebrae have a large vertebral foramen, which forms the vertebral canal for the cervical spinal cord. The segmental spinal nerves exit through the intervertebral foramina. The first and second cervical vertebrae (atlas and axis) join in the atlantoaxial synovial articulation, which participates in the movements of the head. The transverse processes of the cervical vertebrae possess a round opening (transverse foramen), which is for the vertebral artery: the artery runs in these foramina upwards, enters the foramen magnum and the cavity of the neurocranium, to supply the brain. The seventh cervical vertebra has a long spinous process, which is palpable under the skin of the posterior neck: this vertebra is the vertebra prominens (used when counting the vertebrae in a living subject).

THE MUSCLES OF THE HEAD AND NECK

The skeletal muscles form functional muscle groups insert on the skull, the cervical vertebrae, the hyoid bone, the thyroid cartilage, the upper two ribs, the clavicle and the scapula.

1. The muscles of the face (muscles of facial expression).

These muscles are inserted to cranial bones (maxilla, zygomatic bone, and mandible) and to the facial skin. Therefore, the muscles move the skin of the face. More importantly, these muscles move the lips and the wings of the nose, and close the eyes, therefore, participate in important functions (vocalization, eating, drinking, breathing and protection of the eyes). These muscles form the muscular wall of the oral cavity: the muscle in the lips (orbicularis oris muscle), and that in the cheek (buccinator muscle) contribute to the functions of the oral cavity. These muscles are innervated by the facial nerve. Paralysis of the facial nerve hinders (or inhibits) the functions of the muscles.

2. Masticatory muscles.

They are strong, paired muscles, which originate from different skull areas and insert without exception on some part of the mandible. They act in the temporomandibular joint. Innervation comes from the trigeminal nerve.

• A. Masseter muscle: on the lateral surface of the face, from the zygomatic arch to the outer surface of the mandibular ramus. It closes the mouth (elevation of the mandible).

• B. Temporalis muscle: originates in the temporal fossa (above and in front of the ear), inserts on the coronoid process of the mandible. Elevation and retrusion of the mandible.

• C. Medial pterygoid muscle: from the external cranial basis to the inner surface of the ramus of the mandible. It elevates the mandible (mouth closure).
• **D. Lateral pterygoid muscle:** from the external cranial base to the neck of the mandible. Effect: protrusion of the mandible.

3. **Suprahyoid muscles**

Connect the hyoid bone and the mandible. These muscles (mainly the mylohyoideus) form the floor of the mouth (diaphragma oris). They are able to depress the mandible (opening the mouth). Innervation comes from the facial and trigeminal nerves, and the cervical plexus.

4. **Infrahyoid muscles**

Muscles between the hyoid bone and the sternum, hyoid bone, thyroid cartilage and sternum, hyoid bone and the scapula. They move the larynx, and fix the hyoid bone. They are innervated by the cervical plexus.

5. **Sternocleidomastoid muscle**

The largest neck muscle stretching between the external cranial base and the sternum–clavicle. It is important in head movements, innervated by the accessory nerve and the cervical plexus.

6. **Trapezius muscle**

Large, broad muscle covering not only the dorsal surface of the neck, but also the dorsal surface of the upper back. It extends from the occipital bone to the lower thoracic vertebrae and to the scapula and clavicle laterally. It has an important role in the movements of the shoulder girdle. Innervation is from the accessory nerve and cervical plexus.

7. **Deep neck muscles**

Long muscles deep to the trapezius and behind the larynx and pharynx–oesophagus. They move the cervical vertebrae, and innervation comes from the cervical plexus.

8. **Fascia**

Sheaths cover the neck muscles, separating them from each other. Therefore, the neck displays layers, which are important to the surgeon. The fascia layers cover the pharynx, the thyroid gland and separate them from the larynx. The common carotid artery, internal jugular vein and vagus nerve are covered by one of these fascia layers: the carotid sheath.

**BLOOD SUPPLY AND LYMPH DRAINAGE OF THE HEAD AND NECK**

Arterial blood supply is mainly from the common carotid artery (CCA). The lower neck is also supplied by the subclavian artery. The CCA runs on the side of the neck, and bifurcates at the level of the 4th cervical vertebra (or upper edge of the thyroid cartilage) forming the external carotid and internal carotid arteries. The internal carotid artery (ICA) has no branches on the neck, instead it proceeds towards the external cranial base, and enters the skull cavity to supply the brain. The external carotid artery (ECA) is anterior to the ICA above the bifurcation and travels towards the viscerocranium, giving several branches to the organs on the neck and on the face. The ECA supplies the larynx, thyroid gland, large salivary glands, soft tissues and bones of the face, oral cavity, teeth, nasal cavity, pharynx and lower part of the orbit. It also gives branches to the ear. One branch enters the skull to supply the meninges.

The venous blood is drained by three large veins: the internal jugular vein, the external jugular vein and the anterior jugular vein. These three large veins discharge into the vein system of the superior vena cava.

Several groups of lymph nodes are found on the head and neck: the most important are the retroauricular, the parotid, the submental and the submandibular nodes. Deep lymph nodes of the neck are located around the internal jugular vein. The main lymphatic trunk of the neck connects these nodes and is called the jugular trunk. The lymph vessels from the head and neck are drained by lymph vessels (amongst them, the jugular trunk) into the large veins of the superior vena cava system.

**ANATOMY OF THE ORAL CAVITY**

The oral and nasal cavities are parts of the viscerocranium. They are made not only by the bones, but also by some muscles and other soft tissues. The oral and nasal cavities continue in the pharynx, the larynx and finally
the oesophagus and the trachea. The oral cavity contains the tongue and the teeth and the excretory ducts of the large salivary glands. The oral cavity is lined by a thick mucous membrane. The oral cavity is divided into two spaces: the oral vestibule between the lips, cheeks and the teeth; and the oral cavity proper which is a large space, and continues into the pharynx. The oral cavity proper is lined by the dental arches. The roof of the cavity is the palate, and the floor is formed by the suprhyoid muscles (diaphragma oris).

1. **Bones of the oral cavity**

The mandible and the maxilla are the lower and upper jaws. The palatine bones (2) contribute posteriorly. The maxilla is a paired bone: the two bones unite in a midline suture. The jaws have small cavities for the teeth: these are the dental alveoli. The teeth are fixed in them with connective tissue fibers. The maxilla has a flat process which forms the palate. The palatine bones attach to the maxilla from behind, and contribute to the palate with bony processes. The hard (or bony) palate separates the oral and nasal cavities.

2. **The muscles of the oral cavity**

The floor of the oral cavity is made by the suprhyoid muscles (mainly the mylohyoid muscle – this muscle is also known as the diaphragm of the oral cavity). The lateral and anterior walls of the oral cavity are the cheeks and the lips, and they contain strong muscles. Two of them are of particular importance:

A. *Orbicularis oris muscle*: strong, circular skeletal muscle having importance in speech, vocalisation and feeding.

B. *Buccinator muscle*: the muscle of the cheek, its fascicles attach to the orbicularis oris at the angle of the mouth. Both muscles are innervated by the facial nerve.

3. **Anatomy of the tongue**

A. *Parts*: apex, body, and radix. The upper surface is known as the dorsum (back). The inferior surface is attached to the floor of the mouth by means of the frenulum (visible fold of the mucous membrane). The posterior part of the radix is connected to the epiglottis (see at the larynx). The connections between the tongue and the epiglottis are the glossoepiglottic folds.

B. *Muscles*: skeletal-type muscles. Intrinsic muscles form and shape the tongue. Extrinsic muscles connect the tongue to extraoral structures (hyoid bone, mandible, soft palate, and styloid process), and move the tongue.

C. *Innervation*: the epithelium of the tongue contains sensory epithelial cells and nerve endings for the taste sensations. These small structures are the taste buds. They are innervated by the facial and the glossopharyngeal nerves. Other sensations (thermal, pain, touch) are provided mainly by the trigeminal nerve. The muscles are innervated by the hypoglossal nerve.

4. **Anatomy of the soft palate**

Muscular plate attached to the bony palate from behind. The soft palate consists of five pairs of skeletal muscles which connect to the pharynx and the tongue. The soft palate participates in the deglutition: it separates the nasal and oral cavities and prevents the entry of food into the nose.

5. **Anatomy of the teeth (dens, dentes)**

sitting in the dental alveoli, the teeth are fixed by strong but microscopic connective tissue fibers to the alveolar bone. This connection is the periodontal ligament. The periodontal ligament not only fixes but also supplies the tooth radix with blood capillaries. The periodontal ligament is richly innervated with sensory nerve endings.

A. *General morphology of the teeth*: crown (corona), neck (cervix) and root (radix). Inside the tooth there is a cavity (cavitas dentis) which contains a soft tissue, the pulp, capillaries and sensory nerves. Histology: hard, calcified tissues (enamel, dentin, cement). The dentin and the cement contain cells (odontoblasts and cementocytes).

B. *Superior (upper) and inferior (lower) dental arch, divided into quadrants*. Diphyodont teeth: milk teeth (deciduous teeth) and permanent teeth. The permanent teeth in a quadrant: incisors (2), canine (1), premolars (2) and molars (3).
C. Blood supply and innervation: the blood supply is provided by the branches of the maxillary artery (from ECA). Sensory innervation is from the trigeminal nerve (maxillary nerve for the upper; mandibular nerve for the lower teeth). The nerve of the lower teeth (inferior alveolar nerve) runs inside the mandible in a bony canal (mandibular canal). The nerves of the upper teeth reach the teeth in several groups through the maxilla.

6. The anatomy of the throat and the pharynx

The throat (isthmus faucium) is the passage between the oral cavity and the pharynx. It is formed by the radix of the tongue and the soft palate. A muscular process of the soft palate hangs down in the middle: this is the uvula.

Important structures: the tonsillar fossa and the palatine tonsil. The pharynx is a muscular tube connecting the nasal and oral cavities to the oesophagus. The pharynx has three sections, according to the cavities and passages which open into it.

• A. Nasal part: the nasal cavity opens into it. It has a tonsil, the pharyngeal tonsil and the auditory tube opens into it as well (the auditory tube is connecting the tympanic cavity – middle ear – to the pharynx).

• B. Oral part: the oral cavity opens into it with the throat.

• C. Laryngeal part: this section is behind the larynx; it continues in the oesophagus.

7. Large salivary glands

Three pairs of large exocrine glands supply saliva for the oral cavity. Each has excretory duct(s) which open(s) into the oral cavity at visible locations. The glands are activated through parasympathetic innervation (facial and glossopharyngeal nerves bring parasympathetic nerves from the brainstem).

• A. Parotid gland: located in front of and above the ear, the excretory duct runs on the side of the face (on the surface of the masseter muscle) and discharges into the oral vestibule at the level of the second upper molar tooth.

• B. Submandibular gland: located beneath the basis of the mandible in the submandibular trigone. The excretory duct of the gland enters the oral cavity from behind and opens on the floor of the mouth, on the summit of the sublingual papilla.

• C. Sublingual gland: located on the floor of the oral cavity, covered by the thick mucous membrane. The gland has several short excretory ducts which open on the top of a mucous membrane fold, the sublingual fold (on the bottom of the oral cavity).

ANATOMY OF THE NASAL CAVITY

Functions of the nasal cavity are: respiration and olfaction. It is covered by a thick mucous membrane which filters and warms up the air. In the upper part of the nasal cavity, we find olfactory epithelium with special sensory cells for the detection of smells. The nasal cavity is made by bones, cartilages and soft tissues. The cartilages make up the external nose and participate in the formation of the nasal septum. The nose has its own striated muscles (nasalis muscle), which may help the opening of the nostrils.

• A. External nose (nasus): part of the face, above the mouth, made up by the nasal bones, cartilages and muscles. Apex, ala (wing), nostrils and septum.

• B. Nasal cavity: nasal septum in the midline (cartilage and bone), nasal conchae (bony shells on the lateral wall), nasal meatuses (spaces under the conchae). The nasal cavity is surrounded by bones which contain air-filled cavities: the paranasal sinuses (maxillary sinus, frontal sinus, sphenoid sinus and ethmoid air cells).

• C. The paranasal sinuses are covered by mucous membrane and open into the nasal meatuses. The nasal cavity opens into the nasal part of the pharynx: the openings are called choanae.

ANATOMY OF THE LARYNX

The larynx is an important part of the respiratory system. It is not only for respiration, but also for vocalisation and speech. The larynx is in the middle of the neck with some palpable parts under the skin. The skeleton of the larynx is made up of five large cartilages. These cartilages join to each other with movable synovial joints. The cartilages are moving through striated laryngeal muscles. The pharynx is behind the larynx, and the two
structures join to each other by the pharyngeal muscles (thyropharyngeus, cricopharyngeus). The larynx is also connected to the hyoid bone, therefore, it moves during deglutition. The larynx is covered by mucous membrane. Sensory and motor innervations are provided by the vagus nerve.

1. Laryngeal cartilages:
   - thyroid cartilage (1),
   - cricoid cartilage (1),
   - arytenoid cartilages (2),
   - epiglottis (1).

   *The cartilages are connected by ligaments and synovial joints. The thyroid and cricoid cartilages are palpable under the skin of the neck.*

2. Laryngeal muscles: small striated muscles attached to the cartilages. They move the cartilages, therefore, they open or close the cavity of the larynx.

3. Laryngeal cavity: the aditus of the larynx opens behind the tongue and is protected by the epiglottis during swallowing. The epiglottis is partly connected to the tongue, partly to the hyoid bone, partly to the thyroid cartilage. Next to the aditus, we find the laryngeal vestibule, which is behind the thyroid cartilage. The laryngeal vestibule is sensitive to oedema. The glottic space (or glottis) is next, the space between the vocal folds. The vocal folds are stretching between the arytenoid and the thyroid cartilages. The glottis is protected by the thyroid cartilage. The size of the glottic space is regulated by the laryngeal muscles: such as the open and closed glottis positions and the size of the space between the vocal folds. Paralysis of these muscles may cause suffocation.

ENDOCRINE GLANDS ON THE NECK

The thyroid gland is located on the two sides of the larynx. The gland has two lobes and a thin connecting segment. Normally, the glandular tissue is soft and cannot be palpated under the skin. The gland produces iodine containing hormones (thyroxine and triiodothyronine), which stimulate the metabolism. On the posterior side of the lobes of the thyroid gland, four small (pea-sized) glands are present: the parathyroid glands, which produce the parathormone. Parathormone regulates the calcium homeostasis of our body.

2. Development of the craniofacial region (Emil Segatto DMD)

Variety in the size and position of craniofacial structures require all experts working in the field of dentistry to be familiar with development and growth processes in detail. For those who primarily deal with children, it is inevitable to distinguish normal variations from pathologic processes. By selecting adequate therapeutic tools, orthodontists apply this knowledge to manipulate not just the position of the teeth but also that of facial bones in the growth period of the patient in order to avoid well-known adverse effects.

The first pharyngeal arch having separate maxillary and mandibular prominences is part of the pharyngeal apparatus developing during the fourth intrauterine week; it plays a central role in craniofacial development. Development of the face occurs primarily between weeks 4 and 8 of embryonic life, so by the end of the eighth week the face is going to have a “human” appearance. A number of facial prominences fuse between weeks 7 and 10: the maxillary prominences fuse laterally with the mandibular prominences, while the medial nasal prominences fuse with the maxillary prominences and lateral nasal prominences. After this, further facial developments take place more slowly and they are restricted to changes in facial proportions.

Bones of the facial skeleton develop in different ways. The nasal bone, lacrimal bone, maxilla, zygomatic bone, palate and the medial disc of the pterygoid process develop through intramembranous ossification of the maxillary prominence. The mandible develops as a direct condensation of the mesenchyme lying laterally to Meckel’s cartilage of the first pharyngeal arch.

The maxilla develops entirely by intramembranous ossification postnatally. Usually it happens in two ways:

1. by apposition of the bone at the sutures connecting the maxilla to the cranial base;
2. by surface remodelling.

The maxilla grows downward and forward. The mandible grows in a different way due to the cartilage cover of the mandibular condyle. Endochondral activity is typical here, while other parts of the mandible form and grow by surface apposition and remodelling. Translational shift related to growth and to the cranial base of the maxilla takes place forward and downward due to upward and backward growth of the mandibular ramus. Of the growth theories, “functional matrix theory” by Moss is the most realistic one suggesting that the growth of facial skeleton bones is determined by the surrounding soft tissue in which the jaws are embedded.

The growth of the human body is achieved on the basis of an inherited growth pattern characterised by different rates in each age group and affected area. At about the third month of intrauterine development, the head takes up almost 50% of the total body length. At this stage, the cranium is much larger relative to the face. In contrast, the limbs are rudimentary and the trunk is still underdeveloped. By the time of birth, the trunk and limbs have grown fast, so that the proportion of the entire body taken up by the head has decreased to about 30%. The overall pattern of growth thereafter follows this course, with a progressive reduction of the relative size of the head to about 12% in adults. According to the normal growth pattern, the development of body length extends from head to feet, indeed, muscular and skeletal elements grow faster than the brain and central nervous system. This cephalocaudal growth gradient also characterises head and face development, resulting in gradual changes in their relative proportion. When the skull of a newborn is compared proportionally with that of an adult, it is easy to recognize that an infant has a relatively larger cranium and a smaller face. This alteration in proportionality is going to change later in life due to a more significant growth of the face relative to the cranium, which is an important aspect of the pattern of facial development. When the facial growth pattern is viewed against the perspective of the cephalocaudal gradient, it is not surprising that the mandible, being farther away from the brain, tends to grow more and later than the maxilla, which is situated closer to the cranial base.

An important characteristic of the growth pattern is predictability, which refers to proportion development / alteration. On the other hand, variability is one of the features referring to the existence of extreme forms falling within the normal range. It is necessary to have a wide knowledge of extremes as well in order to distinguish normal and abnormal conditions. Finally, it is timing that forms the third main characteristic of the growth pattern. Variants different from normal can also develop due to different timing effects. Such cases may occur during craniofacial development and can be detected by examining the development of skeletal, dental or soft tissue components. All children experience rapid growth at the beginning of adolescence, causing a significant increase in weight and height. This growth spurt can be detected at different times in different individuals, and it does not even show a close correlation with the chronological age; however, of facial skeletal bones it affects the mandible the most. Its precise determination is important to find the right timing of conservative mandibular-orthopaedic interventions influencing mandibular growth. After this spurt, there is a dramatic decrease in the growth of craniofacial structures, and it terminates around the age of 18-20 – earlier in girls than in boys.

3. Teeth morphology (Angyalka Segatto DMD)

DEFINITION, IMPORTANCE

Dental anatomy is defined as the study of the development, morphology, function and identity of each tooth in the human dentition, as well as the way in which the teeth relate in shape form, structure, colour and function to the other teeth in the same dental arch and the teeth of the opposing arch.

The study of dental anatomy, physiology and occlusion provides one of the basic components of the skills needed to practise all phases of dentistry.

FUNCTION OF THE TEETH

Primary function: to prepare food for swallowing and facilitate digestion. Facilitate prehension, incision and trituration of food.

Secondary function: articulation – speech.

Tertiary: important role in socialisation.

CHARACTERISTICS
Diphyodont: primary dentition followed by permanent dentition.

Heterodont: different types of teeth for different purposes.

Thecodont: human teeth are situated in a bony socket.

TERMINOLOGY

Alveolar bone: The bone of the maxilla or mandible that surrounds and supports the teeth

Alveolus: The bony lining of the socket that holds the root(s) of the tooth and is a portion of the maxillary and mandibular process

Anterior: Along or toward the front of the dental arch

Apical foramen: The main opening at the apex of a root for entry and exit of pulp tissue

Arch: In dental anatomy, the arrangement of the teeth in the form of a curve

Buccal: Toward the cheek; the surface of posterior teeth (premolars, molars) in contact with or facing the cheek

CEJ - Cementoenamel Junction: The junction of the cementum and the enamel (visible as the cervical line running along the cervix (neck) of the tooth

Cementum: Hard tissue forming the outer cover of the root of a tooth and surrounding the dentin in the root portion of the tooth

Crown: part of the tooth from the CEJ to the incisal/occlusal surface that is covered by enamel

Crown - anatomical crown: The entire crown from the CEJ to the incisal/occlusal surface is more strictly called the anatomical crown

Crown - clinical crown: The visible portion of the crown is called the clinical crown

Dentin: Portion of the tooth underlying the enamel and cementum and surrounding the pulp cavity. Dentin comprises the bulk of the tooth

Distal: Away from the midline of the arch

Enamel: Hard, mineralised tissue forming the outer cover of the anatomical crown of a tooth and surrounding the dentin in the crown portion of a tooth

Facial: Relating to or involving the face; an inclusive term for the labial and buccal surfaces, both being in direct contact with the face

Gingiva: Part of the periodontium that is composed of mucosal tissue and surrounds a tooth at the cervix and extends to cover the maxillary and mandibular alveolar processes

Incisal: Pertaining to the cutting or tearing surface of the anterior teeth (incisors, canines)

Labial: Toward the lips; surface of the anterior teeth directly contacting or facing the lips

Lingual: Toward the tongue; surface of the anterior and posterior teeth immediately adjacent to or facing the tongue

Mandibular: Relating to the mandible or lower jaw

Maxillary: Relating to the maxilla or upper jaw

Mesial: Toward the midline of the arch. The surface of anterior and posterior teeth facing the midline

Midline: An imaginary line dividing the body into left and right halves; an imaginary line dividing the maxillary and mandibular left and right quadrants
Occlusal: Pertaining to the grinding, crushing, and chewing surface of the posterior teeth

Periodontal ligament: A complex of collagenous fibre bundles that surrounds the root(s) of a tooth and connects the cementum with the alveolar bone

Periodontium: An inclusive term for the supporting and investing structures of a tooth. The main structures are gingiva, alveolar bone, cementum, and periodontal ligament

Posterior: Along or toward the back or rear of the dental arch

Pulp: Innervated connective tissue possessing the formative, nutritive, sensory, and defensive functions essential to the nourishment and vitality of a tooth

Pulp cavity: The space or void housing the entire dental pulp

Pulp chamber: Portion of the pulp cavity in the coronal section of a tooth

Pulp canal: Portion of the pulp cavity that extends from the pulp chamber to the apex of the root of a tooth

Quadrant: Half of the maxillary or mandibular arch when divided by the midline

Root: Portion of a tooth covered by cementum and embedded in a bony socket called alveolus

Root apex: The end or furthest point of the root of a tooth

Vestibule: Portion of the oral cavity bounded on one side by the teeth, gingiva, and alveolar ridge; and on the lateral side by the lips and cheeks. Referred to as buccal, labial and/or facial vestibule

INCISORS

Maxillary central incisor

vestibular (V), oral (O), mesial (M), distal (D) and incisal (I) view

Maxillary lateral incisor

vestibular (V), oral (O), mesial (M), distal (D) and incisal (I) view
Mandibular central incisor

vestibular (V), oral (O), mesial (M), distal (D) and incisal (I) view

Mandibular lateral incisor

vestibular (V), oral (O), mesial (M), distal (D) and incisal (I) view

General features:

• 4 maxillary and 4 mandibular incisors
• centred in the maxilla/mandible
• two on either side of the median line
• the central incisors are the only neighbouring teeth with mesial surfaces in contact (unless a gap or diastema exists)

Characteristics:

• relatively straight incisal edge
• rectangular shape: longer incisocervically than mesiodistally
• taper from contact to cementoenamel junction (CEJ)
• CEJ curves toward apex -lingual surface narrower (best seen incisally)
• marginal ridges converge toward the cingulum
• wedge-shaped (triangular) outline
• bulge greatest in cervical third facially and lingually (cingulum)
• lingual outline is “S” -CEJ curves incisally
• root tapers to apex
• when present, distal root depression is greater than mesial
• concave lingual fossa incisal to cingulum
• labial outline is broader (less convex) than lingual

Characteristics of ALL incisors, EXCEPT Mandibular Centrals:
• distal crown surface is more convex than mesial
• mesioincisal corner is sharper than distoincisal
• distal contact is located more cervically than the mesial contact area
• incisal edges slope cervically in a distal direction

Functions of incisors:
• cut food
• articulate speech
• support lips (aesthetics)
• guide mandible during movement.

Vestibular view:
The Maxillary central incisor has a wider, longer crown. Maxillary central incisor crown is the longest among incisors. Maxillary lateral incisor is relatively narrower mesiodistally. Maxillary lateral incisor is more often missing or peg shaped. Maxillary central incisor has mesioincisal angle at nearly right angle. Maxillary lateral incisor has both mesioincisal and distoincisal angles more rounded than in centrals.

Mandibular central incisor is symmetrical. Mandibular lateral incisor is bigger than central. Mandibular lateral incisor has a distoincisal angle that is more rounded than the mesioincisal; mesio- and distoincisal angles are equal in centrals. Mandibular central incisor has mesial and distal contact at the same level. Mandibular lateral incisor has mesial contact more incisal than distal. Mandibular incisor roots appear to be longer relative to the crown than do maxillary incisor roots.

Lingual view:
Lingual surface narrower. Marginal ridges converge towards the cingulum.

Maxillary laterals are more likely to have deeper lingual fossae and pits than centrals. Maxillary central incisor cingulum is more developed and is off-centred toward the distal (best seen from incisal view); cingulum of lateral incisor is more centred. Maxillary lateral incisor distal ridge appears shorter than mesial (more so than in centrals) due to the sloping of incisal edge shorter distally and distal placement of cingulum. Maxillary laterals are more likely to have deeper lingual pits than centrals. All incisor roots have lingual surfaces that are convex. Roots taper towards the lingual end.

Mandibular incisors distal bend of root tip is more common than mesial bend. Mandibular lateral cingulum is more distal to the midroot axis than in centrals (best seen incisally). All mandibular incisors have minimal marginal ridges and fossa depth (compared to maxillary incisors). Both mandibular incisors have mesial and distal root depression more prominent than in maxillary incisors (better appreciated when comparing mesial and distal views). Mandibular lateral incisor (mesial view) has its distolingual corner of the incisal edge showing lingually due to the distolingual twist; centrals do not. CEJ curves incisally more on the mesial than distal; the most CEJ curvature in the mandibular arch is on the mesial of the central incisor.

Proximal view:
Wedge-shaped (triangular) outline. In all anterior teeth the mesial CEJ curves more than the distal. The largest CEJ curve of all teeth is the mesial of the maxillary central incisor. Both maxillary central and lateral incisors have the peak of contour (facial and lingual) in the cervical third. Maxillary central root has a flatter lingual outline in the cervical third, then tapers. Laterals taper more evenly.

**Incisal view:**

Concave lingual fossa can be seen incisal to cingulum. Both maxillary incisors have mesiodistal surface greater than the faciolingual, but more so for the maxillary centrals. Laterals are closer to equal. Maxillary central has a triangular crown outline (because the facial surface is flatter). Maxillary lateral has a more round oval outline. Maxillary lateral has more labial curvature (is more convex) than central. Maxillary central exhibits a distolingual twist of the incisal edge; the lateral does not. Maxillary central has cingulum distal to root midline. Incisal edge runs between the widest mesiodistal point. Labial outline is broader (less convex) than lingual. Mandibular lateral cingulum is more distal to the midroot axis than in central (best seen incisally). All mandibular incisors have a faciolingual dimension greater than the mesiodistal. Mandibular central incisors are symmetrical; laterals are not. Mandibular lateral incisor has a cingulum distal to mid-root axis; central does not. Lateral incisor has an incisal edge with distolingual twist; central does not.

**CANINES**

**Maxillary canine**

vestibular (V), oral (O), mesial (M), distal (D) and incisal (I) view

**Mandibular canine**

vestibular (V), oral (O), mesial (M), distal (D) and incisal (I) view

**General features:**

- canines are the third tooth from the median line
- 4 canines placed at the “corner” of the mouth
- close resemblance to each other

**Characteristics:**
• canines are the longest teeth in the mouth
• the crown is usually as long as the maxillary central incisors
• have a single root, the longest of all roots

Function:
• support lips and cheeks
• cut, pierce or shear food
• support the incisors and premolars
• “canine guidance” in the intercuspal position.

Vestibular view:
The canine teeth have no incisal edge but a cusp. The incisal ridge is divided into mesial and distal cusp ridges forming the cusp. The facial surface is formed by three lobes: the middle one forms a labial ridge which is more prominent in maxillary canines. The mesial cusp ridge is shorter than the distal. The facial outline is pentagon-shaped. The distal contact is more cervical than the mesial. The crown is more convex on the distal portion. The crown tapers toward the cervical portion. The roots are much longer than the crowns. The mesial crown contour of the maxillary canines is convex in the middle third but nearly flat in the cervical third, whereas the mandibular mesial contour is almost flat or convex and it is almost in line with the root contour. The mandibular canine crown appears to bend more distally relative to the root. The tip of the maxillary cusp is sharper than that of the mandibular. The mandibular cusp is more obtuse (blunter) with its mesial ridge closer to horizontal. The contact areas are more incisal in mandibular canines. The distal contact of the maxillary canine is the most cervical of all canine contacts; it is in the middle third. The length of the maxillary canine crown is almost the same as that of the maxillary central incisor. The mandibular crown is considerably longer than the mandibular incisor crown.

Maxillary canine roots are more likely to bend distally at the apex than mandibular ones; the mandibular apex is blunter.

Palatal/lingual view:
Lingual ridge is more prominent in maxillary canine (with mesial and distal fossae). Maxillary canine cingulum is larger than in mandibular canine. Maxillary canine cingulum and cusp tip are more centred over root axis than in mandibular canine: cingulum is more distal to root axis (like mandibular lateral incisors and maxillary central incisors). Maxillary marginal ridges are more prominent than mandibular.

Proximal view:
Proximal outlines of crowns are wedge shaped (triangular) with lingual “S” shape. Maxillary canines are thicker. Mesial cementoenamel junction (CEJ) is more curved than distal.

Cusp tips of maxillary canines are more labial to midroot axis, whereas mandibular tips are more lingual or centred. Both facial and lingual contours height are in the cervical third. Maxillary canine facial height of contour is farther from CEJ and more prominent than in mandibular canines where contour is more cervical (so minimal that the tooth and crown contour may appear to be continuous). Both maxillary and mandibular canine CEJ curvatures are greater mesially than distally, but the mandibular curve may appear even greater due to narrow facial/lingual dimension. Both have vertical root depression greater than mesial (even more so in mandibular canines).

Incisal view:
From incisal view crowns are greater labioliungually than mesiodistally (even more so for roots) than mandibular incisors. Marginal ridges taper toward cingulum (lingual half narrower than facial). Lingual contour is more curved (convex) than labial. Root tapers toward apex (and lingually). Distolinguial twist of crown apparent in mandibular canines, not in maxillary. Cingulum often distal to centre in mandibular canines compared to maxillary canines so marginal ridge appears longer than distal (especially in mandibular canines). Labial outline
of both canines are more rounded than lingual. Distal half of labial surface in maxillary canine crown is more prominently concave. Lingual ridge on maxillary canines less evident on mandibular.

PREMOLARS (Fig. 7., 8., 9., 10.)

Maxillary first premolar
vestibular (V), oral (O), mesial (M), distal (D) and occlusal (O) view

Maxillary second premolar
vestibular (V), oral (O), mesial (M), distal (D) and occlusal (O) view

Mandibular first premolar
vestibular (V), oral (O), mesial (M), distal (D) and occlusal (O) view
Mandibular second premolar
vestibular (V), oral (O), mesial (M), distal (D) and occlusal (O) view

General features:
• premolars are the fourth and fifth teeth from the median line
• 4 premolars in the upper jaw, 4 in the lower jaw

Characteristics:
• crown is smaller than in molars
• have one root, except for the maxillary first, which have 2 roots

Function:
• mastication - maintaining vertical dimension of face - assist canines with shearing or cutting food
• support cheeks and corners of the mouth.

Vestibular view:
Pentagon outline of crown, contact areas are convex; distal is more cervical than mesial (EXCEPT mandibular first premolar). Mesial cusp ridge shorter than distal (EXCEPT maxillary first premolar).

Maxillary first premolar is larger than second. More convergence from proximal contact to cervical in first than in second. Contacts are in middle third, distal contact is more cervical. Mesial cusp ridge is shorter than distal in maxillary second premolar. Maxillary first premolar has mesial cusp ridge longer than distal (UNIQUE to all other premolar and canine facial cusps). Cusp is more pointed first maxillary premolar than second. Buccal ridge is more prominent in maxillary first premolar than maxillary second. Maxillary first premolar is more likely to have two roots, whereas maxillary second premolar has one.

Mandibular first premolar is longer than the second. Buccal cusp of mandibular first premolar is sharper than second. Cusp notches more common mesially in the first and distally second. CEJ is more curved in mandibular first premolar than in second. Mandibular first premolar is the only premolar (only adult tooth) where the mesial contact is more cervical than distal. A buccal ridge depression can be seen on either side of buccal ridge (when present). It is more likely to be deeper mesially in first premolar and distally in second premolar. Both types of mandibular premolar roots taper toward apex. Distal bend of apex more common.

Lingual view:
Crown outline narrows lingually so some proximal surfaces are visible (EXCEPT some three-cusp-type mandibular second premolars with two lingual cusps).

Maxillary premolars: Lingual cusp is shorter than buccal cusp; more so in maxillary first premolar than second. Lingual cusp is relatively sharper in maxillary second premolar than in first. Both maxillary first and second
premolars have the lingual cusp tip positioned mesially to the midroot axis. Maxillary first premolar root is shorter than buccal root.

**Mandibular premolars** taper lingually (EXCEPT three-cusp mandibular second premolars where lingual half may be wider than buccal. Lingual cusp of mandibular first premolar is a bit shorter than the buccal. Buccal is non-functional. Lingual cusp of second premolar is slightly shorter than the buccal, and is mesial to midroot axis. When the mandibular second premolar has two lingual cusps, the mesiolingual cusp is normally wider and longer than the distolinguinal. The mandibular first premolar is the only premolar (only posterior tooth) to have its mesial marginal ridge more cervically than the distal. Only mandibular second premolars with two lingual cusps have a lingual groove separating them. Most mandibular first premolars have a mesiolingual groove separating mesial marginal ridge from lingual cusp ridge. Root tapers to apex.

**Proximal view:**

Mesial marginal ridges are more occlusal than distal (EXCEPT mandibular first premolar).

**Maxillary premolars** have trapezoid outline. First premolar is the only premolar to have a crown depression (always mesially). Maxillary premolar distal marginal ridge is more cervical than mesial for both maxillary first and second. Lingual cusp shorter than buccal cusp, more so in maxillary first premolar than second premolar. Facial height of contour is in cervical third (like all posterior teeth). Distance between cusps is the same for maxillary first and second premolars. Both buccal and lingual cusp tips are located over the root. Marginal ridge groove is found most often at the mesial marginal ridge of maxillary first premolar. More CEJ curve mesially than distally on both first and second premolars and anterior teeth. Lingual CEJ is more occlusal than buccal. Maxillary first premolar is only premolar with the mesial root groove more prominent than distal; only premolar with a mesial crown depression in line with the deep root depression. Distal root depression more prominent in maxillary second premolar.

**Mandibular premolar** crown shape: Crown tips lingually, more so in mandibular first premolar than second. Outlines are rhomboid. Lingual cusps are shorter than buccal, more so in first with non-functional lingual cusp. Mesial marginal ridge of mandibular first premolar slopes at 45°, nearly parallel to triangular ridge. Lingual triangular ridge is very short. Other posterior marginal ridges are more horizontal. Mesiolingual groove is often present on mandibular first premolars, not second. Both buccal and lingual cusp tips are located over the root. Marginal ridge groove is found most often at the mesial marginal ridge of mandibular first premolar. More CEJ curve mesially than distally on both first and second premolars and anterior teeth. Lingual CEJ is more occlusal than buccal. Mandibular first premolar is only premolar with the mesial root groove more prominent than distal; only premolar with a mesial crown depression in line with the deep root depression. Distal root depression more prominent in maxillary second premolar.

**Occlusal view:**

Most premolars are wider faciolingually than mesiodistally. Occlusal table bound by marginal and cusp ridges. Proximal contacts are located buccally to the buccolingual midline; lingual embrasure spaces are bigger. Triangular ridges join to form transverse ridges (EXCEPT three-cusp mandibular second premolars). A groove extends from mesial fossa to distal fossa (EXCEPT some mandibular first premolars).

**Maxillary** first premolar is likely to be slightly larger than second. Central groove of maxillary first premolar is longer than in second premolar. Maxillary second premolar has more supplemental grooves than first. Both maxillary premolars have noticeably greater dimension faciolingually than mesiodistally (by over 2 mm). Buccal ridge more prominent in maxillary first premolar than in second. Maxillary first premolar outline is more asymmetrical than second premolar. Distal contact of maxillary first premolar is more buccal than mesial; for maxillary second premolar it is more lingual.

**Mandibular** first premolar: has more bulk in distal half than mesial half. Mesiolingual corner is flat (depressed). Diamond-shaped outline. Mandibular second premolar: two-cusped, has round or oval outline. Three-cusped type has more square-like outline; may actually taper buccally (and from distal toward mesial - EXCEPTION to the rule). Prominent transverse ridge may separate mesial and distal fossae (with no central groove). Mesial and distal pits resemble “snake eyes”. Central groove connects mesial and distal pits; no lingual groove. Groove pattern may be H- or U-shaped. Three triangular ridges; no connection so there is no transverse ridge. Only premolar with central fossa/pit. Central groove may be called mesial. Distal grooves join in central pit. L grooves form “Y” pattern. Marginal ridge grooves are not common in mandibular premolars (although the
mandibular first premolar often has a mesiolingual groove, which separates the mesial marginal ridge from the lingual cusp.

Arch traits that differentiate maxillary from mandibular premolars: Mandibular premolars have more noticeable lingual crown tilt than maxillary (and slightly noticeable distal tilt viewed from the facial direction). Mandibular premolars: buccal cusp much longer than lingual cusp (less so for maxillary). Buccal ridge is less prominent than maxillary. All premolars have bucocolingual greater than mesiodistal but mandibular premolar is closer to square (buccolingual slightly greater than mesiodistal). Maxillary premolar has bucocolingual more obviously greater than mesiodistal.

Mandibulars vary in shape more than maxillary premolars. Mandibular second premolar is the only premolar more likely to have three cusps (one buccal and two lingual: mesiolingual and distolingual)

**MOLARS**

Maxillary first molar

vestibular (V), oral (O), mesial (M), distal (D) and occlusal (O) view

Maxillary second molar

vestibular (V), oral (O), mesial (M), distal (D) and occlusal (O) view

Maxillary third molar
Mandibular first molar

Mandibular second molar

Mandibular third molar

General features:

• 3 or 2 molars in every quadrant

• maxillary and mandibular molar widths make up 51% (mandibular) and 44% (maxillary) of the quadrant width

• third molars are the only teeth with no proximal contact tooth

Characteristics:
• Have three to five cusps
• Larger than other teeth but shortest occlusocervically
• Mesiodistal dimensions are greater than occlusocervical
• Crowns taper from buccal to lingual portion (EXCEPT for the maxillary first molar with large distolingual cusps)
• From the occlusal view, molar crowns taper from mesial to distal portion
• All molar crowns taper from mesial to distal, distal cusp are shorter than mesial cusps)
• Facial height of contour is in the cervical third, lingual height of contour is in the middle third.

Mesial contact is located near the junction of the middle/occlusal third (in all molars), distal contact is more cervical, near the middle of the tooth (in all molars)

**Function:**

• Mastication
• Maintenance of vertical dimension of face
• Maintenance of arch continuity for proper alignment
• Support cheeks (aesthetics)

**Vestibular view:**

Maxillary first molar is the largest maxillary tooth. There are two prominent cusps; mesiobuccal wider and longer than distobuccal. Longest mesiolingual cusp tip, may also be seen from buccal view. Buccal groove separates the mesiobuccal and distobuccal cusps, but it is not caries prone. All have mesial contact at junction of occlusal and middle thirds. Distal contact is more cervical near the middle of tooth. Three roots: lingual (longest) shows behind, between mesiobuccal and distobuccal roots; distobuccal (shortest); mesiobuccal (longer and wider than distobuccal). Trunk in first is shorter than second. Roots diverge more in first than second.

Mandibular molars (both) are normally wider mesiodistally than occlusocervically (more so in first). First molar is larger than second. First molar most often have five cusps: Three buccal- mesiobuccal greater than distobuccal, greater than distal (smallest, shortest); two lingual- mesiolingual (longest) greater than distolingual (second longest). First molar has two buccal grooves- mesiobuccal and distobuccal. Second molar has one buccal cusp. 15-20% of mandibular first molars have only four cusps (no distal cusp).

All molars have proximal contact areas located more occlusal on the mesial surface (at the junction of middle and occlusal thirds) than on the distal. Close to straight mesiodistally. Crown tapers more from distal contact to CEJ in first than second. Mesial contour is more straight. Mandibular first and second molars taper distally. Mandibular first and second molars have two roots: mesial and distal. Root trunk is shorter in first than second. Roots are more divergent toward apex in first than second.

**Lingual view:**

Maxillary molars: Little or no proximal surface visible in first due to wider lingual surface, larger distolingual cusp. First molar has largest and longest mesiolingual cusp (often with cusp of Carabelli or groove) and somewhat smaller distolingual cusp. Second molar also has longest mesiolingual cusp compared to much smaller (or missing) distolingual cusp. Lingual grooves and pits are caries prone. Longest lingual root often has longitudinal depression on lingual surface. Distobuccal and mesiobuccal roots are closer to each other in second than first.

Mandibular molar crowns taper from buccal to lingual direction (more so in first) so proximal surfaces are somewhat visible from lingual direction. Usually mesiolingual and distolingual cusps are visible since they are longer than buccal cusp. Mesiolingual cusp is wider than distolingual cusp. Lingual groove, if on lingual surface, is unlikely to be carious. CEJ is relatively straight mesiodistally (but may dip into the bifurcation). Roots narrower on lingual side than buccal. Mesial root is twisted so more of mesial surface is visible.
Proximal view:

**Maxillary molars:** Occlusocervical dimension is smaller than faciolingual. Only two cusps (and Carabelli) visible from mesial direction since distal cusps are shorter. Four cusps visible from distal direction since mesial cusps are longer than distal. Buccal height is in cervical third. Lingual height is in middle third (like all posterior teeth), or even more occlusal if there is a big cusp of Carabelli. From distal view some of the facial and lingual surface may be visible due to crown tapering distally. Mesial marginal ridge is more occlusal than distal, so more occlusal surface is visible from distal view. CEJ is nearly flat faciolingually. Less CEJ curve distally than mesially. Only two roots are visible from mesial direction, lingual root is the longest. Mesiobuccal root has mesial and distal root depression; the distobuccal root is variable.

**Manibular molars:** Buccal height of contour is in the cervical third. Lingual crest is in middle third (as in all molars). Buccal bulge is called cervical ridge, running mesiodistally and more prominent in mesial half. Lingual cusps are more pointed and longer than buccal cusps. From distal direction you can see more of the occlusal surface because it tapers distally. From distal view you can see cusp tips of longer mesiobuccal and mesiolingual cusps over shorter distal cusps. CEJ slopes occlusally from buccal to lingual direction, very slightly curved. Distal marginal ridge is more cervical than mesial marginal ridge (similar to proximal contacts), therefore we can see more of occlusal surface from distal direction. Mesial root is broader faciolingually and longer than distal root, so we cannot see distal root from mesial view. From distal direction wider mesial root is visible behind distal root. Mesial root has mesial and distal root depression. Smaller distal root has mesial root depression but distal surface is variable.

Occlusal view:

**Maxillary** first molar has four cusps plus often Carabelli cusp or mesiolingual cusp. Second molar has three larger cusps: mesiobuccal, distobuccal and mesiolingual, and one smaller or absent (distolinguinal). First and second have greater buccolingual than mesiodistal dimension. In case of first molars buccal half is often narrower mesiodistally than lingual half due to large distolinguinal cusp. Second molar distolinguinal cusp is often quite small so the lingual half is narrower mesiodistally than buccal half. More twisted rhomboid outline (mesiobuccal and distolinguinal angles sharper). Primary cusp triangles.

**Mandibular** first molars most often have 5 cups: mesiobuccal cusp is the largest and distal cusp is the smallest. Mandibular second molars most often have 4 cusps: mesiobuccal is the largest and distolingual is the smallest. If viewed along root axis, more facial surface is visible than lingual due to lingual tipping of the crown relative to the root. Mandibular second is rectangular. Tapers distally and linguually. Buccal cervical ridge is more prominent mesially in second.

**PRIMARY TEETH DIFFERENCES**

1. The crowns of primary teeth are wider mesiodistally compared to crown length than those of the permanent teeth.
2. The roots of primary frontal teeth are narrow and long compared with crown width and length.
3. The roots of primary molars are relatively longer and more slender than roots of the permanent teeth. There is also a greater extension of the primary roots mesiodistally, which allows more room between the roots for the development of premolar crowns.
4. The cervical ridge of the enamel at the cervical third of the anterior crowns is much more prominent labially and linguually in the primary than in the permanent teeth.
5. The crowns and roots of primary molars are more slender mesiodistally at the cervical third than those of permanent molars.
6. The cervical ridge in the buccal aspect of primary molars is much more definite, particularly in maxillary and mandibular first molars, than in permanent molars.
7. The buccal and lingual surfaces of the primary molars are flatter above the cervical curvatures than those of permanent molars, which makes the occlusal surface narrower compared to that of the permanent teeth.
8. The primary teeth are usually lighter in colour than the permanent teeth.
4. Tooth development, anatomy and tooth eruption (Noémi Rózsa DMD)

TOOTH DEVELOPMENT

Humans are diphyodont creatures, they develop two different tooth types during their lifetime: the primary and the permanent dentition. Tooth development starts around the 6th to the 8th weeks of embryonic life. During this period four major developmental stages can be distinguished:

- Proliferation (proliferatio)
- Histodifferentiation (histodifferentiatio)
- Calcification (calcification)
- Eruption (eruptio)

The **proliferation stage** starts in the ectodermal epithelium of the upper and lower dental lamina. The tooth buds of the primary dentition develop on the outer side, those of the permanent dentition on the inner side of the lamina. The development of deciduous teeth starts at the same time for the whole dentition, while the development of permanent teeth occurs at different times for each tooth group. The tooth buds are surrounded by thickened connective tissue, the dental papilla, together forming the tooth germ. Further development, called the cap stage of the proliferation period, is characterised by the differentiation of the three layers of the enamel organ. These are the inner and outer enamel epithelium and the stratum reticulare, the enamel pulp.

**Histodifferentiation** starts around the 9th week of embryonic life; the enamel organ is bell-shaped during this developmental stage, allowing for the production of enamel and dentine. Ameloblast cells are responsible for enamel secretion. In the early bell stage four distinguished layers of the enamel organ can be described: the stratum intermedium is added to the three layers of the cap stage, located between the inner enamel epithelium and the stellate reticulum. The ectomesenchymal cells of the dental papilla, in contact with the inner enamel epithelium form the odontoblasts, the cylindrical cells responsible for dentine development. The dental follicle (folliculus dentis) develops around the enamel organ, thus creating the tooth germ.

During the **calcification** stage the ameloblast cells form the enamel prisms and the odontoblasts develop the dentine structures. This latter group thickens gradually; the odontoblasts move inward and leave behind protoplasmic extensions called Tomes's fibres. This tooth developmental stage corresponds to birth.

The **mineralisation** of primary incisors starts in the 17th week of intrauterine life, that of the primary molars in the 20th. At birth the crowns of primary incisors and canines are almost fully developed, while the mineralisation of primary molar crowns is only 50% complete. The mineralisation of permanent teeth starts much later: at birth only the cusps of the first permanent molars are mineralised. Permanent teeth mineralisation occurs parallel to vertical teeth growth.

During the **eruption stage** root formation starts, induced by Hertwig’s epithelial root sheaths. This process is parallel to deciduous teeth eruption. Cement, root membrane and alveolar bone tissue develop from the dental follicle, while pulp structures are derivates of the dental papilla.

**THE ANATOMY OF PRIMARY AND PERMANENT TEETH**

The morphological characteristics of primary teeth are largely similar to those of the homologous permanent teeth. Deciduous crowns are generally smaller, the crown / root ratios also differ, the roots of deciduous teeth are generally longer compared to the total tooth length.

In addition to the difference in size, there are also differences in the tooth colour. Primary teeth, as their name implies, are whiter or have a bluish tinge, while the permanent teeth are more yellowish. When second dentition is approaching, primary teeth will show a physiological mobility and higher physiological abrasion. As a result of maxillary growth, primary dentition is generally characterised by the presence of gaps between the teeth, a.k.a. physiological diastemas. During the second dentition, the deciduous molars with their higher mesio-distal width, are replaced by premolars. Palatal, Carabelli-like additional cusps can often be observed on the upper second primary molars.
Deciduous teeth are also characterized by Mühlreiter features. Primary molar crowns have a thickened cervical ridge, called tuberculum molare. The molars have divergent thin roots, and the crowns of the premolars are located between them. The physiological root resorption of the deciduous teeth is genetically coded, and it is a precondition for normal secondary dentition.

**ERUPTION OF DECIDUOUS TEETH AND SECONDARY DENTITION**

As a result of major variations in deciduous teeth eruption tooth eruption chronology indicates mean values only. Primary tooth eruption usually begins at 6 months of age and the primary dental arch is completed around 3 years of age. The eruption of deciduous molars is known to cause the most complaints. Children are restless, they have no appetite and their body temperature is slightly increased (subfebrility), accompanied by increased salivation. If eruption occurs earlier than average, the phenomenon is called dentition praecox. A pathological delay of tooth eruption is termed dentito tarda. Problems are more often observed during the eruption of primary teeth (dentito difficilis), such as the frequently occurring eruption cyst.

Secondary dentition starts with the first permanent molars erupting at the distal end of the dental arch. Sometimes this goes unobserved by both parents and children because there is no preliminary loss of primary molars. The first “real” change is the exfoliation of the lower primary central incisors followed by the eruption of the permanent lower central incisors. If the physiological root resorption of the primary incisors is perturbed or too slow, the corresponding permanent incisors will erupt lingual to the dental arch. By 8 years of age all four upper and lower permanent incisors should have erupted. These teeth are followed by the premolars at 9-10 years, and the permanent canines at 11 years of age. If the wisdom teeth are not considered, the final phase of permanent tooth eruption is the completion of the dental arch with the second permanent molars at the age of 12. There are great individual variations in the timing of secondary dentition eruption, and a difference can be observed between the genders as well. In girls the eruption of the permanent canines often precedes that of the premolars.

5. **Pulp and pulp–dentin complex (Zsuzsanna Tóth DMD)**

**MACROSCOPIC APPEARANCE**

The pulp is in the cavity-system of the tooth, which is surrounded by dentin and is located in the pulp chamber and in the root canal (Figure 1.17.). The corresponding near-surface part of the crown cusps are the pulp-horns, which because of the carious process or because of preparation can be easily exposed. The pulp is mainly gelatinous, contains soft connective tissue, in which there are cellular and fibrous elements, blood and lymph vessels and nerves.

The pulp forms an integral, inseparable morphological, histological, and dynamic unit with the dentin. The odontoblast processes creep deep in the dentinal tubules. The formation of the dentin is continuous, altering the size of the pulp space, and consequently the shape and function of the pulp. Primary dentin formation characterizes the developing stage of teeth, and secondary dentin is produced after tooth-eruption in the oral cavity. For stimulus (e.g. caries), tertiary dentin is produced locally in the affected area.

**STRUCTURE OF THE PULP**
Dentin has a tubular structure, its innermost part is the not-yet calcified pre-dentin. Below it, the **odontoblast layer** of pulp can be found, which forms the dentin, arranged in a single cell line. The shape of the odontoblasts (which are dendritic cells) is cylindrical in the pulp chamber and flat in the root canal. There are much more odontoblasts (about 50,000 cell/mm²) in the pulp chamber than in the root canal.

The odontoblast processes extend differently deep into the dentinal tubules, some of them up to the enamel-dentin junction. A **cell-free zone** lies below the odontoblast layer (zone of Weil) and below that a **cell-rich zone** (bipolar zone) can be found. The central zone of the pulp contains the most cells, blood vessels and nerve fibres (the nerve plexus of Raschkow and subodontoblastic capillary plexus) (Figure 1.18).

Aside from the odontoblasts, its cellular components are the fibroblasts, the undifferentiated mesenchymal (reserve) cells, and cells of the immune system: macrophages, lymphocytes, mast cells, etc. The mesenchymal cells can transform into odontoblasts replacing the dead cells of the odontoblast layer. Fibroblasts produce the gel-like basic substance of the dental pulp, which contains collagen and reticular fibres as well. In pathological conditions, but also by aging, symptom-free calcification may occur in the pulp. Beside the histologically diffuse calcification, macroscopically detectable free or fused calcification to pulp wall denticles (pulp stones) can be detected (Figure 1.19.).

Denticles can make the preparation of a root canal difficult. As a reaction to pathological stimuli, inflammation of the pulp, i.e. pulpitis, or transformation into granulation tissue, internal resorption (pink spot) will be formed. The circulation and lymphatic drainage of the pulp is provided by one or two arterioles, venules and lymphatic vessels entering the pulp via the foramen apicale. The arterioles form characteristic capillary loops below the odontoblast layer. Mainly the trigeminal nerve is responsible for the sensory innervation of the dental pulp transmitting mechanical, thermal and chemical stimuli.
DENTIN HYPERSENSITIVITY

The response of uncovered cervical dentin to stimulus is heavy pain, which is a sign of the appearance of dentin hypersensitivity. Due to various reasons (dental erosion, occlusive loading, dental plaque, incorrect brushing technique, etc.), gingival recession will occur, the thin root cement wears quickly, and dentinal tubules will be opened. Dentin sensitivity is explained by the 'hydrodynamic theory' (Brännström and Astra, 1972), the movement of fluid in the dentinal tubules results in the activation of fibres causing sharp pain. Sealing of the dentinal tubules is a successful therapy in most of the cases. Dentin hypersensitivity caused by biofilm is due to the local inflammatory activity of bacterial toxins and enhanced neuronal excitability of released inflammatory mediators.

ROLE OF THE PULP

Thermal, mechanical stimuli and pain arriving through the crown covering enamel and the dentin underneath will be mediated by the pulp to the central nervous system. The third function of the pulp, beside perception and dentin formation, is a protective function, which is manifested in the formation of tertiary dentin, the development of inflammation or immune reactions. The importance of tertiary dentin is decisive when due to the wear of the tooth, caries or trauma the developing layer prevents pulp necrosis.

AGE-RELATED CHANGES IN THE PULP

There are typical changes in the pulp to keep track of age progression (Figure 1.20.). Due to the continued deposition of secondary dentin, a reduction in the size of the pulp chamber will be significant. It is also influenced by the experienced irritations. At the apex of the root cement apposition occurs followed by narrowing of the foramen apicale and decreased circulation. The arteriosclerotic changes in the blood vessels lead to decreased blood supply. Cell death results in a decreased number of cells, and the surviving fibroblasts produce more fibrous matrix as a response to it. The number and proportion of fibrous elements increases, and pulp fibrosis develops. The number of blood vessels and nerves decreases, and dentin permeability is reduced.
Due to the above mentioned facts, the sensitivity test produces weak or no response at all. The pulp has reduced defensive ability, less chance for recovery, e.g. in case of pulp capping. The reduced size pulp chamber, narrow root canal, and calcification processes make root canal therapy difficult and sometimes impossible.

6. Anatomy of periodontium (István Varga DMD, Péter Vályi DMD)

The periodontium, defined as tissues supporting and fixing the tooth, comprises root cementum, periodontal ligament, bone lining the tooth socket (alveolar bone) and part of the gingiva facing the tooth (dentogingival junction) (Fig. 1.21.).
The gingiva is part of the oral mucosa that covers the alveolar processes of the jaws and surrounds the cervical portion of the teeth.

Functions

- covering the cervical portions of the teeth and the alveolar processes of the jaws
- composed of a thin outer layer of epithelium and an underlying core of connective tissue
- provides a tissue seal around the cervical portions of the crowns

Macroscopic anatomy of the gingiva (Fig. 1.22.)

- **Free gingiva (marginal gingiva):** unattached portion of the gingiva that surrounds the tooth in the region of the cementoenamel junction

- **Gingival sulcus:** V-shaped, shallow space between the free gingiva and the tooth surface. The depth is 1-3 mm for a clinically normal gingival sulcus and is measured using a periodontal probe around the tooth. The base of the sulcus is formed by junctional epithelium.
• **Attached gingiva:** Gingiva that is tightly connected to the cementum on the cervical third of the root and to the periosteum (connective tissue cover) of the alveolar bone. It lies between the free gingiva and the alveolar mucosa.

• **Interdental gingiva:** the portion of the gingiva that fills the area between two adjacent teeth apical to the contact area.

  1. *Papilla/Papillae:* the interdental (or interproximal) gingiva consists of two interdental papilla (one facial, one lingual)

  2. *Col area:* Valley-like depression in the interdental gingiva. It lies directly apical to the contact area. The col is not present if the adjacent teeth are not in contact or if the gingiva has receded.

• **Mucogingival junction:** The clinically visible boundary where the pink attached gingiva meets the red, shiny alveolar mucosa. •*Alveolar mucosa:* Movable tissue loosely attached to the underlying bone. Nonkeratinised, thin, smooth and shiny epithelium. The underlying vessels may be seen through the epithelium. It also covers the vestibule and the floor of the mouth and continues in the buckle and labial mucosa.

### Macroscopic anatomy of the periodontium

The colour of the attached and marginal gingiva is generally described as "pale pink" and is produced by the vascular supply and the thickness and degree of keratinisation of the epithelium. The alveolar mucosa is nonkeratinised red, smooth and shiny rather than pink and stippled. The connective tissue of the alveolar mucosa is loosely arranged and the blood vessels are more numerous. The attached gingiva may exhibits varying degrees of brownish pigmentation due to synthesis of melanin by melanocytes located in the basal cell layer of oral epithelium (Fig. 1.23.).

### Pigmented gingiva

The contour or shape of the gingiva varies considerably and depends on different factors (the shape of the teeth and their alignment in the arch, the location and size of the area of proximal contact). The marginal gingiva envelops the teeth in a collar-like fashion and has a scalloped outline on the facial and lingual surfaces.

The gingiva is firm and resilient and, with the exception of the movable free margin, tightly bound to the underlying bone. It presents a textured surface similar to an orange peel and is referred to as being stippled (the attached gingiva). Stippling is best viewed by drying the gingiva. It is absent in infancy, first appears in children at about 5 years of age, increases until adulthood and frequently begins to disappear in old age.
The position of the gingiva refers to the level at which the gingival margin is attached to the tooth (normally strongly attached to the cementoenamel junction).

Microscopic anatomy of the gingiva

The gingiva consists of epithelium and connective tissue (lamina propria). The epithelium covering the free gingiva may be classified into three types:

1. Oral epithelium (OE) which faces the oral cavity
2. Oral sulcular epithelium (OSE) covering the gingival crevice or pocket
3. Junctional epithelium (JE) which provides the contact between the gingiva and the root

The attached gingiva covered by oral epithelium. Characterisation of cell layer (OE, OSE) from basal layer to granular cell layer:

- height of cells decrease
- the number of desmosomes and the number of tonofilaments in the cytoplasm increase
- the number of cell organelles decrease

The connective tissue (CT) consists of

- collagen fibers (60%)
- cells (5%) – fibroblasts, PMN, macrophages, lymphocytes
- matrix (35%) vessels and nerves are embedded in an amorphous ground substances

The supraalveolar gingival fiber bundles attach the gingiva to the tooth surface and to the alveolar bone. Functions of the gingival fibers:

- maintaining of resistance and resiliency of the gingiva
- resistance to force
- stabilizing of the tooth
- supporting of junctional epithelium

The gingival fibers are: circular, semicircular, interpapillary, intercircular, intergingival, transseptal fibers.

The biological width is defined as the dimension of the soft tissue, which is attached to the portion of the tooth coronal to the crest of the alveolar bone. Biologic width is the distance established by "the junctional epithelium and connective tissue attachment to the root surface" of a tooth. The biologic width is essential for preservation of periodontal health and removal of irritation that might damage the periodontium (prosthetic restorations, for example).
Microscopic anatomy of the gingiva


**PERIODONTAL LIGAMENTS**

The periodontal ligament is the connective tissue that surrounds the root and connects it with the bone. It is continuous with the connective tissue of the gingiva and communicates with the marrow spaces through vascular channels in the bone. The average width is about 0.2 mm.

Principal fibres of the periodontal ligament: they are classified into several groups on the basis of their anatomical location. Oblique fibres are the largest group among the periodontal ligaments; they extend from the cementum in a coronal direction obliquely to the bone. They bear the brunt of vertical masticatory forces and transform them into tension on the alveolar bone (Fig. 1.25.).
The periodontal ligaments

Functions of the Periodontal Ligament

Physical functions

- Transmission of occlusal forces to the bone.
- Attachment of the teeth to the bone.
- Resistance to the impact of occlusal forces (shock absorption)

Formative and Remodelling Function
The periodontal ligament is constantly undergoing remodelling. The cells of the periodontal ligament participate in the formation and resorption of cementum and bone, which occur:

- in physiological tooth movement;
- in the accommodation of the periodontium to occlusal forces
- in the repair of injuries.

**Nutritional and Sensory Functions**

It supplies nutrients to the cementum, bone and gingiva by way of blood vessels and provides lymphatic drainage. It contains sensory nerve fibres capable of transmitting tactile, pressure and pain sensations. Nerve bundles pass into the periodontal ligament from the periapical area and through channels from the alveolar bone that follow the course of the blood vessels.

**CEMENTUM**

Cementum is calcified mesenchymal tissue that forms the outer covering of the anatomic root. There are four main types of root cementum:

1. **Acellular, afibrillar cementum** It is found as coronal cementum
2. **Acellular, extrinsic fiber cementum** This cementum is located in the cervical two-thirds of the root
3. **Cellular, intrinsic fiber cementum** It is located in the lacunas of the root at sites of cementum repair.
4. **Cellular, mixed fiber cementum** It is found on the apical third of the root and in furcations

Both consist of a calcified interfibrillar matrix and collagen fibrils.

Thickness of the cementum is increase from the coronal part (50-150 micrometers) to the apical portion (200-600 micrometers). Cementum deposition is a continuous process, most rapid in the apical part of the root. Hypercementosis is prominent thickening of cementum layer.

Cementum resorption may be due to local or systemic causes (trauma from occlusion, orthodontic movement, cysts and tumours, replanted and transplanted teeth) •Cementum resorption is not continuous, it may alternate with periods of repair

**ALVEOLAR PROCESS**

The alveolar process is the portion of the maxilla and mandible that forms and supports the tooth sockets. It consists of compact bone and cortical bone. The existence of the alveolar bone is dependent on the presence of teeth. When teeth are extracted, in time the alveolar bone resorbs so only basal bone remains.

The alveolar process consists of (Fig. 1.26):

- Alveolar bone proper (it is perforated by numerous small canals through which blood and lymph vessels as well as nerve enter the periodontal space)
- Trabecular bone (relatively small amount, often missing at lower incisors, upper canines and upper premolars)
- Compact bone (meet with alveolar bone at margin to form the alveolar crest)
Anatomical areas of the alveolar bone (Fig.1.27.):

- Alveolar crest (the most coronal portion of the alveolar process, located 1-2 mm apical to the CEJs of the teeth)
- Interproximal bone (the area of bone that lies between the proximal surfaces of two adjacent teeth)
- Interradicular bone (bone between the roots of the same tooth)
The contour of the bone normally conforms to the prominence of the roots. The height and thickness of the facial and lingual bony plates are affected by the alignment of the teeth, by the angulation of the root to the bone and by occlusal forces.

Isolated areas in which the root is denuded of bone and the root surface is covered only by periosteum and overlying gingiva are termed fenestrations. In these instances the marginal bone is intact. When the denuded areas extend through the marginal bone, the defect is called a dehiscence (Fig. 1.27.). Fenestration and dehiscence are important, because they may complicate the outcome of periodontal surgery.

The anatomical areas of the alveolar bone. Fenestration and dehiscence

7. Occlusion and mastication (Márta Radnai DMD)

Teeth, as parts of the masticatory system, have to fulfil several functions. The most important functions include mastication, preparation of food for swallowing, phonation and providing an aesthetic appearance. Proper mastication is ensured if a sufficient number of teeth are present in the oral cavity and they meet their opposing teeth at appropriate occlusal contact points. The studying of these occlusal contact points is one of the main areas covered by the science of gnathology.

Gnathology is the study that deals with the biology of the entire masticatory mechanism including morphology, functional anatomy, histology, physiology, pathology, diagnostics and the therapeutics of the masticatory system.

Knowledge of the anatomy of the occlusal surfaces of the teeth and their relationship in closed or other positions of the jaw during function is essential for most dental treatments. Rehabilitation of damaged teeth must ensure that the fillings, crowns or other replacements should not disturb, but improve occlusion and articulation.
In gnathology a distinction is made between static and dynamic occlusions. The latter is also known as articulation. Static occlusion means contact between the teeth when the jaw is closed, while articulation refers to different tooth contacts when the jaw is moving. During chewing the places of tooth contacts change continuously in order to chop the food properly. Occlusal contacts can be examined in the mouth with coloured foil or paper, or by study casts mounted in special equipment, the articulator.

Centric occlusion means the closure of the dental arches ensuring the highest number of occlusal contact points between the occlusal surfaces of the upper and lower teeth, during which the head of the condyle is in the centric relation position in the glenoid fossa. Centric relation is the relationship between the jaw and the maxilla, in which the mandibular condyle takes a centric position in the glenoid fossa, symmetrically related to the median sagittal plane.

In case of healthy teeth specific parts of the occlusal surfaces meet the corresponding parts of the opposing teeth (Figure 1.28.).

The eminences and depths of the occlusal surfaces interlock with each other; this phenomenon is called intercuspation. Functional or holding cusps fit the central fossa or the marginal ridge of the opposite teeth, creating a stable relation between the jaws (Figure 1.29., 1.30.).
In most cases, people who have enough opposing pairs of teeth, close their teeth in the same position as they bite. This position is called **habitual occlusion**, also known as habitual or characteristic intercuspation. The supporting cusps fit the vestibular or oral surfaces of the functional cusps of the opposing teeth, and protect the cheek or the tongue from biting between the opposing occlusal surfaces. The functional/holding cusps are the buccal cusps of the mandibular teeth and the lingual cusps of the maxillary teeth. Their surface is rounded, convex in all directions; their task is to chop food effectively. The marginal ridge is a rounded cylindrical eminence on the mesial and distal parts of the occlusal surface of the cuspal teeth. The cusps are separated by grooves (sulcus, fissure) and pits (fossa, fovea), which are shallow depressions. If we take a look at the closed dental arches, it is obvious that the buccal cusps of the upper teeth cover a little bit of the lower ones; this characteristic applies to the relation of the front teeth as well. This vertical overlapping of the teeth is called "**overbite**", its average extent is 1-2mm. Between the incisal edge of the upper incisors and the labial surface of the lower ones a small distance can be measured. It is known as "**overjet**" (horizontal overlapping). Its average
value is also 1-2mm. In case of certain dental anomalies these measures can vary. If the overlap of the upper incisors in centric occlusion is ≥3mm, deep bite is present. However, in severe deep bite cases the upper front teeth can completely hide the lower ones or some patients even bite into the gum margin of the lower incisors.

**PARTICULAR POSITIONS AND MOVEMENTS OF THE MANDIBLE**

The mandible performs complicated movements in different directions related to the maxilla, whose movements are controlled and at the same time limited by the morphology of the temporomandibular joint, the teeth and the function of the surrounding muscles. The most important mandibular positions during movements are the rest position, centric relation and centric occlusion, intercuspsation, retruded contact position, lateral cusp-to cusp, anterior edge-to-edge position and the end positions of maximum movements, that is the maximum opening, protrusion and laterotrusion. In **resting position** the activity of the elevator and depressor muscles are minimal, the condyles are in a central position in the glenoid fossae (symmetrical on the two sides), there is no contact between the upper and lower teeth, there is a gap of a few millimetres. Most movements start from this position. The **intercuspal position** is determined by tooth contacts and it changes through the lifespan of each individual. In this position the condyles are not necessarily in the centric position. The **retruded contact position** is generally established through an external effect, when the chin is pushed backwards/distally. There is contact between one or more distal teeth; however, as this position is not comfortable, it cannot be maintained for a long time. **Lateral cusp-to-cusp position** develops when the jaw moves to one side and the buccal cusps of the upper and lower teeth contact each other (Figure 1.31.).

Movement of the mandible towards the working side; a gap is visible between the upper and lower teeth on the non-working side.

The side to which the mandible is moving is called the **working side**, while the other one is called the **non-working/balancing side**. **Anterior edge-to-edge position** means that the mandible moves forwards, while the lower incisors move forwards and downwards and the edges of the upper and lower incisors make contact. This is the position, which enables us to bite into food (Figure 1.32. and 1.33.).
Movement of the mandible forwards, while the upper and lower incisors are temporarily in an edge-to-edge position - frontal view

Movement of the mandible forwards, while the upper and lower incisors are temporarily in an edge-to-edge position - lateral view

Normally, when there is no tooth wear, the molar teeth have no contacts in this position; the function of the muscles is concentrated on the front teeth. The positions of maximum opening, protrusion and laterotrusion are the result of the movements with the largest amplitude. The extension of these movements can be decreased (trismus) or increased (dislocation) because of pathological processes of the temporomandibular joints, masticatory muscles or other parts of the masticatory system, therefore the examination and measurement of these movements are important in case of temporomandibular joint disorders.

The movements of the mandible can be described by the movement paths of some characteristic points; these are the incision inferius (the mesial corner of the left lower incisal edge) and the ectocondylare (the most lateral point of the condyle). Gnathology studies the movements in all three dimensions. The path of the border or envelope movements of the incision inferius in the sagittal plane is called Posselt diagram (1952), named after its first describer. Some excursions are free mandibular movements; these are limited only by the joints, their
ligaments and the masticatory muscles. Other movements are guided, articulation or contact movements, which occur during tooth contacts, and are guided and limited also by particular surfaces of some or more teeth. Free mandibular movements include jaw opening and closing, and those forward-, backward and lateral movements which take place when there is no contact at all between the opposing teeth. All these movements – except the lateral movements – are symmetrical; identical movements occur in both temporomandibular joints. During opening the condyles rotate (around an imaginary horizontal axis), then they move forward and downwards (translation); at closing an opposite movement takes place. Besides the anatomy of the joints and the surrounding structures, the forward movement is determined by the shape and relation of the upper and lower incisors, therefore it is called incisal guidance. The lateral movements are guided similarly by the canines (canine guidance, Figure 1.34, 1.35., 1.36.) or the cuspal teeth (group function), while there is no contact between the other teeth.

Canine guidance

The contacting surfaces during canine guiding can be marked by articulating paper or foil - lower arch
The contacting surfaces during canine guiding can be marked by articulating paper or foil - upper arch

During lateral movements of the mandible the condyles move differently in the two joints, these are asymmetric movements. On the side towards which the movement happens, the condyle practically stays in its place and rotates slightly around a vertical axis. This side is called working side, since mastication occurs here. The other condyle has a bigger movement anteriorly, downwards and medially towards the midsagittal plane on the posterior slope of the articular eminence; this side is referred to as the non-working side.

**CONSEQUENCES OF TOOTH LOSS**

The ideal occlusion may be impaired because of caries, tooth migration due to periodontal diseases, tooth loss or as a consequence of dental intervention. The loss of teeth, or even one tooth triggers several processes, as if the masticatory system tries to close the gap left by the lost tooth. The remaining teeth tilt towards the edentulous space, the opposing teeth elongate, as if they were seeking support. Tilting of the teeth may cause traumatic occlusion, as teeth are affected by a load other than the axial force. It may increase tipping, later the periodontal tissues will be affected, and eventually the teeth may become mobile. Tooth loss may destroy the approximal contact point system, with the consequence of food trapping, approximal caries and gingivitis, and without treatment periodontitis will develop. The number of occlusal contact points decreases and because of tooth tipping, non-working side contacts may occur, which may alter articulatory movements. Non-working side contacts are regarded unfavourable.

**CHEWING**

Chewing is the preparatory phase of digestion: during mastication solid food is broken into small pieces and prepared for swallowing. Through crushing the food its surface becomes larger, it becomes soft and mixed with saliva on a great surface; a bolus is formed. The breakdown of carbohydrates by salivary enzymes already begins in the oral cavity. The first step is taking a bite by using the front teeth. During mastication the tongue and the cheeks keep the bite between the opposing teeth and it is ground and pressed together by the intermittent contacts of the upper and lower teeth, and squeezed through the teeth. The upper and lower teeth close and come apart and lateral movements occur as well. This process goes on continuously and is called chewing cycle. Some individuals perform mainly opening and closing jaw movements (temporal type of chewing), while others perform rather horizontal mandibular movements (masseter type chewing). The masticatory process is repeated till the food becomes soft and suitable for swallowing. Chewing consists of rhythmic jaw movements, which is a transition between reflex and conscious movements.

**8. Oral biofilm (Péter Vályi DMD)**

The human body can be defined as an ecological system of 1014 cells of prokaryotics and eukaryiotics consisting of 10% mammalian cells. After birth, a resident microflora develops naturally in distinct organs of
the human body (such as skin, ear, nasal cavity, digestive tract, oral cavity, or vagina), which has a characteristic composition and their habitats play a role in the maintenance of health; however, qualitative or quantitative shifts in microbial flora or an inadequate innate host response can lead to pathological alterations.

More than 700 species have been isolated from the oral microflora but less than 50% of these microbes can currently be cultivated in pure culture in the laboratory. In the dentogingival plaque, 109 microbiota can be detected on the tooth surface, 104 species in the healthy gingival sulci, and 108 microbes in the inflamed periodontal pockets.

Microorganisms can be found in the form of planctonic-like cells in the oral microflora, but they also exist attached to a surface of the oral mucosa, teeth and restorations in the form of a biofilm.

**BIOFILM AS AN ECOLOGICAL SYSTEM**

A biofilm is a complex microbial ecosystem formed by colonies of one or more different species attached to the tooth surface or hard, non-sheding material embedded in an organic polymer matrix in a fluid layer, and it contains nutrients for their habitats. The oral biofilm is a soft, non-mineralised, acquired, gel-like deposit attached to the tooth surfaces, restorations and implants via the pellicle.

The biofilm allows the attachment, growing and multiplication of microorganisms, interactions among bacteria and protects the colonizing species from environmental influence. The biofilm provides the living conditions for their inhabitants. Formation of the biofilm is similar to the development of civilisation: some planctonic form bacteria settle on the surface, and then attach new habitats to the colonies, the microorganisms multiply leading to an increase in the biomass: horizontally than vertically.

The microbes synthesise exopolymers to form the biofilm matrix, and numerous biochemical and molecular interactions play a role in the formation of the plaque. The bacteria may produce nutrients for each other, and may release metabolites that result in altering the environment suitable for the growth of new habitats. These types of processes are synergistic interactions. In other cases, antagonistic interaction takes place between the microbes of the colonies: bacteria produce inhibitory compounds to inhibit neighbouring cells. The structure of the biofilm can prevent the penetration of antimicrobial agents. Between the microcolonies water-channels run, which permit the passage of nutrients and other substances throughout the biofilm.

Microbes of the plaque can communicate with each other by cell-to-cell communication, gene transfer, small diffusible molecules (quorum sensing) and other signal molecules.

Bacteria are able to sense alterations in their environment and respond to these changes leading to reorganization of the structure and composition of the bacterial mass, thus resulting in the detachment of some species from the biofilm. Detachment of bacteria can be seen to achieve the critical mass of the biofilm.

The main characteristics of the biofilm can be seen in Figure 1.37.
FORMATION OF THE BIOFILM

Within minutes after complete cleansing of the solid surface, a pellicle forms from protein and glycoprotein of saliva. These macromolecules alter the hydrophobic solid surfaces: alter the charges and free energy of the surfaces, resulting in an increased efficiency of bacterial adhesion. The first colonisers adhere to the tooth surface initially through weak, reversible adhesion via van der Waals forces and they are removable by waterjet. The pellicle is approximately 1 micron in thickness, and it is a clear, translucent layer, which may be stained by disclosing agents, nicotine, caffeine, etc., becomes brown, greyish or other colour.

After the formation of the pellicle, the supragingival plaque accumulation is starting by the association of the planctonic cell to the pellicle by purely physical forces. Irreversible adhesion involves interactions between the specific components of the cell surface (adhesions) and the complementary special parts (receptors) present in the pellicle. Certain bacteria can attach more rapidly by the help of fimbriae to the biofilms than microorganisms, which have to be growing and synthesise outer membrane components. The first colonisers are the Gram-positive cocci, and then the rods and filamentous bacteria appear.

During co-adhesion, secondary and late colonisers adhere to already attached microbes, and multiplication of the attached bacteria leads to an increase in the mass of the biofilm. The microorganisms synthesise the extracellular polymers to form a matrix. It initiates an intensive metabolism within the biofilm. In the deeper layers, an increased amount of anaerobe bacteria develops because of the alterations of the environment. The outer layers consist of the aerobes or facultative aerobes. The microorganisms produce an extracellular matrix which may protect the colonies against the environmental influences. The colonizer bacteria can be destroyed with 1000 or 1500 times larger difficulty than the solitaire forms of the microbes.

At the critical mass of the biofilm, the microbes in the basal layer die and the rate of growing of the biofilm decreases, which leads to the development of special forms of the colonies such as “corn cob,” “test tube rush” or “rosette” formation. Some habitats get detached from the biofilm moving and forming new colonies.

The subgingival plaque formed from the supragingival biofilms grows into the sulcus, or the bacterial loading leads to the inflammation of the marginal gingiva, and it covers part of the supragingival plaque. There are three forms of subgingival colonies: tooth attached, epithelial attached, and unattached (planctonic “free-floating biofilm) plaques (Figure 1.38.).

The main characteristics of the biofilm

- Colonies of one or more species
- Protection to colonizing species:
  - from competing microorganisms
  - from environmental factors such as
    - host defense mechanisms
    - toxic substances in the environment (lethal chemicals, antibiotics)
- Can facilitate uptake of nutrients (cross-feeding)
- Removal of harmful metabolic products
- Providing of appropriate physicochemical environment (e.g. reducing oxidation reduction potential)
- Biofilm spread horizontally, then vertically
- Protection from sudden harmful changes: providing more stable environment, than individuals in a planctonic or nomadic state
- Communication between bacterial cells within a biofilm (signaling molecules, "quorum sensing" or exchange of genetic information)
The forms of the surfaces covered by the biofilm in the oral cavity are different according to their environmental conditions leading to the different composition of the microcolonies.

**ORAL BIOFILMS OF THE EPITHELIAL SURFACES**
The desquamation (renewing of epithelia) of the epithelial cells leads to relatively light bacterial loading of the mucosa; however, the massive colonies of the tongue may serve as the reservoir of the biofilm forming on the hard, non-shedding surfaces.

The attached microorganisms (bacteria, viruses, fungi and protozoa) of the oral epithelium may be the cause of certain specific origin infections of the oral mucosa, but they may also play a role as aetiological factors of oral cancers.

**Cariogenic Effect of the Dental Plaque**

Cariogenic pathogens can metabolise sugar leading to changes in the environmental conditions such as low pH, resulting in demineralisation, when the caries process is initiated. Acid produced diffusions through the microchannels between the enamel rods lead to dissolving of the tooth minerals, and it creates the subsurface lesions by destroying the organic part of the hard tissue. The spread of infections into the pulp leads to irreversible damage to their tissues.

**Significance of the Subgingival Biofilm**

Despite the presence of dentogingival plaque, the balance of the pathogens and host response results in the maintenance of health in the periodontal tissues. The qualitative or quantitative shift of the microbes or an inadequate host response leads to the reversible damage of the gingiva. After the long-term bacterial loading, in some cases, the progression of inflammation results in an irreversible destruction of supporting tissues. The cause of the progression is mainly unclear.

The oral bacteria may play a role in the aetiology of periodontal diseases described by Black around 1890. It is clear (since Löe et al. published the classic experimental study in 1965) that the accumulation of non-specific supragingival plaque leads to reversible inflammation of the gingival, and removal of the biofilm leads to complete healing. Numerous studies support the theory of “host–parasite interaction” described by Page and Schroeder providing some evidence that cessation of the equilibrium between the bacteria and the host may occur caused by a microbial shift or alteration of host response. It may be due to the destruction in periodontal tissues mainly by the inflammatory and immune processes, and less due to the direct damage of periodontopathogens.

This process has a cyclic nature: a new equilibrium can be developed after the alteration, which can be terminated by new influences leading to the progression of the diseases. The oral biofilm as a potential aetiological factor of the different diseases is described in Chapter 2.

### 9. The defence system of the oral cavity (Péter Vályi DMD)

Numerous protective mechanisms interact to maintain the healthy environment of the oral cavity. Both soft and hard tissues have to be protected against microbial challenge. The first line of defence, the so called “defence line 0” is the saliva.

**The Role of Saliva in Host Response**

The role of saliva is ambivalent: although it has numerous protective antimicrobial properties, pellicle formation results in the attachment of microorganisms with the help of saliva, and it may play a role in the development of calculus through the precipitation of soluble minerals, and saliva also serves as nutrient for some microbes. Saliva plays an important role in the protection against microorganisms, maintains healthy oral environment, digestion and bolus formation. The role of saliva is summarised in the next figure (Figure 1.40.)
Numerous protective mechanisms of the saliva are part of the antimicrobial defence system: it binds the components of microbe metabolism, some salivary proteins destroy bacterial cell walls with the help of enzymes, secretory IgA inhibits microbial adhesion by binding (agglutination) microorganisms. Saliva also controls the adhesion or colonisation of bacteria via its buffering capacity, serves as a catalyst for redox reactions leading to the formation of antibacterial products, proline-rich proteins inhibit the spontaneous precipitation of calcium-phosphate salts and the growth of hydroxyapatite crystals on tooth surfaces and prevent the formation of salivary and dental calculus.

Saliva protects against environmental challenges by maintaining the integrity of the oral mucosa via physicochemical mechanisms, antibacterial effects and the absorption of water.

**THE ROLE OF GINGIVAL SULCUS IN HOST RESPONSE**

Numerous mechanisms serve to maintain clinically healthy conditions in the gingiva; however, continuous host-microbial interactions can be seen in the periodontal environment. The protective processes in the gingival sulcus prevent damage to gingival epithelial layers.

The regular shedding of epithelial cells and the positive flow of gingival crevicular fluid may remove unattached and epithelially attached plaques, as well as toxic products of bacterial metabolism and host response.

Cells of the junctional epithelium continually dissolve and re-establish their hemidesmosomal attachments leading to the migration of defence cells and constituents of the complement system into the sulcus from adjacent venules of the dentogingival plexus. The initial reaction for the microbial challenge is the activation of the complement system in the gingival sulcus by both classical and alternative pathways. The intermediary factors of the complement cascade may play a role in the phagocytosis by opsonisation, activation of mast cells and acting as important chemotactic proteins. The leukocytes (PMN) and the end products of the complement cascade cause osmotic lysis of the targeted microbes, leading to the release of cytotoxic products for epithelial cells. The migrated monocytes remove these metabolites from the sulcus, so a small number of microbes can be eliminated without causing damage to the epithelium. The IgG and IgA of the sulcular fluid may play a role in the opsonisation and removal of toxic bacterial products.

**THE ROLE OF THE GINGIVAL EPITHELIUM IN THE HOST RESPONSE**
The gingival epithelium serves as a physicochemical barrier with its tight intercellular connection, fast regeneration and salivary cover. The fast regeneration of the epithelium and the continuous desquamation of keratinised cells helps to remove microorganisms.

The gingival epithelium is not only a physical barrier characterised by effective innate functions, but it is also strongly associated with host defence processes of the underlying connective tissue and the adaptive defence system. The cells of the epithelium play an important role in the recognition of the commensal flora, differentiate it from pathogen microbes by Toll-like receptors, CD-1 receptors and soluble LPS-binding proteins. Damage to the epithelial surfaces causes the release of chemokines, cytokines and end products of arachidonic acid cascade. The secretion of IL-8 initiates innate immune system- and later on adaptive immune responses. As a result of cytokine expression, vascular changes occur: dilatation of the capillaries facilitate the migration of PMNs, monocytes and the accumulation of immunomodulatory cells in the epithelium. The Langerhans cells have a key role in the epithelial responses: they play a role in antigen presentation, stimulate the release of intercellular adhesion molecules, secretion of cytokines and chemokines and modulation of T-cell responses.

Antimicrobial peptides play an important role in maintaining the balance between health and disease in the oral environment. Human beta-defensins (hBDs) are constitutively expressed in gingival epithelial cells; however the human beta-defensin 2 is an inducible protein; normal uninflamed oral epithelial tissue is activated to express hBD-2. This exposure represents a useful interaction between the commensal bacteria and the tissue, resulting in an enhanced expression of hBD-2, thereby providing an advantage response to other potentially pathogenic organisms. The amount of alpha-defensins released from neutrophil granulocytes in saliva and crevicular fluids proportional with severity of infection. Alpha-defensins have an effect on a wide spectrum of pathogens, both Gram-positive and Gram-negative microbes, similar to Cathelicidin (LL-37) released by PMNs. In addition, they also have an antifungal effect. The antifungal action of histatin has been known: it binds surface proteins of Candida albicans, then penetrates the cytoplasm and destroys microorganisms.

HOST RESPONSE OF THE CONNECTIVE TISSUE OF THE GINGIVA

The inflammatory response leads to the accumulation of immune cells: however, the PMNs migrate through the junctional epithelium to bind to the Fc receptor of opsonising antibodies and destroy harmful agents. Lymphocytes accumulate in the connective tissue of the gingiva, fixed by CD-44 molecules.

The dendritic (Langerhans) cells migrate to the lymph nodes to present antigens to CD4 T-cells, which will result in the migration of the lymphocytes to the places of the injuries: the B-cells transform into plasma cells, while the T-cells play a role in immune reactions; both cellular and humoral responses.

Specific antibodies released in the gingiva and regional lymph nodes migrate to the damage of the periodontal tissues and act in phagocytosis and lysis of microorganisms and microbe aggregation. Host response leads to the release of harmful, destructive, cytopathic, proteolytic enzymes inhibited by alpha-1-microglobulins and alpha-2-antitripsins.

SYSTEMIC IMMUNE RESPONSE

Exposure to several microbes is considered to be pathogenic and it initiates the production of specific antibodies detectable in the serum. IgG and IgM mediated processes result in the protective action of PMNs and the complement system, while IgA-mediated processes do not support the inflammatory reactions.
Chapter 2. PATHOLOGY

1. Developmental disorders of the face and dentition (Emil Segatto DMD)

Malocclusions of dentofacial deformities are either hereditary or environmentally determined. There are several studies available focusing on the ratio determining environmental and genetic factors in the manifestation of malocclusions. Varying results of these investigations, however, have shown that there are rather genetic effects than dental components that assert predominance on skeletal components of the facial appearance. Thus, a strong influence of heredity on facial features is obvious at a glance, it is easy to recognise familiar tendency in relation of the maxilla as well as the shape of the jaw, and the nasal arch. Two of the most likely deformity types that run in families are prognathic mandible and long face pattern. Environmental influences during the growth and development of the face, jaws, and teeth largely affect genetically determined deformities. Identification of the environmental effects is almost impossible as usually they do not affect the developing body in an isolated way. According to the results of a large scale study published recently, there are only less than 10% of the occlusion disorders in which any origin can be identified.

HEREDITARY DENTOFACIAL DISORDERS

Manifestation of genetic effects can be identified easily through their family heredity. Genetic factors play a role in the development of malocclusions in two ways. The first one would be an inherited disproportion between the size of the teeth and the size of the jaws, which would produce crowding or spacing. The second one would be an inherited disproportion between the size or shape of the upper and lower jaws, which would cause improper occlusal relationships. It is hard to determine the contribution of the genetic factors to the development of individual malocclusions.

ACQUIRED INHERITED DENTOFACIAL DISORDERS

A part of the acquired malfunctions develops during the embryonic stage. Physical impacts on the embryo including birth traumas, as well as bone fractures and their consequences experienced in the developing stage, all result in skeletal disorders. Indeed, muscle dysfunctions can also serve as basis for developmental disorders. Developmental disorders of dentition lead to congenital absence of teeth, abnormal or supernumerary teeth, and eruption disorders.

Embryonic developmental disorders

Adverse environmental influences can manifest as early as in the embryonic stage leading to developmental disorders of various severity. Defects in embryonic development usually result in death of the embryo. Only a relatively small number of recognisable conditions producing dentofacial problems are compatible with long-term survival. The time of the environmental effect determines the type of the developmental disorder, which usually belongs to a certain syndrome. From the third week after fertilisation, maternal high alcohol consumption results in embryonic alcohol syndrome with characteristic midface deficiency. Effects on the embryo on the third and fourth weeks are manifested in disorders such as hemifacial microsomia or mandibulofacial dysostosis. Hemifacial microsomia is caused by defective migration of ganglion cells resulting in that lateral areas being farther from the midline of the face remain undeveloped. It is primarily a unilateral and always an asymmetrical disorder, in which the external ear is deformed typically, and both ramus of the mandible and associated soft tissues (muscle, fascia) are deficient or missing. The best evidence suggests that mandibulofacial dysostosis or Treacher Collins syndrome arises because of excessive cell death (cause unknown) in the trigeminal ganglia. People with this disorder have typically both the maxilla and mandible underdeveloped, the abnormality is symmetrical and affects both sides of the face. Both syndromes develop as a result of certain drugs taken in the early stage of the pregnancy.

Various facial clefts represent the majority of inherited developmental disorders affecting the maxilla and the mandible. Their appearance strongly correlates with impacts being present at the time of the fusion. Cleft lips originate in false fusion of the medial and lateral nasal prominences, which is due in the sixth week of gestation in normal cases. Cleft lip may develop only either side (unilateral cleft lip) or they can affect both sides (bilateral cleft lip). Since the fusion of these processes during primary palatate formation creates not only the lip but the area of the alveolar ridge containing the incisors as well, it is likely that a notch in the alveolar process
will accompany the cleft lip. Closure of the primary palate is followed by closure of the secondary palate by the
elevation of the palatal shelves in nearly 2 weeks’ time, which means that an interference with lip closure can
also directly affect the palate. About 60% of individuals with a cleft lip also have a palatal cleft. An isolated
cleft of the secondary palate is the result of a problem that arises after lip closure has been completed. It can be
full or partial, and the exact place of emergence has also close correlation here with the time of the effect
causing the defect. It is not rare that the defect occurs at the very last moment of the bilateral formulation, and it
is manifested in the form of uvula bifida. The width of the mouth is determined by fusion of the maxillary and
mandibular processes at their lateral extent. Failure of fusion in this area could produce an exceptionally wide
mouth, or macrostomia, and in a more severe case, an obliquely directed cleft of the face. Responsibility of
several external factors in which clefts of the lip and palate develop has been clarified, so it is well-accepted by
now that maternal cigarette smoking is one of the aetiological factors in the development of cleft lip and palate
in the form of fetal hypoxia.

Another major group of craniofacial malformations is the various ossification disorders, which are mainly
represented by the individual synostoses. Synostosis syndromes typically develop during the last stage of fetal
life, and their common feature is early closure of the sutures between the cranial and facial bones. Early closure
of a suture leads to characteristic distortions depending on the location of the early fusion. Crouzon’s syndrome
is the most frequently occurring synostosis. It is characterised by underdevelopment of the midface and eyes,
which seem to bulge from their socket. Crouzon’s syndrome arises because of prenatal fusion of the superior
and posterior sutures of the maxilla along the wall of the orbit. Premature fusion in the orbital area prevents
the maxilla from translating downward and forward thus contributing to the severe underdevelopment of the middle
face. The characteristic protrusion of the eyes is largely an illusion – the eyes appear to bulge outward because
the area beneath them is underdeveloped; however, there may be a component of true extrusion of the eyes,
because intracranial pressure increases when cranial sutures become synostosed.

The association between developmental disorders established as the result of teratogenic effects in fetal life and
the time of the impact is described below:

<table>
<thead>
<tr>
<th>Time (post-fertilization)</th>
<th>Related syndromes</th>
</tr>
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<tbody>
<tr>
<td>Day 17</td>
<td>Fetal alcohol syndrome (FAS)</td>
</tr>
<tr>
<td>Days 18-23</td>
<td>Anencephaly</td>
</tr>
</tbody>
</table>
| Days 19-28               | Hemifacial microsomia  
Mandibulofacial dysostosis  
(Treacher Collins syndrome) |
| Days 28-38               | Cleft lip and/or palate  
Other facial clefts |
| Days 42-55               | Cleft palate |
| Day 50 - birth           | Achondroplasia  
Synostosis syndromes (Crouzon’s, Apert’s) |

The links between development disorders established as the result of teratogenic effects proceeding in fetal life
and the time of the impact

Drugs, viruses, harmful chemicals, and radiation are identified as teratogenic agents being the cause of
dentofacial developmental disorders. Here is an incomplete list of the most frequent ones:
Teratogenic agents identified as cause of dentofacial developmental disorders

**Skeletal growth disturbances**

Facial injuries apparent at the moment of birth are the result of a physical impact on the developing face during fetal life or it may be acquired during delivery. On rare occasions, the arm may be pressed into the face in the uterus causing severe growth disturbance of the maxilla. Sometimes the head of the fetus is flexed tightly against the chest in the uterus preventing the mandible from growing forward normally. The result is an extremely small mandible at birth, usually accompanied by a cleft palate. In this case, the restriction on displacement of the mandible forces the tongue upward and prevents normal closure of the palatal shelves. This extreme mandibular deficiency at birth is called Pierre Robin syndrome, which leads to respiratory difficulty due to the disproportion between the reduced volume of the oral cavity and the size of the tongue; it has an adverse effect on the whole neonatal development. Because the pressure against the face causing the growth disturbance is not present any more after birth, there is a possibility of normal growth thereafter. In many cases, however, the underdeveloped mandible requires long conservative therapy and frequently surgical treatment as well. In case of children with an underdeveloped mandible, auxiliary devices used in surgery were blamed for the adverse effects on temporomandibular joints during difficult births. It is, however, refuted by contemporary developmental theories, which declare that the condylar cartilage is not critical for the proper growth of the mandible. The disproof is confirmed by the prevalence of mandibular underdevelopment, since it has not decreased over the past decades in spite of the fact that the use of forceps in deliveries has actually been ceased in clinical practice.

Among childhood injuries, the fracture of condylar neck of the mandible can cause further growth disturbances. Fortunately, the condylar process tends to regenerate well after an early fracture, and the appearance of consecutive mandibular underdevelopment is much less. Overall analysis results suggest that about 75% of the children had normal further growth. In the remaining cases, scarring developing during the therapy or the healing process may cause growth disturbance in the affected side frequently leading to severe asymmetry.

**Muscle dysfunction**

Facial muscles can affect jaw growth in two ways. First, skeletal development at the point of the muscle arisen or connected to bone largely depends on the activity of the muscle. Second, according to functional matrix theory, soft tissue matrix is decisive in the growth of the jaws, which mainly consists of facial muscles. Loss or underdevelopment of the individual facial muscles can occur in the uterus, but it is most likely to result from damage to the motor nerve. The result would be underdevelopment of that part of the face. Muscle weakness syndromes and muscular dystrophy have similar consequences, these pathologies, however, emerge symmetrically, and are also manifested in an increased growth of the total face length beside full open bite. Otherwise, excessive muscle contraction can restrict growth in the same way as scarring after an injury. This effect is seen most clearly in torticollis, a twisting of the head caused by excessive tonic contraction of the neck muscles on one side. The result is severe facial asymmetry because of growth restriction on the affected side.

**Disturbances of dental development**

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Disturbances of dental development may accompany major congenital defects but they are mostly independent from other malocclusions.

*Congenitally missing teeth*

Congenital absence of teeth results from disturbances during the initial stages of tooth formation. The term hypodontia implies the absence of only a few teeth, the term oligodontia refers to congenital absence of many but not all teeth, while anodontia is describing the total absence of teeth. The lack of primary tooth buds results in the lack of the relevant permanent tooth buds, while the lack of the permanent tooth buds does not refer automatically to the lack of primary tooth buds. Anodontia and oligodontia are usually associated with a systemic abnormality, ectodermal dysplasia, which is also characterised by thin, sparse hair and an absence of the sweat glands. Whereas oligodontia may occur in a patient without congenital syndromes. Anodontia and oligodontia are rare, but hypodontia is relatively common. It is a general rule that individual missing tooth buds always belong to the most distal teeth of the given group. If a molar tooth is congenitally missing, it is almost always the third molar; if an incisor is missing, it is nearly always the lateral one; if a premolar is missing, it is almost always the second rather than the first one. Rarely is a canine the only missing tooth.

*Malformed and supernumerary teeth*

Abnormalities in tooth size and shape result from disturbances during the stage of morphodifferentiation. The most common abnormality is a variation in size, particularly of the maxillary lateral incisors and the second premolar. About 5% of the total population have a significant tooth size discrepancy because of disproportionate sizes of the upper and lower teeth. Occasionally, tooth buds may fuse or geminate during their development. Fusion results in teeth with separate pulp chambers joined at the dentin, whereas gemination results in teeth with a common pulp chamber by bud fissure. Normal occlusion is all but impossible in the presence of gminated, fused or otherwise malformed teeth. Supernumerary or extra teeth always result from disturbances during the initiation and proliferation stages of dental development. The most common supernumerary tooth appears in the maxillary midline and is called a mesiodens. Supernumerary lateral incisors, premolars and molars develop very rarely. The presence of an extra tooth obviously has great potential to disrupt normal occlusal development. Multiple supernumerary teeth are most often seen in the congenital syndrome of cleidocranial dysplasia, which is characterised by missing clavicles beside tooth eruption difficulties.

*Interference with eruption*

For a permanent tooth to erupt, the overlying bone as well as the primary tooth roots must resorb. Similarly, gingiva should also promote tooth eruption. Supernumerary teeth, sclerotic bone, and heavy fibrous gingiva can obstruct eruption. In patients with less severe interferences with eruption, delayed eruption of some permanent teeth contributes to malocclusion when other teeth drift to improper positions in the arch. Eruption of permanent teeth can be delayed by radicular resorption of the affected primary tooth, which can also be caused by the ankylosis (ossification) of the roots of the primary tooth. In such cases, delayed permanent tooth eruption usually does not result in malocclusion.

*Ectopic eruption*

Occasionally, malposition of a permanent tooth bud can lead to eruption in the wrong place. Ectopic eruption is most likely to occur in the eruption of maxillary first molars. If the eruption path of the maxillary first molar carries it too far mesially at an early stage, the permanent molar is unable to erupt, and the root of the second primary molar may be damaged and resorbed. The mesial position of the permanent molar results in the shortening of the arch, which will lead to lack of space at incisor level and consecutively to crowding.

Lack of space is frequently the reason for the altered eruption path and ectopic eruption. For example, the position of ectopic canines in the maxilla is to be blamed for the narrowed space due to adjacent teeth, which erupted earlier in most cases.

*Early loss of primary teeth*

Permanent molars are likely to drift mesially and forward during their eruption in the absence of occlusal contacts. Mesial drift of the permanent first molar after a primary second molar is lost prematurely, if this loss occurs more than one year before the eruption of the homologous second permanent molar, consecutively it leads to a shortened dental arch in most cases. Loss of primary first molars or canines provides favourable conditions for the distal drift of the homologous first permanent premolars and canines, which is significantly promoted by the pressure from the lips.
**Traumatic displacement of teeth**

Dental trauma can lead to the development of malocclusion in three ways:

1. Damage to permanent tooth buds from an injury to primary teeth.
2. Drift of permanent teeth after premature loss of primary teeth.
3. Direct injury to permanent teeth.

Trauma to a primary tooth can displace the permanent tooth bud underlying it. If the trauma occurs while the crown of the permanent tooth is forming, enamel formation will be disturbed. If the trauma occurs after the crown is complete, the crown may be displaced relative to the root. Root formation may stop, leaving a permanently shortened or bent root. The usual cause of dilacerations is mechanical trauma to the primary incisors.

**DentoFacial Defects Acquired During Growth**

Environmental impacts may also trigger or aggravate malocclusion in the postnatal developmental period. Some of these effects are associated with physiological functions, and others are the result of bad habits.

Physiological functions are, for example, feeding habits of the baby, holding of the head during sleep, type of the developed swallowing and respiration. Bad habits developed and maintained during life may contribute to the development of the individual deviations by upsetting muscle balance between lips and tongue. Such bad habits are, for example, chewing different objects; sucking and chewing of lips and cheek; bruxism, etc.

Corrective interventions based on a comprehensive and modern approach should correct malocclusion by the elimination of the causes. The stability of adulthood treatments confirms that causes of deviations are derived in the developmental period.

### 2. Temporomandibular Disorders (Zoltán Baráth DMD)

Temporomandibular disorders involve not only the disorders of the anatomical structures in the temporomandibular joint but the disorders of the masticatory muscle as well, and the classification can be seen in the next table:

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Classification of the Temporomandibular joint disorders

**Disorders of the Temporomandibular Joint**

**Disc Displacement (internal derangement)**
It is characterized by the abnormal displacement of the articular disc and so the abnormal movement of the condyle and the disc. The disc is most often displaced anteriorly or anteromedially. For the dislocation of the disc, stretching of the ligaments is needed, and the thinning of the posterior edge of the disc. Disc displacement with reduction refers to the stage in which the disc returns to a more normal position on opening. Disc displacement without reduction (“closed lock”) is characterized by the definitive deformity and compression of the disc, when it remains at the back in front of the condyle on opening limiting the condylar translation. The most characteristic feature of disc displacement with reduction is clicking or popping sounds during mandibular opening and closing. In case of chronic disc displacement without reduction, the severe degenerative lesion may result in crepitation. The pain accompanying acute disc displacement with reduction generally emanates from a strained disc ligament or from condylar pressure against the posterior attachment. The mandibular range of motion is usually normal, and the vertical opening may be greater than normal. Deviation appears on the affected joint that is caused by the translatory obstruction of the displaced disc. When reduction of the disc occurs, the deviation disappears, and the mandible returns to a centred position. When the condyle cannot return to the normal position, the opening of the mouth is intensely limited, and deflexion can be observed during opening on the affected side (20–30). Propulsion and contralateral balanced movements are limited and painful.

The joint affected by acute trauma may cause the anterior disc displacement. (anterior disc displacement-ADD). Acute anterior disc displacement without reduction is painful due to inflammation in the articular capsule, posterior attachment, and disc ligaments. The muscle activity of the temporal and masster on the affected side is usually increased, beside the restricted opening and pain. In an acute phase, no sound effects are audible.

A TMJ hypermobility (instability, subluxation, luxation–dislocation)

It is accompanied by pain in case of chronic condition and it causes wide opening noise.

TMJ hypomobility

Pseudoankylosis of the TMJ with hypomobility may have psychogenic reasons or a lesion in the neurogenic system, in muscles or in bones. True ankylosis is the bony/fibrous ankylosis of the condyle and fossa in the TMJ. The most frequent aetiological factor is trauma. There are many vessels between the condyle and the capsule. In case of trauma, the cortical bone is damaged and the vessels in the capsule are lacerated and the developed haematoma ossifies. The range of motion is getting limited. The osseus/fibrous tissue occupies the space in the joint and the joint space disappears. It is rare in adulthood.

Fibrotic hypomobility of the TMJ may develop after radiotherapy of the head.

Degenerative lesions

They are the most frequent organic diseases of the TMJ. They are the final consequences of many insults influencing the surface of the TMJ. The process may be inflammatory as in capsulitis, synovitis, retrodiscitis, the symptoms of which may be pain, malocclusion and swelling. Patients with gout have too much uric acid in the blood and hard crystals may be formed in the TMJ. The most common sign of gout is a night-time attack of swelling, tenderness, redness in the TMJ and painful in motion. The radiographic signs are very similar to that of the tumorous lesions.

Rheumatoid arthritis is the autoimmune disease of small joints. It affects the lining of the joints, causing a painful swelling that can result in 10–15 % in bone erosion and joint deformity. It impairs the TMJ and the developing granulomatous tissue is rich in vasculisation and limits the motion. Clinical signs are intermittent pain, swelling and loss of function. Most commonly the small joints of the hands and feet are involved. The rheumatic lesions can only be found in the early stage of the disease in the soft tissue, that is why there are no radiographic signs. Later multiple erosions are developed on the disc, subchondral cysts may develop, the joint space is slowly narrowed, and bone decay and osteoporosis can be detected.

Osteoarthrosis (osteoarthritis) is a non-inflammatory, degenerative disease that influences the joint’s surface, but it may result in bone remodelling. The symptoms are similar to that of the other TMJ diseases such as pain, limited motion, and crepitation.

Hereditary and congenital disorders of the TMJ

Congenital hypoplasia–aplasia
**Hemifacial microsomia** is a hypoplasia or aplasia of the TMJ. It is an asymmetrical, progressive defect that affects the bony structure and soft tissue of the skull. The asymmetrical development of the mandible is the first sign, which is getting typical year by year. The mass of masticatory muscles and the subcutis is smaller than on the healthy side. It is frequently accompanied by the paresis of the facial nerve. The ear may be completely healthy or the whole organ may be missing on one side.

**Treacher-Collins syndrome** is a hypoplasia of the TMJ with short ramus and reduced facial height. It is an autosomal dominant congenital disorder, which always appears symmetrically. The physical features are downward slanting eyes, conductive hearing loss, micrognathia, malformed or absent ears and drooping part of the lateral lower eyelids.

**Congenital hyperplasia**

It is the most frequent disorder of the TMJ after birth. It is frequent in women, appears early in adolescence and gets typical with the development. The excessive metabolism of TMJ is supposed to be the main reason. Two growth tendencies may be distinguished: the mandible grows in a vertical direction resulting in long ramus and basis vertically. There is open bite on the involved side. A rotációs növekedési irány esetén, nemcsak nagyobb fejecs és verticálisan hosszabb ramus van jelen, hanem az állkapocs testének konvex megnagyobbodása folytán, keresztharapás és szájnyitáskor deviáció jön létre. The excessive metabolism of involved TMJ can be proved by bonescintigraphy.

**Infections of the TMJ**

Intense pain is indicative of a developing infection. Oedema and redness appear above the TMJ. The presence of a fistula reflects that the disease is becoming chronic. Chronic inflammation may result in deformity, sequester formation and later ankylosis. Predisposing factors are malnutrition, debility and the immunosuppressive processes. Staphylococcus aureus is the main reason for the infection.

**Traumatic injuries in TMJ**

The traumatic injury may be a contusion, haematoma or fracture. According to the situation of fragment to ramus relation, we can distinguish between fractures without dislocation, greenstick fractures with deviation in different level, fractures with dislocation and with or without luxation of the condyle, and sometimes the condyle may protrude into the medial cranial fossa. The later complication may be facial asymmetry, malocclusion in adolescence, developmental disturbance in children, osteoarthritis and ankylosis.

**Tumours**

Benign and malignant tumours and metastases are very rare in the TMJ. The most frequent benign tumour is osteochondroma, which interferes with the functioning of the TMJ and erodes the neighbouring bony structures.

**MUSCLE DISORDERS**

**Acute muscle pain**

Splitting pain is a reflexive, protective response involving the central nervous system. It occurs when a part of the body is injured and it requires rest. It may be a response to a straining activity, from increased tension as seen in clenching, bruxism, prolonged mouth opening or trauma. Myospasmodic pain is manifested as an involuntary sudden tonic contraction causing pain and limited range of motion. Myospasmodic pain may vary from a dull, aching quality to an occasionally sharp one, and can be differentiated from arthralgia because myalgia is more diffuse. Myospasm can be examined by palpation and functional examination.

**Chronic muscle pain**

Myofascial pain is characterised by pain originating from a trigger point within the myofascial structures. The area of perceived pain provoked at the irritable trigger points is known as the zone of reference. The pain can be reproducible by stimulating the trigger points.

Fibromyalgia is a systemic soft tissue disease characterised by aching throughout the body. The pain is accompanied by reproducible tender points at specific anatomic locations. It is frequently associated with sleep disturbances, low plasma level of the neurotransmitter serotonin. Muscle contracture is the clinical shortening of the resting length of a muscle without interfering with its ability to contract further. Myostatic contracture is
kept in a partially contracted state, without fully relaxing for a prolonged period of time. Myofibrotic contracture results in the formation of scar tissue that prevents full restoration of the range of motion.

3. Maxillofacial injuries (Renáta Varga MD)

In the first four decades of life, the leading causes of death include traumatological injuries, and the severity of maxillofacial injuries is directly proportional to increasing work disabilities reported by the patients. The rapid development of motor vehicles and increase in urban violence show intense correlation with the incidence of maxillofacial injuries. This area can be reached easily and can be injured readily due to its anatomical features. Maxillofacial fractures are often associated with severe morbidity, loss of function, disfigurement and substantial financial costs.

Several aetiological factors can influence the incidence of maxillofacial injuries, such as the age, gender, geographic region, cultural aspects, socioeconomic status, the use of alcohol and drugs, compliance with road traffic legislation, domestic violence etc. For instance, maxillofacial injuries are less frequent in developed countries as a result of stricter road traffic rules and regulations (no alcohol consumption, use of seat belts and air bags). At the same time, bicycle accident-induced maxillofacial fractures are increasing because this type of transport is getting more popular in some European countries. In developed countries, the leading cause is violence, whilst traffic accidents are the most common trigger factors of maxillofacial injuries in the developing countries. 30 percent of traumas can be associated with maxillofacial fractures.

CLASSIFICATION OF MAXILLOFACIAL FRACTURES

Fractures can be classified according to different aspects.

Classification based on communication with the external environment

1. Closed fracture
2. Open fracture

The types of fracture

1. Tear – infraction (simplex fracture): this implies a fracture of the ramus or the condyle and does not cause malocclusion.
2. Greenstick fracture: this type frequently occurs in children and rarely in edentulous jaws with no tears in the periosteum and parodontium; therefore it can be regarded as a closed fracture.
3. Compound fracture: it involves complete loss of the continuity of the bone. It is the most common type of the fractures and it is often associated with dislocation and can be regarded as an open type.
4. Impacted fracture: it implies that one bony fragment is forcibly driven into another.
5. Comminuted fracture: it occurs in both of the jaws but generally it is a feature of midfacial fractures.
6. Pathological fracture: this type develops when a fracture results from normal function or minimal trauma in a bone weakened by different pathological conditions, such as tumour, osteomyelitis and osteonecrosis).
7. Complicated fracture: it implies damage to structures adjacent to the bone such as major vessels (facial artery), vein and nerve (inferior alveolar nerve).

Classification based on displacement

1. Displacement and deviation is not present between the fractured fragments (e.g. incomplete fracture).
2. Displacement is not present but deviation occurs (e.g. greenstick fracture).
3. Displacement develops between the proximal and distal bony fragments called dislocated fractures.

Factors affecting fragment displacement are the following:
1. **Muscle force:** the displacement is affected by mandibular muscle attachments, which are divided into two groups based on the direction of the muscle forces: closing muscles (masseter, temporalis, medial and lateral pterygoid muscles) pull the distal fragment superiorly and medially; opening muscles (geniohyoid, stylohyoid, digastric and genioglossus muscles) pull the proximal fragment inferiorly and medially.

2. **The direction of fracture line:**
   - Favourable fracture line is defined as a fracture line which proceeds obliquely from top to down, and in the mediolateral direction it proceeds obliquely dorsoventrally because the distal fragment interferes with the development of the muscle force.
   - Unfavourable fracture line is defined as a fracture line which is the opposite of the above mentioned, where the muscle force predominates completely.

3. **Dental status:** in case of an unfavourable fracture line, tooth-bearing bony portion influences the displacement of the proximal fragment. In case of tooth-bearing jaws, the fracture line proceeds the parodontium therefore, it is regarded as an open fracture. In case of edentulous jaws, closed fracture can develop if the periosteum is not injured. Closed fracture can occur in the fracture of the ramus and condyle process.

4. **Adjacent soft-tissue injury:** in case of comminuted fracture with soft-tissue injury, the soft-tissue attached to the bony fragments can increase dislocation.

5. **The size and the direction of the contact:** direct fracture arises immediately adjacent to the point of the contact of the trauma, whereas indirect fracture arises further away from the site of the fracturing force. An example of this is a subcondylar fracture occurring in combination with a symphysis fracture.

**Classification based on anatomical location**

The lower jaw (mandible) has a strong bony substance, where the compact cortical bone provides resistance against trauma. The thickest part appears anteriorly and the bony thickness decreases towards the angles and condylar process enhancing the risk of fracture.

**The pattern of mandibular fractures:**

1. **Dentoalveolar fracture**

2. **Symphysis and parasymphysis fracture (15%)**

3. **Body fracture (25%)**

4. **Angle fracture (25%)**

5. **Ascending ramus fracture (3%)**

6. **Condylar process fracture (30%):** it can be classified as
   - subcondylar or extracapsular (fracture line below the colllum) and
   - condylar or intracapsular fractures (within the capsule)

7. **Muscular process fracture (2%)**

**Midfacial fractures:**

Midface is defined as part of the facial skeleton which extends between a superior plane drawn through the zygomatico-frONTAL sutures tangential to the base of the skull and an inferior plane at the level of the maxillary dental occlusal surface. Borders: tangential line between both zygomatico-frontal sutures, the sphenomaxillary point - posteriorly, pterygoid process of the sphenoid bone - caudally.
The midface consists of the maxilla, palatal bone, zygoma, nasal bone, lacrimal bone, vomer, ethmoidal bone with its adherent nasal concha and pterygoid process. Midfacial fracture can be regarded as an open fracture affecting the maxillary sinus.

Different Classification Systems:

I. Le Fort Classification System:

1. Le Fort I fracture: the line of fracture starts from the anterior bony aperture of the nose through the facial wall of the maxillary sinus, the zygomatico-alveolar crest, the maxillary tuberosity, the caudal apex of the pterygoid process, the medial nasal wall of the maxillary sinus and then meets the point of exit again at the anterior bony aperture of the nose. The vomer is affected and the cartilaginous nasal septum is often dislocated.

2. Le Fort II fracture: The line of fracture starts above or within the nasal bones, the medial and inferior orbital wall, through the infra-orbital canal, infra-orbital foramen and facial wall of the maxillary sinus, through the infrazygomatic crist and usually splits the pterygoid process in the middle third.

3. Le Fort III fracture: The fracture line in the middle starts similarly to the Le Fort II fracture, but it runs anterolaterally to the lateral orbital margin along the zygomaticosphenoidal suture as far as to the fronto-zygomatic suture below the level of skull base.

II. Topographic Classification System:

1. Central: The fracture line runs from the frontonasal suture, through the medial orbital wall, maxilla and reaches the zygomatic bones bilaterally. (alveolar process bone fracture, Le Fort I fracture, Le Fort II fracture, nasal bone fracture, naso-orbito-ethmoidal fracture)

2. Centrolateral: The viscerocranium (facial skeleton) is completely separated from the neurocranium. (Le Fort III fracture)


III. Classification based on occlusal disturbance:

Fractures without occlusal disturbance:

1. Nasal bone fracture
2. Isolated orbital fracture
3. Naso-orbito-ethmoidal fracture
4. Nasofrontal fracture
5. Zygomatic fracture
6. Isolated zygomatic arch fracture

Fractures with occlusal disturbance:

1. Dentoalveolar fracture
2. Maxillary tuberosity fracture
3. Sagittal fracture of the maxilla
4. Le Fort I fracture
5. Le Fort II fracture
6. Le Fort III fracture
7. Zygomaticomaxillary fracture

ASSESSMENT OF FRACTURES

Fractures can be diagnosed with physical (extra- and intraoral) and radiological (X-ray, CT, MRI) examinations. The suspicion of fracture can be determined according to uncertain signs and symptoms.

Uncertain signs:
1. Pain (spontaneous, during function)
2. Swelling (at the place of the fracture which can be the result of bleeding, oedema or subcutaneous emphysema)
3. Soft-tissue injuries
4. Functioning disturbance (mastication, limitation of mouth-opening)

Certain signs:
1. Detectable disfigurement (e.g. occlusal disturbance, deformity)
2. Pathological mobility
3. Crepitation (between fractured fragments)
4. Radiological examination

4. Inflammations of the head and neck region (Csaba Berkovics DMD)

Inflammation is a response of tissues to harmful stimuli. The goal of the inflammatory process is to eliminate or isolate the initiating stimulus (e.g. an infectious agent) and harmful sequelae (e.g. toxins and necrotic debris). Macroscopic (i.e. visible and readily felt) symptoms of the inflammation are swelling (tumour), redness (rubor), pain (dolor), heat (calor) and function loss in the given organ (functio laesa).

CLASSIFICATION

Based on the causative agent

1. Physical harm: The most frequent of them is mechanical trauma. An inflammation is to be expected subsequently to trauma affecting either bony or soft tissues (even in cases of iatrogenic trauma, like tooth extraction). Chronic, non-physiological load of the stomatognathic system (either in magnitude or direction) can lead to inflammation in the temporomandibular joint and the tissues surrounding the teeth. Improperly fitting prosthetic work causes inflammation through chronic mechanical irritation. Heat: a single burn of the oral mucosa generates quickly, but repeated exposure to high temperatures induces chronic inflammation, which can easily lead to premalignancy and tumours. It is, therefore, of vital importance that during oral surgical interventions requiring bone drilling, proper heating be used to minimise friction heat. Over 45–47 degrees of Celsius, bone dies (necrosis), which may lead to a prolonged inflammation of the operating site. Radiation: Radioactive irradiation of the facial skeleton (orofacial region) may induce a chronic inflammation of the oral mucosa depending on the dose (radiomucositis). Larger doses can even lead to the inflammation of the bone marrow (osteomyelitis) or to its destruction (osteoradionecrosis).

2. Chemical agents: In the practice of oral surgery, inflammation of a chemical origin occurs most frequently as a iatrogenic (acquired during treatment) damage. Anaesthetic tablets placed on the mucosa to alleviate tooth pains often cause irritative inflammation (stomatitis medicamentosa). Endodontic antiseptic irrigants (e.g. NaOCl) may also provoke an inflammation, if proper isolation is not paid satisfactory attention to. Prolonged exposure can lead to mucositis, but these agents can also be passed to the apex to cause an inflammation of the periapical tissues (periodontitis periapicalis acuta). Filling material squeezed through the apex causes inflammation likewise. Bisphosphonates, utilised to treat osteopenia and the bone manifestations of malignant tumours, pose a serious problem today, as a side-effect, they may cause the necrosis of the jaw
bones, and inflammation of the surrounding tissues (BRONJ – Bisphosphonate-Related Osteonecrosis of the Jaws). **Hypersensitivity** (anaphylactic) reactions to certain medications (anaesthetics, antibiotics, antiseptics, etc.) are also to be mentioned here. Such reactions range from skin rash (urticaria), through laryngeal oedema (Quincke-oedema) to circulatory collapse due to an anaphylactic shock.

3. **Biological agents (infection):** The various **microbiological agents** (e.g. bacteria, viruses, fungi, or parasites) are the most common causes of inflammation in the head and neck region. Bacterial inflammation is far the most frequently seen out of them. Henceforth, we focus our discussion on them.

**Specificity of microorganisms**

Inflammations of infectious origin may be classified as specific and nonspecific. Nonspecific inflammations are caused by organisms that are common in our everyday environment (in this case, the oral flora). Specific inflammations are characterised by a specific causative agent (e.g. a given species of bacteria) and a characteristic pathological pattern caused by that agent (see later).

**Spreading of microorganisms**

Infection may take place in several ways:

1. **Through the pulp cavity (transdental, transosseal).** This can happen in pulpitis (due to caries or trauma), when after the necrosis of the pulp, the infectious agents pass through the foramen apicale and get into the periapical space, and they may spread on to the bone (os), bone marrow (medulla ossium), the periosteum or connective tissues.

2. **Through exposed connective tissue (submucosal way).** This is characteristic of periodontal pockets, in infections of the follicular sacs of erupting teeth, and in fractures of the jaw bones.

3. **Through circulation (haematogenic or lymphogenic).** A rare way, but one that is important to remember. An example for it is the spreading of the infection of the upper lip to the sinus cavernosus (in the anterior fossa), via the angular vein. Such an infection may cause the thrombosis of the sinus, and it can lead to the inflammation and infection of other structures of the neurocranium. Abscesses and furuncles of the upper lip should therefore never be compressed during their treatment. Infectious diseases of the childhood (like scarlet fever) sometimes cause osteomyelitis in a haematogenic way, most often in the mandible.

4. **Directly.** Foreign bodies (and also injection needles) can introduce infectious agents into the tissues. A needle that previously contacted the teeth should never be used for injections, as the bacterial biofilm on the surface of the teeth attaches to the surface of the needle!

**Origin of infection**

Inflammations of the pulp (endodontium) and the tooth-supporing tissues (periodontium) are referred to as **odontogenic inflammations.** These can be either of endodontic or periodontic origin (or both). Based on the chronological development of the inflammatory process, they may be described as ”perio-endodontic” or ”endo-periodontic” inflammations.

Inflammatory processes originating in other structures of the head and neck region are **non-odontogenic inflammations.** They include the inflammations of the rest of the stomatognathic system, which are also subject to oral surgery. Arthritis of the temporomandibular joint, inflammations of the salivary glands (sialadenitis) and the lymph nodes are among them.

Otitis, rhinitis and sinusitis are described as **rhinogenic and otogenic** inflammations, but inflammations can also originate in the skin and its accessory structures (dermatogenic inflammations) such as furuncles (infection of the hair follicle), or erysipelas (bacterial infection of the upper dermis and superficial lymphatics).

When microbes enter the body through an injury, it is considered to be of **traumatogenic** origin.

*It must not be forgotten that structures of the head/face are very close to each other, which allows inflammation to spread rapidly from one to the other. Sinusitis, for instance, can be mostly traced back to a rhinogenic cause, but upper teeth infection or an undetected antro-oral fistula (opening of the maxillary sinus by extraction) can also lead to this!*
**Course of process**

According to the course of the process, an inflammation may be:

1. **Hyperacute:** Extremely rapid progression, usually lethal if untreated (e.g. asphyxia in laryngeal oedema due to an anaphylactic reaction).

2. **Acute:** Symptoms of inflammation develop over a course of minutes to hours. Lasts only for a few days.

3. **Subacute:** The process lasts 4 to 8 weeks, does not necessary exhibit all inflammatory signs or not to an extent seen in the acute course.

4. **Chronic:** Only a few of the inflammatory symptoms are present, and this form may last for years.

These types, except for hyperacute inflammation, form a continuum, and they may be transformed into each other.

**According to exudates**

According to the presence of pus production, inflammatory processes may be:

1. **Serous** if the swelling of the inflamed area is caused by plasma extravasation (i.e. leakage from the blood vessels into the interstitium). The common characteristic feature in acute and in subacute inflammations is that they do not form an abscess.

2. **Purulent** if pus is produced in the inflamed area. Pus contains mostly leukocytes, somatic cells and dead microorganisms. Pus is usually collected in a central area of the inflammation (abscess formation, see later), but it may also be drained from the affected area (fistula, below).

**Further signs, manifestations and complications**

1. **Infiltration:** Seen in serous inflammation. Swelling of the soft tissues due to plasma extravasation and redness of the skin (intense capillary circulation). The surface feels harder than that is normal in the given area.

2. **Fistula** Fistula is a (pathological) pipelike connection between the area of the inflammation and the outside world (or body cavity) that drains inflammatory exsudates from the affected area.

3. **Granuloma:** Once a chronic inflammation has developed, the body attempts to confine the affected area. To reach that end, proliferation of connective tissue is initiated, which soon occupies the area of inflammation (granulomatous tissue). There is also strong vascularisation, which permits an ample supply of the interstitium of the granuloma with immune cells. The interstitium itself contains parallel running collagen fibers which are produced by the cells of the connective tissue. Granulomas characteristically appear around openings of fistulas, in the infected periapical space and in inflamed periodontal pockets. The structure is rather fragile and exhibits strong bleeding on touch.

4. **Abscess** Abscess is an accumulation of pus confined by a limiting layer, which is made up of an internal pyogenic membrane (neutrophil-rich granulomatous tissue) and an external membrane of fibroblasts.

5. **Phlegmonous spread** Weak immunity and/or extremely virulent causative agents may result in failure to eliminate the infection. Such a setting might give rise to phlegmonous spread, which means that the causative agents begin to spread all over the body in the connective tissue between various structures causing inflammation along their way. The affected tissue is red and feels rock hard. In the head and neck region, the process usually starts from the infection of the lower molars. High fever and a general feeling of malaise are usually also reported. A specific type of phlegmonous inflammation in the head and neck region is Ludwig’s angina (Angina Ludovici, named after the person who first described it). It is a bilateral phlegmonous process of the spatum submandibulare and the spatum sublinguale, which pushes the tongue up towards the pharynx and causes oedema. It may consequently lead to the death of the patient by suffocation. The inflammation can spread on to the area of the perpendicular portion of the mandible (ramus), between the bone and the muscles. The space between the masticatory muscle (m. masseter) and the ramus is called spatum masseterica. This is a pathway toward spatie around the temporal muscles (spatum temporalis superficialis et profunda). Medially, the spatum pterygomandibulare is found. It is limited laterally by the ramus, medially
by the medial pterygoid muscle, superiorly by the lateral pterygoid muscle, and dorsally by the parotid gland. The inferior alveolar nerves and blood vessels run here, and also the lingual and buccinator nerves; therefore, it is this space where the anaesthetic solution is injected in conduction anaesthesia of the mandible. (Watch out for the danger of infection!) This space can also serve as an entry for infections to the parapharyngeal space, where abscesses may be formed this way, but also toward the mediastinum.

6. Empyema Inflammation of hollow organs may result in pus accumulation in the natural cavity of the organ. Untreated or treatment-resistant infections of the head and neck region may reach the thoracic cavity by phlegmonous spread and cause the inflammation of the thoracic organs and/or the serous membranes surrounding them, causing life-threatening states. Such infections heal with adhesions (e.g. between the two pleurae), which limit the normal functioning of the given organ and deteriorate quality of life to a considerable extent.

7. Osteomyelitis (inflammation of the bone marrow) Once the causative agent reaches the bone marrow of a bone with thick cortex (e.g. mandible) in an odontogenic, traumatogenic or haematogenic way, the defensive processes cannot drive it out of the bone through its thick cortex. It leads to an acute inflammation of the marrow. Acute osteomyelitis is characterised by high fever, a general feeling of malaise, swelling of the face and mobility of the source tooth (later the neighboring teeth as well). Movement of the affected teeth often leads to pus discharging from next to the given teeth. Radiologically the process is either not detectable or only late in its course. Insufficient or late treatment can lead to chronicisation, the acute symptoms disappear, and the inflammation may break out of the bone toward the oral cavity or the skin (fistula). Bone in the affected area dies and gets reabsorbed. The radiological picture is diagnostic: a radiolucent area with a "moth-eaten" perimeter with fragments of necrotised bone (sequesters).

- Two subtypes, focal and diffuse sclerotising osteomyelitis, can cause bone condensation. The focal type is anorganic hyperdensity confined to a small area (a white spot in X-ray images), while the diffuse type affects a larger area, and it is usually traced back to immune deficiency.

- Ostitis alveolaris is the iatrogenic form of osteomyelitis. When a tooth is extracted from an inflamed site or with considerable trauma (however, it was also reported in connection with atraumatic extractions), it sometimes happens that blood clotting in the alveolus is not satisfactory. The blood clot either breaks up because of an infection, or the vasoconstrictor component of the local anaesthetic (epinephrine) prevents satisfactory clotting. In lack of a proper clot, bacteria infiltrate the exposed bone and cause local myelitis. On the first to third day after the extraction, heavy pain that radiates to the neighbouring teeth and bad breath (foetor ex ore) signify the presence of this complication. Usually it lasts for 8 to 10 days, however, under certain circumstances (presence of bone sequesters or a residual root splinter), chronicisation is imminent.

**ODONTOGENIC INFLAMMATION AND THEIR COURSE**

**Acute periapical inflammation (Periodontitis periapicalis acuta)**

In most cases, it is secondary to caries, but chemical irritation, overload and trauma are also among the causes. Usually it follows pulpitis or the decay of the pulp (gangraena pulpaet), but it can also occur as exacerbation of a chronic process.

The affected tooth is sensitive to probing and biting, and the patient characteristically feels as if the tooth were longer than the other teeth. Pressing of the periapical bone is painful, which is a result of plasma exudation between the tooth and the alveolus. This plasma exudate also contains leukocytes, and therefore, pus production sometimes leading to periapical abscess formation is to be expected. (In this phase, the endodontic treatment of the tooth is still possible.)

Unless the causative agent is eliminated, the inflammatory process spreads over the neighbouring areas. In most cases, bone resorption takes place, and the infection breaks out toward the periosteum, giving rise to a subperiosteal infiltration or abscess. It usually happens on the vestibular side, as the bone is the thinnest here, but palatal (upper teeth) and rarely lingual (lower teeth) manifestations are also possible.

Spontaneous, pulsating pain is characteristic, because of the distension of the periosteum. If the inflammation ruptures the periosteum and the accumulated pus is discharged under the mucosa, a submucous abscess is formed. Then the pain usually disappears, as the distension of the periosteum ceases. The swelling, however,
grows, and its surface is red, but it is soft to touch and fluctuates (like a soft plastic sac with liquid in it when pushed).

Weak immunity or a heavy infection might give rise to a phlegmonous spread, and it can even cause osteomyelitis. No specific radiological sign is known, but sometimes a widening of the periodontal gap is seen.

**Chronic periapical inflammation (Periodontitis periapicalis chronica)**

If the cause of the acute inflammation is not eliminated, chronification may occur. It might be due to the proliferation of bacteria in the post-necrotic pulp chamber. The pulp chamber, being relatively unaccessible for the immune system, is optimal for this, especially after tissue necrosis. In such cases, the pulp chamber functions like a depot of bacteria making the tissues susceptible to re-infection. Retained inflammatory exsudate or continuous physical irritation (filling material in the periapical tissue) can also be in the background.

Inflammation induces osteolysis of the periapical area, which is seen as periapical radiolucency in X-ray images (appr. 4–5mm in diameter). The place of the bone is occupied by a granuloma. In the granuloma, pus may be formed, which can break through the bone and the oral mucosa by which a fistula develops. Sometimes it communicates also with the skin of the face. The fistula drains exsudates toward the outside. If the opening of the fistula gets blocked, a small pustula is visible on the mucous membrane. When pus cannot be drained, there is a risk of periapical abscess formation, which can give rise to chronification of the inflammatory process.

The periapical area may contain epithelial cell rests of Malassez. Irritation stimulates these cells to form a cyst wall, whereby a periapical cyst develops destructing the bone (see also in cysts).

Chronic periapical inflammation is poor in symptoms, if there are any symptoms at all. Mild intermittent pain, sensitivity to probing, and fistulae can be tell-tale signs. Infectogenic teeth are always dead teeth, and prolonged inflammation can cause not only local, but also systemic damage.

A small, localised inflammatory focus (e.g. tonsillitis, paranasal sinusitis, periapical inflammation) discharges microbes and microbial toxins, which the blood carries to distant parts of the body causing various symptoms. Glomerulonephritis, rheumatoid arthritis, myositis, neuritis, iritis, alopecia areata (patchy baldness) and eczema may all stem from a single inflammatory focus. All chronic inflammations are considered foci, but the activity of the focus can be verified only if its elimination brings about the disappearance of the systemic symptoms (ex juvantibus). Teeth with intact pulp and periodontium are not considered inflammatory foci, neither are teeth with a full root filling (provided there is no osteolysis periapically). The size of the process is in no correlation with its activity as a focus.

**Pericoronitis**

Inflammation in the tissues around the crown of erupting teeth. It can occur in childhood when the permanent teeth erupt, but it is seen more frequently in case of wisdom teeth (also impacted ones).

The first sign is mild pain, but with the aggravation of the inflammation, the pain gets stronger and radiates into the ear. The tissues around the affected tooth are swollen, and pus is discharged from the pocket around the crown. Swelling of the lymph nodes may be an accompanying sign.

The spread of the process toward the spatium pterygomandibulare can lead to swallowing problems and limited mouth opening.

**SPECIFIC INFLAMMATIONS**

**Actinomycosis**

It can be caused by bacterial species Actinomyces israelii (anaerobic) and Nocardia asteroides (aerobic). These species are often found in the oral flora. They cause granulomatous infection with multiple abscesses. The face, the neck and the oral cavity are the most frequently affected sites.

The infection can start out from the tonsils, from an extraction wound or a periodontal pocket. At the infection site, rock hard infiltration develops. The process often spreads towards the submandibular region. Abscess formation and discharging may also occur. The discharge contains yellow granules, which is typical of this infection.
To establish the diagnosis, however, histological and microbiological methods are necessary. The infection often heals with distorting scars.

**Tuberculosis**

This disease is characteristic in people living in poverty, but it can also be seen in immunosuppressed individuals. It used to be a pandemic disease. Its most prevalent type is the tuberculosis of the lungs, but via haematogenic spread, it can attack the head and neck regions, too, in the form of chronic lymphadenitis and sialitis. Sometimes the skin, the oral mucosa and the bones are also affected.

**Toxoplasmosis**

Toxoplasmosis is a parasitic infection caused by the protozoan Toxoplasma gondii. The organism needs a host cell for its proliferation. As it can pass the placenta, the parasite can also infect the developing embryo. The most frequently reported consequence is chronic lymphadenitis. The diagnosis is set up by serological testing.

**AIDS**

The first symptoms may present in the oral cavity and/or the maxillofacial region, therefore, it can be the dentist who first detects them. Fungal (candidiasis, geotrichosis, histoplasmosis), bacterial (necrotising gingivitis, progressive periodontitis) and viral (especially herpes and papillomavirus) opportunistic infections occur. Beyond recurring abscesses, swelling of the salivary glands and dry mouth (xerostomia), malignancies such as Kaposi’s sarcoma, Hodgkin’s lymphoma and squamous cell carcinoma have all been observed in this patient population.

5. **Cysts of the head and neck region (Csaba Berkovics DMD)**

Cysts are tumor-like structures of developmental or inflammatory origin. As this kind of growth does not originate in cell proliferation, it is not considered a tumor. Structurally, a cyst is a mostly liquid-filled pouch with endothelial lining and connective tissue wall. They are found both in bones and soft tissues.

Bone cysts can be odontogenic cysts, non-odontogenic cysts and pseudocysts (bone cavities without endothelial lining).

**ODONTOGENIC CYSTS**

Their cavity is mostly filled by hay-yellow, thin, serous liquid, and it is not rare that this liquid contains cholesterol crystals visible to the naked eye. The liquid exerts hydrostatic pressure on the surrounding bone, by which the bone gradually grows thin. Odontogenic cysts are made up of stratified squamous epithelium, but the differentiation between the various types is possible only by histology. The cyst is constantly growing, and it can push the roots of neighboring teeth apart, which makes their crowns tilt toward each other. Such a finding, even at the routine examination, must always raise the possibility of a cyst.

Cysts are usually symptom-free, and they are discovered as a by-finding in an X-ray done for some other reason. Exceptions are when the crowns are visibly dislocated, the cyst is superinfected, or when the bone protrudes because of the cyst.

**Odontogenic, non-inflammatory cysts**

Odontogenic, non-inflammatory cysts include perinatal cysts, gingival cysts of adults, primordial cysts, eruption cysts, lateral periodontal cysts and follicular cysts.

**Follicular cysts**

Of these, follicular cysts are the most frequently encountered. According to the most widely accepted explanation, this cyst is the product of the enamel epithelium remaining after the crown of an unerupted tooth has developed. The typical predilection site is around the crown of the unerupted tooth, in the majority of cases a lower wisdom tooth or a canine.
In X-ray images, a round shadow with marked edge is seen around the crown of the impacted tooth. (If the width of the osteolysis is less than 2mm around the crown, it is possibly a normal folliculus.) These cysts are unilobular, but rarely can they be multilobular.

Its treatment consists of the surgical removal of the impacted tooth together with the cyst. Re-occurrence raises the possibility of a keratocyst.

**Keratocysts**

A keratocyst may not be diagnosed without histology. Random histological examination of jaw cysts found 5–10% to be of this kind. These are formed from the remnants of the dental laminae or the epithelium of the enamel organ. In 70% of the cases, they are found in the mandible, especially in the angulus and the ramus. Typically males of 20–40 years are affected. Unlike most of the jaw cysts, they lead to the development of symptoms: patients see their doctor because of a swelling or an intraoral fistula. If multiple keratocysts are found, you should suspect (and exclude) basal cell naevus syndrome. The syndrome is characterised by several keratocysts and basal cell carcinoma of the skin. Anomalies of the vertebrae and the ribs, and intracranial calcification often co-occur.

Root resorption is rare, and keratocysts usually mimic odontogenic cysts in X-ray images. A multilobular appearance should always raise its suspicion.

Re-occurrence is about 55%, which is very high compared to other cyst types. Therefore, the follow-up of these patients is really important including regular orthopantomograms (OPT).

The wall of keratocysts is relatively thin, and small, fragile accompanying cysts may be present. The ultimate rule of treatment is radical excision, that is, portions of the healthy bone are also removed to minimise re-occurrence.

**Odontogenic cysts of inflammatory origin**

Radicular, residual and periodontal cysts belong to this group.

The lining of these cysts is made up of non-keratinised stratified squamous epithelium.

**Radicular cysts**

It is the most frequently seen odontogenic cyst. It is always periapical and of inflammatory origin, usually because of the necrosis of the pulp, but it is not clear why in some cases a radicular cyst, and in others a periapical granuloma is formed. It is assumed that after a given time, all granulomas would turn into cysts, but endodontic treatment or extraction stops this process. The lining is derived from the epithelial islets of Malassez.

Clinically, it is a hard swelling around the apex of the affected tooth, which does not cause any symptoms most of the time. Symptoms occur if the cyst gets superinfected leading to the development of a subperiosteal, submucous or subcutaneous abscess.

A panoramic radiogram and periapical images are prerequisites for the diagnosis. Around the apex, a circumscribed area of radiolucency with a sharp edge is seen, and the periodontium is difficult to differentiate in this area.

The wall of the cyst is made up of collagen-rich connective tissue. Degeneration is extremely rare.

Treatment depends on whether the tooth is preserved or extracted. If the tooth is extracted, the cyst is removed at the same time. If preservation is possible, the cyst is removed, and apical resection is also performed.

**Residual cysts**

If the removal of the radicular cyst is not entirely successful, the remnants of the cyst may get resorbed spontaneously. If they persist (and they are not removed), they are called residual cysts. The X-ray image is similar to that of the radicular cyst without the tooth. Thorough and careful periapical curettage can prevent such complications.

**Periodontal cysts**
It differs from the radicular cyst only in its position. The typical site is the cervical third of the root, and the background condition is periodontal inflammation.

**NON-ODONTOGENIC CYSTS**

This group of cysts originates in epithelial residues that get trapped between the facial processes as they unite in the development of the face. They can be found both in bones and in soft tissues.

**Incisive canal cysts**

Theoretically they are independent of age but seen most often between 30–50 years. A frontal palatal swelling may call our attention to it, but most often it is discovered as an X-ray by-finding. The typical finding of it in an X-ray image is oval or heart-shaped, mostly symmetrical, sharp-edged radiolucency. Sometimes it pushes the roots of the incisors apart, making it difficult to be differentiated from a radicular cyst. An important diagnostic sign is the cold-sensitivity of the affected tooth. Removal is indicated only if it causes subjectively disturbing symptoms, as degeneration has never been reported.

**Medial palatinal cysts**

Practically the same as the previous one, in a more dorsal palatal situation, which makes it questionable if it is an independent pathological entity.

**Globulomaxillary cysts**

Situated between the roots of an upper lateral incisor and a canine, this cyst can dislocate the roots of the affected teeth, which is an obvious indication for surgical removal. In X-ray images, a circumscribed thinning is seen.

**Medial mandibular cysts**

Located in the midline of the mandible.

**Nasolabial cysts (Klestadt’s cysts and also nasoalveolar cysts)**

They are soft tissue cysts and are rare. The peak of its prevalence is between 30–40 years of age. They present as a soft swelling in the superior alveolobuccal groove, or on the floor of the nasal cavity near its opening. The nostril on the affected side may be slightly displaced upwards. Bone alterations can be seen in the X-ray image only in case of a large cyst. Therapy consists of surgical removal.

**Lateral neck cysts**

All ages are affected, but they are most frequent in 21–30 years of age. They are painless, fluctuating structures usually related to upper respiratory tract infections. They may be situated anywhere along the line of the sternocleidomastoid muscle between the mandibular angle and the clavicle. Therapy consists of surgical removal.

**Lateral neck fistulas**

Etiologically the same as the previous one, but here a fistula is formed. The fistula must be resected.

**Medial neck cysts**

Most frequently they develop in the first ten years of life. The cyst is situated in the midline of the neck, near the hyoid bone. It is a soft swelling of 1–2 cm diameter, sometimes with a fistula.

**Dermoid and epidermoid cysts**

These are found in the skin but also in the floor of the mouth. They usually develop from embryonic epithelial tissue, and they have a stratified squamous epithelial lining.

The difference between dermoid and epidermoid cysts is the lack of the accessory structures of the skin in the latter one (e.g., sebaceous glands, hair follicles, sweat glands).
Therapy consists of eradication of the cyst.

**Heterotopic gastrointestinal cysts**

In rare cases, heterotopic gastric epithelium is found in the oral cavity, which can give rise to cysts.

**PSEUDOCYSTS**

They are not real cysts as they have no epithelial lining. The most prevalent form is the simple bone cyst occurring characteristically in young patients and usually in the mandible. In X-ray images, the edge is not as sharp as that of odontogenic cysts, sometimes blurred and lacerated. On the bony wall, there is little fibrotic tissue and no epithelium. As a therapeutic intervention, excochleation is performed.

### 6. Lesions of the oral mucosa (Péter Novák MD)

#### 6.1. Developmental and genetic anomalies

**Congenital lip pits**

*Aetiology:* a congenital genetic anomaly which is occasionally inherited dominantly.

*Symptoms and signs:* occur rarely, and they mostly develop on the lower lip (vermilion border), predominantly bilaterally resulting in cosmetic disadvantages accompanied by increased salivation causing inconveniences.

It may also develop accompanying a cleft lip or palate.

**Fox-Fordyce’s granules**

Heterotopic sebaceous glands located within the oral mucosa.

*Symptoms and signs:* small, yellowish spots which create a yellowish plaque if they are located close to each other. Predominantly, this disorder presents itself on the buccal mucosa, but it may also occur on the mucosal surface of the lips or other mucous membrane surfaces.

*Differential diagnosis:* leukoplakia, candidosis, morsicatio buccarum.

**Upper labial frenulum hypertrophy**

The enlarged upper labial frenulum causes a diastema between the incisors as it protrudes stoutly down to the palatal papilla.

**Ankyloglossia**

“Tongue tie” is present when the lingual frenulum pulls the tip of the tongue partially to the floor of the mouth, but in some cases the whole tongue may be completely fixed to the floor of the mouth. Its causes may be congenital or traumatic, and this disease may also occur in systemic sclerosis. It may be accompanied by disorders of speech or difficulty eating.

**Congenital leukokeratosis (White sponge nevus)**

It is a rare disorder which is inherited in an autosomal dominant trait.

*Symptoms and signs:* present as white, whitish-greyish, peeling. It is a painless and complication-free disorder on the buccal mucosa and the mucosal layers of the nose, oesophagus and the ano-genital region.

These lesions appear first at the time of birth or during childhood. This disorder occurs mostly on the loose mucous membrane of the mouth (buccal mucosa, soft palate, the ventral surface of the tongue, the mucosal surface of the lips, floor of the mouth, etc.). The gingival and the dorsal surfaces of the tongue are usually unaffected.

*Differential diagnosis:* leukoplakia, morsicatio buccarum, lichen oris, fungal infections.
Congenital leukokeratosis (White sponge nevus)

**Lingual thyroid (Lingual goitre)**

Heterotopic thyroid tissue remains in the dorsal third of the tongue, near the foramen caecum. Lingual goitres are more common in women.

*Symptoms and signs:* Lesions various in size and mostly of exophytic form and nodular surface can be observed on the dorsal surface of the tongue. These ectopic, purple-reddish glands may become swollen during menstruation and pregnancy, or they may even bleed.

It may become a life-threatening condition as it may result in an airway or alimentary tract obstruction.

*Differential diagnosis:* lingual tonsils, haemangioma, lymphangioma, tongue cancer.

**Naevus flammeus (“port-wine stain”)**

*Symptoms and signs:* congenital purplish-blue, irregularly shaped stain, which is not or barely elevated from the skin, and it is formed of flat, dilated capillaries. They may be stains of a small diameter or large plaques covering larger parts of the body as well. They may occur anywhere on the skin but most commonly in the regions of the head and neck.

The naevus may be present at birth and may grow in size later on, or may develop into a cavernous haemangioma, but it might as well fade and eventually disappear.


**Down’s syndrome (mongolism)**

Down’s syndrome is a chromosomal disorder and not a hereditary disease. Mongolism is more common in the children of mothers delivering at an advanced age.
Symptoms and signs: short stature, brachycephaly and mental retardation are typical, as well as white spots around the iris in the eye and an expressed medial epicanthic fold.

Those suffering from mongolism are more susceptible to upper airway diseases, while mouth dryness and snoring are explained by the fact that these patients breathe through their mouth because of their nasal obstruction.

The underdeveloped upper jaw causes protruded lower jaw and malocclusions.

The tongue is frequently protruded and deeply fissured (scrotal tongue).

The lips are thicker, dry and crusted, and occasionally lip fissures may also accompany the symptoms.

The eruption of the teeth is delayed, and hypodontia, enamel hypoplasia and the morphological disturbances of teeth are also common signs.

Caries activity is low among these patients, but the progression rate of parodontal diseases is higher. Respiratory, gastrointestinal and dermal infections are characteristic accompanying symptoms of this disease.

6.2. Physical, chemical and iatrogenic oral lesions

PHYSICAL INJURIES

Mechanical injuries

The main cause of traumatic ulcers is usually a single physical damage (for example, fights, sports, epilepsy, bone, sharp, broken tooth and prosthesis, etc.), which causes yellowish, painful, soft changes various in diameter on the affected mucosal tissue. These ulcers are surrounded by a thin, erythematous halo. They heal spontaneously in 6–10 days but leave scarring behind.

Differential diagnosis: specific ulcers (tuberculosis, lues), aphtha or carcinoma.

Epulis fissuratum (denture granuloma)

There are two major factors in the development of a denture granuloma: the pathologically mobile denture and the pressure of its edge on the surrounding mucosal tissue. An epulis fissuratum may most commonly occur in
the middle third of the mandibular bones and is more common on the buccal surface than on the lingual surface. The epidermal layer of the mucosa may develop increased keratinisation. These lesions may become pale, hard and more flexible to the touch where the accumulation of connective tissue fibres is more extensive.

*Differential diagnosis:* gingival cancer, Hydantoin hyperplasia, epulis.

![Epulis fissuratum (on the left premolar region, with denture)](image-url)
Epulis fissuratum (on the left premolar region)

*Morsicatio buccarum et labiorum*

The cause is that nervous, anxious children and young adults may repeatedly chew or bite their facial mucosa, lips or sometimes even their tongue as a bad habit. This constant mechanical trauma causes white desquamation of the effected epithelium, with erosions or even ulcers to develop mainly in the line of dental occlusion and buccal mucosa. This bad habit is commonly accompanied by bruxism.

*Differential diagnosis*: leukoplakia, candidiasis, ulcers caused by inflammations and cancer.

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**THERMAL INJURIES**

*Combustion (burning, hot surfaces)*

Any combustion injury is influenced by the degree and the duration of the thermal contact. Burn injuries may be caused by hot meals, cigarettes, tobacco pipes, hot dental surgery instruments or imprinting materials. Damage to the epidermis, oedema, erythema, peeling and pain (first degree burn) may develop, or in more severe cases blisters may appear which leave crusting when they burst, but no scars when they are healed. A longer lasting burn may heal leaving scars behind (third degree burn).

*Differential diagnosis*: chemical injuries, hypersensitivity.

**CHEMICAL INJURIES**

Chemical poisoning occurs (sodium hydroxid, trichloroacetic acid, Aspirin, carbolic acid, silver nitrate, sodium bicarbonate, hydrogen peroxide, etc.) when these materials get onto the mucosa in higher concentrations during mucosal or dental treatment accidentally or for therapeutic purposes. Erythematous, yellowish-white fibrinous pseudomembrane covered lesions and painful ulcers can occur in the mouth.

*Differential diagnosis*: sore, allergic stomatitis, white lesions.

**IONISING RADIATION**

*Radiogenic mucositis*
Radiotherapy applied as part of a treatment for malignancies may cause early and late onset action depending on the dose and duration on the oral mucosa as well as on the skin.

Among early onset reactions, one must mention erythemas, oedemas, hyperpigmentation appearing on the skin in the first two or three weeks after irradiation, whereas the oral mucosa may develop erythemas, oedemas, ulcers and painful lesions covered by yellowish-whitish pseudomembranes beside a dry mouth. All these may cause problems with eating and swallowing. The patient may lose his sense of taste for a longer period (2–6 months) after these ulcers heal (2–3 weeks). The ulcers heal slowly, leaving a sensitive, atrophic oral mucosa and scarring behind as late manifestations. Xerostomia developed as a result of the irradiation damage of the salivary glands (sensitivity to radiation) may predispose the patient to bacterial (pyostomatitis), fungal (candidiasis) or viral (herpes) infections. Malignancies may evolve on the atrophic, scarred mucocutaneous surfaces.

**Differential diagnosis:** exfoliative leukoedema of the oral mucosa, herpetic and aphthous gingival stomatitis or allergic stomatitis.

**ORAL REACTIONS DUE TO DRUGS**

**Cytostatic drugs**

Erythematous and ulcerative alterations covered by fibrous pseudomembranes (lichenoid reaction) may develop on the oral mucosa following chemotherapy (cytotoxic effects) of patients suffering from cancer. Erosions and ulcer are usually diffuse and multiple. They may occur anywhere but must commonly on the lips, buccal mucosa and the tongue. Symptoms of mucositis are the most severe in the period of 10–15 days after chemotherapy and may persist for weeks. These lesions may be a result of toxic pharmaceutical effects, but mostly they are the results of secondary neutropenia or thrombocytopenia (bleeding).

**Differential diagnosis:** allergic stomatitis, pseudomembranous candidiasis.

**Lichenoid reaction**

This lesion may be caused by several drugs (anti-diabetic drugs, anti-hypertensive drugs, beta blockers, penicillamine, allopurinol, non-steroidal anti-inflammatory drugs and anti-malaria medication), chemotherapy, some metallic fillings non-metallic materials used in dental treatment (amalgam) as well as by graft-versus-host reactions.

These reactions resemble true lichen, vulgar pemphigus, pemphigoid and lupus erythematos oral symptoms very much. The oral mucosa develops erythemas, and multiple erosions, burning, painful ulcers covered with fibrous pseudomembranes may evolve. Metallic and newly introduced composite dental materials and prosthesis structures may cause lichenoid lesions on the oral mucosa. After the cessation of the use of these drugs, the lesions may heal, but exchanging the fillings involved may not always yield proper results.

**Differential diagnosis:** contact allergy, chemical and drug-induced injuries.

**FOREIGN BODY INJURIES (METAL TATTOOS)**

**Amalgam**

The most common among these alterations are amalgam tattoos, which are created when corrosion, diffusion or a trauma causes metallic particles to diffuse into the mucosa. It is more common on the mandibular gingival surface than on the maxilla or around the alveoli.

**Differential diagnosis:** mucocele, haemangioma, malignant melanoma.

### 6.3. Infectious diseases

**BACTERIAL DISEASES**

**Folliculitis**

**Pathogen:** Staphylococcus aureus
Superficial pyoderma of the hair follicles is frequent on the skin.

**Symptoms:** the yellowish pustule is pierced through by a hair. Superficial folliculitis is not surrounded by an inflammatory ring, while deeper ones have a red, hyperaemic, painful base. It is frequent on markedly hairy skin that is prone to irritation. It heals without leaving a mark or a scar.

**Differential diagnosis:** Furunculus, acne.

**Furunculus**

*Pathogen:* Staphylococcus aureus

Frequently painful, deep pyoderma developing in hair follicles. Clinically, the yellowish pustule is pierced through by a hair.

**Symptoms:** face, upper lip, nape, perianal region. Squeezing the upper lip is strictly prohibited. After healing a mark or a scar is usually left behind.

**Differential diagnosis:** Furuncle, phlegmon, actinomycosis.

**Acute necrotising ulcerative gingivostomatitis**

*See in Chapter 2.15.4. Acute periodontal diseases*

**Tuberculosis (TB)**

Chronic infectious disease

*Pathogen:* Mycobacterium tuberculosis

Primary tuberculosis is rare in the oral cavity.

**Symptoms:** it is most frequent on the tongue, palate, cheeks or on the lips. Apparently it is an excavated ulcer with irregular, undermined edges, covered with a greyish-yellow fibrinous pseudomembrane. The erythematous, solid ulcers with prominent edges are usually painless.

The final diagnosis is set up on the basis of sputum culture, chest X-ray, PCR and histological examination. As a consequence of lymphogenic spread, regional lymphadenomegaly is an accompanying symptom.

**Differential diagnosis:** Lupus erythematous, syphilitic ulcer, tumorous ulcer, aphthous ulcer (Sutton).

**Primary syphilis (lues)**

Sexually transmitted disease.

*Pathogen:* Treponema pallidum

Bacterial invasion takes place in the genital area but sometimes on the lips or the oral mucosa as well, as a consequence of oro-genital intercourse.

**Symptoms:** after 3-5 weeks of incubation, a primary lesion (primary chancre) develops on the lower lip, tongue, palate, tonsil or other parts of the oral mucosa. At the site of the invasion, first dark red erythema (1-2 cm in diameter), then a brownish-red papule develops. The primary painless, solid, dark red ulcer (ulcus durum) is usually solitary, but might be multiple, too. The evolved ulcus durum heals spontaneously with scar formation after 4-6 weeks. Painless regional rubbery lymphadenomegaly (indolent bubo) is present.

The diagnosis is based on dark field and fluorescent microscopic examinations (smear). Serological examinations are needed.

**Differential diagnosis:** Herpes, aphtha, tuberculosis, carcinoma.

**Secondary syphilis**
Between 6 weeks and 6 months after the infection, the second phase of syphilis develops.

The first and the second phases are the early stages of syphilis. As a consequence of bacterial dissemination, secondary syphilis is characterised by generalised dermal and mucosal signs and generalised lymphadenomegaly.

**Symptoms:** multiple, slightly prominent, initially dark red roseolas on the oral mucosa are typical. Later they transform into papules and then into opalescent, greyish white mucous patches with a red base. The most common sites are the tongue, palate, angle of the lips.

Serological examinations are positive.

There are many bacteria in the dermal and oral lesions; as a consequence, the lesions are highly contagious.

**Differential diagnosis:** Candidosis, allergic stomatitis, stomatitis aphthosa-herpetica, oral pemphigus, lichen oris.

**Tertiary syphilis**

Late syphilis evolves 3-10 years after the initial infection.

**Symptoms:** painless solid gumma infiltrating the dermal or deeper tissues is characteristic. This is a special granuloma that can appear on the skin, mucus membrane, bones, blood vessels or in the nervous system, too. The size of the gumma varies from some millimetres to some centimetres and they can be solitary or multiple. The typical saddle nose develops due to the destruction of nasal cartilage and bones. The filiform and fungiform papillae become atrophic and the tongue becomes “smooth”.

Gummas rarely contain Treponema bacteria, thus, they are less dangerous from the point of view of infection. They finally heal with scarring.

**Differential diagnosis:** pernicious anaemia, carcinoma.

**PROTOZOAN DISEASES**

**Toxoplasmosis**

An infectious disease caused by the protozoan Toxoplasma gondii.

Toxoplasma can mainly be found in cats, rodents and birds, and it gets into the human body with food contaminated with the faeces of these animals. Human Toxoplasma infection is common; however, it rarely causes clinical symptoms and actual disease. The disease mainly affects children and young adults.

**Symptoms:** the onset may be slow with hardly any general symptoms; only a painless swelling of the lymph glands draws the attention to the disease. In acute cases; however, solitary or generalised lymphadenopathy is preceded by fever, headache, sore throat and muscle weakness. On rare occasions, blisters, papules and ulcers may present on the oral mucosa. The disease may be self-limiting with a slow course, but it can lead to severe complications as well (uveitis, myocarditis, and encephalitis). Toxoplasmosis in pregnant women may cause foetal damage, including chorioretinitis and consequential blindness.

**Differential diagnosis:** Infectious mononucleosis, leukaemia, Hodgkin’s diseases.

**VIRAL DISEASES**

**Recurrent herpes simplex infection (herpes labialis, cold sore)**

**Pathogen:** Herpes simplex virus

After the primary infection, the virus becomes latent in the epithelium and in the ganglia of the trigeminal nerve.

**Symptoms:** it usually affects the lips or the boundary between the lips and the skin. Predisposing factors include exposure to sun, fever (fever blisters), upper respiratory tract infections, pneumonia, malaria, meningitis, physical or emotional stress, menstruation, dental treatment, stress, gastroenteritis, AIDS, pregnancy, trauma, local irritation, etc.. Before the development of blisters, fever and enlargement of lymph glands may develop. At
the onset of the disease an itchy, burning sensation (prodrome) and redness are present. A few hours later clusters of vesicles are formed, which are 1-3 millimetres in diameter. Vesicles become coalesced, and after two or three days they rupture and become ulcerated with yellowish crusts. The surrounding skin is erythematous. Skin lesions usually heal in 8-10 days without scarring. Sites and frequency of recurrences vary from patient to patient.

_Differential diagnosis:_ Herpes zoster, pemphigus.

_Herpes labialis_

**Primary herpetic gingivostomatitis**

*See in Chapter 2.15.4. Acute periodontal diseases*

**Herpes zoster (Shingles)**

_Pathogen:_ Herpes virus varicellae.

Shingles typically affects adults and the elderly. Children rarely develop shingles.

In case of endogenous or exogenous (salivary droplet) re-infection, the latent virus is re-activated due to various causes, such as immunosuppression, carcinoma, trauma, corticosteroids, cytostatic drugs, Hodgkin’s disease and leukaemia.

_**Symptoms:**_ herpes zoster frequently affects the orofacial area, along the first (eye) or the second or third (oral mucous membrane and face) branches of the trigeminal nerve. Fever, headache and enlarged regional lymph glands are common. Pain can be mild, but mostly it is excruciating (neuralgic). The affected mucous membrane becomes erythematous. Typical signs and symptoms include unilateral, very painful, vesicular skin and oral mucosal lesions. The ulcerations heal in 5-10 days without scarring.

_Differential diagnosis:_ Herpetic gingivostomatitis, aphthous stomatitis, allergic stomatitis.

**Infectious mononucleosis (Pfeiffer's disease)**

_Pathogen:_ Epstein–Barr virus.
This acute infectious disease is most common in adolescents and young adults.

The infection is typically transmitted through direct contact with saliva – especially kissing can spread the virus (it is also called “kissing disease”) – and through salivary droplet infection.

**Symptoms**: headache, fatigue, fever, pseudomembranous angina, primarily cervical, postauricular and generalised painful lymphadenopathy and hepatosplenomegaly. Maculopapular skin rash similar to the spots in measles, rubella and scarlet fever may occur. Intraorally, erythema, purpura and petechiae (diagnostic) develop on the uvula and on the soft palate. Uvular oedema, gingival bleeding, ulceration and painful aphthous ulcers can also occur. Pharyngitis and tonsillitis also contribute to the clinical picture.

**Complications** may include hepatitis, myocarditis, haemolytic anaemia, splenic rupture, encephalitis, neuritis and sialoadenitis.

**Differential diagnosis**: Aphthous and herpetic stomatitis, secondary syphilis, AIDS, leukaemia.

**Morbilli (measles or rubeola)**

**Pathogen**: morbilli virus (paramyxovirus).

It is transmitted through salivary droplet infection.

**Symptoms**: highly contagious infection that causes a range of symptoms including fever, chills, malaise, cough, conjunctivitis and rhinitis after a two-week incubation period. Then, maculopapular, grey-reddish exanthemas appear on the face, neck (behind the ears), trunk and extremities. Irregular, small (1-3 millimetres in diameter) lesions develop on the erythematous oral (buccal), genital and conjunctival mucosa. Oral lesions usually precede skin lesions. Bilateral cervical lymphadenitis is also characteristic.

**Complications** may include otitis media, bronchial pneumonia, and in severe cases subacute sclerosing panencephalitis. The most effective form of prevention is vaccination.

**Differential diagnosis**: Infectious mononucleosis, herpetic and aphthous stomatitis, white lesions.

**Verruca vulgaris (common wart)**

**Pathogen**: human papillomavirus (HPV) – a member of the papovavirus family.

The wart occurs less frequently in the oral cavity than on the skin, but more commonly than previously believed.

**Symptoms**: they usually appear on the lips, tongue and palate, but any other location in the oral cavity may occur. They appear abruptly and grow rapidly (viral origin), they are broad based and have a whitish-grey, papillary surface (usually feel hard on the skin).

Verrucae may appear as multiple and confluent lesions.

**Differential diagnosis**: Papilloma, verrucous leukoplakia.
Rubella (German measles)

It is a moderately infectious viral (RNA virus) disease usually transmitted by salivary droplets and affects primarily children and young adults.

Symptoms: may begin with milder symptoms (fever, headache, common cold and general malaise). Swollen, tender lymph nodes, usually in the back of the neck (cervical), behind the ears (retroauricular) or in the suboccipital area are characteristic. A maculopapular skin rash appears on the face and spreads downward to the trunk. Erythematous, red, petechial spots occur on the palates and the pharynx, especially on the soft palate.

In the first 16 weeks of pregnancy, rubella infection may trigger congenital rubella in the developing foetus (resulting in heart, eye, ear and brain damage).

Differential diagnosis: Morbilli, infectious mononucleosis, scarlet fever.

AIDS (Acquired Immune Deficiency Syndrome)

HIV (Human Immunodeficiency Virus) retrovirus is transmitted through infected bodily fluids.

High-risk groups include homo- and bisexual men, heterosexual women and men, (intravenous) drug users and people with haemophilia.

Symptoms: the disease does not have any characteristic symptoms. Initial symptoms include swollen lymph nodes (generalised lymphadenopathy), weight loss, fatigue, diarrhoea and night sweats. All types of infections may occur in the oral cavity, including fungal (acute pseudomembranous candidiasis, acute atrophic or chronic hyperplastic candidiasis), bacterial (periodontitis, necrotising ulcerative gingivitis), viral (herpetic stomatitis, herpes zoster, verruca vulgaris and condyloma acuminatum) and protozoan (toxoplasmosis) infections. Furthermore, non-Hodgkin’s lymphoma and carcinoma may also develop. Two of the pathognomic oral symptoms of AIDS are diagnostic: hairy leukoplakia and Kaposi’s sarcoma.

Differential diagnosis: Hairy leukoplakia has to be differentiated from real leukoplakia, lichen planus, cheek chewing, while Kaposi’s sarcoma from malignant melanoma and hemangioma.

Varicella (Chickenpox)
Chickenpox is an acute, highly contagious, generally mild disease in children, which occurs mainly in autumn and winter.

*Pathogen:* herpesvirus varicellae.

Chickenpox is a salivary droplet infection.

*Symptoms:* the disease begins with nasopharyngitis, headache, fever and pain in the extremities, followed by itchy maculopapular exanthemas and vesicles on the trunk, extremities, face and scalp. The disease usually heals in 7-10 days without a skin mark. The ruptured vesicles on the skin form scabs and scale off, but in case of secondary bacterial superinfection pustules, and after healing small, round scarring may develop. The lesions mainly affect the hard and soft palates and the lips, but the tongue, the gingival and other mucosal areas are also at risk. Complications may include pneumonia, bilateral parotitis, TB activation and encephalitis.

*Differential diagnosis:* Herpetic and aphthous gingivostomatitis, infectious mononucleosis, herpangina.

**Epidemic parotitis (Mumps)**

*Pathogen:* mumps virus – a member of the paramyxovirus family.

The acute infection spreads through salivary droplets.

Mumps mainly affects children, it rarely occurs in adults.

*Symptoms:* the parotid gland gets painfully swollen. Trismus sometimes develops. Swelling might be unilateral, but in most cases (70%) it affects both sides. Depending on the severity of subsequent xerostomia, fungal, viral or bacterial infection may appear in the oral cavity. The orifice of the parotid duct may swell and become red (papillitis), but no pus can be squeezed from the parotid gland. Complications may include meningitis, nerve palsy, pancreatitis, orchitis in men and oophoritis in women.

*Differential diagnosis:* Acute bacterial parotitis, sialolithiasis, parotid tumor.

**FUNGAL DISEASES**

**Acute pseudomembranous candidosis (thrush)**

The commonest Candida albicans-caused oral disease.

*Predisposing factors:* inappropriate oral hygiene, heavy smoking, xerostomia, radiotherapy, the administration of antibiotics, corticosteroids or cytostatic drugs, diabetes mellitus, chronic systemic diseases, autoimmune diseases, leukaemia, AIDS, immunosuppression. It mostly presents in newborns and children suffering from chronic illnesses.

*Symptoms:* the appearance of cream-coloured or pearl-white, bluish-white plaques which leave a slightly burning, erythematosus surface if wiped off is characteristic of this type of candidiasis. Predilection sites include the bucca, the palate and the tongue. It is common in newborns and infants. In case of acute pseudomembranous candidiasis in adults, an underlying systemic disease has to be considered.

*Differential diagnosis:* Lichen, leukoplakia (cannot be wiped off), morsicatio buccarum, allergic stomatitis.
Acute pseudomembranous candidosis (thrush)

Acute atrophic candidosis

Untreated pseudomembranous candidiasis can turn into the atrophic form, but the latter can develop independently as well, without a previous pseudomembranous-type candidiasis.

Acute atrophic candidiasis is the commonest type of candidiasis in the oral cavity.

_Predisposing factors:_ overuse of antiseptics or wide-spectrum antibiotics (topical or systemic), cytostatic and immunosuppressive drugs and HIV infection.

_Predilection sites:_ the dorsum of the tongue, but it can present anywhere in the oral cavity.

_Symptoms:_ the mucous membrane becomes oedematous, smooth (atrophy), erythematous, painful, and a burning sensation develops. In comparison with other clinical types, only acute atrophic candidiasis causes pain.

_Differential diagnosis:_ Pernicious anaemia, iron-deficiency anaemia, Sjögren’s syndrome, chronic atrophic candidosis, migratory glossitis.

Chronic atrophic candidosis (denture stomatitis)

It is a common chronic disease of the mucous membrane especially in patients wearing upper dentures.

Tight fitting dentures provide favourable circumstances: they practically function as a substrate for the growth of _Candida_.

_Symptoms:_ well-circumscribed, red, oedematous, sometimes eroded mucous membrane corresponding with the plate of the upper denture is characteristic of the disease. Chronic atrophic candidiasis is symptom-free.

_Differential diagnosis:_ contact allergy of the palate.
6.4. Immune-based diseases

Seborrheic eczema

The same allergen can cause allergic dermatitis in acute cases, and eczema in chronic cases.

Eczema might run in the family. Seborrheic eczema is usually based on sensibilisation due to microbes (bacteria, fungi) and chemicals. It is a frequent dermal disease.

Predilection sites: the forehead, the edge of the scalp, the perioral, perianal, periauricular and presternal regions, but it might also be present on the lips.

Differential diagnosis: herpes simplex.

Urticaria

Urtica (nettle rash) is the oedema of the upper part of the dermis as a consequence of increased vascular permeability. Drugs (penicillin, non-steroidal anti-inflammatory drugs, sulphonamides, salicylates, and opiates) usually play a role in the development of acute allergic urticaria.

Symptoms: the skin is highly itching when pink or white nettle rashes or nodules with a sharp edge appear on its surface. Diameter from some millimetres to some centimetres.

Differential diagnosis: lip furuncle.

Quincke’s oedema (angioneurotic oedema)

The disease has two forms, hereditary (rare) and non-hereditary (acquired).

The latter one is an early type of a hypersensitivity reaction caused by a drug, food, endocrine disorder, focal infection, insect bite or emotional stress, but in most cases the origin remains unknown.

Urticaria and its “giant” form (urticaria gigantea), Quincke’s oedema can develop.

Symptoms: swelling of the face, eyelids, lips, tongue, soft palate and pharynx. The swelling has a normal or slightly pink colour and might suddenly, unexpectedly increase in size. Sometimes it may cause burning, or
slightly itching sensation. In severe cases the oedema of the epiglottis or that of the larynx may lead to dyspnoea (suffocation, death). Angioneurotic oedema might also turn into an anaphylactic shock.

**Differential diagnosis:** granulomatous cheilitis, glandular cheilitis, lymphoedema.

**Allergic contact stomatitis**

**Metal**

The contact allergy of the oral mucous membrane is less frequent than that of the skin, because allergens can act only for a shorter time due to the cleaning effect of the saliva. Not physical, but immune reactions account for contact allergic reactions. The metal bridge of the partial prothesis may induce contact allergy on the hard palate resulting in bright red oedematous enanthemas, sometimes erosions. Foreign body sensation, impaired gustatory sense and burning, itching sensation are among the subjective complaints. Nickel, cobalt, chromium, copper and gold tend to trigger contact allergy. Among these, nickel is the most frequent sensitising agent.

**Differential diagnosis:** candidosis (atrophiac).

**Acrylate denture**

True allergy to acrylate denture is rare. The mucous membrane turns red and oedematous according to the denture, besides the patient complains of a burning sensation.

**Differential diagnosis:** atrophic candidosis.

**Drug-induced allergy (stomatitis medicamentosa)**

After drug administration (hypnotics, analgesics, antibiotics, etc.), a bulla is developed, and later an erosive allergic enanthema accompanied by diffuse erythema and subepithelial burning sensation. The lips, the tongue, the palate and the gingiva are the predilection sites of allergic reactions.

**Differential diagnosis:** acute pseudomembranous candidosis, chemical injuries, white lesions, pemphigus.

**Recurrent oral ulcers**

**Recurrent aphthous ulcer (Mikulicz’s aphtha; minor aphtha)**

The aetiology is not yet explained. It might be an autoimmune, viral, bacterial (Streptococci), genetic (familial) or allergic disease or could be influenced by mechanical injuries, gastrointestinal diseases (Crohn’s disease), psychic stress, menstrual cycle (hormonal factor), walnut or chocolate consumption, etc., or by hypovitaminosis (B12, folic acid).

It is the most frequent disease of the oral mucous membrane. Recurrent aphthae are more frequent in adulthood and in women. It is rare among smokers (mucosal hyperkeratinisation).

**Symptoms:** initially erythema and oedema develops on the corresponding area, accompanied by a nipping, burning sensation or by paraesthesia. It can be either solitary or multiple. The development of ulcers is not preceded by vesicle formation. Well-demarcated lenticular (3–10 mm) painful ulcers on erythematous bases, covered with yellowish fibrous pseudomembranes are present. After 1 or 3 weeks, they heal without leaving scars. They might persist for years, but their frequency varies from person to person. They might be accompanied by lymphadenomegaly, too. Predisposed areas are the vestibular surface of the lips, the floor of the mouth, the ventral surface of the tongue and the cheek (non-keratinized mucosal epithelium). It is rare on the hard palate, the gingiva and the back of the tongue; these areas are usually affected by gingivostomatitis herpetica.

**Differential diagnosis:** herpetic stomatitis, allergic stomatitis.
Recurrent aphthous ulcer

Major aphtha (Sutton’s aphtha)

Recently major aphthae have been considered to be the rare severe form of aphthous ulcer.

Symptoms: 1–4 cm in diameter, usually solitary, but might have multiple forms, too (giant aphtha). Very painful crater-like ulcers of variable depth, covered with yellowish-grey fibrous pseudomembranes. The lesion usually heals with deforming mucosal scarring after epithelisation. Women are usually more affected. Predilection areas are the oropharyngeal region, the soft palate, the cheeks and the tongue. It causes severe pain, fever, trismus, lymphadenitis and difficulties in swallowing or eating. It may persist for several months or even for one or two years and tends to recur. Biopsy is mandatory.

Differential diagnosis: tumorous ulcer, decubital ulcer, specific ulcer: TB, lues.
Systemic autoimmune dermal and oral diseases

Lupus erythematosus

The aetiology has not been fully revealed, but it is mostly regarded as an autoimmune disease. More women are affected, and untreated lupus erythematosus might lead to death after a shorter or longer time.

Two types:

- systemic lupus erythematosus (SLE) includes both mucocutaneous and internal symptoms,
- discoid type (DLE) is a local dermal and oral disease.

Symptoms: cutaneous symptoms: symmetrical butterfly-shaped red plaques on the face, maculopapular exanthes, plaques, petechiae, vesicles, pigmentation on the thorax, arms, hands and nail folds and alopecia. Fever, leucopenia, thrombocytopenia, anaemia and other internal signs (nephritis, myocarditis, polyarthritis, lupoid hepatitis, epilepsy, etc.) might also accompany the disease. Sjögren’s syndrome or Raynaud’s syndrome might be secondary to lupus. The seriously ill patients with SLE die of renal, cardiac or pulmonary dysfunction.

The discoid form usually affects the lips and the cheeks, while the systemic form is more frequent on the hard palate and the cheeks. The slightly prominent, bright red, painful plaques appear on the erythematous mucous membrane; they might transform into erosions or ulcers. There are whitish hyperkeratotic ray-like striae on the slightly prominent edges of the plaques. Lupoid oral signs are spot-like and usually bilateral, contrary to lichen oris, where the lesions are more diffuse and symmetrical. Lupoid foci heal with atrophic scar formation.

The disease is lethal in 15% of the cases.

Differential diagnosis: erosive lichen oris, erythroplakia, erosive leukoplakia, pemphigus vulgaris.

Sjögren’s syndrome

It is a chronic and systemic autoimmune disease, which causes the destruction of exocrine salivary glands. It is accompanied by xerostomia and keratoconjunctivitis sicca (primary form, or sicca syndrome).
The disease is of unknown origin, but the factors may include genetic susceptibility, environmental predisposing factors, pathologic immune reaction, viruses and sex hormones.

**Symptoms:** during its progression, the tissue of salivary glands is destroyed by the increasing lymphocyte infiltration. Raynaud’s phenomenon (whitened and cold fingers), lymphadenopathy, vascular purpura and complications affecting the kidneys or lungs may also coexist. Predominantly it affects middle-aged women. Increased dryness of the mouth is often accompanied by (unilateral or symmetrical) rarely painful swelling of the large salivary glands, especially the parotid ones. Xerostomia may cause circular caries. The tongue is pale or red, atrophic, and lobular. The red and painful, in patches eroded or ulcerated tongue is often infected by Candida. Dryness of the eyes may bring about conjunctivitis.

The diagnosis is helped by the examination of saliva secretion, sialography, scintigraphy, histopathology (lip and bucca biopsy), ophthalmologic (Schirmer test), haematological and immunologic examinations as well as sonography, CT and MRI.

**Differential diagnosis:** pernicious anaemia, iron-deficiency anaemia, atrophic candidosis, diabetes mellitus, irradiation, mumps, inflammatory diseases and tumours of the salivary glands.

### 6.5. Vesiculobullous, granulomatous skin and mouth diseases

**VESICULOBULLOUS SKIN AND MOUTH DISEASES**

**Pemphigus vulgaris**

Pemphigus vulgaris is a chronic, life-threatening autoimmune skin, mouth and other mucous membrane disorder characterised by blistering of the skin and oral mucosa. Sometimes pemphigus appears in reaction to a viral infection, certain drugs (D-penicillamine, antihypertensive Captopril), thymoma or myasthenia gravis.

**Symptoms:** it usually affects elderly women. Blistering starts intraepithelially, the surrounding mucosa is not erythematous. The thin-walled intraoral bullae easily burst leaving painful erosions covered with whitish-grey fibrinous diphtheroid coating. The erosions may also bleed. Painful erosions may cause difficulty swallowing and eating and increased salivation. Erosions heal without scarring. Sites of greatest involvement include the buccal mucosa, tongue, palates and the gingiva. Nikolsky’s sign is positive (when the surface of the unaffected skin and oral mucosa is rubbed, and the skin and mucosa separate easily).

The diagnosis is established on the basis of skin symptoms and the histopathological examination.

In order to adjust the drug therapy, hospitalisation is necessary.

**Differential diagnosis:** herpetic gingivostomatitis, erosive lichen oris, aphthous stomatitis.
Pemphigus vulgaris

GRANULOMATOUS SKIN AND MUCOSAL DISEASES

Hand-Schüller-Christian disease (Langerhans cell histiocytosis)

The disease is one of the histiocytosis X syndromes. It is a chronic disease that is manifested in three classical signs in 25% of the cases: osteolytic bone lesions (“cloudy skull”), diabetes insipidus and unilateral or bilateral exophthalmus.

The aetiology of the syndrome is not clear.

Symptoms: may begin to develop in childhood, but the severe complaints appear only in older ages and mainly in male patients. It may be accompanied by fever, lymphadenomegaly, otitis media, brown scaly papules on the skin (mainly on the head, trunk, and on the anogenital area) and other internal signs. Oral symptoms are frequent, and the most characteristic sign is the destruction of the alveolar process of the jaw resulting in loose teeth or in the loss of teeth. The wound of extraction heals very slowly. Painful, bleeding, necrotic ulcers covered with fibrous pseudomembrane evolve usually on the gingiva, on the alveolar mucus membrane or on the palate, or sometimes on other neighbouring mucus membranes. It may be accompanied by fetor ex ore.

The diagnosis is set up by X-ray imaging (skull, long bones, and jaw) and by histological examination.

Differential diagnosis: malignant primary and secondary carcinoma.

Foreign body granuloma

Any insoluble, non-absorbable foreign body (metal, wood, thread, talcum, bone, glass, etc.) that gets into the skin or into the oral mucous membrane (injury, operation) causes smaller or larger brownish-red nodules (giant cell granuloma).

Differential diagnosis: tumour, sarcoidosis.

6.6. Oral manifestations of systemic diseases

ENDOCRINE DISEASES

Hyperthyroidism

It is the hyperfunction of the thyroid gland.

Symptoms: the enlargement of the thyroid gland (goitre), a staring gaze, protruding eyes (exophthalmus), increased heart rate (tachycardia), weakness, sweating, loss of weight, nervousness, diarrhoea and shaking. It is more common among women than men. No specific oral symptoms are observed. It may present with shaking and burning sensation in the tongue accompanied by the early eruption of deciduous and permanent teeth. Swelling of the gingiva may also be a common symptom.

Differential diagnosis: thyroid tumours.

Hypothyroidism

It is the hypofunction of the thyroid gland.

Symptoms: it causes cretinism in infancy and myxoedema in childhood and adulthood. Congenital hypothyroidism causes severe mental and physical retardation, neonatal jaundice and a characteristic, hoarse crying of the infant, apart from these, wide, flat facial appearance and macroglossia (the tongue protrudes from the mouth), thick skin, delayed dental eruption and the dysplasia of the dental enamel. In adulthood, mucopolysaccharide infiltration causes macroglossia, macrocheilia and dry, reddish mucosa. The enlarged tongue makes speech and feeding more difficult. The face may become swollen as well. The appearance of the skin is determined by the myxoedema, the skin becomes dry and yellowish due to carotin deposits. Mental retardation, bradycardia, sensitivity to cold or hypothermia and the predisposition to ischemic cardiac disease are also typical. Hypothyroidism may be accompanied by chronic mucocutaneous candidiasis.
**Differential diagnosis:** amyloidosis, acromegaly.

**Diabetes mellitus**

Hyperglycaemia develops due to the decreased insulin production of the beta cells within the islets of Langerhans of the pancreas. Recently this disease has been considered a metabolic disorder.

**Symptoms:** apart from the dry skin being more susceptible to bacterial infections (furuncles, carbuncles), xerostomia (dry mouth), glossodynia and disorders of taste sensation are characteristic. Chronic candidiasis and angular cheilitis are also common in patients with diabetes. Swollen parotid glands (sialosis) and gingival (gingivitis, parodontitis) anomalies, as well as itching of the gingival may develop. Lichen oris and lichenoid reactions (caused by antidiabetic drugs) are also common.

**Differential diagnosis:** candidosis, Sjögren’s syndrome, disorders of the salivary glands.

**VITAMIN DEFICIENCIES, NUTRITIONAL AND METABOLIC DISORDERS**

**Riboflavin (vitamin B2) deficiency**

Causes: may be malnutrition, malabsorption, alcoholism, conditions leading to protein malnutrition. This deficiency is most common among children not consuming any milk.

**Symptoms:** the most common oral symptom is angular cheilitis. Seborrhoeic dermatitis may develop on the skin of the face, on the nasal apertures and within the nasolabial folds. In the beginning, the corners of the mouth are covered by yellowish, scaly crusts, and then they become red, deeply and painfully fissured (pebbly appearance) and they bleed easily. Apart from that, the tongue becomes atrophic, red in colour, painful and fissured as well.

**Differential diagnosis:** anaemia caused by iron deficiency, candidiasis (chronic, atrophic).

**Niacin, nicotinic acid (vitamin B3) deficiency (pellagra)**

Deficiency of the vitamin group B3 used to be caused by nutrition poor in proteins, but it is most commonly caused by gastroenteritis, anacidity, congenital metabolic disorders and chronic alcoholism nowadays.

**Symptoms:** may be loss of appetite, weakness, and irritability. In more evolved cases dermatitis, diarrhoea, or dementia may occur. The filiform papillae of the tongue become atrophic, the tongue becomes swollen, painful, resembles burning red “raw meat”. Thick, greyish coating may appear on the dorsal surface of the tongue. The gingiva may become swollen, ulcerated and erythemas may also appear on it. Increased salivation is also characteristic, and the inflammation may develop into ulcerative, necrotic stomatitis or gingivitis.

**Differential diagnosis:** pernicious anaemia, anaemia caused by iron deficiency, Sjögren’s syndrome.

**Folic acid deficiency**

The lack of folic acid damages protein synthesis and cell division. The primary cause of folic acid deficiency is imbalanced nutrition (vegetables and fruits); however, malabsorption (coeliac disease), alcoholism, anorexia, cancerous diseases, pregnancy, haemolytic anaemia, medications (contraceptives, phenytoin, anticonvulsive drugs) may all result in folic acid deficiency.

Lack of folic acid on its own is uncommon, as this deficiency is mostly accompanied by lack of vitamin B12 as well.

**Symptoms:** it commonly causes erythemas and painful, atrophic lesions (filiform and fungiform papillae disappear) on the tongue, as well as aphthous ulcers, similarly to the symptoms of vitamin B12 deficiency. Angular cheilitis may also be a characteristic symptom.

**Differential diagnosis:** anaemia, other vitamin B deficiencies.

**Vitamin C deficiency (scurvy)**

This deficiency is most commonly caused by insufficient nutrition (due to poverty, elderly age or chronic alcoholism).
Scurvy, the disease caused by vitamin C deficiency, is now quite uncommon.

**Symptoms:** the classical clinical symptoms of scurvy are hyperkeratotic hair follicles and perifollicular bleedings. Occasionally, ecchymoses may appear on the skin. The oral symptoms are most significant in case of patients with bad oral hygiene. The gingiva of these patients becomes extremely swollen, it may cover most of the dental crown and may very easily, even spontaneously bleed. Pain in the mandibular bones is quite common. Necrosis may develop on the site of gingival bleedings. An unpleasant oral odour and severe fusospirochetal stomatitis may also accompany this disease. Any inflammation caused by bacteria in vitamin C deficiency may cause rapid periodontal destruction and thus loss of teeth due to disturbances in connective tissue regeneration. Due to the haemorrhage of the temporomandibular joint, opening the oral cavity is painful.

**Differential diagnosis:** ulcerative and necrotising, or herpetic gingivostomatitis, leukaemia, agranulocytosis.

**LIVER DISEASES**

**Hepatitis C viral infection**

This type of viral hepatitis is mainly transmitted by transfusions, but needle pricks and dental treatments are less common causes of transmission than in the case of hepatitis B infections.

Since hepatitis C viral infections are in more than 60% of the cases accompanied by atrophic and erosive lichen lesions, today it is a recommended procedure to examine patients with erosive lichen for hepatitis C virus. According to some authors, other types of lichen alterations (reticular, bullous, plaque lichen, etc.) may refer to hepatitis C viral infections in the background as well. Chronic hepatitis C viral infections may eventually cause cirrhosis or even hepatic cancer.

**Differential diagnosis:** lupus erythematosus, lichenoid reaction, erythroleukoplakia.

**Hepatic cirrhosis**

**Symptoms:** typical symptoms are hepatomegaly, jaundice, later splenomegaly, ascites, fever and encephalopathy. Patients with hepatic cirrhosis may develop gastrointestinal bleeding, spider naevi, suggillations and hyperpigmentation on the skin (hepatic chloasma). Haemorrhagic tendency is caused by the metabolic disorders of vitamin K due to liver damage. Bleeding tendency demands careful, prudential surgical interventions. Typical oral symptoms are atrophic, smooth ("lacquered"), painful, red alterations of the tongue and angular cheilitis, which are due to the decreased protein intake, chronic gastritis (consequent vitamin B-12 and folic acid deficiency) and mainly vitamin B deficiency of alcoholic patients. The absorption of iron is also affected, which may also cause aphthous stomatitis. Patients suffering from hepatic cirrhosis often complain about halitosis (hepatic fetor). The atrophy of mucosal tissue may cause leukoplakia and carcinoma. Hepatic cirrhosis is often accompanied by sialadenosis (sialosis). The development of hepatic cancer is more common among patients suffering from hepatic cirrhosis.

**Differential diagnosis:** acute or chronic atrophic candidiasis, pernicious anaemia and anaemia caused by iron deficiency.

**DISORDERS OF THE CARDIOVASCULAR SYSTEM**

**Varicosity of the tongue**

It is a diffuse dilation of the veins occurring in an elderly age (varix). The dark blue varicosity on the ventral surface of the tongue or sometimes on the floor of the mouth is often called caviar lesion. The varicosity of the tongue is most common among patients suffering from cardiopulmonary diseases. Due to its tumour-like surface, it may cause cancerophobia.

**Differential diagnosis:** haemangioma, Kaposi’s sarcoma, malignant melanoma.
**Varicosity of the tongue**

**DISEASES OF THE AIRWAYS**

**Bronchial asthma**

The pathogenesis of this disease is still not clear, and allergic, non-allergic, physical, chemical and psychoneurotic factors are involved.

Bronchial asthma is characterised by cough and dyspnoea with a sudden onset during which the alterations of the bronchial and pulmonary capillaries and the thickening of the walls of the bronchioles cause deficiencies in the oxygen supply of the blood (cyanosis). Chronic asthma will cause elevated pulmonary blood pressure, hypertrophy and insufficiency of the right cardiac ventricle, and eventually, cor pulmonale.

**DISEASES OF THE UROGENITAL TRACT**

**Uraemia**

Patients suffering from severe acute or more commonly, chronic renal diseases (glomerulonephritis, pyelonephritis) may develop increased levels of serum uric acid.

*Symptoms:* general symptoms of uraemia (nausea, vomiting, breath smelling like urine, hypertension anaemia (pale skin), infections, bleeding, and prolonged wound healing). A characteristic, brownish-yellowish-faint colour (xanthochromia) appears on the skin, especially in the facial region. The sclera has a yellowish discoloration. Erythematous, pseudomembranous ulcerations and hyperkeratotic alterations may appear on the oral mucosa. Stomatitis accompanying uraemia (erythematous, pseudomembranous or ulcerative) as well as the white lesions respond well to dialysis. Apart from the symptoms mentioned above, xerostomia, taste deficiencies, oropyrosis, metallic taste sensation, gingival bleeding, purpuras (hemorrhages) of the oral mucosa, bacterial plaques, candidiasis, lingua pilosa, cheilitis, angular cheilitis and hairy gingival hyperplasia may occur. Salivary glands may become swollen as well. Due to anaemia, the oral mucosa and facial skin become pale. Hyperpigmented lesions may also occur on the oral mucosa.

*Differential diagnosis:* leukoplakia, morsicatio buccarum.
DISEASES OF THE GASTROINTESTINAL SYSTEM

Gastroesophageal reflux disease

Primary motility problems of the oesophagus are common.

Symptoms: apart from acidic regurgitations, patients complain of heartburn, chest pain, cough and nausea. Due to irritating chemical effects, the oral mucosa of the pharynx and the posterior dorsal lingual surface become erythematous and atrophic. The tongue of patients suffering from reflux becomes coated (whitish-yellowish), and hairy tongue may also accompany this condition. Patients often complain about burning sensation in the oral mucosa (oropyrosis) and the tongue (glossopyrosis). Permanent dental erosions (damage to the enamel and dentin) may also evolve apart from the symptoms of the oral mucosa.

Differential diagnosis: acute atrophic candidiasis, anaemia, Sjögren’s syndrome

Crohn’s diseases

It is a chronic, inflammatory, presumably immunopathological disease with unknown origin which may affect the entire digestive system from the oral cavity to the rectum, but extraintestinal, metastatic Crohn’s disease may also occur. This disease primarily affects the ileum.

Symptoms: apart from internal symptoms (enteritis, diarrhoea, constipation, malabsorption, anaemia, arthralgia). Periorificial erythema may occur on the face. These alterations may develop anywhere on the oral mucosa but most commonly on the bucca. The mucosa becomes erythematous and swollen, and the surface is ulcerative, deeply fissured. Diffuse, red, indurated and granulomatous nodules evolve on the lips (especially on the upper lip) and gingiva. Multiple fissures may develop on the lips. Granulomatous cheilitis may be the first sign of Crohn’s disease; therefore, if swelling of the upper or lower lip occurs, examination of the intestinal system may be recommended. Aphthous ulcers may occur on the oral mucosa, most commonly on the palate. Angular cheilitis may occur in the commissures.

Differential diagnosis: granulomas due to candidiasis, Quincke’s oedema, granulomatous cheilitis.

Coeliac diseases (enteropathy caused by sensitivity to gluten)

This disease is caused by the pathological function, malabsorption and motility disorders of the small intestine. This disease is explained by genetic factors, gluten toxicity, intestinal peptidase deficiency and immune response.

Symptoms: characteristically, gastrointestinal protein loss, vitamin deficiencies and the malabsorption of lipids, carbohydrates, water and iron are common. Usually it begins in childhood, however, its symptoms may persist in adulthood as well. Symptoms associated with this disease are anaemia (usually microcytic, hypochromic), anorexia, sore bones, eczema of the skin, bleeding tendency and finger clubbing. Symptoms affecting the oral mucosa are aphthous ulcers mainly due to iron and folic acid deficiency. Small aphthas may also accompany this disease. The tongue becomes erythematous, atrophic and painfully burning. Angular cheilitis is another common symptom. Coeliac disease in childhood causes dental hypoplasia.

Differential diagnosis: anaemia, vitamin deficiencies.

ORAL SYMPTOMS OF HAEMATOLOGICAL DISEASES

Iron deficiency anaemia

Anaemia is a decreased amount of haemoglobin and of red blood cells within the circulation.

This disease is caused by extensive loss of blood, decreased iron intake or iron absorption. The idiopathic form of the disease is called Plummer–Vinson syndrome.

Symptoms: anaemia, dysphagia, broken, concave fingernails (koilonychia), glossitis and cheilosis. It is more common among women. Patients suffering from iron deficiency are usually listless, pale, their hair is turning grey, and they tend to have headaches. The tongue becomes atrophic, pale or sore, red, enlarged and smooth. Sometimes the surface of the tongue is fissured. The oral mucosa is pale. Painful erosions, angular cheilitis may also accompany this disease. Aphthous ulcers are more common on the oral mucosa. The resistance to infections
decreases, therefore, osteomyelitis is more common after a tooth extraction. The lips become thin. The tongue of the patient suffering from iron deficiency is more likely to develop candidiasis and leukoplakia, which may even transform into a malignancy.

**Differential diagnosis:** pernicious anaemia, Sjögren’s syndrome, acute atrophic candidiasis.

**Pernicious anaemia**

Anaemia which is caused by the lack of vitamin B12 or folic acid and the lack of intrinsic factor required for the absorption of vitamin B12.

**Symptoms:** pallor, fatigue, breathlessness, tachycardia, palpitations, diarrhoea and paraesthesia of the extremities are common. Apart from these, headaches, vertigo, nausea, weight loss, yellowish, pale, dry skin, nervous system problems, such as paraesthesia, or the numbness of the limbs may be the accompanying symptoms. The most characteristic oral symptom is Möller–Hunter glossitis, which involves the atrophy of lingual papillae, thus, the tongue becomes smooth and reflective. The tongue of these patients is “beefy red” and the painful tongue may develop paraesthesia or may become lobulated (due to decreased muscle tone) later on. The tongue resembling “cobble-stone appearance” is similar to the lingual alterations that can be observed in patients suffering from Sjögren’s syndrome. The oral mucosa becomes pale and yellowish, disturbances of taste sensation, xerostomia, burning oral sensation (oropyrosis) may also occur. Chronic mucocutaneous candidiasis, angular stomatitis, leukoplakia, or carcinomas are also possible complications. Recurrent aphthous ulcers may develop.

**Differential diagnosis:** iron deficiency anaemia, acute atrophic candidiasis

**Haemorrhagic diatheses**

**Von Willebrand’s diseases**

Clinical symptoms resemble haemophilia rather than thrombocytopenia. This disease is inherited in an autosomal, dominant trait. The disease occurs in childhood and affects girls mostly, which is not characteristic of haemophilia. Apart from the pathologic alterations of factor VIII and the capillaries, the functional disorders of thrombocytes and thromboplastin secretion are also typical. Bleeding petechiae, purpuras and bleedings (hematomas) occur on the oral mucosa.

**Symptoms:** petechiae and minor ecchymoses appear on the bucca and on the lips. Purpuras are mostly located on the hard and soft palates. Gingival bleeding is more common than one would expect during gingivitis. Prolonged and severe bleeding should be expected after tooth extraction. Apart from oral symptoms, only this latter one requires any therapy, and this symptom is alleviated by proper oral hygiene and the cessation of gingivitis.

**Differential diagnosis:** haemophilia.

**Haemorrhage caused by anticoagulants**

Anticoagulant treatment is common among patients receiving renal dialysis, or suffering from thromboembolic diseases, myocardial infarction, etc. Patients taking anticoagulants containing cumarin (e.g. Syncumar) and indandione compounds may develop haemorrhagic purpuras on the oral mucosa. The continuous monitoring of prothrombin time is required during oral surgical interventions.

**Differential diagnosis:** Werlhof’s disease, Osler’s disease

**6.7. Diseases of the lip (Péter Novák MD)**

**Exfoliative cheilitis (“cracked lips”)**

**Symptoms:** exfoliative periods alternate with erosion. The lips become dry, swollen, erythematous, and in rare cases painful vertical fissures occur. Scaling, yellowish-brown crusts recur periodically in stressful conditions. This chronic disease usually affects adolescent girls and women. Predisposing factors include licking and biting of the lips, irritation caused by protruding maxillary incisors, fever, nervous tongue protrusion, tension of the lips, and cold or extremely hot weather. Licking of the lips may cause perioral inflammation and secondary bacterial or fungal infection. In some cases, the cause of the disease remains unidentified.
**Differential diagnosis:** acute and chronic actinic cheilitis and cheilitis glandularis.

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**Angular cheilitis (perlèche)**

In children and young adults Staphylococcus or Streptococcus infection results in pyoderma of the labial commissure.

**Symptoms:** the condition causes maceration fissures and burning, painful, red infiltration. Later, greyish-white, hyperkeratotic thickening occurs on the denuded areas. The lesions may spread to the surrounding skin and honey-coloured crusts, similar to those seen in impetigo, appear. The disease may persist for months or years. Adults and the elderly are primarily infected with Candida albicans (Candida-associated angular cheilitis). It may also develop due to iron deficiency anaemia (perlèche), vitamin B12 deficiency, Plummer-Vinson syndrome, pellagra, diabetes mellitus and malnutrition. Other risk factors include overbite, congenital lip fistula, increased salivation (ptyalism), sagging face in the elderly, edentulous persons, deepened labiomandibular fold due to a decrease in the proper vertical dimension of the teeth among denture wearing patients, which may be accompanied by maceration caused by saliva and secondary Candida infection. Secondary Staphylococcus aureus infection may exacerbate the condition.

**Differential diagnosis:** pemphigus vegetans.
Angular cheilitis

Cheilitis glandularis

Cheilitis glandularis is a relatively uncommon and chronic lip disease, which affects middle-aged men more frequently than women. The aetiology of the disease is poorly understood, familial predisposition may occur, and trigger factors possibly include exposure to sun, emotional stress, tobacco smoking and poor oral hygiene.

Symptoms: primarily the lower lip is affected. Red spots appear mainly on the vestibular surface of the lips and the ductal orifice of small mixed salivary glands. Chronic inflammation and the hyperplasia of the salivary glands cause swelling of the lip and drooling due to increased salivation,. The dilated ductal orifices pose a high risk of pyogenic infections, which are characterized by suppuration of the salivary glands (suppurative cheilitis glandularis), and the swollen, infiltrated lip becomes painful, ulcerated and crusty (apostematous cheilitis glandularis) with mucinous purulent secretion expressible from the ductal openings. The lesion can be considered as a potential predisposing factor for the development of lip cancer.

Differential diagnosis: granulomatous cheilitis, chronic cheilitis, Quincke’s oedema and Crohn’s disease.

Fissured cheilitis

The cause of the disease is not clarified, but different exogenous predisposing factors such as tobacco smoking, mouth breathing, climatic factors, Crohn’s disease and Down’s syndrome may play a role.

Symptoms: men are more frequently affected than women. Painful, denuded fissures appear on the lower and upper lips, which heal with difficulty. The lesions are mostly infected with Staphylococci.

Differential diagnosis: mechanical injury.

Acute actinic cheilitis

Acute actinic cheilitis is an acute inflammation of the lips caused by exposure to strong sunlight (“solar cheilitis”). Acute cheilitis frequently develops on the beach („summer cheilitis”), during skiing, and among
people with light complexions. Photoallergic and phototoxic cosmetics (lipstick cheilitis) or medications may also cause acute contact cheilitis.

**Symptoms:** swollen, oedematous lips, redness, tension and itchy sensation. Single sun exposure results in vesicular-bullous, painful, oozing lesions, ulcers, followed by brownish-red drying crusts appearing mainly on the lower lip.

**Differential diagnosis:** exfoliative cheilitis, eczema, chronic actinic cheilitis and lip cancer.

**Cheilitis due to diabetes**

Chronic, unhealing, burning, distending cheilitis or a lip wound may indicate diabetes. In such cases, a laboratory test may confirm the diagnosis. Chronic ulcer may mimic lip cancer, thus a biopsy is necessary.

**Differential diagnosis:** chronic cheilitis.

### 6.8. Diseases of the tongue (Péter Novák MD)

**Median rhomboid glossitis**

The disease used to be classified as a developmental disorder (the persistence of tuberculum impar), but today the role of chronic Candida infection and decreased vascularity is emphasized. It is common among smoking men and among diabetic patients. The lesion occurs more frequently in denture-wearing patients.

**Symptoms:** an oval or rhombic, red, smooth, non-protruding or verrucous, prominent lobulated patch with a depapillated surface presents in the middle of the back of the tongue, in front of the foramen caecum. It is usually asymptomatic, except for patients wearing full upper dentures.

**Differential diagnosis:** Tongue cancer, migratory glossitis, verrucous leukoplakia.

**Allergic glossitis**

Various local (dental materials, medications – drug-induced glossitis) and systemic factors (medications, alimentary factors, etc.) may cause vesiculobullous lesions on the tongue that later turn into painful ulcers covered with a whitish-yellowish, fibrinous pseudomembrane.
Differential diagnosis: Migratory glossitis, decubital ulcer, pemphigus vulgaris, or atrophic candidiasis.

Coated tongue

A coating (epithelial cells, neutrophil granulocytes, mucin, fibrin and food remnants) of various colour and thickness develops together with changes in the condition of the filiform papillae of the tongue. Possible causes include upper respiratory (tonsillitis) and lung diseases, smoking, febrility, gastrointestinal and liver diseases, inappropriate oral hygiene, soft foods, denture wearing and oral diseases (e.g. stomatitis). It can be an accompanying symptom of glossitis and it may develop as a result of decreased salivation, too. The colour of the coating may vary from white to brown (smoking, tea, coffee, etc.).

Differential diagnosis: Leukoplakia, candidiasis.

Lingual papillitis

The red lesion presents in elderly, denture-wearing patients and involves the inflammation and hypertrophy of the papillae, first on the tip of the tongue, then at the back of the tongue as well. The fungiform papillae appear in the form of red spots.

Symptoms: inflammatory papillitis causes a burning sensation and moderate pain.

Vitamin deficiency (A, B, C), iron deficiency, alcohol consumption, smoking, hot and spicy foods and mechanical irritation may also play a role in the development of the disease.

Hypertrophy of the circumvallate papillae

The epithelium of the circumvallate papillae contains many taste buds, thus these papillae play a significant role in the sense of taste. The circumvallate papillae are situated on the posterior third of the back of the tongue, resembling the limbs of a letter V inverted.

Symptoms: in case of inflammatory hypertrophy, the papillae protrude more than normal and become lumpy, red and moderately painful during eating or swallowing. Frequently recurring upper respiratory infections or chronic irritation contribute to the inflammatory hypertrophy of the papillae, and even doctors or dentists might be deceived by the nodular-appearing ‘tumour-like’ lesion. Such lesions on the tongue often lead to the development of cancerophobia.

Differential diagnosis: benign and malignant tumours, or median rhomboid glossitis.

Geographic tongue

The cause of the disease is not clarified; familial predisposition, psychological factors, vitamin deficiency, malnutrition, exudative diatheses and endocrine disorders may play a role in the development of the lesion.

It can be an accompanying symptom of psoriasis, reactive arthritis and diabetes mellitus.

Symptoms: Red patches of round, oval or indeterminate shape appear on the tongue (or rarely on the lips, buccal or gingiva) and migrate (“wander”) to other locations after some time, or occasionally persist at the same place for a longer period. Filiform papillae disappear but fungiform papillae persist at the site of the red patch, which has a diameter ranging from a few millimetres to some centimetres. The patch is bordered by a white, hyperkeratotic, zigzag-like and elevated margin. The condition can develop at any age. It often coexists with fissured tongue and might be accompanied by a stinging, burning sensation. Patients suffering from benign migratory glossitis are not uncommonly mentally unstable and cancerophobic.

Differential diagnosis: Leukoplakia, oral lichen planus, secondary syphilis, annular lichen planus of the mouth, or candidiasis.
**Geographic tongue**

*Lingua villosa (hairy tongue)*

The condition is considered to be a multi-aetiologial disease, which presents more frequently in elderly denture-wearing patients. Hairy tongue is often idiopathic, but it can also be triggered by antibiotics, corticosteroids, vitamin A or B deficiency, radiotherapy, chemotherapy, inappropriate oral hygiene, emotional stress, Candida albicans, heavy smoking, gastrointestinal disorders or hydrogen peroxide.

**Symptoms:** the hairy surface is caused by the elongation, thickening (hypertrophy) and keratosis of the filiform papillae. The yellowish-greenish, brownish-black colour can be explained by the presence of pigment-producing bacteria and Aspergillus species (not Candida albicans), foods, medications, consumer goods and smoking.

**Differential diagnosis:** Coated tongue.
Lingua villosa (hairy tongue)

Glossodynia

In the majority of the patients organic changes cannot be detected on the tongue, so probably psychogenic causes lie in the background of the unpleasant tongue burning.

Symptoms: the condition is more frequent in women and it presents mostly during menopause. In some cases, the fungiform papillae are slightly elevated on the tip of the tongue and they become erythematous. Burning and itching develops mainly on the tip and lateral border of the tongue, but it can present on other mucous membranes as well. Glossopyrosis is often accompanied by cancerophobia.
Glossodynia may also present in iron-deficiency and pernicious anaemia, xerostomia, candidiasis, geographic tongue, lichen planus, herpes simplex, allergic reactions, diabetes mellitus, gastro-oesophageal reflux disease and hypertension.

6.9. Diseases of the salivary glands (Péter Novák MD)

**Sialolithiasis**

Salivary calculi develop mostly inside the submandibular or sublingual glands or within Wharton’s duct.

Salivary calculi may be composed of calcium (phosphorous or carbonic acid) compounds, bacteria, epithelial cells, foreign bodies or thickened saliva.

The development of calculi is uncommon in the parotid and minor oral salivary glands. It is more common among men than women.

**Symptoms:** complaints are due to the calculi partially or completely obstructing the aperture. Pus may be evacuated through Stenson’s duct when applying pressure to it. Before meals, but especially during eating, bursting, intense pain occurs due to increased saliva production, the affected glands become swollen and tender. Salivary calculi within Wharton’s duct on the floor of the mouth are easily palpable, furthermore, the swollen gland becomes visible.

**Differential diagnosis:** ranula, tumours of the floor of the mouth.

**Recurrent parotitis in childhood**

The causes of this disease are unclear, however, apart from otitis or tonsillitis, some researchers presume that allergic or mycotic factors may also play a role in its development.

**Symptoms:** the disease starts with uni- or bilateral, painful parotid swelling, which may be accompanied by swollen lymph nodes, fever or subfebrile periods. When pressure is applied to the parotid glands, purulent or white, thick saliva empties from Stenson’s ducts. Recurrences may occur on a monthly or yearly basis. Relapses spontaneously cease after puberty.

**Differential diagnosis:** epidemic parotitis, salivary calculi.

**Sialoadenitis of the submandibular gland**

Inflammation of the submandibular salivary gland may be caused by direct bacterial infections, salivary calculi or tumours. Bacterial sialoadenitis may be caused by staphylococcus or streptococcus infections, but dental, periodontal or mechanical irritation of the orifice (denture) may also play an important role.

**Symptoms:** the salivary gland becomes painfully swollen and swallowing causes pain as well. The swelling of the salivary glands may become permanent as a result of long-term involvement due to fibrosis.

**Differential diagnosis:** sialolithiasis, dermoid cysts, metastases.

**Xerostomia**

Xerostomia is not a disease but a symptom. Xerostomia may be caused by a wide range of conditions, the main cause being temporary or permanent cessation of salivary secretion.

**Causes:** xerostomia most commonly presents as an adverse effect of pharmaceuticals (minor and major tranquillisers, antihistamine products, antihypertensive drugs, Parkinson’s medication, cytotoxic pharmaceuticals, cytokines, narcotics, retinoid drugs, sympathomimetics and other anticholinergic drugs). Apart from these, xerostomia may be caused by congenital anomalies of the salivary glands or dehydration (diabetes mellitus or insipidus, vomiting, diarrhoea, bleeding), elderly age (menopause), psychogenic conditions (depression), salivary gland conditions (Sjögren’s syndrome), iron deficiency anaemia, irradiation, etc.

**Symptoms:** the oral mucosa becomes dry and red while the epithelial tissues themselves become atrophic. Patients suffering from these symptoms usually complain of swallowing, eating, speaking and tasting.
difficulties, as well as a burning sensation of the tongue (glossodynia) and the mouth (oropyrosis). The predisposition to caries and oral candidiasis is also increased.

6.10. Oral symptoms of neurological and psychiatric diseases (Péter Novák MD)

Trigeminal neuralgia

Facial pain may be an accompanying symptom of some underlying diseases (multiple sclerosis, tumors of the brain stem, etc.), or may be of idiopathic origin as well.

Symptoms: sudden, flash-like, paroxysmal, sharp and stabbing, intense facial pain is also characteristic, and it may be provoked by touch, facial mimics, eating, toothbrushing, cold air or draught. As the disease progresses, attacks of radiating pain occur more frequently and spontaneous remission becomes increasingly rare. Symptoms are unilateral and usually affect areas served by the second or third branches of the trigeminal nerve.

Differential diagnosis: toothache, pain associated with herpes zoster.

Bell’s palsy (idiopathic facial palsy)

The paralysis of the facial nerve may be idiopathic or it may occur due to inflammation in the area of the stylomastoid foramen, or due to inflammation, tumour, trauma or vascular spasms within the osseous facial canal. In the past few years, the role of herpes simplex and zoster-varicella viral reactivation, draught or cold trauma have been considered as trigger factors; however, idiopathic forms of the disease occur as well.

The disease may be accompanied by hypertension, diabetes or lymphoma and may develop due to lower peripheral nerve block anaesthesia before tooth extraction. Facial palsy may be preceded by mandibular pain and numbness of the tongue on the affected side. Facial palsy usually develops suddenly, the patient feels stiffness in the face, one side of the face may be drawn, the commissure becomes loose, the closure of the ipsilateral eyelid may be compromised, the corneal reflex is missing and patients complain of increased (unilateral) lacrimation during meals (“crocodile tears”). Typically, the eyeball deviates upwards when the
eyelids are closing. When patients attempt to smile, the commissure of the lips on the affected side is drawn downwards instead of upwards, and peri orbital wrinkles do not appear on the paralytic side. Disturbances of taste sensation, difficulties with eating, speaking, whistling may accompany the disease, the paralysis may persist for several days or weeks or sometimes it may even become permanent.

**Oropyrosis (oral dysesthesia)**

Burning sensation (stomatopyrosis) or pain (stomatodynia) inside the oral cavity may affect the tongue alone (glossopyrosis, glossodynia).

The patients are commonly middle-aged or elderly women, who experience dysesthesia on waking up, and this sensation progresses over the course of the day. Affected patients often have cancerophobia or other causes of anxiety.

The symptoms are more common after menopause. The complaints may be caused by localised, systemic, neurological or psychiatric factors. The common local causes include geographic tongue, fissured tongue and medial rhomboid glossitis. According to some experts, candidiasis may be the most common cause, especially when it is accompanied by excessive smoking or alcohol consumption.

As a general cause, vitamin B deficiencies and consequent pathological conditions may be considered, such as pernicious anaemia, hypoacidity, gastro-oesophageal reflux disease, iron deficiency anaemia, diabetes mellitus, oral lichen planus or long-term treatment with antibiotics or other pharmaceuticals (ACE inhibitors). Xerostomia is a common predisposing factor. Unilateral symptoms suggest neurological conditions. If there are no evident local, systemic or neurological underlying causes, the diagnosis is idiopathic glossodynia. In such cases depression, cancerophobia and neurosis may be the explanation of these complaints.

### 6.11. Traumatic dental injuries (Ildikó Pinke DMD)

Traumatic dental injuries affect mainly the front teeth and may occur with high frequency in preschool and school age children and young adults (comprising 5% of all injuries for which people seek treatment).

Luxations are the most common traumatic dental injuries in the primary dentition, whereas crown fractures are more commonly reported for the permanent dentition. Dental traumas are often accompanied by soft tissue and other associated injuries, which may require other surgical intervention. Injuries may involve the teeth, lips, jaws, tongue, gum, cheeks (rarely the roof of the mouth, palates, neck, or tonsils).

**PATIENT EXAMINATION:**

- *Where did the injury occur?* – the possibility of contamination
- *When did the injury occur?* - the extraoral storage condition (avulsion)
- *How did the injury occur?* – any sign of child abuse
- *Was there a period of unconsciousness and how long?* – Amnesia, nausea and vomiting are all signs of brain damage.
- *Is there any reaction in the teeth to cold and/or heat exposure?* – exposed dentin or pulp
- *Is there any disturbance in the bite?* – any alveolar fracture, jaw fracture or a fracture of the condylar region

**DIAGNOSTIC SIGNS**

**Visual signs**

- not displaced (concussion, subluxation)
- appears elongated (extrusion)
- displaced in palatal/lingual or labial direction or axially
- visible fracture line
Percussion test

- tender to touch or tapping (concussion, subluxation)
- tenderness (extrusion, crown-root fracture)
- ankylocytic sound (lateral luxation, intrusion)
- not tender (infraction, enamel fracture)

Mobility test

- no increased mobility (contusion)
- immobile (intrusion)
- increased mobility (subluxation)
- excessively mobile (extrusion)
- normal mobility (infraction, enamel fracture)
- coronal fragment mobile (crown-root fracture)

Pulp sensibility test – cold test and/or electric pulp test

The purpose of the test is to determine the condition of the pulp. Extrusion, intrusion and lateral luxation injuries have high rates of pulp canal obliteration (PCO). PCO occurs more frequently in teeth with open apices which have suffered a severe luxation injury.

- Usually a negative result: contusion, subluxation, extrusion, intrusion, root-fracture
- Usually a positive result: infraction, enamel-fracture, enamel and root-fracture

Radiographic test

- No radiographic abnormalities: contusion, subluxation, infraction
- Periapical enlargement of periodontal space: extrusion, lateral luxation,
- No periodontal ligament space: intrusion
- The fracture line is usually visible

The diagnostic protocol can be seen on the next figure (Fig.2.23.)
**TREATMENT**

**Avulsion**

Treatment guideline for permanent teeth depends on

- **extraoral dry time:**
  1. extraoral dry time
  2. extraoral dry time longer than 60 min. *Delayed replantation has a poor long-term prognosis.* less than 60 min.

- **storage media** (tooth has been kept in physiological storage media or osmolality balanced media as milk, saline, saliva or Hank’s Balanced Salt Solution)

- **maturation of tooth** (open apex or closed apex)

**Splinting** *(Fig. 2.24.)*

Short-term, non-rigid splints (usually orthodontic arches) are used (passively) in luxated, avulsed and root-fractured teeth. The duration of splinting is significantly related to healing outcome of the pulp (2 to 4 weeks). The splint may maintain the repositioned tooth in correct position, provide patient comfort and improved function.
Fixation of injured tooth with flexible fiber-reinforced composite splint

*Restore with resin composite and/or glass-ionomer*

In case of enamel fracture, enamel-dentin fracture

*Antibiotics*

Use of antibiotics is limited

- use of systemic antibiotics is recommended in the management of avulsion (the first choice is Doxycycline 2x per day for 7 days in appropriate dose for patient age and weight; in young patients Amoxicillin in appropriate dose for age and weight)

- there is no evidence that antibiotic therapy is effective in root fractured teeth.

*Patient Instructions*

- a flexible splint for 2 weeks to stabilise the tooth;
- brush your teeth with a soft toothbrush after each meal;
- oral hygiene and rinsing with an antibacterial solution (alcohol free chlorhexidine gluconate 0.1%) for 2 weeks;
- patient compliance with follow-up visits and home care contributes to better healing;
- advise regarding care to patients and parents of young patients of the injured tooth/teeth for optimal healing;
- follow-up controls are required to make a pulpal diagnosis;
- prevention of further injuries (avoidance of participation in contact sports or mouth guards should be worn as an oral protector by the players).

*Dental Box*
Utilizing for the emergency care of acute dental emergencies. It can be used quickly and easily in the care of fractured teeth, subluxations, luxations, avulsions, dry sockets, deep caries, loose appliances and bleeding mucosa. Contents can be seen on the next figure (Fig. 2.25.)

### Contents of Dental Box

**Save-A-Tooth**

It is a kind of emergency tooth preserving system. Use of this appliance increases the survival rate of a knocked-out tooth. Ingredients: water and Hank’s Balanced Salt Solution (active ingredients)

#### 6.12. Deposits forming on the teeth (Zsuzsanna Tóth DMD - Péter Vályi DMD)

For several centuries, the importance of good oral health has been emphasised in the literature. There is mediaeval evidence originating from more than 5,500 year old findings of the Babylonians on the removal of deposits formed on the teeth. The role of the deposits causing periodontal diseases and caries are proved by scientific studies. A wide variety of materials of different hardness, colour and adhesiveness originating from the oral milieu can be deposited on the natural and artificial hard surfaces in the mouth:

- acquired pellicle
- dento-gingival plaque
- calculus
- debris
- materia alba

The dental plaque is a strong adhesive layer on the surface of the teeth, restorations and dentures, which develops by the help of acquired pellicle, and it can be removed only by rubbing and not by mouth rinsing or water jet (Figure 2.26.). The main components of the dental plaque are bacterial strains colonising in the intercellular matrix, which are located in the biofilm (See Chapter 1.8). The supragingival plaque adheres above the gingival margin, and the subgingival plaque adheres under it.
Dental plaque (from Prof. S. Kneist)

The *materia alba* is a greyish-white-yellow soft layer that is clearly visible also by the naked eye on the hard surfaces. It is less adherent in comparison with plaque, and it can be removed using a water jet. Its main mass contains bacteria, salivary proteins, detached epithelial cells, disintegrated leukocytes, and often food remnants as well.

Debris may be found on the teeth, between the teeth, and possibly also on the surface of soft tissues, which is food remnant in the mouth washable by water, but usually they are removed by the mechanical effect of muscle function and salivary flow.

**THE ROLE OF DENTAL PLAQUE IN GINGIVITIS**

There are bacterial colonies organized in a biofilm, as members of the natural bacterial flora are present in the healthy oral cavity without causing any disease. Small amounts of bacteria causing diseases can often be detected, but in healthy conditions the human organism’s defence mechanisms are able to maintain a balanced state without any damage to the tissues. Due to strengthening of the bacterial effects or weakening of the body’s defence mechanisms, the balance will be tilted and pathological changes, pathoses occur.

The biofilm forming microorganisms can be classified as *symbiotic* (apathogenic) and pathogenic microorganisms. Against the members of the symbiotic flora the organism will not trigger the tissue destruction causing, damaging defence mechanisms. The members of the *pathogenic flora* cause inflammation and promote the development of disease characterized by direct and indirect (inflammatory and immunological) damage of tissues. Certain risk factors influence the development and the process of the pathosis significantly (see chapter 2.15.1.).

The increase in the amount of dental plaque results in gingivitis. Gingivitis without any treatment will be followed by periodontal disease. In most of the cases the regular and efficient dental plaque removal prevents or heals the gingivitis. Frequently experienced gingivitis in pregnant women developing as a result of the hormonal changes is of great importance, but it is also related to the formation of dental plaque.

According to the *non-specific plaque theory* of Loesche W. (1976) and Theilade (1986) the quantity of accumulating dental plaque is the cause of the consecutive gingivitis. In larger amount of dental plaque more harmful bacterial decomposed products will be produced leading to inflammation of gingival tissues and growing worse to inflammation of periodontal tissues. In some cases however excessive mass of dental plaque of the neglected mouth does not result in serious medical consequences. Therefore scientific examinations have focused on the bacterial composition of the dental plaque, and it was found that certain well-defined pathogenic strains can be isolated, which have important role in the periodontal pathological processes, they cause disease and their removal causes the healing. That is the explanation also for the experience whereas the same large
amount of plaque in the oral cavity of neglected patients can cause inflammation of different stages: mild or quite severe as well. According to the specific plaque theory (Loesche W. 1979) the disease of the periodontium can be explained not by the quantity of the dental plaque, but by the presence of some pathogens. Among the disease causing parodonto-pathogen bacteria there are only a few species, which can not be found or only in trace amounts (exogenous pathogens) in the healthy mouth. Bacteria also of the healthy organism cause very often opportunistic infections due to a change in circumstances (constitutional factors, quantitative and qualitative changes of the composition of dental plaque, risk factors etc.). This is the endogenous infection, which plays an important role in the development of inflammatory and destructive disease of the periodontal tissue (Wirthlin and Armitage, 2004).

There is not still correct answer received what a role is played by the mostly obligate anaerobic parodonto-pathogen bacteria strains in the aetiopathogenesis of the disease, since they can not be the initiators of the disease because of their nature, they are likely responsible for the progression of the disease.

Formation and properties of dental plaque see in chapter, 1.8.

THE ROLE OF DENTAL PL AQUE IN CARIES

In the chapter on biofilm is already mentioned that the composition of various colonies found on the different surfaces, mainly due to the different environmental conditions is significantly varied. While the bacterial strains at the gingival margin and below them play a role in the inflammatory processes of gingival and deeper periodontal tissues, the bacteria of dental plaque accumulating on the smooth surfaces and in pits and fissures of the coronal part of the teeth are the etiological factors of dental caries. The metabolism of cariogen plaque’s components produces acidic milieu which initiates demineralization. If they can not be neutralised and compensated by remineralizing processes, the dental hard tissue will be damaged.

The role of dental plaque bacteria in caries aetiology detailed can be found in the next chapter.

THE FORMATION OF DENTAL CALCULUS

In mature dental plaque accumulating on the surface of the teeth mineral salts can be precipitated and calcifying the plaque forming supra- and subgingival type of dental calculus.

The supragingival calculus

In the beginning the supragingival calculus is a yellowish-white porous deposit above the gingival margin (Figure 2.27.). The characteristic places are the predilection sites, where they appear very early and easily. Typical localization of dental calculus formation is as follows: the opposite tooth surfaces of the salivary glands’ outflow, for example lingual surface of the lower front teeth, and vestibular surface of upper molars. Typical experience is, that in neglected subjects chewing only on one side (unilateral chewing), also the occlusal surface of the teeth of opposite side will be covered by calculus. Without any oral hygiene deposits can be formed not only on the surface of the teeth, but on the surface of the restorations and dentures, prosthesis as well. The cleaning of the dental calculus can not be perfect, because of its rough outer layer. At first it is porous, but in the course of time it will be harder and harder, its removal needs considerable effort. Its colour can vary regarding the meals and beverages and depending on the by-product of bacterial metabolism originated on its surface developing plaque. Its colour can vary from yellowish-white to dark brown or black. Note that the role of dental calculus is determining in the process of development of periodontal diseases because it is an important plaque-retentive factor. Beside this it widens the gingival sulcus that is why the forming of subgingival biofilm will be possible. The bacterial toxins originated from the plaque of the calculus surface cause pathological tissue destruction. Every roughness of the tooth surface promotes the formation of dental plaque and calculus. In addition the accumulation will be facilitated by gaps, leakage, margins of fillings, restorations, prosthesis and orthodontic appliances beside the original dental conditions.

The subgingival calculus

Subgingival calculus can be formed only in the gingival sulcus of inflamed gingiva. There is no predilection place for its development, but there is no subgingival calculus below the healthy gingival margin (Figure 3). In case of thin gingival margin its dark bluish purple discolouration is visible also by naked eye. Its diagnosis is possible by periodontal probe, by removal of inflamed marginal gingival with air, and its interproximal localization is visible in the X-ray picture as well. Subgingival calculus develops due to the mineralization of subgingival dental plaque, its colour is black, and its surface is very rough. Its calcifying is originated from sulcus secretion and blood, that is why the supragingival calculus is from saliva and contrast with it.
Subgingival calculus is originated from the gingival sulcus. It adheres very strong to the tooth, stronger than supragingival calculus. It can be explained by the roughness of the root surface and by the fact, that not only dental plaque but also the acquired pellicle will be mineralized.

Supra- (yellowish-white) and subgingival (brownish-gray) calculus

In the inter-bacterial matrix of dental plaque some components (proteins, fats) are centres of crystallisation, here starts the crystallisation from the supersaturated salt solution of the saliva. Dental calculus mostly contains inorganic salts (octa-calcium-phosphate, tri-calcium-phosphate, magnesium-carbonate, sodium carbonate) and later hydroxyl-apatite crystals. Supragingival calculus has a layered structure, the mineral content of the layers are different but the average value is lower than in case of subgingival calculus.

The supra- and subgingival calculus should be fully removed from the tooth surface. In case of subgingival calculus, frequently, it cannot be performed only with the accidental attenuation of the root cement.

6.13. Dental caries (Zsuzsanna Tóth DMD)

DEFINITION OF CARIES

Caries is damage to hard tissue in the presence of cariogenic microorganisms on erupted tooth surface covered by dental plaque. The chronic process is reversible in the initial stage but will be irreversible following cavitation. It extends from the coronal enamel surface towards pulp as a result of demineralising and remineralising processes depending on the frequency of the carious attacks. As a result of aging and recession of the gingival, caries can develop on the cementum and on the nude dentin as well. Over time the deep penetrating destruction results in pulp pathosis.

EPIDEMIOLOGY OF DENTAL CARIES

Epidemiology as part of medical sciences deals with pathological changes and epidemic diseases occurring on a large-scale. The existence, severity, frequency, development, spread and conditions of diseases are investigated regarding different parameters. These parameters can be e.g. the following: age, sex, occupation, qualification, behavioural and dietary habits, and geographical, social and economic conditions. After the evaluation of statistical data we may come to a conclusion about pathological factors, risk groups, therapeutical and preventive options, and about the effectiveness of therapy and prevention. Discovering the caries preventive effect of fluorides is also a result of epidemiological investigation.

Dental caries – besides inflammatory diseases affecting the periodontium – is the most frequent chronic disorder. Its therapy is expensive for the society although only a few patients visit their dentist regularly. The right solution is prevention.
Dental caries has been present since the start of human history. Its prevalence has increased to 90-100% nowadays. In the 1970’s and 1980’s in some industrialised countries the increase stopped and then decreased, e.g. in the US, in western European countries, in New Zealand and in Australia. The decline can be attributed mostly to the presence of fluoride in the drinking water and later in the products of oral hygiene, first of all in toothpastes. Dental caries is a civilisational disease. In developing countries there used to be generally low values which rapidly increased with civilisation. There is a close correlation between disadvantageous dental caries conditions and dietary and oral hygiene habits.

**Epidemiological survey**

The participants in epidemiological studies are the population. Caries frequency (caries prevalence) shows the percentage of persons with carious teeth. The number of diseased teeth is expressed by caries intensity (caries experience). Horizontal or cross-sectional surveys provide information about the actual conditions, in a follow-up survey, or data of a longitudinal study are registered periodically (in case of caries: yearly) in the same way. The changes in caries conditions can be determined. Caries increment reports new carious lesions, caries incidence indicates the number of persons with a new carious lesion.

**DMF scores** are individual quantitative data regarding caries; DMF index shows data of a group. It is a quotient: the sum of all DMF scores in the group should be divided by the number of subjects. DMF is an acronym, D means decay, M means missing and F means filled. DMF-T index (T means tooth) refers to carious teeth, DMF-S (S means surface) refers to tooth surfaces. The increasing depth of caries in the enamel is indicated by D1-D2 scores, dentinal lesions are indicated by D3-D4 scores. For primary teeth the nomination is the same but it is written with lower case letters: dmf-index. The indices df and def are widespread; in the second one e means deciduous tooth indicated for extraction. The df-t index refers to teeth and df-s index refers to tooth surfaces.

In comparative epidemiological caries surveys the data of the same age groups will be compared. Successful prevention and conservative therapy result in more remaining teeth in elderly patients although they have more periodontally involved teeth. The frequency of root caries on the exposed tooth neck is increasing due to gingival recession. RCI, root caries index expresses the quotient of carious root surfaces and exposed root surfaces. Recent epidemiological studies demonstrate an increasing number of root caries depending on age and remaining teeth. Root caries is more frequent in males and in lower teeth with the exception of the incisors.

**DENTAL NOTATION**

There are many nomenclatures in the literature for the definite identification of the 20 primary and 32 permanent teeth. In Hungary the Zsigmondy system is used the most frequently: the teeth are put in the Zsigmondy-cross. The patient is sitting opposite the dentist, the mouth is divided into four quadrants and each permanent tooth is assigned a number from 1 to 8 starting at the midline.

Each primary tooth is numbered similarly but with Roman numbers from I to V starting at the midline.
In scientific publications the two-digit notation of the World Dental Federation, FDI (Federation Dentaire Internationale) is adopted.

**FDI notation of teeth (ISO system by WHO)**

In the US the teeth are numbered simply following the quadrants clockwise from the upper right side numbering them continuously:

**Universal numbering system "American method"**

**AETIOLOGY OF CARIES**

Hundreds of theories explaining the development of caries have developed in the past centuries, many of which contain plausible mechanisms according to our present knowledge.

**Modern theory of caries development**

Dental caries is a disease of dental hard tissues with a multifactorial aetiology. Besides the four essential (primary) factors, secondary factors (e.g. biological, environmental, geographical, socio-economic, etc.) play an important role in the development of tooth decay.

*The primary conditions for the formation of dental caries are (Figure 2.31.):*

1. The surface of erupted tooth in the oral cavity
2. The microbial flora adhering on it
3. The substrate providing nutrients for microorganisms
4. The time factor
The primary factors for the development of dental caries

Tooth surface as host indicates the importance of systemic factors. The outer cover of the crown, the enamel is the hardest tissue of our body, but it is still susceptible to environmental harmful effects. The enamel is of ectodermal origin, has an acellular structure, no circulation, and it is unable to defend itself or to reverse already existing tissue damage. In the oral cavity after eruption, the tooth surface as host is covered by a biofilm (dental plaque). The dental plaque is a strongly adhesive bacterial aggregate on the surface of oral structures which can only be removed by intensive mechanical cleaning. In the dental plaque there are microorganisms and nutrients as well.

Biochemical reactions taking place between the enamel and the dental plaque are in a dynamic equilibrium. This balance is disturbed during food intake. After carbohydrate intake (carious attack) microorganisms produce organic acids (such as lactic acid) by the degradation of carbohydrates. This decreases the pH of dental plaque.

The mineral components of hard tissue (e.g. calcium, phosphate) are dissolved, this process is called demineralisation. With the disappearance of carious attacks under the influence of saliva, pH is increased and remineralisation processes predominate. By the diffusion of calcium and phosphate (mostly in the presence of fluoride) the demineralised surface is remineralised.

Demineralisation and remineralisation processes occur many times a day. The frequency of carious attacks leads to a dominance of demineralisation, to macroscopic lesions and irreparable cavitation (Figure 2.32.).
The significance of the time period is closely related to frequency. The more often the surface is exposed to carious attacks, (demineralisation) (e.g. due to frequent snacking), the less time is available for remineralisation (Figure 2.33.).

In addition to primary aetiological factors, many biological, behavioural, environmental, geographical, socio-economic factors play a role in influencing the likelihood of caries development.

**Microbiological background of caries**

The complex and dynamic relationship between bacterial plaque, host and diseases in the oral cavity is the result of a working ecological system. The oral microflora of a toothless baby is poor, it will be rich in the presence of
teeth, but under elderly edentulous circumstances the situation is similar to childhood. Only a limited number of bacteria of the 400-500 types existing in the oral cavity have an important role in caries development (cariogenesis). Dental plaque accumulates on the tooth surface in a determined sequence. Most of the microorganisms leave the mouth by swallowing and only a few of them are able to stay and adhere to soft and hard tissues. At different places of their retention there are different complex colonisations. That is why it is hard to prove the direct casual relation between the caries process and one single pathogen, but the role of Streptococcus mutans and Lactobacilli is definitive in caries development and progress.

The role of saliva in the development of caries

Saliva is particularly important in cariogenesis. The water and mucin content of saliva moistens the mucous membranes and helps taste and swallowing. An adequate amount of saliva (appropriate level of flow rate) cleans the oral cavity from a significant part of food remains, microorganisms and dissolved metabolites by its mechanical washing and diluting effect. This role inhibits the development of caries. The lubricant components of saliva facilitate speaking. Mucin and mucoid content increases viscosity and thereby facilitates the formation of dental plaque and consequently, the formation of caries as well. Enzymes of the saliva already start to digest nutrients in the oral cavity; carbohydrate breakdown results in an acid pH in the mouth promoting caries development. In contrast to this, however the caries inhibitory properties of saliva predominate. The high number of bicarbonates has a buffering effect; its efficiency is enhanced by phosphate and to a certain extent by the protein buffer system as well. The antibacterial activity of saliva is provided partly by immune proteins and enzymes. Growth factors in saliva promote wound healing; inorganic ions (calcium, phosphate, fluoride) promote the remineralisation of enamel. Decreased salivary secretion (xerostomia) reduces the amount of protective proteins (protective function) and reduces acid and carbohydrate clearance. A decreased salivary flow rate is a physiological phenomenon of aging, menopause, symptoms of some diseases, for example psychic disorders, autoimmune diseases of the salivary glands (e.g. Sjögren's syndrome) or diabetes mellitus. It can be associated with anaemia, dehydration, vitamin deficiency or pregnancy as well, and can also occur as a result of drugs. The reduced production of saliva leads to caries increment.

Nutrition and caries

Food intake is of crucial importance in dental caries and in periodontal diseases because it provides the essential nutrients for the microorganisms in the dental plaque. The composition and consistency of food, the way, the quantity, the frequency of food intake and the duration of residence in the mouth are all important factors. Its effect can work in two ways:
• praeresorptive effect prevails in the oral cavity before the absorption in contact with teeth and other oral surfaces during chewing.

• postresorptive effect is a systemic effect after absorption, as in the developmental period of teeth before eruption (praeruptive).

Other factors affecting caries development

In addition to the primary factors in the aetiology of dental caries there are also non-negligible secondary factors playing a role in the process, for example macroscopic and microscopic properties of the teeth, physique (genetic background), age, sex, hormonal and immunological factors, and some geographical, social and economic factors.

THE LOCATION OF CARIES

Caries develops if in the presence of bacteria, the balance of demineralisation and remineralisation processes is upset, leading to increased demineralisation. The anatomical location of caries is on the crown and on the root. Morphological and histological differentiation is based on enamel, dentin and cement tissue. Predilection sites are areas susceptible to caries development. The predilection sites are the non-self-cleaning surfaces, so-called habitually unclean areas. As a result of self-cleaning, the dental plaque disappears from tooth surfaces which are in contact with the lips, tongue, and bucca due to speaking and chewing or due to the abrasive effect of food. To clean the non-self-cleaning or habitually unclean areas is often very difficult. The caries susceptible predilection sites are as follows (Figure 2.34.):

1. pits, grooves, fissures,

2. smooth surface interproximally below the contact point,

3. smooth surface at the gingival border,

4. root surface.

Caries predilection sites (Wannemacher 1963)
Crown caries

Crown caries means circumscribed carious lesions developing on the tooth crown in pits, fissures or on the smooth surface (Fig. 2.35.).
To clean deep and narrow fissures is often impossible; in this case fissure sealing in due time is possible to prevent caries formation (Fig. 2.36., see under IV. Prevention, in chapter 5. Fissure sealants). Pits and fissures are to be found on the occlusal surface of premolar and molar teeth and on the palatal surfaces of the upper incisors (foramen caecum).
Fissure sealing

Areas between the gingival margin and equator of the crown are not self-cleaning smooth surfaces, although it is easy to clean them because of good access. Caries in these areas draws the dentist’s attention to the lack of oral hygiene or disorder of saliva production (Figure 2.37.).
Caries at the gingival margin

The approximal smooth surface of the teeth below the contact point belongs to habitually unclean areas. The cleaning of this area requires some manual skill and the use of tools for oral hygiene besides the tooth-brush (e.g. dental floss, interdental brushes) (Fig. 2.38.).

Root caries

Nowadays more and more elderly people have their own teeth, but often the root surface is exposed. This area is not self-cleaning, plaque formation is promoted also by the decreased saliva flow rate. Due to the deterioration of manual skills, cleaning is problematic. At the enamel cement junction a soft, irregular, often discoloured lesion develops and extends relatively quickly.
THE EXPANSION OF CARIES

Dental caries is a pathological process extending from the enamel (root cement) surface through the dentine toward the pulp into the depths. Because of the prismatic structure and the different formation of enamel prisms, the cross-section of fissure caries and smooth surface caries are different in the enamel. There is no difference in the dentine, which has a tubular structure, and in both cases the cross-section is similar (Fig. 2.39.).

Smooth surface caries and cross-section of groove

Primary dental caries (caries primaria) occurs on an intact tooth surface which is not self-cleaning. Secondary caries (caries secundaria) develops along the margin of crowns, fillings or inlays placed in the teeth. In the stage of caries incipient the enamel surface is carious without any clinically or histologically macroscopic cavity formation. There is a chalky white spot on the tooth surface, and it becomes porous (Fig. 2.37.). From the occlusal surface at the bottom of the fissure, developing incipient caries appears to be a dark or an opaque area. The therapy of incipient caries is remineralisation. Superficial caries is a cavitated lesion progressing to the dentine. Caries media extends to the dentine, the deepest part of the process is far away from the pulp chamber. The differences in colour, transparency and the changes in the surface and the contour are visible to the naked eye as well, the break in continuity is tactile by a dental probe. Caries profunda is an extensive carious process, at this stage there is only a thin intact dentin layer between the pulp chamber and the carious process. In the case of caries penetrans the carious process has extended into the pulp, which communicates with the oral cavity. The contamination of the pulp leads to inflammation.

THE TIME COURSE OF CARIES

Dental caries is a chronic disease. Between the incipient stage and the clinically diagnosed cavitated the development takes about 18 ± 6 months. The process extends faster in the pits and fissures than in the smooth surface. If poor oral hygiene is connected with snacking, the frequency of daily carbohydrate intake is high, incipient enamel caries can develop in three weeks. X-ray irradiation leads to xerostomia, which can cause dental caries in three months. In healthy subjects the development of caries is slower. Depending on the time, an acute and a chronic type of caries can be distinguished despite the chronic character of the process. The disintegration of the enamel and cavity formation in children develops rapidly; it is caries rapida (florid, or rampant caries). The lesion is white coloured (caries alba), it is filled with crumbly pasty mass (caries humida). At older age, when the dentinal tubules are narrower due to calcification, the process advances more slowly (caries tarda). The lesion itself is drier and harder (caries sica), and the area has a dark brown or black discolouration (caries nigra). Stationaer or chronic caries means a carious process, which does not show further progress after the cessation of the cariogen attacks (insistens caries, arrested caries). During remineralisation, the enamel surface gets dark brown and black due to exogenous discolouration.
The acid solubility of the enamel will be reduced, its surface will be more acid resistant compared to the original condition if remineralisation occurs in the presence of fluoride. This situation can be seen, for example in approximal caries after the extraction of the adjacent tooth. Latent or hidden caries is difficult to diagnose. In this case the carious lesion involves the dentine as well, and the process extends into the depth although the enamel surface has been remineralised due to the fluoride content of oral hygiene products. In the case of a cavity, which is to be found under an extensive occlusal caries, the intact enamel does not have enough support and may break due to chewing forces. The softened carious dentine becomes exposed, and in the course of time it can be worn off by intensive chewing. Due to the increasing tubular obliteration of the dentine, a smooth, hard surface is formed (dentinal sclerosis).

**Caries Diagnosis**

In the earlier history of dentistry caries diagnosis was the disclosure of a carious cavity, and the only therapeutic option was filling. Nowadays we know a lot about the complex pathological process, about the extent of caries risk, so we can determine the appropriate treatment planning and methods of prevention as well. Caries diagnostics is part of the patient’s medical examination. In the case of dental caries, symptoms may include sensitivity to sweet, cold, interproximally inserted remains of food, gingival papilla bleeding, discolouration, cavity formation, damaged aesthetics, bad taste or unpleasant odour. For the oral examination and treatment the supine position is used. The dried tooth surface has to be investigated under appropriate illumination by inspection and palpation with a dental probe. The sensitivity of the tooth has to be tested with thermal or electrical stimuli. Do not forget to take an X-ray if it is needed.


**Pathological changes in the dental pulp**

Pulp diseases can be caused by the following aetiological factors:

- microbial irritants / infections most commonly due to decay (Figure 2.40.),
- mechanical / physical irritants, for example damage due to preparation without coolant,
- chemical irritants, for example due to cavity cleaning with alcohol.

Extensive caries refers to bacterial irritation of the pulp.
Injury of the pulp by these irritants may cause inflammation. There is no possibility for dilatation of the inflamed tissue encased in rigid walls in the cavity system of the tooth; therefore, significant pain can develop. From a clinical point of view, it is important to know whether the health of the pulp can be completely restored or not. The exact condition of the dental pulp and the seriousness of physiological and pathological lesions can be determined only by histological examination.

The vitality of the pulp means that pulp is a living tissue, which can be proved by the vitality test. The vitality test should be called correctly sensibility or sensitivity test, because it results in information about the condition of the pulp and not only about its living or necrotised character. The most common sensitivity test is carried out by a small, hard cotton pellet soaked with a drop of ethyl chloride. First, an isolated healthy tooth should be examined at the tooth neck as reference, and after that the questionable tooth to compare the results. If the tooth responds to cold stimulus, the pulp is living. In addition to the cold stimulus by heat and electrical stimulation, or by drilling test-cavities, it is possible to collect some information about the status of the pulp.

The clinical experience does not correspond unambiguously to the histological condition. The task of the dentist is an effective therapy. Treatment planning is related to the diagnosis. The diagnosis is based on the patient’s complaints, symptoms, and the results of the clinical investigations. The diseases of the pulp can be classified as follows:

- Reversible pulpitis
- Irreversible pulpitis
- Hyperplastic pulpitis (pulp polyp)
- Pulp necrosis
- Pulp calcification
- Internal resorption.

In case of **reversible pulpitis**, the inflammatory process is reversible, and the circulation of the pulp can become settled after the interruption of damaging stimulus (caries). If the stimulus is answered by the development of localised, sharp but only transient pain, the pulp is vital and sensible. There is no percussion tenderness, and tooth decay can be seen in the X-ray without any periapical lesion. The proper treatment is the removal of the carious tissue and restoration.

In the case of **irreversible pulpitis**, the pulp suffers permanent, irreversible damage. In the closed pulp chamber, intense sharp or dull pain can be generated by stimuli (e.g. cold stimulus) or even spontaneously, continuing for minutes or even for hours. The localisation of the pain is often very difficult because of its diffuse character. If the pulp chamber communicates with the oral cavity, the process is asymptomatic or only mild pain is reported.

The pulp is in vital condition, and as the inflammation is limited to the pulp, there is no percussion tenderness. There is no periapical lesion in the radiogram, but caries or deep filling can be seen. While the integrity of the pulp is impossible to recover, the correct therapy is root canal treatment or tooth extraction. The root canal filled tooth should be checked by X-ray after half a year, and then annually for 4 years. By systematic check-ups, we can intervene in due time in case of the development of apical lesions to avoid the possibility of focal infection.

In **hyperplastic pulpitis** (pulp polyps), the damaged pulp tissue of deciduous molars with good blood flow and circulation, and the first permanent molars in development increases and occupies the carious cavity. There is chronic inflammation, the tissue bleeds immediately but the inflammatory exudates can leave, thus, the asymptomatic disease, which might even be long-term, is discovered accidentally. Rarely signs of irreversible pulpitis with remaining pain can be found. The pulp is tender, inflammation is limited to the pulp, no percussion tenderness or a periapical lesion can be found, but caries can be visualised in the X-ray. While it is impossible to maintain the health of the pulp, root canal treatment or the extraction of the tooth is the correct therapy. In the case of permanent immature molars, pulpotomy can be performed.

Collapse of the circulation originated from inflammation, or damage of blood vessels supplying the pulp due to trauma (accident, aggressive orthodontics) results in **pulp necrosis**. The case can be characterised by sensitivity to heat, and later discoloration of the tooth, but pulp necrosis is poor in symptoms. Regarding multi-rooted teeth, a diagnostic mistake can be made if a canal has necrotised pulp, but another one contains vital tissue.
Neither percussion tenderness is detected, nor a periapical lesion is seen in the X-ray, the adequate alternatives for the therapy are endodontic treatment or the tooth should be removed.

**Pulp calcification** may develop with ageing, or due to long lasting low grade irritation in the pulp. The fibrous elements are accumulated, the number of cell elements is reduced, the circulation is low, and the chance of healing in pulp damage is significantly lower. In case of extensive diffuse calcification, the crown of the tooth has yellowish discolouration. Pulp stone or denticulus may also develop freely in the pulp, respectively fixed to the dentin wall. These are asymptomatic conditions. The reaction after a stimulus is decreased, however, the pulp is vital. There is neither percussion tenderness detected, nor a periapical lesion found, and sometimes pulp stone can be recognised in the X-ray picture. Irritants should be removed, and no other treatment is needed. If the root canal of the tooth should be treated for other reasons, calcification and pulp stone can make it complicated.

In case of **internal resorption**, the pulp tissue is transformed into granulation tissue, and because of the dentinoclast activity, the hard tissue of the root canal will be resorbed advancing from the centre to the periphery. The pulp tissue is vital. The highly vascularised pulp involving the pulp chamber seems to have a pink spot. The progressive process may be clinically asymptomatic, but discolouration of the tooth, or a round shadow in the X-ray or an unexplainable fracture can help us in setting up the correct diagnosis. As soon as possible, preferably before the root perforation, endodontic treatment should be performed, but it has a dubious outcome in advanced stages.

The pulp is spread without any anatomical limit to the apical periodontium. Therefore, the pulp inflammatory processes through bacteria, respectively toxins can spread quickly into the periapical space across the apex. In contrast to the poor circulation of the pulp, the periapical space is rich in blood and lymphatic vessels. It favours the development of protracted, indolent chronic processes, which are poor in symptoms. An acute periapical process is characterised by its rush evolution, by pain that spreads quickly and swelling, and sometimes it is also associated with general malaise. **Classification of the periapical diseases** is the following:

- Acute apical periodontitis,
- Chronic apical periodontitis,
- Condensing osteitis,
- Acute apical abscess,
- Chronic apical abscess.

In the case of **acute apical periodontitis**, pulpal inflammation spreads into the periodontal space and causes intense pain during biting, but it also happens if the tooth is only touched or pressed. The tooth seems to be elongated because of the production of inflammatory exudates, and that is why the occlusion is painful. The pulp is non-vital, but because of the expansion of gases in the root canals, heat sensitivity can be possible. Similar symptoms may be caused by instruments, irrigants and root canal filling materials, and also due to a flare-up of existing chronic apical periodontitis. Periapical radiographic examination shows thickened periodontal ligament space or a frank periapical lesion according to the chronic inflammation, and coronally caries, deep filling, crowns, or another restoration. The process may be accompanied by swollen lymph nodes, shivering or fever. Root canal treatment or tooth extraction can be the appropriate therapy. After obturation of the root, the tooth is to be controlled regularly. An acute abscess or a chronic lesion can develop without appropriate treatment, depending on the intensity of the infection and the resistance of the organism.

Depending on the type and number of pathogenic bacteria, the infected root canal flora causes smaller or larger periapical lesions, which refer to the development of **chronic apical periodontitis**. Balance is established between the organism and the infection. This condition is poor in symptoms, the tooth is often discoloured, does not respond to cold, but may respond to heat stimulus, and the patient may describe a slight discomfort feeling. Percussion causes some pain, the X-ray shows either thickened periodontal ligament space or a significantly large radiolucency (Figure 2.41.).
Chronic apical periodontitis with significant periapical radiolucency at 46 tooth before treatment and one year after healing

The periapical lesion is a granuloma or a cyst lined by epithelium. The correct differential diagnosis is possible based only on histological findings. Treatment of the disease can be root canal treatment or extraction. In the case of a cyst, after root canal treatment, additional apical surgery has to be done and regular control has to be performed.

**Condensing osteitis** presents with a characteristic radiopaque lesion which may develop either from irreversible pulpitis or from pulp necrosis. Inflammation results in enhanced osteoblast activity, and therefore, bone formation. Because of the necrosis, the calcification of the periapical lesion will cause a similar circular radiopaque phenomenon. Symptoms develop according to the pathological origin with painful or painless tooth, positive or negative response to thermal stimuli. Its therapy is the endodontic treatment (or extraction) with regular control after the obturation.

**Acute apical abscess** is accompanied by a severe general condition due to bacterial infection. It can develop directly from acute apical periodontitis, but more frequently is it produced by a flare-up of the chronic apical periodontitis as it is confirmed by the X-ray findings. The patient presents with malaise, has fever and a characteristic swelling on the face (Figure 2.42.). The necrotised tooth will be raised, it is loose and painful during biting or even if it is touched.
Acute apical abscess of the 26 tooth with swelling of the face

Inflammatory exudates can be cleared and solved by tooth extraction, but in order to save the tooth, root canal treatment should be performed. If exudates are not excreted via root canal, an incision should be performed and/or antibiotics may be administered, and daily inspection is needed. In case of deterioration in the condition, institutionalised oral surgery care is needed.

In chronic apical abscess or suppurative apical periodontitis a fistula develops more frequently after the occurrence of chronic apical periodontitis, which has formed an abscess. There is pressure and pain until the inflammation product, the pus, breaks to the surface (Figure 4), and leaves through the fistula. When the fistula can be localised by inserting a gutta percha point before taking an X-ray, and the problematic tooth can be identified. The evidence of a successful root canal treatment is that the fistula is closed.
6.15. Periodontal diseases (Péter Vályi DMD - László Párkányi DMD)

6.15.1. Aetiology of periodontal diseases

Periodontal diseases are mainly inflammatory diseases. If only the gums are affected, it can be reversible, but if it proceeds to the supporting structures, the process becomes irreversible (in part). The aetiology is diverse, but the main reason of cause is bacterial infection. The following figure (Fig. 2.44.) summarizes aetiological factors of periodontitis:

As far as we know today, excessive tissue destruction is not the direct effect of bacteria, but the destructive effect of proteolytic enzymes released as a course of immune reaction. Different alterations in the immune system can be spotted either in defects of the neutrophil defence line, which is reversible, or by infection related, excessively released inflammatory cytokines induced over reaction processes. Both situations result in tissue destruction and pocket forming, which provides favourable circumstances for sub-gingival Gram negative anaerobic bacteria. These lead to further progression, which was well demonstrated by the Offenbacher (1996) model on the pathogenesis of periodontitis:
Pathogenesis of periodontitis (modified from Offenbacher 1996)

The processes above are modified by genetic and non-genetic factors as well, as we described earlier. The complex process is demonstrated in the image below:

Current model of periodontal disease (modified from Page and Kornman 1996)

In the following, we will examine the role of each factor in the process.
Direct damaging effect of bacteria

Destructive processes of bacteria are the result of the release of proteolytic enzymes and harmful metabolic products. Damage affects the tissues (connective tissue fibres, fibrin, fibronectin), the components of the immune system (immunoglobulins, complement system), and structural proteins of soft tissues. Endotoxins of Gram-negative bacteria disturb blood clotting, damages the bone and complement system.

Indirect damaging mechanisms of bacteria

Bacterial toxins can force cells to produce cytokines directly. However, their endotoxins (lipo-poli-sacharides - LPS), and cell wall antigens (gingipain, fimbrillin, or stress proteins) can initiate pronounced immune reactions. Proteolytic enzymes released by PMN cells cause tissue destruction. This is followed by epithelial in-growth and granulation tissue forming, which is rich in plasma cells at the destructed area. Plasma cells continuously release inflammatory cytokines, which leads to further destruction. Enzyme release of PMN cells can be inhibited by alpha-2-macroglobuline and alpha-1-antitripsine, but a bacterial antigen of Porphyromonas gingivalis called gingipain can even destroy enzyme inhibitor proteins.

Role of cytokines in periodontal destruction

Cytokines are small molecule weight glycoproteins. Their role is information flow among cells, immune response, cell differentiation, and regulation of growth. A mediator released by a specific cell can affect neighbouring cells (paracrine) or even itself (autocrine). Cytokines can be either proinflammatory, anabolic cytokines or growth factors. The same cell is capable to produce both, depending on the triggering effect. Specific receptors are responsible for their effect.

Inflammatory cytokines express adhesion molecules, which help PMN cells migrate towards the inflammation, and fixate macrophages and lymphocytes in the connective tissue matrix. Their role is to initiate the immune response. They also play a major role in connective tissue and bone catabolism. Bone resorption is activated directly and indirectly: PGE2 production, macrophage transformation into osteoclasts, stimulating collagenase MMP enzyme production. They also inhibit new bone formation. Further cellular effects are the stimulation of T-cell and macrophage response, increasing the number of circulating PMN cells, promoting their chemotaxis, and stimulating MMP production.

Anabolic (anti-inflammatory) cytokines basically unite innate and adaptive immune subsystems by modulating T-helper cells. They reduce macrophage activity, and their production of cytokines. Anabolic cytokines also block phagocytosis and intracellular destructive processes, and antigen presentation. Through T-helper cells, they can inhibit inflammatory cytokine production, cytotoxic T-cells, and chemotactic function of the rest of T-helper cells. However, they promote activated B-cell growth and differentiation.

Growth factors play a role in the healing processes, attract differentiating cells (fibroblasts, mesenchymal cells, osteoprogenitor cells), promote differentiation, increase collagen, glucose-amino-glycane and other growth factors production and secretion. If these reparative procedures overweigh destructive procedures, regeneration (tissues as original) or reparation (tissues replacing original) takes place.

Products of the arachidonic acid cascade

As a result of insults damaging the membrane, phospholipids of the membrane are transformed into arachidonic acid by phospholipase-A2 enzyme. This metabolises further into leukotrienes (lipoxigenase), prostaglandines and tromboxanes (cyclooxygenase/COX). Enzyme COX-1, is responsible for protective measures (protection of mucosa, blood clotting). The other COX enzyme (COX-2) is responsible for pain sensation by releasing prostaglandins, and for fever, by affecting the heat control centre of the hypothalamus.

Prostaglandins induce oedema by vasodilatation, potentiate the effects of cytokines, stimulate MMP production, cause bone resorption in a direct and indirect way, and reduce the production of antibodies. Patientsh a hyper-reactive monocyte genotype are prone to extensive prostaglandin release, even under low bacterial insult.

Role of cellular elements

The main role of PMN cells is to eliminate opsonized material by phagocytosis. In order to achieve this, it has to react adequately to chemotactic stimuli, move through blood vessels, exit the blood stream, and destroy the opsonized foreign material by phagocytosis at the location of immune response. Any functional disorder can
lead to severe periodontal destruction. Hyper-reactive PMN genotype comes with increased release of proteolytic enzymes, which ends up in severe tissue destruction.

More types of macrophages take part in the defence mechanisms of the periodontium. Some examples are the myeloid originated Langerhans-cells, and lymphoid originated natural killer (NK) cells. They play an important role in adaptive immune response. They use phagocytosis and proteolytic enzymes for antigen elimination. Besides bacteria, they eliminate decomposed cells, too. They bind to immunoglobulins and complement proteins with surface receptors. They also produce inflammatory anabolic cytokines, prostaglandins, and leukotrienes.

**Periodontal bone loss**

A continuous remodelling takes place in bones: building and resorption are in balance. This makes it adaptive to altering loading forces (occlusial, orthodontic forces). The main cells in this process are osteoblasts and osteoclasts. Activated osteoclasts induce reapprortion, which stimulates osteoblasts to rebuild bone. Systemic (Vitamin-D, parathormone), and local hormonal factors modify these processes. In a paradox way, osteoblasts are also responsible for bone resorption by producing RANKL (receptor activator ligand), which activate osteoclasts. This process is inhibited by osteoprotegerin (binding to RANK receptor, taking the place of RANKL), which synthesises in both osteoblasts and gingival fibroblasts. The RANKL-OPG connection is regulated indirectly through cytokines. This determines whether resorptive processes dominate, or there is a stable balance. Inflammatory cytokines promote RANKL production.

Several locally produced paracrine growth factors influence bone formation, matrix formation, and mineralising procedures. These factors are PDGF, IGF, TGF-beta, FGF, BMP, and EMD.

**RISK FACTORS IN THE AETIOLOGY OF PERIODONTITIS**

**Role of plaque microorganisms in periodontal diseases**

In Chapter 2.12., the role of plaque bacteria has been mentioned in the aetiopathogenesis of periodontal diseases. From oral cultivated bacteria, 12 species have shown connection with periodontal diseases. Bacteria are considered to be periodonto-pathogenic, if they fulfil the Koch-postulates modified by Socransky:

- Connection – has a greater probability of existence in periodontal patients
- Eliminating – eliminating the bacteria causes remission
- Host response – provokes immune response, serum and salivary antibodies are increased in number
- Virulence factor – production and facilitation of enzyme production, which make changes in the function of immune system elements
- Animal study model – proven disease inducing effect, bacteria can be found in case of experimentally induced destruction.

The figure below describes periodonto-pathogenic bacteria and their relationship with aetiopathogenesis of periodontitis (Fig. 2.47.):
Causal relation between an infectious agents and disease (modified from Wolf HE)

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Association</th>
<th>Elimination</th>
<th>Host response</th>
<th>Virulence factor</th>
<th>Animal studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aggregatibacter actinomycetemcomitans (Aa)</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Porphyromonas gingivalis (Pg)</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Prevotella intermedia (Pi)</td>
<td>+++</td>
<td>++</td>
<td>++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Fusobacterium nucleatum (Fn)</td>
<td>+++</td>
<td>+</td>
<td>+++</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>Tannerella forsythensis (Tf)</td>
<td>+++</td>
<td>++</td>
<td>+</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Campylobacter rectus (Cr)</td>
<td>+++</td>
<td>++</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eikenella corrodes (Ec)</td>
<td>+++</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peptostreptococcus micros (Pm)</td>
<td>+++</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Selenomonas sp (Ss)</td>
<td>+++</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eubacterium sp</td>
<td>++</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spirochetes</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+</td>
</tr>
</tbody>
</table>

Besides the mentioned Streptococcus milleri can be found in a greater amount during the progression of the disease. Pink coded ones resemble close relationship, while yellow code stands for a more distant relationship with the disease.

Another group of bacteria can also influence periodontal diseases. These bacteria are not part of the natural oral flora and sub-gingival plaque (enteric bacteria, staphylococcus), but can be the initiators or contributors of periodontal diseases. They can be found in those who did not receive or did not react well to treatment (Klebsiella pneumoniae, Klebsiella oxytoca, Enterobacter agglomerans, and staphylococci).

We are deliberately speaking about microorganisms as the causes of disease. Recently viruses have been found to influence periodontitis. Viruses (Herpes simplex, Epstein-Barr, Human Cytomegalovirus and Papilloma virus) can alter the host response to sub-gingival plaque bacteria. They can be found more frequently in the periodontally compromised than in healthy patients.

The elements of sub-gingival flora are not randomly distributed, they are well organised spatially. Socransky and co-workers examined more than 13000 plaque samples to determine the spatial orientation of bacteria in the sub-gingival plaque. They were divided into colour coded groups named complexes.
Microbial complexes in subgingival plaque (Socransky 1992)

Members of the blue, yellow, green, and purple complex are called early colonising bacteria. Gram-negative bacteria of the orange and red complex can bind to them.

Pathogenic effect of bacteria is influenced by the effectiveness of oral hygiene, plaque retentive factors, smoking, hormonal changes, and diet.

**Genetic factors**

Monogenically transmitted, autosomal dominant and recessive illnesses, which come with PMN function disorder, or collagen metabolism disturbances, can be the cause of rapidly progressing, severely destructive periodontitis.

In case of aggressive periodontitis, no systemic background can be revealed, and the family history is positive for the disease. Genetic polymorphism is a common sequential alteration in the genome structure. It can be the result of the transition of a base or insertion, deletion. Periodontitis is a multifactorial disease; therefore, several genes play a role in its development, both environmental and behavioural factors. These gene alterations are not always manifested, only a connection between the disease and the gene alteration can be discovered. Cytokines (IL-1, IL-6, TNF), HLA-antigens, Fc-receptors and structural protein coding genes can be made responsible for the altered immune reactions. Genetics can explain the differences in likeliness of periodontal diseases among the various races.

**Hormonal factors, smoking, systemic diseases influencing periodontitis have been discussed in Chapter 5.13.**

**Other risk factors**

The beginning of this chapter, the immune status of the body has already been discussed. In case of occlusal overloading, it has to be mentioned that it cannot cause attachment loss by itself, but it can change the resistance to plaque in the periodontium, and increase in damage and tissue destruction of the inflammatory process.

Regarding nutrition, some nutrition deficiencies can only influence plaque induced illnesses: protein, Vitamin-D, calcium, Vitamin-C withdrawal can be mentioned as risk factors.

**Psychological conditions**: Indirectly, depression can cause negligence of oral hygiene, and antidepressant drugs can induce mouth dryness. Cytokines isolated from the saliva proved the influencing effect of negative stress in the pathogenesis of periodontitis.
In poor **social status** population, several risk factors are combined (environmental and behavioural), therefore being influencing factors of periodontal diseases. Low degree of education comes usually with poor oral hygiene; however, high degree education does not necessarily come with appropriate oral care.

### 6.15.2. Classification of periodontal diseases

Until the 1920s, periodontal diseases were classified by clinical signs and symptoms. Later, till 1977, the classification was based on classical pathological point of view. In this aspect, periodontal diseases were classified as inflammatory diseases (gingivitis, periodontitis), degenerative processes with severe hard tissue loss (parodontosis), atrophy, hypertrophy, and periodontal trauma.

The rationale behind modern classification is the aetiopathogenesis of the disease. Miller, W.D. suspected irritating factors, host predisposition, and mixed infection in the background of periodontal diseases, but his theory was not approved at his time, in 1880. In the mid-1960s, a study on experimental gingivitis (Löe, H., Theilade, E., and Jensen, S.B), in the mid-1970s the infection-host response principle, and the discovery of leukocyte dysfunction (Page, R.C., Schroeder, H.E) led to the classification of our days. Classification today considers the role of immune status, immune responses, and even behavioural factors linked to the disease. Classification in the 1980s recognized the following groups of diseases:

<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Gingivitis</td>
<td>I. Adult periodontitis</td>
</tr>
<tr>
<td>2. Adult periodontitis</td>
<td>II. Early-onset Periodontitis (EOP)</td>
</tr>
<tr>
<td>3. Juvenile periodontitis</td>
<td>a. Praepubertal Periodontitis (PP) -</td>
</tr>
<tr>
<td></td>
<td>generalized, localized</td>
</tr>
<tr>
<td>4. Systemic disease related periodontitis</td>
<td>b. Juvenile periodontitis (JP) -</td>
</tr>
<tr>
<td></td>
<td>generalized, localized</td>
</tr>
<tr>
<td>5. ANUP</td>
<td>c. Rapidly Progressive Periodontitis (RPP)</td>
</tr>
<tr>
<td>6. Refractory periodontitis</td>
<td>III. Periodontitis associated with systemic</td>
</tr>
<tr>
<td></td>
<td>disease</td>
</tr>
<tr>
<td></td>
<td>IV. ANUP</td>
</tr>
<tr>
<td></td>
<td>V. Refractory periodontitis</td>
</tr>
</tbody>
</table>

Classification of periodontal diseases in the 1980's years

These classifications did not cover all pathological conditions, but the main issue was the classification of periodontitis. On the one hand, classification according to the patient’s age is not very fortunate because both typical cases of periodontitis can develop in any age, even if most cases are covered by the above mentioned criteria. On the other hand, it is hard to determine the development of the disease because many patients only come in contact with the specialist years after developing the disease.

The latest classification from 1999 tried to fix the problems mentioned above and classified diseases as follows:

- restricted to gums or affecting periodontal apparatus
- relationship with plaque or not
- separate groups:
  1. abscesses
  2. endodontic lesions
  3. developmental and acquired diseases
- can local factors give an explanation for it?
  1. chemical, physical, thermal insults
2. occlusal trauma
3. developmental or acquired defects, conditions
4. endodontic lesions
5. allergic factors

• in relationship with systemic factors?
  1. endocrine factors internal diseases (endocrine, haematological)
  2. dermal and mucosal deformities
  3. genetic factors, syndromes
  4. infectious diseases by specific germs

New classification, 8 classes:

| I. | Gingival diseases |
| II. | Chronic periodontitis |
| III. | Aggressive periodontitis |
| IV. | Periodontitis as a Manifestation of Systemic Diseases |
| V. | Necrotizing Periodontal Diseases |
| VI. | Abscesses of the Periodontium |
| VII. | Periodontitis Associated With Endodontic Lesions |
| VIII. | Developmental or Acquired Deformities and Conditions |

Main groups of classification of periodontal diseases, AAP 1999

Diseases of the gums, further classified:

| Dental plaque induced gingival diseases |
| Gingivitis associated with dental plaque only, with, or without local irritative factors |
| Gingival diseases modified by systemic factors |
| endocrine (female sexual hormones, diabetes mellitus) associated with blood dyscrasias (leukemia, other) |
| Gingival diseases modified by medications |
| drug influenced gingival enlargements |
| drug influenced gingivitis |
| Gingival diseases modified by malnutrition |

Non-plaque induced gingival lesions

Gingival diseases of specific origin

1. Bacterial (N. gonorrhoea, Treponema pallidum, Streptococcus)
2. Viral (primary gingivostomatitis herpetic, secunder, recidiváló gingivostomatitis h., Herpes zoster)
3. Fungal (candida alb., lineáris gingivalis erythema, histoplasmosis, other)

Gingival lesions of genetic origin (hereditary fibromatosis gingivae, other)

Gingival manifestations of systemic conditions

Mucocutaneous disorders (lichen oris pemphigoid, pemphigus vulgaris, erythema multiforme, lupus erythematosus)

Allergic reactions (dental materials, oral hygienic material, other)

Traumatic lesions (physical, chemical, heat)

Foreign body reactions

Other disorders

Diseases of gingiva
This is followed by the two main types of periodontitis: chronic and aggressive forms. Periodontitis with systemic background also stands for several diseases:

<table>
<thead>
<tr>
<th>Periodontitis as a Manifestation of Systemic Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Associated with hematological disorders</td>
</tr>
<tr>
<td>1. Acquired neutropenia</td>
</tr>
<tr>
<td>2. Leukemias</td>
</tr>
<tr>
<td>3. Other</td>
</tr>
<tr>
<td>B. Associated with genetic disorders</td>
</tr>
<tr>
<td>1. Familial and cyclic neutropenia</td>
</tr>
<tr>
<td>2. Down syndrome</td>
</tr>
<tr>
<td>3. Leukocyte adhesion deficiency syndromes</td>
</tr>
<tr>
<td>4. Papillon-Lefèvre syndrome</td>
</tr>
<tr>
<td>5. Chediak-Higashi syndrome</td>
</tr>
<tr>
<td>6. Histiocytosis syndromes</td>
</tr>
<tr>
<td>7. Glycogen storage disease</td>
</tr>
<tr>
<td>8. Infantile genetic agranulocytosis</td>
</tr>
<tr>
<td>9. Cohen syndrome</td>
</tr>
<tr>
<td>10. Ehlers-Danlos syndrome (Types IV and VIII)</td>
</tr>
<tr>
<td>11. Hypophosphatasia</td>
</tr>
<tr>
<td>12. Other</td>
</tr>
<tr>
<td>C. Not otherwise specified (NOS)</td>
</tr>
</tbody>
</table>

Periodontitis as a manifestation of systemic diseases

The fifth group involves acute diseases. These are acute necrotising ulcerative gingivitis, or if the bone is affected, acute necrotising ulcerative periodontitis. Abscesses of the periodontium are gingival abscess, periodontal abscess and pericoronal abscess. The seventh main group contains endo-periodontal lesions. The last group also covers several deformities:

<table>
<thead>
<tr>
<th>Developmental and acquired periodontal deformities and conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis</td>
</tr>
<tr>
<td>1. Tooth anatomic factors</td>
</tr>
<tr>
<td>2. Dental restorations/appliances</td>
</tr>
<tr>
<td>3. Root fractures</td>
</tr>
<tr>
<td>4. Cervical root resorption and cemental tears</td>
</tr>
<tr>
<td>B. Mucogingival deformities and conditions around teeth</td>
</tr>
<tr>
<td>1. Gingival/soft tissue recession</td>
</tr>
<tr>
<td>2. Lack of keratinized gingiva</td>
</tr>
<tr>
<td>3. Decreased vestibular depth</td>
</tr>
<tr>
<td>4. Aberrant frenum/muscle position</td>
</tr>
<tr>
<td>5. Gingival excess</td>
</tr>
<tr>
<td>6. Abnormal color</td>
</tr>
<tr>
<td>C. Mucogingival deformities and conditions on edentulous ridges</td>
</tr>
<tr>
<td>1. Vertical and/or horizontal ridge deficiency</td>
</tr>
<tr>
<td>2. Lack of gingiva/keratinized tissue</td>
</tr>
<tr>
<td>3. Gingival/soft tissue enlargement</td>
</tr>
<tr>
<td>4. Aberrant frenum/muscle position</td>
</tr>
<tr>
<td>5. Decreased vestibular depth</td>
</tr>
<tr>
<td>6. Abnormal color</td>
</tr>
<tr>
<td>D. Occlusal trauma</td>
</tr>
<tr>
<td>1. Primary occlusal trauma</td>
</tr>
<tr>
<td>2. Secondary occlusal trauma</td>
</tr>
</tbody>
</table>

Developmental and acquired periodontal lesions

Though the classification above has received many critiques from its first moments, it can be stated that it is evidence based. It means that every disease has been classified based on the aetiology of the disease, which can be a great help in the treatment plan provided the correct diagnosis has been established.

6.15.3. Diseases of the gingiva
Gingival diseases can be divided into two groups based on plaque being an aetiological factor or not. Plaque induced gingival diseases can be further divided, based on plaque being the only reason of diseases, or other factors (systemic diseases, medications, malnutrition) contributing to that.

SOLELY PLAQUE INDUCED GINGIVAL DISEASES

Gingivitis caused by plaque accumulation. There is a correlation between the aetiological factor and the clinical appearance. Symptoms are swollen gingival margins, reddish/purplish discolouration, bleeding (in the beginning to mechanical irritation, in severe cases spontaneously). Changes start at the papilla and proceed to the gingival margin. The orange skin texture of the attached gingiva can be still present. If the inflammation persists, papillae become rounded, the gingival margin and papillae can get fibrotic, pseudopockets can develop. The bacterial flora changes, but no specific strain grows its proportion.

Plaque induced gingivitis - result in gingival enlargement

Gingival inflammation cannot only attack intact periodontium. After periodontal attachment loss or recession, inflammation free soft tissues can get re-infected. This is called reduced periodontium gingivitis. It differs from the re-establishment of periodontitis by not resulting in further attachment loss.

In case of re-established oral hygiene, usually even fibrotic gingival margins can go through complete healing.
Reduced periodontium gingivitis

Solely plaque induced gingivitis, combined with local irritative factors

In this case, local factors stimulate plaque formation, or inhibit adequate oral hygiene. Local factors can be natural or artificial. Artificial factors are restorations with improper marginal closure, micro-mechanical elements of removable partial dentures, or orthodontic appliances. Natural factors are tooth with malformations (enamel pearl, prolonged fissures) or malpositioned, crowded teeth.

A special condition is found in mouth breathers, which restricts to surfaces not covered by the lips while breathing, like incisor buccal surfaces.

Treatment in these situations is the same, with the difference of the need to eliminate local irritative factors.
Patient with gingivitis due to mouth breathing

Gingivitis due to mouth breathing - typical signs

GUM DISEASES WITH SYSTEMIC BACKGROUND
This group gathers gum diseases influenced by endocrine and haematological conditions. Most of the time, endocrine modifying factors restrict to sexual hormones and diabetes mellitus, but parathyroid and adrenal cortex hormones can also influence these diseases.

**Plaque induced gingivitis modified by sexual hormones**

The most common modifying factors are oestrogen and progesterone hormone level changes in women. These changes occur in puberty, during menstruation and pregnancy, and after menopause. It is important to emphasise that these changes do not develop gum diseases, they just amplify the symptoms.

Sexual hormones cannot only affect the mucosa of the uterus, but all other mucosa, including the oral mucosa. By increasing vascular permeability, the tendency for oedema and bleeding increases as well. Through biologic mediators, they can alter immune reactions, and secreting in the sulcular fluid, some specific periodontopathogen strains use them as growth factors, this way changing bacterial composition of the plaque. Aetiologic consequences of sexual hormones will be discussed in detail in Chapter 5.13.2.

**Puberty gingivitis**

During puberty, hormonal changes are dramatic. This explains gingival inflammation on the papillae, even in case of relatively good oral hygiene. The gingival margin can get oedematous, and the tendency for bleeding increases. The consistency of the gums is rather tender, due to accumulated fluids. After settlement of the menstruation cycle, symptoms moderate. Incidence is very high, close to 100%, according to some authors. By re-establishing proper oral hygiene, the inflammation goes through complete remission. Interdental cleaning is of great importance with the help of dental floss, or interdental brushes (Chapter 4.1).

![Puberty gingivitis](image)

**Menstruation gingivitis**

It is most commonly accompanied by the ovulation cycle. In more than 2/3 of women, sulcular fluid increases by 1/5 during ovulation. In rare cases, inflammatory symptoms come with the menstruation cycle, even under non-compromised oral hygiene.

Symptoms are not pronounced, rather restricted to increased tendency to bleed. The condition can be eliminated by good oral hygiene.

**Pregnancy gingivitis**
It was discovered in the late 1800s that during pregnancy, pre-existing minor gingivitis gets more and more severe till the end of the 8th month of gestation. The changes are periodical. In the first trimester, the changes of gonadotrope hormones, at the end of the third trimester, oestrogen and progesterone hormone changes facilitate the symptoms.

Vascular changes are even more pronounced than in other hormone influenced gingivitis. Therefore, gingival swelling is more severe, bleeding tendency is greater, discoloration of the gums leads to vivid reddish/purplish colour. Papillae can even have berry-like surface.

Symptoms become minor after delivery, but will still be present until the end of nursing. Complete remission can only be achieved by re-establishing appropriate oral hygiene, and eliminating all plaque retentive factors.

Pregnancy gingivitis

Vascular changes during pregnancy can get to a level, where localised, or even generalised tumour-like gingival growth can develop. This is called pregnancy granuloma, or pregnancy epulis. Other synonyms in the literature are pyogenic granuloma, granuloma gravidarum, epulis angimatosa, nodular gingival hyperplasia, lobular capillary hemangioma, etc. Surgical intervention during pregnancy is contraindicated due to the high recidivation. After delivery, it can heal even spontaneously, or after non-surgical therapy entirely.
Pregnancy epulis

*Mucosal changes in menopause*

With lowering oestrogen levels, the mucosa becomes thinner, layers of stratum basocellulare and stratum spinocellulare become atrophic. This causes subjective symptoms on the mucosa, sometimes on the gingiva, which are more pronounced. Clinical signs are burning sensation on the mucosa, dry mouth with tingling sensation, taste dysfunction. Symptoms react well to hormonal treatment.

*Diabetes mellitus*

Diabetes mellitus – especially in adults – can enhance symptoms of periodontitis. In type I diabetes mellitus, with an insufficient metabolic control, children can develop severe gingivitis, which cannot be explained by local irritative factors.

*Haematological diseases manifesting on the gums*

All cellular components of the blood are important to maintain gingival health. White blood cells are responsible for both specific and non-specific immune reactions. Red blood cells are responsible for oxygen supply of the tissues, and platelets are responsible for clotting. Most common and most severe diseases are related to leukocytes, but in myelo-proliferative diseases (leukaemia), generally all three types of blood cells become defected in production and function.

*Leukaemia*

As we mentioned before, in leukaemia besides leukocytes, erythrocytes and thrombocytes also suffer disturbances in production. This makes insufficiently oxygenised tissues more vulnerable to infections, which develops more easily due to deficient leukocytes, and tendency for bleeding also increases as a result of thrombocytopenia and coagulopathy.

Typical symptoms: gingival swelling, ulcerations (orally), severe bleeding, petechiae on the skin and mucosa. Symptoms resemble gingivitis ulcerosa.

For differential diagnosis, localisation and spreading of the ulceration, existence of an inflammatory margin can be helpful.
1. Localisation of the ulceration: gingivitis ulcerosa affects the front teeth and wisdom teeth, ulcerations can be found on the buccal aspect. In leukaemia, ulcerations can be found at every tooth, more pronounced orally.

2. Spreading of the ulcerations: Leukaemia related ulcerations spread into deeper layers, while gingivitis ulcerosa stays superficial in the tissues.

3. In gingivitis ulcerosa, ulcerations are surrounded by an inflammatory margin, but in leukaemia, this margin cannot be found.

Chronic myeloid leukaemia (from Prof. I. Gorzó)

Chronic myeloid leukaemia with deep ulceration in the oral side (from Prof. I. Gorzó)
Agranulocytosis

Agranulocytosis is the complete disappearance of neutrophil granulocytes from the blood, while neutropenia is the reduced number of them. Aetiological factors can be the following:

- Bone marrow diseases
  1. aplastic anaemia isolated leukocyte aplasia
  2. congenital - rare
  3. cyclical neutropenia
  4. medications (antibiotics, procainamide, phenytoin, chlorpromazine, methimazol, etc.)
  5. chronic benign

- Peripheral diseases
  1. hypersplenia
  2. sepsis - immune
  3. Felty syndrome

Diseases accompanied by decrease in leukocytes result in severe destructive periodontitis with large ulcerations not only on the gingiva, but on other areas of the mucosa, and even the tonsils.

![Ulcers due to agranulocytosis](image)

The rest of the haematological diseases can be found detailed in Chapter 5.13.13.

**GUM DISEASES RELATED TO MEDICATIONS**
Medications taken for systemic conditions/diseases, can modify gingival inflammation, or in most cases, cause gingival overgrowth. Many of the medications used today, can cause gingival overgrowth. The major uncommon aetiological factor though is dento-gingival plaque. Gingival growth begins with the papillae, but in extreme cases, can cover the whole tooth. The three most common medications causing gingival overgrowth are hydantoine, Cyclosporine A, and Calcium-channel blockers. Uncommon properties among these are immunosuppressive and Calcium-antagonist effects. There are differences in effect mechanisms, but all provoke gingival fibroblast activity, and prevent connective tissue break down (collagenase activity, phagocytosis).

Typical histological changes are present in hyperplasia. The "rete peg", the wave-like run of the oral epithelium becomes long and deep. Acanthosis can be observed in the epithelium. The amount of collagen increases, the run of fibres becomes irregular. Vascularisation increases, new vessels develop. The amount of fibroblasts increase as well, plasma cell, or lymphocyte infiltration is detectable. The relative amount of collagen type III increases, while type I decreases. Other, non-collagen protein proportions also increase. Proteoglycan, hexose-amine, glucose-amino-glycane amounts increase in the connective tissues. The amount of glucocorticotoid receptors and activity of certain cytokines (TGF-β, bFGF) increase. Collagenase activity decreases.

Hydantoin is no more used in epilepsy therapy, therefore, hydantoine induced hyperplasia is rarely observed. Cyclosporine was used following organ transplantations for immunosuppression to prevent organ rejection. It is also used to treat several autoimmune diseases (Crohn-disease, ulcerative colitis). Calcium-channel blockers are the most common drugs, which are used in anti-arrhythmia and anti-ischaemia therapy. In some rare instances, erythromycin (antibiotic) and bleomycin (cytostatic drug) can also lead to gum hyperplasia.

Clinical symptoms are typical: hard consistency, discoloured swollen gums ranging from pink to red colour. Connective tissue fibres, and matrix are both overproduced, and even the epithelium becomes thicker. Sometimes tissue growth is so advanced that only diathermical knife or high power lasers can remove the excess.
Post op. 6 weeks after gingivectomy by electrosurgery

Taking of contraceptive drugs (early generation, high hormone dose) can also cause gingival inflammation accompanied by hyperplasia. Symptoms are really similar to pregnancy gingivitis, since the hormone levels are relatively the same. When taking modern, low hormone dose pills, these side-effects are no longer present.

MALNUTRITION RELATED GINGIVAL DISEASES

Nowadays, severe Vitamin-C malnutrition (scurvy) is rather historical. Malnutrition of Vitamin-C is not the direct cause of the disease, but the disturbance of collagen metabolism, which enlarges the symptoms of plaque related gingivitis. No other vitamin malnutrition results in periodontal symptoms, because these diseases are not present today. Vitamin-B malnutrition can cause typical mucosal and skin lesions (see Chapter 2.6).
Malnutrition of folic acid is related to systemic diseases with gingival symptoms. Protein malnutrition can only lead to ulcerative gingival lesions in developing countries.

**NON-PLAQUE RELATED GINGIVAL DISEASES**

These diseases have no relationship with dento-gingival plaque, they can develop irrespective of the oral hygiene level.

**Gingival diseases of specific origin**

Bacteria, as well as viruses and fungi can lead to specific infections on the gums and oral mucosa.

**Specific bacterial infections** are caused by neisseria gonorrhoea, which resembles the clinical appearance of gingivitis ulcerosa. The TB ulceration (caused by Mycobacterium tuberculosis) has an irregular shape, is not painful, has a red surface, and is covered by a greyish-yellowish membrane. The symptom of syphilis (Treponema pallidum) is a regular, round shaped, painless lesion with a hardened base.

Most common **specific viral infections** are primary and secondary gingivostomatitis herpetica. The disease is discussed in details with acute periodontal diseases. Varicella zoster infection appears more infrequently. Symptoms of primary infection resemble gingivostomatitis herpetica. In secondary infection, reactivating viruses released from the ganglions induce one sided vesicular lesions on the gums and mucosa with a neuralgiform pain. Tearing and joining of the vesicles end up in painful, ulcerative lesions.

**Candida albicans related diseases** were mentioned in Chapter 2.6. Manifestation in gingival lesions only occurs in severe immunodeficient conditions, like AIDS. Xerostomia, smoking, steroid treatment, antibiotic therapy, chemotherapy, and radiotherapy can facilitate pseudo-membranous fungal infections.

**GUM DISEASES WITH GENETIC BACKGROUND**

Hereditary fibromatosis gingivae is an autosomal dominant hereditary deficiency, which manifests in regular overgrowth of the papillae, and marginal gingiva. Its localised form is called tuberal fibrosis.

![Hereditary fibromatosis gingivae](image)

**GINGIVAL DISEASES RELATED TO SYSTEMIC CONDITIONS**

These conditions gather mucosal diseases, which affect the gingiva causing desquamative gingivitis. Diseases together called desquamative gingivitis are characterised by bullous formation, erosions, atrophical spots, which in severe cases, peel off the underlying connective tissue, leaving a greyish-bluish unepithelialised area behind.
They create major subjective complaints, especially in connection with irritative food and drink consumption (spicy foods, carbonated drinks, acidic foods, etc). This is the oral manifestation of Pemphigus vulgaris, Pemphigoid, Lichen planus, Erythema multiforme, Lupus erythematosus.

**TRAUMATIC LESIONS, FOREIGN BODY REACTIONS**

Gingival trauma can be caused by chemical irritation (iatrogenic: chemicals, medications, bleaching agents, acids, bases applied in dental treatment), mechanical irritation (improper oral hygiene, food particles), and thermal irritation (hot foods, drinks, dental instruments).

Foreign body reactions can be caused by dental materials, like piece of amalgam, or metal crown margin, etc.

**OTHER, NON-PLAQUE RELATED GINGIVITIS**

These are mainly tumours of the gingiva, and heavy metal intoxication related gingival lesions (bismuth, lead, mercury)

Melanosis gingivae is a physiological dark spot on the gingiva in some dark ethnical groups, which are the result of high amount of melanocytes in the gingiva. A demonstrative image can be found in Chapter 1.6.

**DIFFERENTIAL DIAGNOSIS OF GINGIVAL OVERGROWTH**

Gingival overgrowth can be due to inflammation related to oedematous swelling, or hyperplasia caused by overproduction of connective tissue fibres and matrix, together with thickening of the epithelium. In chronic infections, besides oedema, connective tissue producing processes can also affect the gingiva.

**Gingival overgrowth**

1. infection related
2. medication related (Ca-antagonist, hydantoine, Cyclosporine-A, Erythromycin, Bleomycin)
3. modified by systemic conditions (pregnancy, puberty, Vitamin-C malnutrition plasma cell infiltration, non-specific pyogenic granuloma)
4. provoked by systemic conditions (leukaemia, granulomatous diseases, sarcoidosis)
5. tumours (malignant, benign)
6. hereditary fibromatosis gingivae

7. idiopathic

**6.15.4. Acute periodontal diseases**

Acute periodontal diseases are usually accompanied by pain, which brings many, previously untreated patients to the dental practice. The most common acute diseases of the periodontal structures are the following:

**NECROTIZING PERIODONTAL DISEASES**

*Acute necrotising ulcerative gingivitis (ANUG)*

During the world wars, it was an endemic disease, also called „trench disease‖. This was the first disease linking a specific pathogen to a periodontal disease. Fortunately today, the prevalence of this disease is really low, due to disposing factors being less and less common.

Disposing factors are categorised as local and systemic ones. Systemic factors are the sympato-mimetic effect of nicotine in smokers, stress, seasonality: the disease had higher occurrence in autumn and winter due to nutritional factors. It is most common among young adults with poor general health (e.g. HIV), or on the ground of malnutrition. Local predisposing factors are poor oral hygiene and the presence of plaque retentive factors. The plaque is dominated by Fusobacterium species. Tar present in cigarettes also facilitates the development of the disease.

The onset of symptoms is typically sudden. It comes with severe pain, necrosis of the papillae: blunt papillae besides the incisors and wisdom teeth, which are covered with a greyish pseudo-membrane. Necrotic ulcerations are present beside them. Ulcerations are typically present on the labial side. Bad breath is also a typical characteristic of the disease. Bleeding starts on mechanical irritation, in later stages even spontaneously. Systemic symptoms are rare, sub-febrility can occur in rare cases. More severe cases involve swelling of the regional lymph nodes.
In adverse cases, contact ulcerations may be present on the pharyngeal mucosa, tongue, lips (Vincent angina), and in the most severe cases, on the buccal tissues, which can lead to perforations (cancrum oris or noma). In severe cases, systemic symptoms can develop. The disease may involve the attached gingiva and alveolar bone (Acute Necrotizing Ulcerative Periodontitis).

It is important to differentiate it from leukaemic ulcerations: this can be done by determining the spreading, location of the ulcerations, and whether the inflammatory margin is present or not (see also Chapter 2.19.!). The differential diagnosis distinguishes the disease from gingival carcinoma and gingivostomatitis herpetica.

**GINGIVAL DISEASES OF SPECIFIC ORIGIN**

*Herpetic gingivostomatitis*

It is a form of gingival inflammation caused by specific pathogens. This is Herpes Simplex virus 1. Primarily it develops in 2–6 year old infants. From 10,000 acute diseases, 1.6 cases are identified as this disease. It is foregone by a prodromal phase: 1 week before the development of the actual disease, an upper-respiratory catarrhal disease precedes it. In parallel with oral symptoms, systemic symptoms develop: weakness, high fever, and painfully swollen lymph nodes. Small coloured blisters develop in the oral cavity with reddish surrounding, which are soaked within 24 hours. By the time patients visit their doctor, round ulcerations are present, covered by yellowish-whitish fibrin pseudo-membrane. The marginal gingiva is scalloped. The ulcerations can later converge. It is accompanied by bad breath.

![Herpetic gingivostomatitis](image-url)
PATHOLOGY

Typical signs of herpetic lesions on marginal gingiva: scalloped formation

Healing is spontaneous in 10–14 days. Immunity does not develop against is, the viruses survive in ganglia, and can be reactivated by external or internal changes. Secondary disease develops in young adults, and the symptoms are similar to that of the primary disease. Later on, labial herpes is more likely to develop.

It has to be differentiated from ANUG and aphtha (more likely on the mucosa), herpes zoster (typically unilateral), perhaps erythema exudativa multiform, based on the typical symptoms.

*Coccal gingivitis*

It is also caused by specific pathogens, but instead of viruses, Streptococcus bacteria are the triggers. It is most likely to develop in immunocompromised patients with poor oral health, typically on the ground of chronic periodontitis.

Besides systemic symptoms (sub-febrilitiy, fever, swelling of regional lymph nodes), it is likely to be accompanied by reddish swollen gingiva with ulcerations on the papillae and marginal gingiva. Pus forms in the pockets, which can be drained by pressure. The teeth and gums are tender and painful.

**PERIODONTAL ABSCESSES**

Based on the classification by AAP in 1990, they form a separate group. They are divided into three main categories: gingival, periodontal, and pericoronal abscesses. The most common form of these is the periodontal abscess, which needs to be distinguished from similar diseases (periapical abscess, infected ateral cyst), to have the opportunity to provide the adequate therapy.

*Gingival abscess*

It develops typically on attached the gingiva due to mechanical trauma (e.g. food remnant, oral hygiene instrument, or braces). After 1–2 days of reddish shiny deformity, spontaneous melding occurs. Pus develops on the border between the connective tissue and the epithelium, or within the connective tissue. Fluctuation can be observed. It opens without treatment spontaneously.

*Periodontal abscess*

It is the third most common acute periodontal disease (8–14% of all cases). Its significance is shown by the fact that it is present in 60% of untreated periodontal patients. Many patients first get in contact with the specialist because of this disease. Periodontal abscess is the purulent inflammation of the periodontal connective tissue. Pathogens need to infiltrate the connective tissue of the periodontium directly or indirectly. This happens if inflammatory products cannot be drained from the pocket: either the virulence of bacteria increases the amount of inflammatory infiltrates under open orifice pockets, or obstruction of the pocket occurs, possibly related to treatment (post-therapy abscess).

Based on the aetiology, we distinguish between periodontal infection related and non-related abscesses. The latter can be caused by a foreign body (food remnant, oral hygiene instrument, or dental material) incorporation into the gingival sulcus, or anatomical or pathological (resorption, perforation, or fracture) alteration of the root.
In connection with periodontal infection: chronic infection exacerbation or post-treatment forms (after scaling, antibiotic therapy, or as a result of surgery) may develop.

Thorough history and clinical examination is required to set up a diagnosis. There is no sign on radiograms, only general signs of a periodontal infection can be visualised. A radiogram with a Guttapercha point leading into the fistula can help us in the differential diagnosis.

The nature of the pain is an important feature of the history, as well as the presence of facial swelling, and information regarding aetiology (previous treatments, incorporation of food remnant, etc.).

The first steps of clinical examination are localisation of the swelling, probing of the tooth, palpation of the tooth, evaluation of sensibility and occlusal discrepancies. Based on the above mentioned symptoms, a periodontal abscess may result in:

- dull pain
- lateral palpation tenderness
- swelling of marginal gingiva, reddish, shiny gingiva above
- no face deformity in most cases
- pocket on probing, pus formation
- teeth are generally intact, sensible
- teeth often become supra-occlusal, resulting in early contacts
- no radiological sign (besides general periodontal infection)
- systemic symptoms are unusual
Periodontal abscess

As differential diagnosis, **pulpal originated subperiostal abscess** is relevant. This comes with vertical palpation tenderness, sharp pain, and often facial deformity. The tooth is not vital. The crown is usually severely decayed or restored in some way. Systemic symptoms can be pronounced (sub-febrility and swollen lymph nodes). The swelling is localised to the root apex, there is no pocket on probing, or if it drains through the pocket, the base is always wider than the orifice. This is the sign of primary endodontal origin endo-periodontal lesions. The radiogram shows signs of a periapical infection.

![Periapical abscess - fistula with opening](image)

A **lateral periodontal cyst** can also show the signs of a periodontal abscess. In case of infection, there is shiny, reddish swelling besides the root, and a fistula may often develop. This disease has its typical radiographic appearance: sharp edged, radiolucent picture, wide periodontal space around the root.
Pericoronal abscess

It develops around partially erupted wisdom teeth, infiltrating the coronal part of the covering gingiva, on the ground of pericoronitis. It comes with pain, swelling of the gums, partial trismus, as it is often accompanied by systemic symptoms (fever and swelling of the regional lymph nodes). The real danger is that the inflammation can spread to the neighbouring connective tissue spaces (spatium), which can lead to even life-threatening conditions.

6.15.5. Periodontal diseases (Vályi P.)

The irreversible, destructive inflammatory disease of the structures supporting the teeth is called periodontitis. The obligatory symptoms are inflammation and destruction of the supporting tissues, which leads to attachment loss and pocket formation. Periodontal diseases also have secondary symptoms, which are not present at every stage of the disease. Such symptoms are: pathologic mobility, furcation involvement, abscess formation, bad breath, changes in position and angle, hypereruption, pus formation, gingival recession, masticatory dysfunction.

CLASSIFICATION OF PERIODONTAL DISEASES

Before the introduction of the classification described in chapter 2.15.2., periodontal diseases were classified according to the age of disease development. Early onset periodontitis is considered to be fast progressing, while adult periodontitis (starting around the age of 35-40), is considered to be of slow progression. The drawback of the classification is that early onset periodontitis can also be of slow progression with its typical appearance, and adult periodontitis may progress fast with its typical clinical appearance. On the other hand, determining the time of disease development is difficult or sometimes impossible even with a precise history.

According to the classification approved by AAP, chronic and aggressive periodontitis are separate disease entities, just like the periodontal manifestation of systemic diseases. Acute ulcerative gingivitis is also classified separately. It can progress to acute ulcerative periodontitis and spread to the supporting structures. If periodontal lesions develop together with endodontic lesions, they are classified as endo-periodontal lesions.

Identifying different forms of periodontitis is not pointless; however, the outcome of the disease is quite similar: destruction of periodontal supporting tissues. When setting up a diagnosis, it is important to identify the
aetiological factors which influence therapeutic methods, as well as the genetic and environmental factors which determine the prognosis.

**Chronic periodontitis (ChP)**

Chronic periodontitis (earlier known as adult periodontitis) is most common among middle aged and elderly people, but it can develop at any age. Typical clinical symptoms are shown in the next image:

<table>
<thead>
<tr>
<th>Main characteristics of chronic periodontitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Changes in the gingival margin – color, texture, size</td>
</tr>
<tr>
<td>(2) Bleeding on probing</td>
</tr>
<tr>
<td>(3) Peri-odontal probe penetrates into the junctional epithelium under low pressure</td>
</tr>
<tr>
<td>(4) Clinical attachment loss</td>
</tr>
<tr>
<td>(5) Gingival recession – <em>in moderate and severe cases, late symptom</em></td>
</tr>
<tr>
<td>(6) Bone resorption – typically horizontal, advanced cases can be vertical</td>
</tr>
<tr>
<td>(7) Furcation involvement – <em>in moderate and severe cases, late symptom</em></td>
</tr>
<tr>
<td>(8) Tooth mobility – <em>in moderate and severe cases, late symptom</em></td>
</tr>
<tr>
<td>(9) Tooth malalignment, inclination, extrusion – <em>fan-like spreading of anterior teeth is a typical sign</em></td>
</tr>
</tbody>
</table>

**Typical symptoms of chronic periodontitis**

During the evaluation of periodontal parameters and clinical symptoms, it can be stated that aetiological factors (plaque, calculus, plaque retentive factors, smoking, stress, systemic factors) are directly proportional to the severity of the disease. A mixed subgingival flora, which varies from tooth to tooth, surface to surface is typical. Subgingival calculus is present in all cases.
Radiological signs of chronic periodontitis

**Progression** is typically slow. While Socransky believed in alternating active and passive stages, others consider it as a continuous destructive process. Recent publications have led to a consensus among researchers: the constant destructive process is interrupted by stages of fast progression and severe destruction. Among patients undergoing treatment, untreated or poorly treated areas (individual or professional oral hygiene) are at risk of further attachment loss.

**Classification** can also be based on severity or location. Mild (1-2 mm), moderate (3-4 mm) and severe (5+ mm) forms are distinguished based on attachment loss. Localised forms are confined to 30% of the dentition at most. If affected tooth surface exceeds 30%, the disease is generalized.

Chronic periodontitis has a high prevalence in developing and developed countries as well. The prevalence of the moderate form (WHO standards, large study sample) among 35-44 year-old patients is 42% (Denmark), 61% (Finland) and 73.6% (Canada). Some countries have extreme low 1.5-21.4% (India, France) or extreme high 91% (Chile) prevalence. The severe form is present in 5-21% of the same age group. Prevalence is higher in high-risk groups (smokers, Afro-Americans, Hispanic population, low social status and education) and elderly people.

Chronic periodontitis is more likely to develop in people with one or more risk factors present: persistent gingivitis (due to poor oral hygiene, constant “bacterial load” of periodonto-pathogenic strains), elderly age, smoking, systemic diseases (diabetes mellitus, PMN disorders, leukaemia, medications associated with gum hyperplasia), stress. Although there is no sound evidence for genetic background, some twin studies support that genetic factors play a role in half of the cases. Research on gene polymorphism suggests that in the chronic form of the disease IL-1 polymorphism (forced cytokine production) is an aetiological factor: patients with IL-1 beta polymorphism are very likely to develop the disease.

**Aggressive periodontitis (AgP)**

Aggressive periodontitis usually develops at a young age, it is typically present in the family history and has a fast progression. Lang et al defined primary and secondary characteristics, which can be found in the figure below:
Aggressive periodontitis is a term including Local Juvenile Periodontitis and General Juvenile Periodontitis, also known as Early Onset Periodontitis (EOP).

It is important to rule out the presence of systemic diseases, because periodontitis as a symptom of certain systemic diseases can lead to similarly fast-progressing, destructive periodontal diseases by compromising the immune system. Typical features are fast progression and vertical bone resorption starting proximally, resulting in intra-osseal periodontal pockets. Due to the fast progression and destructive manner, secondary symptoms (hypermobility, pocket formation, abscess formation, changes in position and angulation, hypereruption) develop quickly. After thorough questioning, the presence of the disease in the family is often revealed.

Secondary signs of AgP: tipping, movement, diastema forming between central incisors, deep bite

The aggressive form is a heterogenic disease, which can be divided into localised and generalised forms. The generalised disease itself has a variable manifestation. Characteristic features of the two forms are shown in the following image:
Characteristics of forms of aggressive periodontitis

<table>
<thead>
<tr>
<th>Localized form</th>
<th>Generalized form</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Develops in puberty</td>
<td>- Develops in early adult age (&lt; 30 years)</td>
</tr>
<tr>
<td>- Affects at least two permanent teeth, one of which is first molars. No more than two teeth other than incisors or first molars</td>
<td>- Affects at least 3 teeth other than incisors and first molars</td>
</tr>
<tr>
<td>- Typical interproximal attachment loss</td>
<td>- Obvious cyclic nature of disease</td>
</tr>
<tr>
<td>- Robust serum antibody response against infectious agents</td>
<td>- Poor serum antibody response against infectious agents</td>
</tr>
</tbody>
</table>

Patient with AgP
Aggressive periodontitis affects about 1-5% of the population. Tinoco and Albandar published an article on racial distribution in 2002:

- Caucasian: 0.1-0.2%
- African, African-American: 1.0-3.0%
- Hispanic, South-American: 0.5-1.0%
- Asian: 0.4-1.0%

The prevalence of the localised form in puberty and youth age is 0.1-0.2% among Caucasian people and 2.6% in the African-American population. Screening is important and increasingly easy to perform as a result of more widespread orthodontic treatment and x-ray facilities. The distance between the CEJ and the marginal bone is considered pathological if it exceeds 2 mm. The following results were published based on these measurements:

- Deciduous teeth showed destruction on one or more surfaces in 2% at the age of 7, in 3.1% at the age of 8 and in 4.5% at the age of 9 (Sjödin, 1994)

- In retrospective studies patients with Juvenile Periodontitis who had more than 2 teeth affected, had detectable radiographic resorption in 52% of the cases already around deciduous teeth. This percentage was 20% in patients who had destruction around one tooth only, and 5% in the control group, who had no radiographic bone loss around permanent teeth (Sjödin, 1993)

- Patients with Juvenile Periodontitis had bony defects around deciduous teeth in 71.4% of the cases and in 85.7% of the cases in mixed dentition according to a retrospective analysis. (Cogen, 1992)

The simple analysis described above can help identify patients at high risk for periodontal infections. This way an individualised oral hygiene program can prevent or at least postpone the development of destructive diseases in these patients.
Specific bacterial flora plays a major role in the destructive, fast-progressing, aggressive form of periodontitis. Host responses are generally compromised, mostly due to genetic abnormalities. Bacteria produce virulence factors and they also have special properties: leukotoxins damage PMN cells and macrophages of the immune system, the production of endotoxins stimulates the production of catabolic cytokines, bacteriocins prevent the growth of normal bacterial flora, immunosuppressive factors inhibit the production of IgG and IgM, collagenase production damages connective tissue fibres and inhibits the chemotactic migration of neutrophils preventing them from reaching the defence line.

Changes in host immune response (local, systemic) play a major role in the development of destructive diseases. A certain degree of PMN function disorder is noticeable, which prevents them from migrating and performing antibacterial functions, but it only manifests in periodontal diseases, not in any other disease. A similar dysfunction is lower mixed lymphocyte response with increased B-cell response, increased pro-inflammatory cytokine production of PMN-s and macrophages. While systemic antibody response is pronounced in the localised form, antibody levels in the blood are relatively low in generalised aggressive periodontitis.

**Periodontitis as a manifestation of systemic diseases**

Several (rare) systemic diseases show symptoms similar to aggressive periodontitis. These are mostly parts of syndromes. The table below gives a list of these diseases:
Periodontitis as a manifestation of systemic diseases

The necrotising form was mentioned in the previous chapter, the form linked to endodontic disease will be mentioned in the following chapter.

6.15.6. Non-bacterial lesions of the periodontal tissues and peri-implant lesions (Vályi P.)

PERIODONTITIS ASSOCIATED WITH ENDODONTIC LESIONS

Aetiology

Since periodontal tissues are connected to the pulpal tissues through the apical foramen, a pulpal disease can influence the periodontium and vice versa: periodontal diseases can affect the health of the pulp. Endo-periodontal lesions can develop in different ways. Lateral root canals, which can be found in the apical and middle third of the root, are on average 4-250 micrometres in diameter. Most of the time they have capillaries inside them. Through these, periodontal vessels can carry nutrients into the pulp chamber. Pulpal and periodontal processes can communicate through these connections. The other form of connection is the dentine tubules. Excessive root planing is not proven to cause pulp necrosis, but it is a possibility. Iatrogenic harm is much more common: when a false way is created during root canal treatment, it enables irrigating solutions to pass through to the periodontal space, thus creating an infection. Root fracture can be another cause of problems. Root perforation can also occur as a result of internal resorption. Trauma can disrupt the neurovascular connection of the pulp chamber, which can also lead to endo-periodontal lesions, sometimes even without infection.

Inflammation of the vital pulp does not result in periodontal lesions. Most bacteria causing pulpal infections are also periodontal pathogenic bacteria, although the bacterial composition of the necrotic pulp is much less complex than that of periodontal pockets. Necrotic pulp is a good substrate for these Gram negative bacteria.

From the pulp chamber there are two ways for the infection to proceed to the periodontal space: the apical foramen and the lateral root canals. In most cases, it is diagnosed as periapical periodontitis and damages the periodontal tissues around the apex. Infections exiting lateral root canals lead to lateral periodontitis. Infections exiting at the furcation level imitate the clinical picture of true furcation lesions.

There are three ways in which the infection can proceed: through the periodontal space and bone, under the periosteum reaching the marginal periodontium and passing through both the bone and the periosteum (and mucosa) into the vestibule through a fistula.

The degree of damage depends on the amount of bacteria, their virulence factor and host defense system. Inflammation can go two ways, depending on host defense reactions. Acute periapical inflammation (purulent,
serous) will develop, if host defense is insufficient. If the immune system functions properly, granulation tissue containing inflammatory cells can embed the inflammation, which can be present asymptotically for years. If the inflamed area is surrounded by a fibrocellular connective tissue cover, bacteria from the pulp chamber rarely get to the surrounding tissues.

When acute inflammation is present, the excessive amount of inflammatory products spreads fast. When reaching the periodontal pocket, it is hard to differentiate from a periodontal abscess.

For differential diagnosis patient records, evaluation of clinical symptoms (location of swelling, configuration of pocket, direction of percussion tenderness, nature of pain, vitality test, degree of tooth destruction) and radiological examination can be of help. Previous chapters discussed the difference between a periodontal and a pulpal abscess. The morphology of the pocket can be helpful when probing: an apically narrowing pocket indicates a periodontal lesion, while an apically widening pocket is a sign of pulpal disease.

Continuous resorptive and rebuilding processes of the bone are separated from the dental hard tissues by periodontal ligaments and cementoblasts. If this barrier is injured and the root cementum gets in direct contact with the active bone cells, a resorptive process begins on the root. It only shows clinical signs when it is accompanied by an infectious or inflammatory process. Minimal surface resorption is present in case of occlusial trauma or smaller injuries on the cementum, caused by orthodontic treatment. The damage caused by osteoclasts in these cases is quickly repaired by cementoblasts. Replacement resorption can take place when periodontal ligaments get injured following tooth trauma or if autografts (containing vital bone cells) are used to fill periodontal pockets. This resorption ends with large resorption lacunas, which cannot be repaired by cementoblasts, therefore, osteoblasts gradually replace the defect by creating bone. This is most commonly recognised on radiographs without any clinical signs.

External inflammatory resorption is caused by granulation tissue developing around the root. This can happen after major trauma, accompanied by pulpal infection and necrosis. This scenario is associated with continuous irritation caused by the infection, as well as replacement resorption. The same process takes place after periodontal therapy, when treated root surfaces are populated by epithelial cells instead of fibroblasts.

**Classification of endo-periodontal lesions**

Several classifications are present, but the most recognised one is the Guldener and Langeland classification:

1. **primary endodontal lesion** The necrotised pulp is causing the periapical or lateral periodontal lesion. The inflammatory products can deflect into the gingival sulcus and to the vestibule through a fistula.

2. **primary periodontal lesion** Periodontal inflammation and consequent attachment loss can lead to the opening of accessory root canals. Pulp can get infected through these canals.

3. **combined endo-periodontal lesion** Pulp necrosis and periodontal lesion caused by persisting severe periodontitis develop at the same time.

**DEVELOPMENTAL AND ACQUIRED PERIODONTAL DEFECTS AND CONDITIONS**

Periodontal conditions or mucogingival deformities which influence the development and progression of periodontal inflammatory diseases, but can also occur independently of dento-gingival plaque.

**Localised periodontal conditions, modified by tooth-related predisposing factors**

**Anatomical factors of the tooth**

Changes in the shape of the tooth can modify interdental spaces and shapes of the papillae or lead to their absence. Interdental spaces might be too narrow (crowded teeth) or too wide (pronounced curvature of clinical crowns). These deformations can be the result of rare anatomical structures, fissures engaging the root or enamel pearls.

**Effects of restorations**

Incorrect marginal closures of crowns, fillings, micro-mechanical retentive structures can irritate the gums. Besides facilitating plaque accumulation, their direct traumatising effect can harm the periodontal tissues.
**Root fracture**

Infections caused by traumatic root injuries

**Cervical root resorption**

Root resorption was mentioned above

**Mucogingival deformities**

**Gingival recession**

In case of alveolar dehiscence or fenestration, the lack of bony support makes the gums more susceptible to infections and mechanical trauma, which may lead to gingival recession.

**Lack of keratinised gingiva**

Periodontal health can be maintained in the lack of keratinised gingiva with good oral hygiene. However, lack of keratinised gingiva may inhibit appropriate oral hygiene, which can lead to inflammation. When performing restorations with crowns, a minimum of 3 mm keratinised gingiva is necessary to preserve periodontal health, especially in thin biotype.

**Shallow vestibule**

Shallow vestibule may prevent proper oral hygiene

**Pronounced frenule**

Pronounced frenule can prohibit proper oral hygiene. Highly attached frenule can damage the marginal gingiva.

**Gingival overgrowth, pseudo-pocket, irregular gingival contour**

These conditions make appropriate individual oral hygiene impossible. They support plaque-induced inflammatory diseases and their progression.

**Gummy smile**

Gummy smile is mostly an aesthetic concern, but gums can be mechanically irritated as a result of mouth breathing.

**Gingival overgrowth, pigment disorders**

These disorders were mentioned in detail at the beginning of this chapter.

**Mucogingival deformities on the edentulous ridge**

The edentulous ridge suffers constant involution following teeth loss. If the tooth was affected by periodontal disease, cyst, surgical trauma or developmental disorder, its loss leads to various tissue deficiencies.

The ridge may lose volume horizontally as well as vertically. Seibert, Allen and Wang classified defects into

- Horizontal (H, Class I, Type A),
- Vertical (V, Class II, Type B) and
- Combined (C, Class III, Type C) defects.

Studer determined the severity of the horizontal defect relative to the dentate arch, and that of the vertical defect based on the papilla height of the adjacent tooth: 0-3 mm: moderate, 3-6 mm: advanced, 6 mm severe bone resorption.

According to the studies of Seibert and Allen, defects are combined in more than half of the cases, horizontal only in one-third of the cases, and isolated vertical defects are extremely rare. In less than 1 of 10 there is no deficiency at all.
Correction of the edentulous ridge is inevitable for both fixed partial dentures and implant restorations.

However, not only tissue deficiency, but also shallow vestibule, gingival overgrowth, pronounced, highly attached frenules can cause problems associated with the edentulous ridge.

**Occlusal trauma**

All adaptive and pathological changes in the periodontium caused by overload of the masticatory function are called occlusal trauma. Unfavourable forces can appear after the adjustment of occlusion, early tooth contact and parafunction. Early contact can be the result of tooth loss, periodontitis, changes in dentition (elongation, tooth malposition or mobility) or poorly designed restorations. In case of primary occlusal trauma, the tooth is periodontally healthy, but is subject to increased loading forces. In secondary occlusal trauma, the force might even be normal, but affected teeth are periodontally compromised.

Occlusal trauma can lead to qualitative and quantitative changes in periodontal tissues, which can regenerate in part or completely if the damaging forces no longer exist. As a result of the pathological process, the periodontal space gets wider, the alveolar bone starts to resorb, vascular changes take place in the ligaments, degenerative changes in the cementum and the amount of collagen decreases in the alveolar bone. The pathological mobility of tooth can be divided into two stages: progressive (increasing) and stabilised (persistent).

Clinical signs of occlusal trauma: high tooth mobility, increasing tooth mobility, tooth wearing, facets on teeth, tooth migration, tooth tilting, failure of restoration, pain. The radiographic image shows extended periodontal space, which is „U”; „V”; or sandglass shaped. Cementiculi can develop within the periodontal space and hypercementosis can occur on the apical portion of the root.

Two types of forces can reach the tooth. One way, or orthodontic type forces are present if resorptive and rebuilding processes induced by orthodontic movement are not evenly distributed, because of over forcing. Jiggling type forces create resorptive forces on both sides of the tooth. Widening of the periodontal space and bone destruction (together with increased mobility) will go on until adaptive measures compensate for high forces.

Occlusal trauma is not responsible for periodontal inflammatory diseases by itself, but forces exceeding adaptive capacity of the periodontium may trigger inflammatory processes. Several human studies proved that occlusal trauma contributes to the severity of plaque induced inflammation, and it even influences the success of the therapy in a negative way.

**LESIONS OF PERI-IMPLANT TISSUES**

Implant failures can be classified as early and late. Early failures develop around the implants before osseointegration. Late failures can affect implants in function. Implant loss can be due to overloading (improper size, surface characteristics, bone quality) or infection.

Alberktsson and Izidor applied the following definitions on implants, based on definitions related to natural dentition:

**Peri-implant mucositis**: reversible soft tissue lesion around implants

**Peri-implantitis**: inflammation affecting osseointegrated implants in function, resulting in supporting bone loss

**Incidence**

The longest study on periimplantitis was conducted by Roos and Jansaker (implants placed 9-14 years ago). They found that 16% of study subjects and 6.6% of implants were affected. According to Zitzmann and Berglundhs, who refined the criteria, over 56% of patients and 28% of implants revealed a lesion. Periimplantitis had a 2-10% prevalence in a 5-year period. Peri-implant mucositis was present in more than two-thirds of the patients with implants in function for 9-14 years.

**Risk factors**

Based on available data, peri-implant and periodontal infections have a lot of risk factors in common. Peri-implant diseases are much more likely to occur in patients with a history of periodontitis, smoking, inadequate
oral hygiene or local disturbing factors present. Further risk factors are diabetes mellitus and genetic factors (IL-1 gene polymorphism). Overloading does not cause bone resorption by itself but facilitates inflammation.

**Characteristics of the lesion**

Plaque induced changes in the peri-implant mucosa are similar to gingival inflammation in the first three weeks. Following this, fibroblasts appear in a much higher number in the inflamed gingiva than in the mucosa. Apical and horizontal spread of the lesion is much wider, reparation is much less pronounced. Inflammatory cells are widespread in the mucosa, inflammation is much less contained. (bordered)

A large portion of the soft tissues gets infiltrated. A large number of inflammatory cells (macrophage, plasma cell, lymphocyte) appear in the histological specimens. The biofilm is not separated from the connective tissue by an epithelial barrier. A large number of PMN cells are present in the lesion, even further away from the implant surface, perivascularly. Elastaze-activity and lactoferrin-concentration is higher in the sulcus in periimplantitis compared to the healthy mucosa.

The composition of the biofilm is similar in periodontal and peri-implant lesions.

**Symptoms:**

Bone resorption on the radiographic image. Inflammatory signs (swelling, reddish peri-implant mucosa, bleeding on probing, suppuration) are noticeable in peri-implant soft tissues. Stability of the implant is maintained for a long time.

It has been suspected since the time of Hippocrates that oral diseases can have an effect on systemic diseases. From the late 19th century till the beginning of the 20th century, „oral sepsis” and „focal infection” were well known terms, but pointless and brutal tooth extractions did not fulfil expectations: systemic conditions did not improve in most patients after the treatment. As a result of modern diagnostic procedures and broader immunological knowledge, in the late 20th century studies confirmed that oral infections can cause damage to distant organs and can influence the development of systemic diseases.

DENTAL FOCUS OF INFECTION

Focal infection is a chronic, isolated, symptomless inflammation, from which bacteria or their toxins and antigens spread to distant organs, where they provoke inflammatory and immune reactions, causing secondary diseases. Focal infections can be categorised based on their relation to the oral cavity (open, closed) or by aetiology (periodontal, endodontic). There are major differences between periodontal and endodontic infections in duration, possibility of spreading (bacteraemia-involved surface area) and incidence (bacteraemia can develop from periodontal pockets during daily oral hygiene or even chewing), as well as in number and types of bacteria. Some bacteria spread directly to specific organs (e.g.: T. forsythensis and P. gingivalis prefer the endothelium); this is called tropism. There are also differences in the frequency of clinical symptoms. The spread of bacterial products can be direct or indirect. Classification of focal infections, ways of spreading and target organs are listed in the table below:
The damaging effect of focal infections is also due to the special anatomical conditions: bacteria can get from the oral cavity directly to the heart through veins, and form there directly into the systemic circulation without passing through the liver, which plays a major role in immune responses. Adhesion to Kupffer cells (fixed macrophages) in the liver causes increased adhesion of granulocytes and release of chemotactic substances, cytokines, which activate further components of the immune system. Passing by this mechanism, pathogens can easily get to targeted organs. In the targeted organs they need to attach and reproduce. Previous chapters mentioned the virulence factors of bacteria which can dodge immune responses and secure their survival in targeted tissues.

Focal dental infections trigger destructive procedures not only by the spread of bacteria themselves (metastatic infection) but also by releasing toxins (metastatic damage)). In this case, these toxins can be isolated, and linked to specific bacteria, and their antigens provoke immune response and inflammation in the target organs (metastatic infection). Antigens of these toxins act as super-antigens (toxic shock syndrome), furthermore they can lead to autoimmune diseases by cross-reactions, stimulating the production of autoantibodies.

SECONDARY DISEASES IN TARGET ORGANS

Quite rarely, severe infections can spread from the head and neck region directly between cervical muscles, mediastinum, cranium (cerebral abscess) and sometimes to masticatory muscles. Infection in the sinuses and deeper facial tissues is much more common.

From digestive system diseases, Helicobacter pylori can persist in periodontal pockets maintaining underlying diseases, e.g. duodenal abscess. Periodonto-pathogenic bacteria may be the aetiological factor of autoimmune inflammatory intestinal diseases (P. gingivalis).

Long-term artificial respiration or aspiration among frail elderly patients can lead to respiratory infections, caused by oral pathogens. Relationship between pneumonia, lung abscess, COPD and periodontitis is confirmed by epidemiological studies.

Inflamations originating from the head and neck region can lead to autoimmune diseases of the kidneys through antigen-triggered immune complexes in the target organ. Possible relationship between dental infections and liver abscesses have also been mentioned in some studies.

Ophthalmological (uveitis, endophthalmitis) and dermatological (erythema nodosum, pustulosus palmo-plantaris) diseases have also been linked to dental infections as aetiological factors, with some supporting
evidence. **Joint diseases** have also been related to dental infections, but evidence is lacking to prove this hypothesis. Relationship with **cardiovascular, genital diseases** and **diabetes mellitus** will be detailed in Chapter 5.13.

### 6.17. Benign tumours of the oral and maxillofacial region (Péter Novák MD)

**Papilloma**

Papilloma is a benign epithelial tumour, which frequently appears on the oral mucosa. Local irritation and infection, especially viral infection (human papilloma virus) may play a role in its development.

**Symptoms:** a pedunculated or sessile, white, or whitish-grey, papillary surfaced tissue growth, which does not cause any symptoms. A slowly proliferating, smaller or larger epithelial tumour may also develop. It occurs mostly in the palates, tongue, gingiva and lips.

**Differential diagnosis:** verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

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**Fibroma**

Fibromas are benign, slow-growing tumours of the oral cavity composed of connective tissue. Fibroma is the most common tumour of the oral mucosa, which particularly occurs on the buccal mucosa and on the tongue. In the oral cavity, fibromas are reactive proliferative lesions, which develop secondarily to irritation (by sharp tooth edges, artificial teeth or calculus) or low grade infection. The lesion feels solid or soft on palpation. It is sessile or pedunculated, smooth-surfaced, reddish or pale and covered with intact mucosa, but as a result of mechanical irritation it may become whitish leukoplakia due to keratinisation or may become ulcerated. No other clinical sign or pain can be observed.

**Differential diagnosis:** verruca vulgaris, condyloma acuminatum and verrucous carcinoma.
epulis, pyogenic granuloma, granuloma fissuratum and neurinoma.

Fibroma

**Lipoma**

They are rare, painless and benign tumours in the oral cavity, which arise from the fat tissue and grow slowly. Lesions mostly involve the bucca, the tongue and the floor of the mouth. The growth is mainly single, rounded or oval, mobile, pedunculated, soft, yellowish translucent and painless. The surface is vascularised.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

mucocoele, ranula, dermoid cyst, lymphangioma and adenoma.

**Pleomorphic adenoma**

It is also called a “mixed tumour”. The epithelial tumour mainly originates from the small and large salivary glands, it is hard, springy and lobulated, and grows slowly without causing any pain. Extraorally, it mainly arises from the parotid gland. Small and large (giant and weighty) tumours occur alike. In the oral cavity, in addition to other localisations, the tumour predominantly appears on the hard palate, which occasionally interferes with eating and speaking.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

cystadenocarcinoma, epulis.

**Papillary cystadenoma lymphomatosum (Warthin’s tumour)**

Warthin’s tumour or adenolymphoma is a rare and benign condition, which almost exclusively develops in the parotid glands. It rarely involves the submandibular glands or the intraoral small salivary glands. Usually men are affected.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

lymphoepithelial cyst (branchial cyst) and other benign and malignant tumours.
**Capillary haemangioma**

The benign tumour develops as a result of vascular proliferation. The congenital form is considered to be rather a congenital disorder (malformation) than a real tumour, since it may regress spontaneously. The capillary form is composed of a capillary network, and is soft, small and bluish-red. Manual compression of the lesions expresses blood, leaving behind an empty sac (vitropression), thus temporarily they fade away.

**Cavernous haemangioma**

This type of haemangioma is a hamartoma (not a neoplasm), which histopathologically does not have a capsule. It mostly involves the tongue, lips and the bucca, and reaches an extreme size (macrocheilia, macroglossia), thus hindering speech and swallowing. It occurs as a flat or exophytic, painless growth with a raspberry-like appearance. Its colour ranges from red to dark blue.

**Differential diagnosis:** verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

**Haemangioma**

**TUMOUR-LIKE LESIONS**

**Pyogenic granuloma**

The tumour-like reactive tissue proliferation may develop in response to trauma or infection, but it may also occur as a result of hormonal changes (pregnancy, menopause, or puberty), the administration of oral contraceptives, steroid drugs and anti-diabetics. Most frequently it develops on the gingiva, bucca, lips and tongue. It is a raised, pedunculated, flat or wart-like lesion with a dark red or pink colour depending on the vascularisation of the affected area. In case of ulceration, it is covered with yellowish fibrin. Its diameter ranges from a few millimetres to a centimetre. It is a painless tumour, which easily bleeds.

**Differential diagnosis:** verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

epulis, papilloma, fibroma, haemangioma, granuloma gravidarum and malignant tumours.
Pyogenic granuloma

*Granuloma gravidarum*

After the first trimester of pregnancy, a single or rarely multiple, dark red, ulcerated tumour-like lesion which is prone to bleeding develops mostly on the gingiva. It mostly involves the vestibular gingiva at the level of the upper premolars, but it may also appear in other areas. During pregnancy, it may recur despite its removal, but after delivery it may spontaneously disappear. The clinical and pathological picture is similar to that of pyogenic granuloma.

*Differential diagnosis*: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

pyogenic granuloma, epulis and haemangioma.

*Postextraction granuloma*

This reactive, inflammatory tumour-like lesion develops after tooth extraction as a result of retained root, broken pieces of bones, amalgam or other foreign body.

*Differential diagnosis*: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

epulis and gingival tumour.
Postextraction granuloma

_Epulis granulomatosa_

It is not a real neoplasm, but a reparative tissue proliferation arising from the mucoperiosteum of the tooth socket or from the periodontal membrane. The diagnosis of "epulis" may be applied only clinically. The growth is a painless, red lesion, which is prone to bleeding and occurs mostly on the vestibular surface of the front and premolar teeth. It is mostly brought about by chronic inflammation (tartar, excess filling, or crown).

_Differential diagnosis:_ verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

pyogenic granuloma and granuloma gravidarum.
Epulis granulomatosa

**Fibrosus epulis**

It is the most common type of epulis. It is a light red or yellowish-white, stiff tissue proliferation, which grows slowly.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

**Peripheral giant cell epulis**

It is a soft, bluish-red, often ulcerated growth covered with yellowish, fibrinous pseudomembrane, which may reach 2–3 cm in diameter. The teeth may loosen due to the destruction of the surrounding bones. In rare cases, the tumour becomes malignant. It develops less frequently than the granulomatous or the fibromatous forms.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

**Pyogenic granuloma, granuloma gravidarum, central haemangioma, gingival cancer, malignant melanoma.**

**Pigmented naevus (intradermalis)**

Intradermal naevus is the most common lesion of the skin. It is usually elevated and as a result of fibrous tissue, stiff, brown or brownish-black, in rare cases colourless (amelanotic), occasionally papillary-surfaced or pedunculated develops. Malignant degeneration may result from irritants (collar, bras, or shaving) or insufficient treatment. It appears as a flat or slightly raised, brownish or brownish-black macule on the bucca or lips.
Pigmented naevus

**BENIGN ODONTOGENIC AND NON-ODONTOGENIC TUMOURS**

**Ameloblastoma**

It is benign, locally aggressive and grows invasively. It is four times more frequent in the mandible than in the maxilla. Ameloblastoma may present in all ages, however, regarding the average age it is the most common in the fourth decade. The tumour grows slowly, and in 80% of the cases, it does not cause pain. A tumour in the jaw bone may cause facial deformation of various severity.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

fOLLICULAR Cyst, keratocyst.

**Odontoma**

Odontomas should be considered hamartomas rather than real tumours. This relatively common odontogenic tumour develops in the jaw bones mainly in children and young adults. The tumour has few symptoms and leads to swelling of the jaw bones, sometimes with a diameter of 5–6 centimetres.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

ameloblastoma, fibrous dysplasia.

**Osteoma**

Osteoid osteoma is a rare, benign, non-odontogenic tumor of the jaw bones. It occurs more frequently in women than in men, and it presents more commonly in the mandible than in the maxilla. It is a slowly-growing, usually painless, hard tumour that is bone-like to the touch and causes facial deformity.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

osteosarcoma, maxillary tumour, exostosis.

**Exostosis**
The disease should be differentiated from osteoma, which is a real osteogenic tumour. The cause of exostosis is unknown. It is not a real neoplasm but a reactive lesion.

**Torus palatinus**

It is a developmental malformation of unknown origin, which presents as a slowly growing, bony growth (exostosis) in the middle of the hard palate, covered with intact mucosa. The growth is hard and feels like bone to the touch.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

palatal abscess, nasopalatine cyst.

**Torus mandibularis**

The disease can be manifested on the lingual surface of the mandible, unilaterally or bilaterally, at the site of the premolars, and may reach various sizes. The bony outgrowth is clearly visible in the radiogram.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

osteoma, mandibular cyst.

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**6.18. Precancerous states and lesions (potentially malignant lesions) (Péter Novák MD)**

All morphologically altered tissues that have a greater predisposition to develop cancer than surrounding, apparently normal tissues are considered to be precancerous. The term leukoplakia is merely a clinical designation, which refers to any white lesions of the oral mucosa that cannot be wiped off and cannot be classified as any other well-defined disease.

*Leukoplakia simplex (homogeneous)*
Causes: the underlying cause for leukoplakia is unknown, however, predisposing factors may include genetic factors, inappropriate oral hygiene, vitamin A or C deficiency, smoking, alcohol, trauma, candida albicans, viral infections (HPV), systemic diseases (syphilis, sideropenia, hepatopathy), galvanism and immunological alterations, but it may also occur idiopathically.

The general prevalence of leukoplakia within the population is 0.1–5%. Carcinoma has already developed at the time of detection in 5% of the cases on average. 3–8% of all leukoplakias develop into malignancies, and most of them occur in mostly men at the ages of 40–60 or over. Malignant transformations are more common among women and non-smoking patients with leukoplakia. Leukoplakias on the tongue and the floor of the mouth are more likely to develop into malignancies. Leukoplakias may occur at any location within the oral cavity, but they are the most common in the retrocommissural triangle, bucca, tongue, palate, lips, gingiva and floor of the mouth.

Leukoplakia simplex (homogeneous)

Non-homogenous leukoplakia

Verrucous leukoplakia

These are greyish-white, white and asymptomatic alterations that protrude from the oral mucosa similarly to verrucae and have a wrinkled or corrugated surface and a "cobblestone" display. Chronic inflammations and keratinisation of the oral mucosa is irreversible due to its intensity, and reacts only to the proper clinical therapy.

Differential diagnosis: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

Lichen oris, oral candidiasis, morsicatio buccarum.
Verrucous leukoplakia

Nodular leukoplakia

These alterations occur mostly in the retrocommissures or on the bucca and appear as white nodules on an exophytic, erythematous background. Histologically, results may include epithelial dysplasia or carcinoma in situ or even invasive carcinoma.

Differential diagnosis: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

Hyperplastic candidiasis.

Erythroleukoplakia

Keratinised mucosal tissues may develop ulcers; clinically, the surface of these alterations may become nodular and speckled with red and white patches. The most common location of these alterations is the oral mucosa behind the commissure of the lips. Most of the patients seek medical help because of the increased tenderness during eating caused by the erosions, or less commonly, because of the pain these alterations may cause. Malignancies evolve from erosive areas without epithelium more easily and more frequently than in case of simple or verrucous alterations.

Differential diagnosis: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

Erosive lichen, chronic discoid lupus erythematosus, pemphigus vegetans

Proliferative verrucous leukoplakia

This alteration occurs rarely, effects non-smoking women more commonly, and it is clinically similar to verrucous leukoplakia; however, a major difference is that its surface is not homogenous, it resembles “soaked skin”, while its surface is crinite and deeply fissured and it is visibly multifocal and frequently covers a wide area. It is more common on the gingival mucosa, bucca, tongue and lips. This chalk-white, exophytic alteration is free of any clinical symptoms. This condition has a marked tendency to become malignant.

Differential diagnosis: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.
Lichen oris, verrucous carcinoma.

**Erythroplakia**

Similarly to leukoplakia, erythroplakia is a clinical term, which describes a vivid, velvety, soft, well-circumscribed, flat alteration that is slightly indented or less commonly, slightly elevated from the surrounding tissues, but it does not resemble a plaque. These alterations cause no symptoms for the patient; however, during meals, a slight burning sensation or tenderness may infrequently occur. Erythroplakias develop into malignancies commonly and easily. These rare alterations occur at the ages of 50–70, the most common locations are the soft palate, bucca, tongue and the floor of the mouth. White, hyperkeratotic areas appear in the lateral regions of these alterations. Histology often shows severe epithelial dysplasia, carcinoma in situ and in more than 50% of the cases, squamous cell carcinoma.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

Erosive lichen, atrophic candidiasis.

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**Erythroplakia**

**Chronic actinic cheilitis**

This form of chronic inflammation caused by sunshine (solar cheilitis), wind or agrochemical compounds (sprays) occurs mostly on the lower lip in patients working outdoors (agricultural workers). It is more common among men than among women. The lower lip becomes crusty to some extent, and later, it also becomes swollen and hard due to the accumulation of connective tissue (macrocheilia). It may easily crack, bleed or become painful. Symptoms of this chronic inflammation of the lips become worse during the summer, and they improve in the autumn and winter. After some years, these alterations may evolve into lip cancer, thus, they should be treated as precancerous conditions.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

Multiform exudative erythema, Quincke’s oedema, lip cancer, lupus erythematosus.

**Lichen planus**

**Reticular lichen**
This condition of unknown origin is a result of chronic inflammation, it reoccurs quite commonly and affects the skin and mucosal tissues. Autoimmune causes and psychological factors are more and more in the foreground nowadays as underlying causes of this disease. Diabetes mellitus, hypertension and hepatic lesions may increase the predisposition to this condition. These conditions occur most commonly in the medial and posterior third of the bucca bilaterally, but they may also appear on the tongue, lips, gingiva; however, they are quite rare on the palate. This is a whitish-grey, slightly protruding, pearlescent, reticular and hyperkeratotic alteration, which appears as a branch-like structure (Wickham’s striae). This is the most common clinical type. The alterations themselves cause no pain, and patients usually feel a slightly uneven spot on their oral mucosa which may be firmer than the surrounding mucosa.

Reticular lichen

*Atrophic lichen*

Atrophic, lively red, painful spots appear mostly on the bucca and tongue, which are surrounded by reticular alterations (Wickham’s striae). This condition is hard to differentiate from erythroplakia (the latter is not surrounded by a white contour) and discoid lupus erythematosus (which is characterised by dermal symptoms and distinctive histological and immunofluorescent findings).

*Ulcerative lichen*

In most of the cases, ulcerative alterations are preceded by atrophic lichen. Ulcers lacking epithelium and partially covered by yellowish-white, fibrinous pseudomembranes appear on the mucosa, and these ulcers cause pain spontaneously as well as during meals. Establishing the proper diagnosis is aided by the fact that reticular lichen is observable surrounding the ulcers on both buccal areas. Similarly to atrophic lichen, these alterations have a low risk (1–3%) of developing into malignancies.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma, erythroplakia, oral pemphigus, allergic stomatitis.

*Actinic keratosis*

This is a precancerous condition, which evolves primarily in elderly agricultural workers due to exposure to sunlight; however, it may also accompany immunosuppression, chronic ulcers or human papilloma virus (HPV) infection. According to recent views, solar keratosis is a superficial form of epithelial cancer. These alterations
are mostly flat or slightly protruded, multiple, their surface is rough, hyperkeratotic, red or yellowish-brown (and pigmented). These precancerous alterations occur mostly on the face, forehead, nasal crest, ears, neck and back of the hand. After years or even decades, these keratotic lesions may transform into carcinomas in about 25% of the cases.

**Differential diagnosis:** verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

Basalioma, seborrheic verruca, and dermal cancer

### 6.19. Malignant tumors of the head and neck region (Péter Novák MD-Róbert Paczona MD -József Pifkó MD, DMD)

**INTRODUCTION**

Head and neck cancer describes a range of tumours that arises in the head and neck region, which includes the oral cavity, pharynx, larynx, nasal cavity, paranasal sinuses, thyroid gland, and salivary glands.

**INCIDENCE AND PREVALENCE**

The worldwide incidence of head and neck cancer exceeds half a million cases annually, ranking it as the fifth most common cancer worldwide. Most commonly “head and neck cancer” refers to epithelial squamous cell carcinomas that arise from the mucosal surfaces of the upper aero-digestive tract. However, the impact of this disease on society is not measured solely by its relatively low absolute mortality, but also by the acute and chronic cosmetic, functional, and psychological morbidities experienced by all patients. Head and neck cancer thus remains a feared disease associated with significant rates of both morbidity and mortality.

**RISK FACTORS**

**Alcohol and tobacco**

Alcohol and tobacco containing products remain the most significant risk factors for this disease. All fermented and distilled alcoholic beverages contain carcinogens that promote the development of head and neck cancer. Tobacco-containing products have carcinogenic effects on the upper aero-digestive tract identical to those reported in the lungs. Important to the head and neck region, the local effects of alcohol and tobacco carcinogens are both dose-dependent, and synergistic. Smokeless tobacco abuse results in a local exposure to carcinogens that promotes cancers of the oral cavity and to a lesser extent, the oropharynx. Leukoplakia and erythroplakia are pre-malignant mucosal lesions associated with a risk for transformation to in-situ and invasive cancers. Both lesions are commonly found in the oral cavity and oropharynx, where chronic exposure to alcohol and tobacco carcinogens is most significant.

**Nutrition**

Dietary factors may also contribute to their development. The incidence of head and neck cancer is highest in people with the lowest consumption of fruits and vegetables. Recognising the latter association, clinical trials of various vitamin and anti-oxidants supplements have been conducted. To date, however, micronutrient supplementation has not led to a reduction in the risk of developing head and neck cancer.

**Viruses**

Exposure and prolonged local infection by Epstein–Barr virus or the high risk HPV subtypes (16 and 18) are associated with cancers of the epithelial cancers of the nasopharynx and oropharynx, respectively. For both viruses, transient infection without long-term sequelaes is common. Persistence of the viral genome and induction of malignant transformation is a rare event. Nasopharyngeal cancer occurs endemically in some countries of the Mediterranean and Far East.

**Irritating factors**

Other significant risk factors for this disease include prolonged exposure to asbestos (larynx cancer), textile fibers, nickel refining, and wood dust and leather tanning (nasal cavity and paranasal sinuses, adenocarcinomas).

**Genetic predisposition**
Perhaps the single greatest risk factor for developing a squamous cancer of the head and neck region is having had one at an earlier time. Similar to the situation with breast, colorectal, bladder, and lung cancer, having one cancer is associated with a significant risk for the development of a second cancer. Indeed, following the curative treatment of a primary head and neck cancer, the annual risk for developing a second unrelated squamous cancer of the upper aerodigestive tract is approximately 2%.

**CLINICAL SIGNS AND NATURAL COURSE OF THE DISEASE**

Squamous cell cancers of the head and neck region are occasionally discovered during routine dental or ENT (Ear-Nose-Throat) examinations. More commonly, they are discovered after weeks to months of relatively non-specific symptoms arising from the primary site, such as altered speech or swallowing, bleeding, localised pain or referred otalgia. Alternatively, many patients will present with new onset cervical adenopathy of unknown origin. Such patients are often treated empirically for infection, and later discovered to have head and neck cancer after the adenopathy persists or enlarges and the primary physician or a surgical consultant consequently performs a detailed examination of the head and neck region. Distant metastatic disease at presentation is unusual, and symptomatic disease related to such is rarely seen.

Squamous cell cancers of the head and neck region are locally aggressive and carry a moderate risk of spread to regional lymph nodes and a low probability of spread to more distant sites. This common natural history has several site-specific exceptions. For example, certain primary sites are more likely associated with regional disease at presentation (e.g. the nasopharynx, hypopharynx, base of tongue, and supraglottic larynx). In contrast, other sites have a relatively low risk for regional spread (e.g. paranasal sinuses, small lesions of true glottis).

**SITE SPECIFIC CLINICAL SIGNS**

Oral cavity, pharyngeal and laryngeal cancer’s typical complaints:

- odynophagia
- foreign body sensation
- hoarseness haemoptisis
- cough
- solitary or multiple, painless neck lymph node enlargement

Nasopharyngeal or nasal, paranasal sinus tumours:

- blocked nose
- unilateral hearing loss (serous otitis media)
- epistaxis
- facial sensory or motility disturbances
- visual problems
- solitary or multiple, painless neck lymph node enlargement

*Nota Bene: in case of hoarseness or dysphagia lasting for more than two weeks, thorough ENT examination should be performed.*

**MALIGNANT TUMOURS OF THE ORAL AND MAXILLOFACIAL REGION**

**MALIGNANT EPITHELIAL TUMOURS**

**Cancer of the lower lip**

Cancer of the lower lip is one of the most frequent malignant epithelial tumours of the orofacial region. It is a mature-cell (well-differentiated) epithelial cancer in 90–95% of the cases. Besides smoking a pipe or cigarettes (heat and mechanical), sunlight (ultraviolet light), wind, viruses (HPV, herpes simplex), candidiasis, syphilis as
as genetic factors, malnutrition, vitamin A or C deficiency, immunosuppressed state (HIV, organ-
transplanted patient), agrochemical substances and alcohol are listed among the predisposing factors. It is the
disease of elderly men working in the agriculture. The frequency ratio between cancers of the upper and lower lips is 20:1.

Three types can be differentiated clinically: exophytic, ulcerative and verrucous types. The commonest is the
exophytic type.

Lower lip cancer forms metastasise rarely and only in advanced stages. The tumour is symptomless for a long
time. The development of labial cancer is usually preceded by chronic cheilitis or leukoplakia.

The prognosis of lower lip cancer is good, the recovery rate ranges from 90 to 98%, depending on the stage of
the tumour.

Differential diagnosis: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.
keratoacanthoma, actinic cheilitis, specific (tuberculotic, syphilitic) ulcers.

Cancer of the lower lip

Cancer of the upper lip

While cancers of the lower lip account for 90–95% of oral cavity cancers, upper lip cancers represent 3–10%. Clinical types of upper lip cancer correspond with those of lower lip cancer. The prognosis of the upper lip cancer is worse than that of the lower lip cancer, and it metastasises earlier and more frequently.

Differential diagnosis: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.
pyogenic granuloma, keratoacanthoma.

Tongue cancer

The frequency of tongue cancer follows that of the lip cancer, but its prognosis is worse. It is the most frequent
site within the mouth; 35–45% of malignant oral tumours affect the tongue.
The **predilection site** is the middle third of the lateral border of the tongue, and the ventral surface and base of the tongue (pharyngeal tongue).

Primarily, alcohol (liver damage – cirrhosis) and smoking have a role in triggering the disease, but chronic irritating factors, poor oral hygiene, sideropenic and syphilitic atrophic glossitis, Candida albicans, viruses (HSV, HPV) and hot and spicy food may also contribute to its development.

The tumour is symptom-free at the beginning, thus patients present late, when the diameter of the thick-feeling tumour is more than 2 centimetres (endophytic type). At this time, regional metastases may also be palpated in 60–70% of the cases. Tumours in the posterior third of the tongue (pharyngeal tongue), and the ulcerated form cause pain (‗sore throat‘), bleeding and difficulty swallowing, eating and speaking more frequently and earlier than other forms. Tongue cancers are mainly squamous cell carcinomas (90%).

Only 24–51% of patients with tongue cancer survive for more than five years following the diagnosis, and the prognosis is even worse in case of tumours in the posterior third of the organ.

**Differential diagnosis:** verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

decubital ulcer, specific (tuberculosis, syphilis) and non-specific ulcers.

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**Cancer of the floor of the mouth**

The **frequency** of the disease follows those of lip and tongue carcinomas (20–25%). Usually it affects elderly, alcohol consuming (liver cirrhosis), and smoking men.

The tumour develops in the anterior third of the floor of the mouth more frequently than in the posterior third. Pain appears only in a late stage, when the tumour becomes thick and ulcerated and infiltrates its surroundings (bone).

The **prognosis** of early-stage tumours is better than that of tongue cancer and it metastasises later and less frequently. However, in case of tumours developing from the middle and posterior sublingual region and infiltrating the periosteum of the mandible, the bone and the tongue (anaesthesia, trismus), the prognosis is also poor.
The tumour metastasises primarily into the submandibular lymph nodes, unilaterally or bilaterally.

* Differential diagnosis: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

leukoplakia, ranula.

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**Buccal cancer**

Buccal cancer accounts for 10% of carcinomas of the oral cavity. Cancers situated closer to the angle of mouth have a better prognosis than those in the middle third of the bucca, and especially than those located in the posterior third.

Among triggering factors, smoking and traumas play crucial roles. Buccal carcinoma frequently develops from leukoplakia or erythroleukoplakia that has persisted for a longer time.

Lymph node metastases occur in the submandibular and jugulodigastric lymph nodes.

* Differential diagnosis: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

verrucous carcinoma, specific (tuberculotic, syphilitic) ulcers, large thrush.

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**Gingival cancer**

The disease develops mostly on toothless alveolar ridges, where it spreads from the gingiva to the alveolar mucosa and to the surrounding oral structures. Gingival cancer accounts for 8–12% of oral cavity tumours.

Beside smoking and alcohol, mainly chronic irritating factors (odontolith, prosthesis, etc.) may play a role in triggering gingival cancer.

The disease destroys the periosteum and the bone if it reaches them, and loosens the teeth. Gingival cancer is more frequent on the mandibular gingiva than on the maxillary, and mandibular gingival cancer metastasises regionally more frequently. Metastases occur mainly in the submandibular region. Similarly to oral cavity cancers located elsewhere, it is mainly the disease of the elderly (50–70-year-old) men, and histologically most of the cases are squamous epithelial cancers. Incisional biopsy is necessary.

* Differential diagnosis: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

epulis, pyogenic granulomas.
Palate cancer

Palate cancer is rare among cancers of the oral cavity. It is more frequent on the soft palate (15–20%) than on the hard palate (5%).

The prognosis of hard palate cancer is better than that of soft palate cancer, which metastasises regionally more frequently. On the soft palate, squamous cell carcinomas, while on the hard palate, adenocarcinomas are more common. Clinical differentiation is supported by the fact that while squamous cell palate carcinomas are almost always ulcerated, adenocarcinomas rarely show ulceration. Ulcerated palate cancers may penetrate into the paranasal sinus, then to the orbit, due to destruction of the palatine bone. Incisional biopsy is necessary.

Differential diagnosis: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

specific ulcers, abscess, cylindroma.

Basal cell carcinoma

Basal cell carcinoma is the most common skin cancer on the skin of the head and neck (80%). The ratio between basal cell carcinomas and squamous cell carcinomas is 5:1. Basal cell carcinomas usually appear in the periorificial region, and only exceptionally occur in the oral cavity, on the oral mucosa and on the lips. The disease is more common in elderly patients, especially among those working in the agriculture (sunlight). The tumour grows slowly and invasively, however, if it reaches the cartilage or the bone, it destroys them. Metastasising is extremely rare, it occurs mainly if the basal cell carcinoma is transformed into a keratinising squamous cell carcinoma.

Differential diagnosis: verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

seborrheic verruca, squamous cell carcinoma.

MALIGNANT TUMOUR OF THE SALIVARY GLAND

Adenoid cystic carcinoma (cylindroma)
Among malignant salivary gland tumours of the oral cavity, the infiltratively growing adenoid cystic carcinoma (cylindroma) is the most frequent one. It may develop from the small accessory salivary glands of the hard or soft palate, or it may appear in the parotid gland and in the submandibular salivary glands as well. Cylindromas originating from the submandibular salivary glands almost always affect women and, besides rapid progression, they have a poor prognosis.

It is a tumour with a firm and elastic touch, sometimes leading to facial paresis (parotid gland) accompanied by pain and ulceration. Usually it is regarded as a slowly growing tumour; however, rapidly infiltrating tumours should also be counted on. Regional metastases develop rarely and in late stages. It is a malignancy prone to recurrence. The prognosis of cylindromas is unfavourable.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma. pleomorphic adenoma, palate cancer, malignant lymphoma, palatal abscess.

**Mucoepidermoid carcinoma**

It is the most frequent malignant tumour of the salivary glands. It develops mainly in the parotid gland (89%), but it may also appear in the submandibular and small salivary glands (palate, tongue, bucca, etc.). In children, it is the most common malignancy originating from the salivary glands. Its low-grade malignity form grows slowly and painlessly, feels like a fluctuating cyst on palpation and rarely becomes large. It develops on the palate, tongue and bucca and lips. Its high-grade malignant form shows rapid, painful and infiltrative growth and may cause facial paralysis (parotid gland), reach large sizes and ulcerate.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma. malignant lymphoma, pleomorphic adenoma, mucocele, cysts of the jaw bones.

**Malignant melanoma**

Malignant melanomas are life-threatening malignancies with a poor prognosis, originating from the melanocyte precursors and naevus cells of the skin and arising as a result of solar radiation; however, genetic factors may also play an important role. They are most commonly located on the skin, there are only a few rare cases originating from the oral cavity, however, in these cases the prognosis is even worse than in case of melanomas of the skin.

The most common locations: the palate, (maxillary) gingiva, bucca, and the lips.

Alterations preceding melanoblastomas (pigmented naevus, dysplastic naevus, juvenile melanoma, blue naevus, circumscribed preblastomatous melanosis) or lentigo maligna may become malignant. Therefore, any trauma or insolation of these alterations as well as hormonal changes caused by age may pose a threat.

If any pigmented lesion on the skin or oral mucosa shows signs of accelerated growth, becomes ulcerative, and crusty or it is surrounded by an inflamed halo, becomes indurated, asymmetric with an irregular border, darker in colour, itchy, or it bleeds, one should inevitably think of a malignant transformation (starting symptoms). The deeper these malignancies invade the surrounding connective tissues (the thicker they are), the worse the prognosis will be. These tumours may metastasise into regional and remote lymph nodes (lungs, liver) via lymphatic and blood vessels easily and at an early stage.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma. haemangioma, pyogenic granuloma, epulis, haematoma.

**MALIGNANT ORAL TUMOURS OF MESENCHIMAL ORIGIN (SARCOMA)**

**Kaposi’s sarcoma**

This is a vascular, relatively rare malignancy dominantly effecting male patients. Since the appearance of AIDS, it has become the most common type of sarcoma of the oral cavity. In most of the cases, dermal symptoms are followed by rare symptoms in the oral cavity; however, the earliest manifestations of the disease may evolve directly within the oral cavity. It is more common in patients suffering from AIDS, lymphomas or leukaemia,
and Kaposi’s sarcoma may develop following an immunosuppressive treatment as well. Multiple, pink or dark blue maculae, plaques, nodules and tumours occur on the skin. The most common localisations within the oral cavity are the palate, tongue, lips and bucca.

_Differential diagnosis:_ verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

haemangioma, malignant melanoma, pyogenic granuloma

**ORAL MALIGNANCIES OF THE LYMPHORETICULAR AND HAEMOPOIETIC SYSTEM**

**Hodgkin’s lymphoma**

The aetiology of Hodgkin’s disease and non-Hodgkin’s lymphoma is still unclear. Hodgkin’s lymphoma is less common than non-Hodgkin’s lymphoma. Painless, swollen lymph nodes within the neck region may be the first warning signs, while another characteristic sign is that these lymph nodes become painful after alcohol consumption.

Oral symptoms rarely occur; however, they may precede and thus indicate the systemic disease itself. Oral symptoms occur as the swelling of soft tissue in the beginning, and then painful ulcers may develop, which may actually cause bone destruction beneath them.

Lymphomas may transform into leukaemias if they continue to exist for a long period of time.

_Differential diagnosis:_ verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

_non-Hodgkin’s lymphoma, cylindroma, tuberculosis_

**Non-Hodgkin’s lymphoma**

It develops from the proliferation of the lymphocytes of the immune system. Non-Hodgkin’s lymphomas usually evolve in lymph nodes accompanied by painless swelling. Tumorous infiltration occurs mainly in the lymph nodes of the neck region; however, the alterations may develop in the lymph nodes of other regions or even in extranodal regions (skin, mucosa, gastrointestinal tract, kidneys, bones, or parotid gland).

This disease is quite uncommon within the oral cavity, however, when it does occur there, the extranodal type is more common, and in these cases, the most common locations are the Waldeyer’s lymphatic ring, the palate, gingiva, tonsils and root of the tongue.

The tumours themselves are usually painless, soft to the touch and may become ulcerative at an early stage. Apart from the soft tissues, these alterations may erode surrounding osseous structures (teeth may become loose). The _prognosis_ is worse than in case of Hodgkin’s lymphoma.

_Differential diagnosis:_ verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

Hodgkin’s disease, eosinophil granulomas, epithelial cancer.

**Acute myeloblastic leukaemia**

Leukaemia is actually the appearance of young, immature white blood cell formations showing signs of atypical signs. Currently, this condition is considered to be an oncogenic bone marrow disease. The disease itself can be divided into acute and chronic forms according to differentiations between groups of leukaemias arising from the pluripotential marrow stem cells.

The aetiology is unknown, however, there is a number of factors held responsible for causing this disease (genetic causes, exposure to radiation, chemical substances, pharmaceutical products, etc.). Apart from pathologic leukocytosis, anaemia and thrombocytopenia may also accompany this disease. Oral symptoms are more common in the acute form, but may occur in chronic cases as well.

_Symptoms:_ initially they resemble infectious diseases (fever, tonsillitis, pharyngitis, malaise, fatigue, etc.). Generalised swelling of the lymph nodes is quite rare; however, the swelling of lymph nodes in the neck and submandibular regions may be important concomitant signs. One particular sign of this disease is the increase in the number of immature white blood cells. It is particularly common among adult patients.
Extensive, profound ulcers develop in the oral cavity and commissure of the lips of leukaemic patients due to fusospirochetal infections, candidiasis or other infectious causes. The most common oral symptom is gingival bleeding, however, purpuras, petechiae and ecchymoses due to thrombocytopenia may develop in other mucosal regions as well.

Due to the risk of haemorrhagic diathesis, no tooth extractions or any other types of oral surgeries are recommended. Oral symptoms may be the primary signs of this disease, thus, dentists should be quite aware of this condition. Gingival and mucosal ulcers are profound; the resorption of parodontium may cause the teeth to become loose. Gingival hyperplasia may accompany oral symptoms.

When establishing the correct diagnosis, laboratory examinations are quite decisive. The prognosis is quite poor. *Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

Ulcerative gingivitis, ulcer-gangrenous stomatitis, infectious mononucleosis, haemorrhagic diathesis, agranulocytosis, Hodgkin’s disease

**Acute lymphoblastic leukaemia**

Acute lymphoid leukaemia is particularly more common in childhood (80%) than myeloid or monocytic leukaemia, however, the lymphoid type is the least common to cause any oral symptoms. The prognosis is better than it is for myeloid leukaemias (60% of these cases result in full recovery).

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

the same as in myeloid leukaemia

**Chronic myeloid leukaemia**

After the manifestation of the clinical symptoms, the disease will prospectively progress for another 3–6 years. The disease usually starts with a latent period. Myeloid leukaemia is more common among middle-aged men, while lymphoid leukaemia particularly affects elderly male patients.

Symptoms: general symptoms causing complaints usually include stabbing or painful swelling of the spleen; however, hepatomegaly may also occur. Headaches, fatigue and occasional high temperature may develop. Patients usually seek medical care due to gingival bleeding. Lymph node swelling is quite uncommon in myeloid leukaemia, while it is common in the lymphoid type of the disease. Oral symptoms are rarely primary, and these manifestations are not pathognomic, however, gingival hyperplasia and haemorrhage may persist and even profound, persistent ulcers or the loosening of the teeth may occur. Parotid swelling is more common than it is in patients suffering from acute leukaemia. Concomitant herpes zoster, pemphigus and candidiasis may also occur. The prognosis of chronic lymphoid leukaemia is better than that of the myeloid types of the disease.

*Differential diagnosis:* verruca vulgaris, condyloma acuminatum and verrucous carcinoma.

epulis, ulcerative gingivostomatitis, hydantoin hyperplasia, or agranulocytosis

**Chronic lymphocytic leukaemia**

This is the type of leukaemia, which shows the slowest rate of progression (with an overall survival of 6–10 years). A characteristic sign of chronic lymphocytic leukaemia is lymph node swelling at an early stage of the disease. Generalised lymphadenopathy is commonly accompanied by splenomegaly. This disease usually occurs at an elderly age. Apart from these symptoms, other, non-specific dermal symptoms may occur as well, such as papulous eczema and the formation of vesicles and bullae. Skin infections, advanced periodontitis, ulcers and bleeding may occur within the oral cavity.
Chapter 3. ASSESSMENT

1. Patient history (Péter Vályi DMD - László Párkányi DMD)

The aim of taking patients’ history is to evaluate their activity, lifestyle and to get information about their physical, emotional, intellectual and social status as related to oral health indicators and patient needs.

When taking the history, the parameters of five aspects are evaluated. physical state and development

- emotional state
- social status
- intellectual status
- mental status

Physical condition and development

The most important aspect is the patient’s view of his/her own health. Besides this, information about past and current diseases and information regarding their treatment is necessary to help evaluate the patient’s general health and its relationship with oral conditions and necessary treatment interventions. It is important to determine risk factors, the level of physical activity and coordination as well as systemic conditions. Progress needs to be evaluated: developmental status (maturity), if any inherited or acquired deficiency affects development, if the developmental status corresponds to the patient’s the age, or if it is below/above that.

We have to determine if the occupation of the patient affects his/her health condition in any way. Family history has to reveal all hereditary diseases that run in the family. The next step is the evaluation of the patient’s self-sustaining ability (ADL classification – Activity of Daily Living see chapter 3.5.).

Emotional state

Evaluation of the behavioural and emotional state is also important regarding treatment planning, motivation and cooperation. We have to pay attention to the patient’s self-concept, body-concept, this way revealing some disorders that can have oral consequences (anorexia, bulimia), and also the reasons for some unrealistic expectations from the patient. We have to evaluate the patient’s mood, willingness to take challenges, and the existence or lack of emotional support. These circumstances are all related to the progression of the disease and effectiveness of the treatment.

Social status

Health and its preservation are of high financial value, therefore preventive measures and therapeutic procedures require financial investment. Financial status needs to be carefully evaluated to be able to create a treatment plan which is affordable for the patient.

Special attention needs to be paid to patients’ recreational activities, because they can affect their physical or emotional well being, which also affects oral health. Good treatment results can be achieved with patients leading a healthy lifestyle, in contrast with patients living a self-destructive life.

When evaluating social status, the influence of the environment needs to be analysed, too. The cultural setting can influence health status; treatment can be hindered by cultural obstacles (Muslim women can only be treated by a female doctor). Environmental risk factors also have to be analysed, just like the social network and support of the patient.

Intellectual status

We have to evaluate the intellectual potential of the patient, how high the level of education and problem solving is. Evaluating communication and concentration skills, as well as long- and short-term memory is important when choosing the appropriate instruction and motivation strategies.
Mental status

When examining the mental status, it is important to pay attention to faith, religious experience, being part of a religious community. It can also affect health maintenance and medical treatment. Anamnestic data need to be evaluated together with personal observations. While recording the patient’s history, signs of the patient status need to be noticed. These are the following:

- emotional state (happy, depressed, anxious, scared, confused...etc.)
- physical signs (injuries, signs of illness, functional disorders..etc.)
- skin colour alterations (pale, cyanotic, greyish, yellowish...etc)
- eyes (focusing, redness, blurry, pupil size..etc)
- clothing, personal hygiene

We have to pay attention to patients’ responses, if they are adequate and coherent. It should be noted if the patient needs any medical device, dependent on it, or if any other help is needed. Any signs which could physically disturb appropriate individual oral hygiene should be observed. Signs of substance abuse (drugs, alcohol), which can lead to inadequate orientation in time and space should be noted. Emotional stability needs to be evaluated to see if the patient poses any threat to himself/herself or to surrounding people. And last but not least, cultural and linguistic obstacles of patient communication need to be assessed.

For patient history a questionnaire can be applied, but the written answers have to be confirmed in the form of an oral interview. This interview can be divided into three parts. The first, orientation phase is for gathering information about the cause of the visit, personal information of the patient, and this is the time when we explain why our questions are necessary, and we can also introduce the first steps of oral health education. It is important to handle confidential information properly and respect patients’ rights. During the working phase, easy, properly oriented questions need to be asked. Feedback is very important, the patient has to be confirmed that he/she has our total attention and we have understood everything that has been said. With additional questions and short explanations we can indicate what kind of information we consider relevant in connection with the disease and treatment planning. Without feedback, this information might get lost. Finally, in the terminal phase, we indicate the end of the discussion. (e.g.: My last question is...). Without the right control, information can be missed, the conversation can get too long, which can lead to data synthesising difficulties.

Four types of questions can be used in a questionnaire:

- **organ system oriented questions** are for getting information about the specific diseases of different organs
  (specific disease, time discovered, duration)

- **disease oriented questions** inquire about certain diseases in the questionnaire (time, severity, outcome): they focus on diseases which influence oral diseases or their treatment in any way

- **symptom oriented questions** are easy to understand questions about symptoms recognisable for the patient
  (e.g.: dry mouth, frequent urination can indicate diabetes mellitus)

- **culture oriented questions** are to rule out sexual or ethnic risk factors, but the application of alternative medicine can also influence the progression of oral diseases and even treatment modalities to be chosen

1.1. Chief complaints

During patient history directed questions are asked, which give information about primary or secondary symptoms of the disease or reveal relevant information for differential diagnosis. Questions regarding the nature, duration and trigger factors of pain are always the most important. Besides objective signs, such as swelling, ulceration, discoloration, surface deformities, subjective signs (bad breath, mobile teeth, difficulty chewing, tooth malpositioning, elongation) can indicate certain diseases, in this case periodontitis. Asking about current complaints tells the patient that we are concerned about his/her condition. After discussing this, questions start to focus on general medical conditions, then oral health situation, and finally we discuss preventive measures.
1.2. Medical history

After taking the history and evaluating complaints, questions about the general medical condition should be asked. The following questions need to be answered:

1. Can the oral status be linked to any systemic disease, condition?
2. Can the treatment of systemic diseases influence oral deformities?
3. Can the oral deformities be a late consequence of a systemic condition or disease, are increased preventive measures necessary?
4. Does the systemic disease influence the overall treatment plan?
5. Are any precautions necessary, which prevent the adverse effects of dental treatment, related to the pre-existing systemic disease or its treatment
   - Can excessive bleeding occur? Is there a risk of infection?
   - Can the systemic disease worsen leading to a life threatening condition?
   - Is the patient endangering the treating staff or other patients?

The most common life threatening condition is allergic reaction, followed by anaphylactic shock. When taking patients’ history, all medications, food and other allergies (e.g.: latex) need to be discussed, which can lead to this reaction. Allergy to iodine is not uncommon, and it is used as an antiseptic, disclosing agent in dental practices. Fortunately PVP-iodine is much less likely to cause allergic reactions than its alcohol based, crystalloid solution. Cross-reactions can happen quite often: allergy to kiwi is often accompanied by latex allergy.

Cardiovascular diseases are quite frequent in the Hungarian population. Several risk factors need to be considered. Medications taken by the patient can influence the gingiva (Calcium-channel blockers), anticoagulant medications (cumarine derivatives) and thromboocyte aggregation blockers (NSAID) increase the risk of excessive bleeding. In case of implanted devices (e.g.: artificial valve), which can accumulate bacteria, antibiotic prophylaxis needs to be applied. Dental treatment can also put the patient through a lot of stress. Within one year after an acute myocardial infarct, the risk of (possibly lethal) seizures is very high. This should be considered throughout treatment planning, during the selection of medications, and while determining treatment time.

Diabetes mellitus is one of the most common diseases in developed countries. When taking the history, even in case of a negative answer, one needs to make sure that none of the symptoms of the disease are present (see above). If the disease is present in the family history, especially combined with other risk factors (obesity, dietary habits), consulting with the patient’s physician is recommended. In diagnosed cases, assessment has to be made to check how well-controlled the disease is. High blood sugar levels can lead to adverse effects (visual, renal, cardiovascular, nervous system), but prolonged wound healing and the risk of infections also makes antibiotic prophylaxis necessary.

Presence of infectious diseases (Hepatitis B, C, TBC, HIV, STD) puts danger on the treating staff, other patients and other members of the dental team (technicians, receptionists). Besides sticking to rules of infection control, increased precautions should be made (barriers) while treating these patients (see chapter 6.3.). It should also be considered that medications used to treat systemic diseases can affect our choice of medications. In case of Hepatitis B and C infection, liver dysfunction is present. This leads to immune dysfunction (risk of infection), decreased level of clotting factors (risk of excessive bleeding), dysfunction of detoxification. The dysfunction of detoxification means that medications broken down in the liver must be avoided. This applies for other liver diseases as well.

Of endocrine disorders, hyperthyroidism and adrenal dysfunction are significant. In these cases the use of epinephrine in local anaesthesia should be avoided. Female sex hormones influence the metabolism of the periodontal tissues and affect the composition of biofilm. In puberty, due to hormonal changes, the gums are less resistant to dento-gingival plaque, therefore bleeding or hyperplastic overgrowth may occur. Similar symptoms are present during pregnancy, where even epulis can develop. This can spontaneously go into regression after delivery. For pregnant patients in their third trimester treating position is also important (see
After menopause osteoporosis may be present. Bisphophonates are often used for its treatment, and they can have serious side-effects (see chapter 3.1.3.). Physiological changes are also possible. (e.g.: women after menopause have higher blood pressure than men in their age).

Patients with renal diseases may experience increase in blood pressure. Dialysed patients may have clotting disorders due to the treatment, but also platelets and clotting factors can be dysfunctional, and the potential for infection is typically higher. Patient undergoing treatment for a longer time may be infected with Hepatitis C.

Patients with malignant tumours are often treated with radio therapy. This causes the contraction of vessels in the head and neck region, which results in reduced circulation and the risk of infection is also higher. This can lead to radio-necrosis. Because of the applied medications antibiotic prophylaxis may be necessary. Tumorous diseases of the haematopoietic system can affect all three cell lines: red blood cells, white blood cells and platelets, therefore they present high potential for infections and uncontrollable bleeding. Treatment can cause mouth dryness (medication, irradiation) which promotes both cariological and periodontal disease progression. Breast cancer treatment may also include the use of bisphophonates.

In case of lower respiratory tract diseases, lying position of the patient should be avoided as well as the use of sprays. Drug interactions should also be considered. Steroid medications used in asthma patients can interfere with the healing of viral diseases (e.g.: herpes simplex, varicella zoster). Patients should always carry their medications with them in case of seizures, but the dental office must also have antihistamine and steroid drugs in case of emergency.

Medications used in gastrointestinal diseases can reduce saliva production. Inflammatory bowel diseases often require immunosuppressants. Antibiotic prophylaxis is mandatory. Medicinal treatment of these can only be performed after consulting the patient’s physician.

Epilepsy drug hydantoin, can facilitate plaque induced gingival growth. A frequent side-effect of drugs used for psychiatric diseases is mouth dryness.

Autoimmune diseases can have oral manifestations. Therapy related drugs cause immuno-suppression, which increases the risk of infection.

When ophthalmological diseases are present, eye protection is required. The use of epinephrine is contraindicated in glaucoma.

1.3. Pharmacologic history

The point of collecting information about drugs of regular use from the patient is to reveal possibly related oral side-effects, toxic reactions, allergic reactions and to rule out drug interactions. The list of applied medications can help us discover relevant information about the patient’s diseases not mentioned during history taking.

Drug effects on desired organs are called therapeutic effects, effects on all other organs are called side-effects. Taking drugs can have oral and systemic side-effects. The most common drug related side-effects in the mouth are shown in table 3.1.3-1. Dry mouth is the most common oral side-effect of drugs. More than 500 medications can cause this. Besides cariological and periodontal consequences, dry mouth makes eating, speaking and wearing removable dentures more difficult. Opportunist infections can also develop on its ground.

Another common side-effect is gum hyperplasia. Drugs with this affect are Calcium-channel blockers (cardiac medication), cyclosporine (immunosuppressant), phenytoin (epilepsy drug), erythromycin (antibiotic), bleomycin (anti-tumour drug).
The most common oral side-effects of medications

During drug metabolism interim products (metabolites) are produced, which can damage certain tissues. This damage can be both macroscopical or microscopical. Damage to the most important organs can be extremely dangerous. In most cases, toxic reactions are the sign of drug overdose. When the gap between therapeutical and toxic dose is narrow, side-effects are more frequent. Even slight overdose of over-the-counter paracetamol can lead to damage. Chemicals can be toxic to the liver (hepatotoxic), kidneys (nephrotoxic), nervous system (neurotoxic) and heart (cardiotoxic).

Some medications can harm the developing foetus (teratogen drugs). We have to be extremely cautious when prescribing medicine to pregnant women. Consultation with the patient’s gynaecologist is required.

When two medications are used at the same time and they affect each other, it is called drug interaction. One drug can increase or decrease the effect of another drug, but the two together can lead to side-effects, too. It is important to know that antibiotics used in dental treatment decrease the effect contraceptives. In case of regular drug use, interactions with the pre-existing drug should always be considered before applying new medications. Interactions of non-steroidal anti-inflammatory drugs used in dental therapy are shown in next figure.
During pharmacological diagnosis previously discovered allergic reactions must be revealed. If it is only present in the history but has not been investigated yet, an allergy test must be performed before the use of the drug. One must not forget that not only the active agents of drugs can cause allergic reactions, but also the preservatives used in them. If someone is allergic to Ultracain DS-Forte, it does not mean that the allergy applies to Ubistesin or Septanest as well. Allergic reactions to amide-type anaesthetics are extremely rare, therefore it is worth testing for preservatives used in these medications.

1.4. Assessment of risk factors

Before the treatment all anamnestic data and personal observations have to be evaluated to see if the planned treatment includes any potential risk. Risk factors can be uncontrollable bleeding, postoperative infection, allergic reaction or acute, life threatening conditions (circulatory, respiratory dysfunction) induced by systemic diseases.

In case of known systemic diseases, consultation with the patient’s physician is recommended regarding the treatment, medications and prophylactic measures. Consultation can be verbal or written and it may require the general examination of the patient.

Based on basic vital functions, the American Anaesthesiologist Association (ASA) created a classification which evaluates the risk of a planned treatment, and sets standards for systemic pre-treatment and prophylaxis. According to the ASA classification:

- ASA I.: No organic pathology, or patients in whom the pathological process is localised and does not cause any systemic disturbance or abnormality.

- ASA II.: A moderate but definite systemic disturbance, caused either by the condition that is to be treated or caused by other existing pathological processes.

- ASA III.: Severe systemic disturbance from any cause or causes. It is not possible to state an absolute measure of severity, as this is a matter of clinical judgment. The following examples are given as suggestions to help demonstrate the difference between this class and Class 2.

- ASA IV.: Extreme systemic disorders which have already become an eminent threat to life regardless of the type of treatment. Because of their duration or nature there has already been damage to the organism that is irreversible. This class is intended to include only patients that are in an extremely poor physical state. There
may not be much occasion to use this classification, but it should be useful when separating a patient in very poor condition from others.

• ASA V.: Emergencies that would otherwise be graded Class 1 or Class 2

Though the ASA classification is a good predictor of adverse events, it cannot consider important modifying factors as patient behaviour (smoking, alcohol consumption), metabolic disorder, unrevealed relevant information about medical condition.

Several systemic diseases and their medications can be a risk of infections. To avoid this, antibiotic prophylaxis is used according to a strict protocol. Patients that need antibiotic prophylaxis to avoid infectious endocarditis: Only recommended for high risk patients regarding IE:

1. artificial valve or artificial material used for valvoplasty
2. previous IE
3. congenital cardiac diseases
   - cyanotic congenital cardiac diseases without surgical correction, with residual defects, palliative shunts, conduit
   - congenital cardiac diseases with full surgical correction, with artificial arterial implantation, within 6 month of surgery
   - residual defect surrounding an implant

There is no need for antibiotic prophylaxis in inherited cardiac diseases and other forms of valve disorders. Other conditions may also need antibiotics: dialysed patients and other kidney diseases, liver diseases, uncontrolled diabetes mellitus, bisphosphonate use, immunosupression, severe neutropenia.

Dental treatments requiring antibiotic prophylaxis:

• interventions on the gums
• treatment of the periapical region
• therapy with the perforation of the oral mucosa

Oral surgical interventions:

• Anaesthesia of inflamed tissues,
• extraction, sculpting,
• excocleation,
• circumcision (operculectomy),
• incision,
• excision,
• frenulectomy,
• apicoectomy,
• syalolith removal from the duct,
• sinus-closure,
• suturing,
• dental implantation (surgical part)
Periodontal interventions:

- deep pocket probing,
- sub-gingival scaling,
- gingivectomy, gingivoplasty,
- open and closed curette,
- all flap access surgeries

Besides this, all maxillo-facial interventions with oral involvement require prophylaxis. Applicable antibiotic prophylaxis protocol can be found in next figure.

<table>
<thead>
<tr>
<th>Situation</th>
<th>Agent</th>
<th>Single dose, 30-60 minutes before intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Adult</td>
</tr>
<tr>
<td>Oral</td>
<td>Amoxicillin</td>
<td>2 gramm</td>
</tr>
<tr>
<td>Unable to take oral medication</td>
<td>Ampicillin or</td>
<td>2 gramm i.m., or i.v.</td>
</tr>
<tr>
<td></td>
<td>Cefazolin or</td>
<td>1 gramm i.m., or i.v.</td>
</tr>
<tr>
<td></td>
<td>Ceftriaxone</td>
<td></td>
</tr>
<tr>
<td>Allergic to Penicillin or ampicillin, Oral medication</td>
<td>Cephalexin or Clindamycin or Azithromycin or Clarithromycin</td>
<td>2 gramm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>600 mg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>500 mg</td>
</tr>
<tr>
<td>Allergic to Penicillin or ampicillin, Unable to take oral medication</td>
<td>Cefazolin or Ceftriaxone or Clindamycin</td>
<td>1 gramm i.m., or i.v.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>600 mg i.m., or i.v.</td>
</tr>
</tbody>
</table>

Antibiotic prophylaxis protocol

1.5. Dental history

The purpose of dental history is to reveal patients’ oral hygiene behaviour, identify previous dental problems and solutions, assess the condition of dental restorations. The time and cause of earlier tooth loss gives us valuable information about the nature of the disease.

The first questions have to reveal the frequency of dental visits. Sometimes the actual truth will be found out through answers to later questions, because direct questions may urge the patients to satisfy our expectations instead of revealing the truth.

Questions have to be asked very carefully, because in most cases, the present dental status revealed during examination is a result of insufficient funds, not the negligence of the patient. In many cases, information acquired and possibly misunderstood during previous dental visits can influence the current dental status.

The next step of the dental history is to reveal the cause of previous tooth loss. Tooth loss due to carious lesions is the result of high caries frequency and/or the lack of preventive methods during childhood, dietary problems,
inadequate or even completely lacking individual oral hygiene. Early loss of mobile teeth indicates early onset, fast progressing periodontal infection.

Questions regarding orthodontic treatment can clear the background of observed developmental or positional deformities. Answers can determine whether earlier attempts at the orthodontic correction of the deformities were made, or whether the deformities (e.g.: recession) were initially the result of an orthodontic treatment. Getting information about orthodontic treatment processes can also tell us a lot about the patient’s cooperation skills.

Questions about existing restorations are to learn more about the indications and modifying circumstances of the treatment (restoration type, used materials), as well as the time of the restoration to be able to evaluate the relative condition of the restorations.

1.6. Oral hygiene history

The role of dental hygiene anamnesis is to evaluate the patients knowledge, attitude, by asking about individual oral hygiene performed at home. From the information received, motivation and instruction can be individualized, which is inevitable in planning of patients individual oral hygiene. First questions should aim at regular tooth brushing habits. Here, we can obtain information about frequency of brushing, the type of brush used (manual, electric), hardness of bristles, hand used for brushing, and order of surfaces brushed. From obtained answers, we can clear out, whether the frequency, time spent, or the technique is insufficient, leading to periodontal disease.

The next set of questions are to evaluate the information the patient has about accessory dental hygiene instruments, whether any is used, and if the technique applied is correct. Demonstration equipments are useful, to let the patient show, how he/she uses dental floss, interdental brush, Superfloss, or any other instrument. This method reveals the manuality of the patient, which either needs to be improved, or other instrument need to be used as replacement.

During dental hygiene anamnesis, use of antiseptics need to be considered. If the patient uses mouthwashes, the active substance of it must be revealed. It is important to determine if the agent is of any use during the treatment at all.

1.7. Family history

Family history can provide valuable information about the development of the disease, but it can also draw attention to conditions, diseases which need to be examined before the treatment. Questions can be directed to address developmental disorders, cariologic aspects. If family history reveals multiple cases of periodontal disease and cancer, applying preventive measures as part of the treatment has a major importance. Especially in young patients, diseases in the family history can be relevant, because they may not have manifested yet, but their later development can be expected, so the patient is at risk. Such diseases are e.g.: bleeding disorders, liver and kidney disorders (which are associated with the risk of infection), bleeding and drug metabolism disorders. Diabetes mellitus, circulatory diseases, endocrine disorders in the family history may necessitate a consultation with an internal specialist, even if the patient has no specific complaints.

2. Clinical diagnosis (Márk Antal DMD - András Forster DMD - Péter Vályi DMD - László Párkányi DMD)

2.1. Extra-oral examination (Péter Vályi DMD - László Párkányi DMD)

The first step of the clinical examination is extra-oral examination, which is an important part of the stomato-oncological screening. The examination of the patient starts with observation of the patient. Alterations in vital signs (posture, nourishment, gait, voice, hoarseness, respiratory rate, cough, sweating, or hyper-movements) have to be discovered already at that time.

Extra-oral examination follows the steps of the basic physical examination:
1. inspection
2. palpation
3. percussion
4. auscultation

During inspection, all areas of the head and neck region have to be well examined in particular to the face and neck, but disorders on the hair-covered skin should also be analysed. Asymmetries and swellings are easy to discover, but discolorations of the skin, spots, surface alterations (injuries, ulcerations, or blisters) should also be noticed. One of the exceptional regions is the epithelial transition of the lips, where like in other histologically similar regions, neoplasms may develop with a higher chance. Certain movements are evaluated visually, which apply for TMJ disorders in case of the mandible, but movement alterations of the lips, eyelashes, losing of their tone may indicate neurological abnormalities. Examination of the eyes is also very important, the typical symptoms discovered can be exophthalmus, scleral colour changes, and hyper-vascularisation, size alterations of pupils, their reaction to light, or eye movement dysfunction.

Palpation can reveal any resistance, tissue growth, or tissue deficiency. Both bone and soft tissue discrepancies need to be examined. However, palpation by itself is not enough for establishing the diagnosis: fixation, consistence, and tenderness on palpation of the examined volumes are important factors. Palpation can reveal swelling of the lymph nodes, alterations in salivary glands, and part of the temporomandibular joint disorders. Examination can be done by one finger (digital) for detection of torus mandibularis, two fingers on the same hand (bidigital) to examine the lips, one finger on both hands (bimanual) to examine the floor of the mouth, or two sided (bilateral) palpation to examine symmetrical structures on both sides like cervical triangles.

Percussion is used to detect changes in the sinuses. A typical sign of acute inflammation is tenderness or even pain on percussion.

Auscultation signs are also typical for several diseases. No instruments are needed to hear the sounds of temporomandibular joint disorders, or broken jaws, or even certain inflammatory reactions. The typical sound is crepitation or clicking. Endoscopes are recommended as well to detect joint disorders.

Besides the above mentioned examination, our nose is also important diagnostic tool. Bad breath (foetor ex ore, malodour) can refer to the presence of oral diseases, but it can also be related to diseases in the lower parts of the digestive tract. Smelling can detect if the patient is smoking, but it can also reveal diabetic acidosis, as it has a typical smell. Alcohol consumption also has typical signs in smell.

Description of disorders

Examinations are done according to regions, and documentation also needs to be done in this manner. The following image shows regions of the head and neck:
During documentation, the following aspects should be considered, which apply to oral mucosal disorders as well:

- **Localisation and distribution**
  1. anatomical position (anterior-posterior, lateral-medial, inferior-superior, ipsilateral-contralateral)
  2. symmetry (unilateral-bilateral, midline)
  3. distribution (solitary-multiple, localised-generalised, separated-converging)

- **Size and shape**
  1. size is given in units or in pathological terms (pepper-sized, pea-sized)
  2. shape (regular–irregular, sharp–uneven edges, infiltrated, round, oval, etc., the surrounding structure is inflamed, fibrotic, etc.)

- **Colour** (pink colour can turn into reddish, livid, bluish–purplish. It can be covered by yellowish fibrin, can be adipose, purulent, necrotic, hyperkeratic, etc.)

- **Surface structure** important differential diagnostic factors: crater-like, bark-surfaced, invaginated, papillary, pseudo-membranous, smooth, warty.

- **Consistency**– resistance of mass: soft, hard, compact, or related to pathological terms: wood-hard, horse fur-like, etc. Regarding liquid content, it can be fluctuating, or empty.

- **Spreading and connectivity**– it can spread superficially or deeply, can be connected to its base or to a stalk.

- **Mobility or fixation**– connection to surrounding tissues: mobile or fixed.
• **Tenderness**—insensitive or painful (the pain can be spontaneous, constant, or develop on palpation)

### 2.2. Intraoral examination (Péter Vályi DMD - László Párkányi DMD)

During intraoral examination, the oral cavity and its surrounding structures (palate, tongue, floor of the mouth, pharynx, lips, and cheeks) are evaluated. For this, the patient has to be prepared (rinsing with antiseptics, protection of lips and cheeks with non-oil based lubricant, and removal of removable dentures), and appropriate instruments (two dental mirrors, air/water syringe) are needed. Some superficial disorders can only be evaluated after drying the mucosa. A gauze slab is required for the examination and fixation of the tongue.

#### THE CHEEKS

The inner surface of the cheeks is covered with pink non-keratinized squamous epithelium. The deeper layers, i.e. fat tissues, masticatory muscles, and the parotid glands can be palpated bimanually. The papilla is normally bulky at the excretion ducts of the parotid gland. A typical surface irregularity seen at the line of closure of dental arches is called morsicatio buccarum.

The area between the lips, attached mucosa and the cheeks is called the vestibule. The connection between the attached gingiva and the non-attached mucosa is described as the muco-gingival junction. Iodine can visually express it. The inferior and superior labial frenula can be found on the inner surfaces of the lips, in the midline, which connect the lips to the attached gingiva. They can be underdeveloped, or can be attached far coronal, this way mobilizing the midline papilla. Similar frenula can be found in the premolar and molar regions as well.

#### THE GINGIVA

Gingiva is tooth dependent structures which cover the alveolar process, where the teeth are anchored by ligaments. After loosing the teeth, it is called mucosa that covers the edentulous alveolar ridge. Two main parts are the attached gingiva and the marginal gingiva. Its coral-pink colour can have pigmented spots even under healthy conditions. The most common disorders are swelling, discoloration, ulceration, or changes in contour. Papillae fill the interdental gaps under normal circumstances. Periodontal attachment loss or positional disorders can lead to loss in the papillary structures. The papillae can reside in ulcerative gum diseases. They can go through complete healing or remain in crater like defects in chronic diseases.

#### THE PALATE AND PHARYNX

The palate can be divided into hard and soft palate depending on the underlying structures. The hard palate is supported by bony base, whereas the soft palate is made up of muscles underneath the mucosa. The soft palate leads towards the pharynx. The hard palate is covered by light pink, keratinized squamous epithelium. The frontal part of the hard palate contains irregular mucosa (rugae palatinae) and papillae indicative of the incisival foramen behind the central incisors. The hard palate turns into the gingiva of the upper teeth without transition.

The border between the hard and soft palate is well distinguished by making the patient pronounce certain vowels (e.g. while taking an impression). A mucosal tongue-like structure is found in the midline at its distal border. Its swelling or discoloration indicates inflammation, and deviation to one side indicates a neurological disorder.

The pharynx is a muscular, mucosal structure distal to the oral cavity. Pharyngeal tonsils are important to be examined. In inflammatory processes, they get swollen and show discoloration, and in some cases purulent follicles can be seen (quinsy tonsillitis).

#### TONGUE

The dorsal surface of the tongue is covered by a special papillary mucosa with a fissure in the middle. When examining the tongue, the inspection of the lower surface and the floor of the mouth are important. Holding the tongue with a gauze slab can provide visual access. Bimanual examination of the tongue can reveal resistance or tissue growth within the tissues. Examining the movements of the tongue can help diagnose some muscular and neurological disorders.

#### FLOOR OF THE MOUTH
To examine the floor of the mouth, the tongue needs to be retracted in a way previously described. Under healthy conditions, sublingual and submandibular salivary gland excretions can be discovered in the frontal part, which are excreted together as caruncula sublingualis. In the frontal part, mucosal structures continuing on the tongue are visible, besides bluish transparent veins. A hard bony structure on the inner surface of the mandible called torus mandibulæ can have a pronounced bulkiness.

### 2.3. Dental clinical examination (Márk Antal DMD - András Forster DMD)

With the clinical examination, we evaluate the status of the hard dental tissues – erupted through the soft tissues, the possible coronal restorations and the sensibility of the pulp. As a matter of course, examination of the occlusion is also involved, which we discuss in another chapter (3.2.5).

#### EXAMINATION OF HARD DENTAL TISSUES

Teeth must be dried separately or in groups for the clinical detection of carious lesions. In case of hard tissues, we can use inspection, palpation, and apply different types of dental explorers or transillumination, which is based on the fact that the transmission of light in carious lesions is worse than in sound enamel structures.

We differentiate between carious- and non-carious lesions of hard dental tissues. Non-carious lesions of the enamel are attrition, erosion, hypoplasia, abrasion and any kind of fracture of the hard tissues.

Carious lesions on the smooth, self-cleaning surfaces can be detected easily in an early stage (c. incipient), while the lesions of approximal contact points, even in an early condition, can only be identified with the help of a radiogram (Bite-Wing). By the progrediation of this approximal caries, it is possible to visualise or to palpate with the dental explorer, but only the breakage of the approximal ridge and involvement of the occlusal surface can be considered deep caries. Dental probe is essential to the assessment of fissure caries. Carious lesions are documented according to the surface of the tooth on which they are detected.

Non-carious lesions, in several cases, can relate to severe problems with general health condition of the patient, e.g. an erosion can be intrinsic because of vomiting of patients in pregnancy or eating disorders, while environmental damage or problems with supplement intake can be extrinsic causes of it. Attrition can indicate occlusal alterations, even more the most common sign of bruxism. It provides important information to set the treatment plan up.

Damages of hard dental tissues are classified into 5 groups:

- lesions involving the enamel only
- involving enamel and dentin
- involving enamel, dentin and pulpal tissues
- fracture of the full clinical crown
- fractures spreading under the bone level

Accidental injuries of teeth require special documentation on which the circumstances of the accident, signs, symptoms, the status of the teeth, treatments and check ups are precisely documented.

#### ASSESSMENT OF THE RESTORATIONS

Marginal integrity and the possible carious lesions along marginal gaps of existing fillings, inlays, partial or total dental crowns should be evaluated. The extended surfaces of the restoration, secondary caries and notes on marginal opening should be documented. It is necessary to examine the anchors of removable dentures as well.

#### SENSIBILITY TESTS

By the means of sensibility tests it is possible to get information on the current status of the pulp. We evaluate the pain reaction as a response of the test. The stimulus can be thermal, such as cold (cold spray or ice) or heat (by plastic polishing burs), or even electric. These responses reflect more on the status of the circulation than the integrity of the pulpal nerve complex. It can be influenced by the degenerative progresses in the pulp, the
threshold and sensibility of pain in the patient, depending individually on gender, age, emotional status, stress and medications taken, or nerve blocks and neighbouring metal restorations. On general dental examination, only the vitality of the pulp is documented, the type, the length, the triggering factors and the alterations in time period are evaluated as part of the differential diagnosis of pulpal diseases.

**EXAMINATION OF THE OCCLUSION** – see chapter 3.2.5

**DOCUMENTATION**

Documentation can be paper based or electronic. Information gained by intraoral examination can be documented by means of systems, using graphical or stylized tooth pictures or even number identification. Picture 3.2.3.-1. shows a sample electric patient chart.

![Sample electric patient chart](image)

18 dissected tooth 17 és 16 extracted teeth 15 c. penetranst on the MO surfaces , pulp necrosis, radicular cyst. 14 tooth is root filled, granuloma on the palatal root, 13 tooth with porcelain crown, abutment tooth, root filled with post cored 12 resected tooth, porcelain crown, abutment tooth, root filled, after apectomy with retrograde root filling, para-post cored, 23 tooth is abutment, 24 tooth: temporary filling of root canal, temporary filling of occlusal surface, 25 radix, 26 tooth: para-post cored build-up with composite resin, 37 tooth: metal inlay, 36 tooth: gold-ceramic crown, 35 tooth: composite resin inlay, 34 tooth: gold inlay, 43 tooth composite resin direct restoration of mesial surface, 44 tooth: amalgam restoration of occlusal surface, 46 tooth: dental implants restored with porcelain crown, 47 tooth: metal inlay on mesial, occlusal and distal surfaces.

**2.4. Examination of periodontal parameters (Péter Vályi DMD - László Párkányi DMD)**

Periodontal destruction is described with reproducible, comparable parameters during the clinical examination. These basically describe the level of destruction and inflammation, but they also aim to determine prognosis and treatment by evaluating mobility, furcation involvement...etc. Monitoring oral hygiene parameters gives a fair description of patients’ cooperation and motivation.

**ORAL HYGIENE INDICES - Measuring patient cooperation and motivation**

Dentogingival plaque is the main aetiological factor of periodontal diseases. The biofilm contains living microorganisms, which can be removed by scrubbing. Examination can be done in two ways: either by scraping with the tip of the probe or by using a disclosing agent. Showing the coloured plaque to the patient is part of the motivation and instruction. It can demonstrate poor individual oral hygiene, but correct hygiene procedures can also be demonstrated. Home use is also an important part of the instruction and motivation.

Several agents can be used as disclosing agents. Crystalloid iodine, merurochrome, Bismarck brown, Merbromine, Eritrozin, etc. are used as the basis of pharmaceutical products. There are fluorescent dyes visible under photopolimerisation, and ones which change the colour of fresh and mature plaque in a different way. The form of the product is either a pill (chewed by the patient) or a fluid, which can be applied to the tooth surface with a cotton pellet. The lips should be protected with a cream to prevent permanent discoloration. The patient rinses after plaque dying, this way only the attached biofilm will change its colour.
The most important indices of oral hygiene

The quality of individual oral hygiene is measured by plaque and calculus index, which are presented in the table above. Today the role of calculus index is insignificant. The most relevant value is the plaque score, which measures plaque-covered surfaces in percentage of total tooth surfaces. This value is acceptable under 20%. Some authors recommend a fair percentage of 15-25% for acceptable oral hygiene.

DETERMINING THE EXTENT OF INFECTION

Bleeding index

If the gingiva is inflamed or the sulcular epithelium is atrophic or ulcerated, periodontal probing induces bleeding in the sulcus. Probing healthy gingival tissues will not provoke bleeding. Bleeding together with color changes is an early sign of inflammation. Bleeding score is recorded during probing or 30-60 seconds after, if present.

The accuracy of bleeding scores is questionable, but the existence of bleeding is an important prognostic and risk factor in the treatment of patients receiving periodontal therapy. On the other hand, the absence of bleeding usually indicates healthy conditions. Some modifying factors (e.g. smoking – sympathetic effect of nicotine) can mask inflammatory signs (bleeding, color changes).

Increase in sulcular fluid suppuration

Increase in the production of sulcular fluid indicates active inflammation. It can be measured with paper points or a photo-spectrometric device called Periotron.

Purulent suppuration on palpation or spontaneously also indicates acute diseases. One form of this is periodontal abscess, which develops as an acute periodontal infection.

PARAMETERS DESCRIBING TISSUE DESTRUCTION

Probing depth
As a result of periodontal destruction, epithelial attachment shifts apically, the gingival sulcus becomes deeper, and a periodontal pocket is formed. Pocket formation prevents proper individual oral hygiene and creates a perfect environment for periodontal pathogens for reproduction and for sub-gingival biofilm formation. When pocket depth is to be measured, two terms must be familiar: histological pocket depth is the distance between the most coronal cells of the junctional epithelium and the gingival margin. Clinical pocket depth or probing depth is the distance from the gingival margin to the tip of the probe inserted with standard applied force (0.75 N, equivalent to 25 grams of pressure). These two values are different because they are dependent on the resistance of the tissues (probes can easily penetrate inflamed epithelium, but they will not reach the base of the pocket when fibrotic gingiva is present). The examiner and the probe used also influence the measured parameters. For this reason, standardisation is necessary in case of clinical studies so that the difference between the examiners does not become intolerable. This decreases the chance of recording different values for the same situation. Even if there is a single examiner, calibration is recommended by measuring values on the same patient at different times. The difference must not be significant.

Regarding probing depth, normal (0-3 mm), moderate (4-6 mm) and deep pockets (>6 mm) are distinguished. Classification is important to determine tooth prognosis and applied therapy.

Pocket probing is recommended to be performed as “walking measurement”: the probe is guided around the perimeter of the tooth and pocket depths are recorded in mm. The probe is guided into the pocket parallel to the tooth (image 3.7.). Values are recorded on 6 surfaces as far as contact points allow it in order that they will be comparable: mesiobuccal, mesiolingual, distobuccal, distolingual, midbuccal, midlingual.

Bone sounding is also a way of measuring periodontal bone levels. When performing this, the area is under anaesthesia and the probe is forced through the attachment right until the marginal bone level. This examination provides a more accurate understanding of the morphology of bony defects.

Clinical attachment level – clinical attachment loss

Clinical attachment level describes the amount of periodontal destruction and/or the level of regeneration. Like in the case of pocket depth, 2 terms are distinguished regarding this value as well: histological and clinical attachment level. Histological attachment level is determined on a histological segment, while clinical attachment level is measured from the base of the pocket to the cemento-enamel junction.
Mild attachment loss is present if the distance between the base of the pocket and the CEJ is 4-5 mm. This indicates about 1-2 mm actual tissue destruction, because a value of 3 mm is still considered healthy. Moderate attachment loss is present if the measured value is 6-7 mm. Values over this are considered advanced clinical attachment loss.

Clinical attachment loss can also be calculated if the measurement of recession and probing depth is performed at the same site. Recession values are negative if the gingival margin is below the CEJ, 0 if it is at the level of the CEJ and positive if it is above that (hyperplasia).

Clinical attachment level gain shows the result of reparation or regeneration. Epithelial attachment level becomes more coronal compared to the baseline level. This figure does not indicate whether it was regeneration (fibre attachment) or reparation (long junctional epithelium). Probing depth reduction in itself does not indicate the level of healing because in case of unchanged attachment level, by the regression of inflammation (absence of swelling), probing depth can reduce through apical shifting of the gingival margin (recession). The following image (Fig. 3.8.) shows the relationship between probing depth, gingival recession and clinical attachment level:

**Gingival recession**

Recession stands for the distance between the gingival margin and the cemento-enamel junction (crown margin in case of restored teeth). Miller classified recessions based on their relationship to the mucogingival junction and the condition of the adjacent papillae. They were divided into 4 groups (Fig. 3.9.)
Miller’s classification of recessions

**Width of attached gingiva**

It stands for the distance between the base of the sulcus and the mucogingival junction (gingival margin – MGJ distance minus probing depth). Although perfect oral hygiene can maintain periodontal health even if attachment level is at the MGJ, a sufficient amount of attached gingiva is required to support periodontal health, especially around restored teeth (biologic width). Width of attached gingiva is also an important factor when planning periodontal surgical interventions.

**PARAMETERS DESCRIBING PROGRESSION OF PERIODONTAL INFECTIONS**

**Tooth mobility**

Periodontal tissues are built up in a way to allow some oro-vestibular movement of the teeth. The extent under physiological conditions is 0.1 mm, but it can increase to about 0.5 mm during pregnancy. Physiological movement has two components: spatial organisation of rigid periodontal ligaments and mechanical flexibility of supporting bony housing. Pathological mobility is the result of quantitative and qualitative changes in the supporting tissues. Supracrestal fibres of the gingiva are destroyed by inflammatory processes. Composition changes also take place within the fibres. As a result of the spreading infection, periodontal ligaments and supporting bone gets damaged, compromising tooth anchorage. Occlusal overload can also result in tooth mobility because periodontal tissues try to adapt to the change in loading forces and neutralise the load. Widening of the periodontal space and increased mobility are typical in this condition. Anatomical characteristics of the roots can also influence the degree of mobility, not only pathological processes.

Several classifications were introduced to describe tooth mobility (Miller, Genco, Schluger, Carranza FR, Abrahms&Potaschnik, Graces&Smales, Kieser). The classification used today is shown in the table below. Some machines are able to measure mobility in an objective way: in the mid 1980s the Periotest was introduced, but laser-Doppler instruments were also invented. Figure 3.10. shows bidigital measurements compared to Periotest values.
Severity of tooth mobility

Furcation involvement

Periodontal attachment loss may reach the level of the furcation in multi-rooted teeth, which can be an important prognostic factor. It also determines the method of therapy to some extent because these lesions are almost inaccessible by oral hygiene measures, therefore the patient will need surgical correction in most of the cases.

Furcation involvement is evaluated in a horizontal plane according to the Hamp classification (1975), which can be supplemented with the Tarnow and Fletcher classification, which defines subclasses of lesions based on vertical extent. (table 3.2.4.-2). Probing is done with a furcation probe. The number and distribution of roots determine the direction of probing (upper molars: distopalatal, mesiopalatal and buccal, lower molars: lingual and buccal, upper first premolar: mesial and distal).

<table>
<thead>
<tr>
<th>Level of mobility</th>
<th>Manual examination</th>
<th>Periotest value</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Physiologic movement $\mathbf{M} = 0 - 0.2\text{ mm}$*</td>
<td>8 - 9</td>
</tr>
<tr>
<td>I</td>
<td>Horizontal, bucco-oral movement: Physiologic $&lt; \mathbf{M} &lt; 1\text{ mm}$</td>
<td>10 - 19</td>
</tr>
<tr>
<td>II</td>
<td>Horizontal, bucco-oral movement: $1\text{ mm} &lt; \mathbf{M}$</td>
<td>20 - 29</td>
</tr>
<tr>
<td>III</td>
<td>Horizontal, bucco-oral movement: $1\text{ mm} &lt; \mathbf{M}$ Also in axial direction</td>
<td>30 - 50</td>
</tr>
</tbody>
</table>

*can reach up to $0.5\text{ mm}$ during pregnancy
Furcation involvement alone does not determine the prognosis of the tooth or the treatment of choice. Other factors also play a major role: length of root, shape of root, root fusion, different anatomical variations, vertical extent, cervical extent of enamel.

**EVALUATION OF MUCOGINGIVAL MORPHOLOGY – BIOTYPES, SMILE LINE, PAPILLA INDEX**

Mucogingival morphology, besides its functionality, has a great influence on aesthetics, which is important for the patient’s psycho-social health: as a result of the lack of aesthetic satisfaction, smiling may be avoided, non-verbal communication compromised.

Mucogingival morphology is basically determined by genetic parameters – the biotype of the patient. Being familiar with the biotype can help predict the changes caused by inflammation and tooth extraction. The biotype also determines indication, surgical technique and prognosis of mucogingival surgeries. Characteristics of different biotypes are shown in the figure below:
Biotypes and their characteristics

Determining the smile line is important to achieve nice pink aesthetics and also when preparing restorations. Jensen et al (1999) created a classification which describes aesthetics in relation to the smile line. This can help with determining indications for smile line correction.

Classification of smiles based on smile line (Jensen et al, 1999)

The condition and position of the papilla is important when planning restorations. Missing papillae in the interdental region can cause major aesthetic issues, so called “black triangles”. The aesthetic compromise is dependent on the position of the smile line. Relationship between the papilla and the interdental area is based on the Jemt (1997) classification.
Epidemiological studies, indices

Epidemiological studies play an important role in investigating the spread and severity of a disease within a population. They reveal the aetiology of diseases and their relationship with other diseases. These help to determine the need for treatment and required resources. Epidemiological studies work on large samples, therefore they need measures that convert subjective symptoms into objective, numerical data.

Although large sample size, multicenter studies are mostly accepted, some indices for evaluating individual oral hygiene and severity of inflammation are subjective to the examiner. Indices describing periodontal and gingival inflammation are presented in figure 3.15.. Indices measuring the level of oral hygiene are described in previous chapters. The standard Russel and Ramfjord indices have been replaced by indices of precise clinical parameters (probing depth, clinical attachment level).
Gingival indices describing the degree of periodontal inflammation

<table>
<thead>
<tr>
<th>Index</th>
<th>Examined area</th>
<th>Evaluation</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gingival Index (GI)</td>
<td>On four surfaces of teeth</td>
<td>discoloration bleeding on probing</td>
<td>0: no visible inflammation 1: discoloration 2: discoloration, bleeding 3: severe inflammation, bleeding gums</td>
</tr>
<tr>
<td>Papilla Bleeding Index (PBI)</td>
<td>Papillae, 1. and 3. quadrant oral surface, 2. és 4. quadrant facial s.</td>
<td>Bleeding on probing</td>
<td>0: no bleeding 1: A single discreet bleeding point 2: single line of blood appears 3: interdental triangle fills with blood 4: Profuse bleeding occurs after probing</td>
</tr>
<tr>
<td>Gingival Index (GI-S)</td>
<td>On four surfaces of teeth</td>
<td></td>
<td>present (+) non-present (-)</td>
</tr>
<tr>
<td>Gingival bleeding index (GBI)</td>
<td>On surfaces of teeth</td>
<td>Bleeding on probing</td>
<td>$\frac{\text{Bleeding surface}}{\text{all surfaces}} \times 100 = %$</td>
</tr>
<tr>
<td>Bleeding On Probing (BOP)</td>
<td>On surfaces of probing</td>
<td>Bleeding on probing</td>
<td>present (+) non-present (-)</td>
</tr>
</tbody>
</table>

Indices evaluating the need for treatment

Indices evaluating treatment needs in large populations started to spread in the 1970s. Such indices are: PTNS (Periodontal Treatment Need System – Johansen), PSE (Periodontal Screening Examination) and CPITN (Community Treatment Index of Treatment Needs) index. The most commonly accepted PSR (Periodontal Screening and Recording) index is not only for determining the necessary treatment for the patient (dental hygienic, general dental, periodontal) but it is also capable of monitoring periodontal conditions by evaluating and probing every single tooth. Values are recorded for every sextant.

Regular ball-end, two-striped probes are sufficient for measuring. Signs indicate 3.5 mm and 5.5 mm. (Figure 3.16.)
PSR (CPITN) probe

The index considers aetiologic factors (plaque retentive factors), presence of inflammation (bleeding), severity of destruction (probing depth). Additional signs are used to indicate furcation involvement, hypermobility, mucogingival defect, pronounced recession. Criteria of PSR index are shown in Figure 3.17, below:

<table>
<thead>
<tr>
<th>PSR code</th>
<th>Probing</th>
<th>Bleeding</th>
<th>Surface</th>
<th>Restorative margin</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>&lt; 3.5 mm</td>
<td>-</td>
<td>smooth</td>
<td>-</td>
<td>Individual oral hygiene, instruction, motivation</td>
</tr>
<tr>
<td>1</td>
<td>&lt; 3.5 mm</td>
<td>+</td>
<td>smooth</td>
<td>-</td>
<td>Individual oral hygiene, instruction, motivation</td>
</tr>
<tr>
<td>2</td>
<td>&lt; 3.5 mm</td>
<td>+</td>
<td>rough, calculus</td>
<td>irregularity</td>
<td>Professional oral hygiene, finishing restorative margins</td>
</tr>
<tr>
<td>3</td>
<td>3.5 &lt; P &lt; 5.5 mm</td>
<td>+</td>
<td>rough, calculus</td>
<td>irregularity</td>
<td>Full periodontal examination and complex treatment</td>
</tr>
<tr>
<td>4</td>
<td>5.5 mm &lt;</td>
<td>+</td>
<td>rough, calculus</td>
<td>irregularity</td>
<td>Full periodontal examination and complex treatment</td>
</tr>
</tbody>
</table>

* Stands for furcation involvement, hypermobility, mucogingival defect, or recession in that sextant. If the values are 0, 1, or 2: specific treatment of the lesion is necessary. If the values are 3 or 5, complete periodontal examination and treatment is necessary.

Criteria of PSR index and treatment needs

DOCUMENTATION
Monitoring the health of the periodontium needs continuous registration of periodontal parameters. Registration may be electronic or paper based, whichever is more suitable to demonstrate changes in parameters. Lengthy examinations can be reduced in time by using electronic probes and their documentation systems, which were already mentioned in chapters concerning therapy and periodontal instrumentation.

2.5. Occlusal examination (Márk Antal DMD - András Forster DMD)

Maximal intercuspation (occlusion) is the position of the jaws when maximal contact occurs between the upper and the lower dental arch.

Articulation (dynamic occlusion) is the name of occlusal contacts that occur when the teeth move away from the static occlusal position.

**STATIC OCCLUSION**

During intercuspation, the functional cusps of the molars contact the marginal ridges and central pits of the antagonist teeth, creating bi- and tripodistic contacts as seen in Figure 1-2. Bipodistic contacts - also named cusp-ridge contacts - are formed when the functional cusps of the molar teeth contact the marginal ridge of the antagonist teeth. Tripodistic contacts are formed by the contact of the functional cusps and the central pit of the antagonist tooth. In the central pit, the three triangular ridges contact the convex surfaces of the occluding cusp. During occlusion of the front teeth the incisal edges of the lower front teeth contact the palatal marginal ridges of the upper front teeth.

If the occlusion of the molar functional cusps is examined from an orovestibular cross section, three contact points can be defined: A - supporting-functional cusp contact, B - functional-functional cusp contact, C: functional-supporting cusp contact. For the stable occlusion of a tooth, minimum a B plus an A or C point is necessary. The most important is point B, which applies axial load to the antagonist tooth, thus stabilising the position. If a point B is not present, the occlusion is to be considered unstable. So the essential requirement of stable occlusion is the formation of these contacts. In the everyday practice, the idealistic occlusal scheme is not always obtainable, but it is essential to obtain a similarly composed stable situation.

If a tooth does not contact on at least two points as described above, its position is to be considered unstable, thus its position will change as a sum of the mesialisation, eruption, and occlusal forces until an equilibrium is reached. The uncontrolled proceeding of this movement can take part in the development of TMD (TemporoMandibular Disease). If the masticatory organ is not healthy, the attention of the patient should be drawn to it, and the patient has to be referred to a gnathologist or a prosthodontist. The signs and symptoms of TMD are listed in Chapter 2.2.

**DYNAMIC OCCLUSION**

According to the description of the most important gnathology schools, during dynamic occlusion, a phenomenon named anterior guidance is considered ideal. This means that during the frontal movement of the mandible (protrusion) the contact of the incisal edges of the lower incisors and the upper incisor’s palatal surface will guide the movement of the mandible, while the posterior teeth are not in contact (disclusion). During lateral movements of the mandible (laterotrusion), contacts between the maxillary and mandibular canines guide the movement of the jaw (canine guidance), while the molar teeth are discluded. The typical scheme of anterior guidance is presented in Figure 4. It has to be noted that anterior guidance is present in only a part of the population, but the description of all other possible articulation schemes exceeds the goals and limitations of this textbook. During the fabrication of prosthetic appliances, several different occlusal schemes are applied, taking into consideration the type of the prosthesis, the needs of the patient and the goals designed by the dentist. The diagnosis and modification of these schemes is not the task of the dental hygienist.

**ASSESSMENT OF OCCLUSION**

The most important instrument for occlusal diagnosis is the articulation paper/foil. This appliance is accessible in several colours and thicknesses. The selection of articulation foil is determined by the goal of the treatment. For small dental interventions or the diagnosis of a physiological state, thin (8-14 μm) foils are ideal. The application of more colours provides a possibility to differentiate between the static and dynamic occlusal properties. Besides the foils and papers, colour sprays or impression materials can also be utilized for the
examination of the occlusal properties. In case of an all-round gnathologic examination or treatment planning process, anatomical models are mounted into an articulator for precise diagnosis.

Fremitus is also a simple method for occlusal diagnosis. Fremitus is the palpable vibration and movement of the teeth. By examining this phenomenon, occlusal differences can be diagnosed. Method of examination: put a finger on the gingival third of the maxillary teeth in a sitting, unsupported posture of the patient and ask the patient to “click/knock” the teeth. Examine all the teeth systematically and note the degree of fremitus according to your subjective sensation, based on the following classification:

- N=normal (without vibration or movement)
- += One-degree fremitus; only slight vibration can be felt
- ++=Two-degree fremitus; the tooth is clearly palpable but movement is barely visible.
- +++= Three-degree fremitus; movement is clearly observed visually.

This technique offers an easy and reliable detection of contacting pairs with a premature or traumatic contact.

*Examining the occlusion - step-by-step*

1. After opening the mouth the teeth are dried with the three-way-syringe, and an 8-micron articulation foil is inserted between the dental arches in the posterior region on both sides. The patient is then asked to knock the teeth firmly and fast. This process will most likely show the contact points of maximal intercuspation. If we want to check whether an occluding pair or one part of the arch contacts firmly, the articulation foil should be inserted between the examined teeth and the patient is asked to firmly close. Then the foil is pulled, in order to remove it, while the firm closure of the patient is visually controlled. If the foil can be pulled out, it is possible that the examined pair of teeth is not contacting sufficiently and the bite is not in equilibrium.

2. For the examination of the dynamic occlusal properties, a different colour foil is inserted between the posterior teeth on both sides, and the patient is asked to produce laterotrusive (“slide/grind to the side”) and protrusive (“slide/grind forward”) movements. Then the foil is inserted between the front teeth and the same process is repeated. As a last step, the points of maximal intercuspation are marked with the previous colour, as described above. This way colour 2 will show the dynamic occlusal properties, while colour 1 will present the static contacts of the teeth.

**WARNING:** Even the slightest occlusal modification can lead to the development of irreversible and severe temporomandibular and skeletal diseases. Thus the correction or modification of the occlusion is not the responsibility of the dental hygienist and should be carried out by a dentist.

*2.6. Study cast (Márk Antal DMD - András Forster DMD)*

Plaster models are essential for diagnostics and treatment planning. To document the current, starting situation, a study impression is taken, that is the negative copy of the oral structures. Such impressions are mainly performed, using alginate, but for a more precise result, silicone impression materials can also be used. Because of financial reasons, the C silicones are more advised here than the A silicones used mainly for precision impression. Impressions taken from the mouth need to be evaluated carefully (source: Szabó I, Prágai G. Protetikai Propedeutika):

1. Do we have all relevant information in the impression?
2. Is the impression deformed or without any mistakes? Are there any bubbles or rough deformities?
3. An impression with non-sharp, fuzzy edges is unacceptable.

Most impressions are to be filled with gypsum. The well mixed, sour-cream like paste shall be layered on the highest point of the impression. With continuous shaking the whole impression needs to be filled. A base has also to be formed, in order to avoid fractures. For more precise diagnostics and longer usability, super-rigid gypsum is suggested.

The produced study-cast is the positive copy of the oral structures including hard and soft tissues. It should be mentioned that some soft tissues will be documented in a static phase. Impressions taken for removable dentures
can contain dynamic properties as well, but this is not a need in case of a study-cast. It shall mainly serve as a basic diagnostic tool for the patient, documentation and in some cases can be used as a template for temporary denture.

2.7. The basics of dental photography (Andras Forster DMD - Mark Antal DMD)

The basic goal of dental photography is the reproducible documentation of all work phases. To achieve this, special instruments are required. The range of instruments consists of 3 main parts: 1. Digital Single Lens Reflex (DSLR) camera frame 2. Fix focus 60 or 100-105 mm macro lens 3. Macro flash (ring-flash or twin-flash). This armamentarium is completed by a range of photographic (battery, memory card, and cases) and dental (retractors, contractor, and mirrors) accessories. To be able to process and use the captured images, appropriate hardware and software is also necessary.

The reproducibility and comparability of the captured images is provided through strict following of photography protocols. These protocols can be developed to be different for each field of specialty and dentist, as influenced by certain needs and goals. After developing and learning the protocol, strict correspondence with the pre-defined rules and objectives makes dental photography a really versatile and powerful tool. A complex photographic protocol may consist of 30 or more images occasionally. In this chapter, a simplified photographic protocol is presented, which will allow for sufficient documentation of the dental hygiene treatment phases.

9 IMAGE PHOTO DOCUMENTATION PROTOCOL FOR DENTAL HYGIENISTS:

General rules: Every image is captured in landscape format. All images should be free of debris, blood, dirt, saliva and all visually disturbing factors. A non-distracting, homogeneous (grey, black, or blue) background should be used. The crop factor of the utilised camera should be taken into consideration when setting the magnification ratios.

Full face

Camera settings: lens f: 8.0, shutter speed: 1/60 s. The nose of the patient is the middle of the image. The vertical midline of the image is the midline of the face, while the horizontal midline is determined by the bi-pupillary plane. The patient presents a full smile. The optical axis of the camera is perpendicular to the facial plane. The focus is set to the lateral incisors.
1:2 ratio full smile

Camera setting: lens f: 22, Shutter speed: 1/125 s. The centre of the frontal image (2) is the approximate contact point of the maxillary central incisors. The horizontal midline is determined by the bipupillary plane (do not compensate for the canting of the teeth!), while the vertical midline is determined by the midline of the face. The centre of the two lateral images (3,4) is the lateral incisor. The horizontal midline is determined by the incisal plane of the maxillary teeth, and the vertical midline is parallel to the midline of the face. The patient shows a full smile with closed teeth for all three pictures. The focus is set to the lateral incisors.
Camera settings: lens f: 22, shutter speed: 1/125 s. To capture the described images, the retraction of the soft tissues with a suitable retractor is essential. All three images are captured with slightly parted teeth, so the lower incisal edges become visible. The retractors should be held symmetrically with the contour of the gingiva visible on all surfaces. Focus is determined at the lateral incisors. This image should be captured from frontal (5) and semi lateral 45 degree (6–7) angle.
Camera settings: lens f:22, shutter speed: 1/125 s. A retractor and an occlusal dental mirror warmed to body temperature is necessary to obtain correct images. The mirror is in 45 degree angle to the occlusal plane of the dental arch with the distal part of the mirror touching the second molars, and the picture showing the buccal surfaces of the central incisors. The distal marginal ridge of the first molar should be visible in the image. The optical axis of the camera should be set at 45 degree angle to the mirror, this way being parallel to the occlusal plane. The two quadrants should be symmetrical. The focus is set to the occlusal surface of the premolars.
3. Radiographic examination (Zoltán Baráth DMD)

The aim of x-ray examinations is to diagnose pathological conditions in an early stage, to differentiate the disorder from other diseases with similar symptoms, to localise malfunctions, to define the prognosis of the disease and to check dental therapy as well as their therapeutic effects.

The fact that the objects on radiographs are distorted, superimposed on each other and some processes mimic others should be considered in every case.

![Distorted and superimposed dental implant on OPG](image)

Distorted and superimposed dental implant (14) on the OPG, but the real situation on the periapical film in the upper left corner

**BASICS OF X-RAY TECHNIQUES**

**Production of X-ray**

X radiation is a form of very short wave electromagnetic ionising radiation. When a wolfram spiral (cathode) with high melting point is heated electrically in an x-ray tube, electrons are emitted. The electrons accelerate within a vacuum containing a high-voltage electric field and then strike the effective focal spot of the anode composed of wolfram. During this impact, ionisation, excitation, characteristic x radiation and braking radiation are formed and 1 % of the original energy turns into x radiation (photon). The remaining energy is transformed into heat.

X radiation cannot be detected by our senses. They spreads spherically, linearly in all directions at the speed of light from the point of origin and they cross, scatter, or absorb either wholly or partially, in material depending on its characteristics and density.

The radiation ionises matter and may cause scintillation and fluorescence as well as biological effects in living tissues. X-rays will darken a light sensitive emulsion and may cause chemical effects by breaking chemical bonds.

**X-ray machine**

**X-ray tube**

A. The **tube head** is a made of heavy metal, contains the x-ray tube and minimises primary radiation.

The x-ray tube is an air-evacuated glass envelope or tube that is composed of a **cathode** soldered to one end, and the **anode** to the other. It is covered with insulating oil for filtration of the x-ray beam.

- The **cathode** is a rod shaped wolfram spiral which emits electrons when a heating current is applied.
• The cathode is placed in a molybdenum cathode cup.

• The anode is made of wolfram and is perpendicular to the electron beam.

• The tube window is a window from which the beam exits the X-ray tube.

The aluminium filter is a disk 0.5 mm in diameter that absorbs soft x-rays which do not play a role in imaging, but increase the exposure to skin and soft tissue.

The collimator narrows the beam coming through the x-ray gate, thus decreasing the size of the irradiated area.

B. The aiming cylinder directs the beam precisely to the examined area and provides the fix focus film distance.

The power supply provides the anode voltage and heating current as well as allowing for proper operation.

X-ray image and film

X-ray film is a light and radiation sensitive material suitable for making x-ray images. Its largest area is the polyester film carrier layer. It has a gelatine and plastic fixing layer on both sides, which fixes the light sensitive emulsion layer to the carrier layer. The emulsion layer contains silver halogen particles suspended in gelatine and it is where the x-ray image is formed upon radiation. There is a hard gelatine protective layer on both sides of the surface.

Processing conventional x-ray film begins with development, followed by rinsing, fixing, washing and drying. During development, the silver ion is reduced to metal silver particles in exposed silver bromide crystals, which appear black in the emulsion. During fixing, the fixing liquid releases the non-exposed silver bromide particles. Sharpness is an important characteristic of x-ray images and an image is regarded sharp if the object point corresponds to the image point. It is affected by geometric factors, movement, emulsion of the film, intensifying screens and subjective factors.

Characteristics of x-ray image

Film contrast describes the difference between dark and bright areas of an image. If there are black and white areas in the x-ray image, the photo is contrasted, and is termed short-scale of contrast. If the image is grey scale and is less contrasted, it is termed long-scale of contrast.

Object contrast is caused by differences in radiation absorption of the different parts of the object that have various densities. It is affected by the amount and quality of radiation, as well as by scattered radiation.

Film contrast influences how the object contrast appears on the film.

The contrast of the X-ray image depends on the characteristics of the film and object contrast.

Digital techniques

We need traditional x-ray equipment to make x radiation when a digital technique is used take the image. The image is taken on a sensor or detector that is similar in size to the analogue film. The final image is made by digital conversion of the analogue data and is composed of pixels and 256 shades of gray. The image can be displayed and examined on a computer monitor.
Digora Optime for intraoral diagnostics
Digora PCT for extraoral diagnostics

Advantages of digital techniques

- Easy to retrieve
- No need for film development (equipment is expensive, large and hazardous waste is generated)
- Stores large quantities of data
- Transferable
- Long-lasting quality
• All data are stored in an electronic archive •Analogue-digital conversion is necessary, latent image turns into an analogue one

**Types of digital techniques**

• When using CCD, the image recording semiconductor is coated with luminescent material and the optical image has the same light intensity as the x-ray. The CCD receives it and light intensity is converted into electric charge in the semiconductor light sensor. The analogue signal voltage belongs to each pixel and is proportional to the charge and radiation. After readout, the signal voltage is coded to greyscale by an analogue to digital converter.

• When using PSP, the electrons of europium absorb the x-rays and reach the conducting line of the BaFX crystals. The number of electrons is proportional to the x-rays. When the laser light scans, luminescent photons are emitted. The light intensity corresponds to a particular signal voltage. Associating the proper shade of grey to the corresponding signal voltage forms the digital image.

**Radiation protection**

The ALARA standards regulate the reduction of radiation exposure, ensuring the reduction of the individual dose, the number of exposed people and probability of exposure. The benefit of x-ray imaging should balance by the possible risk of radiation. Utilizing more sensitive film and enhancing intensifying screens are suggested in case of extraoral images. Proper processing technique, an appropriate size of x-ray gate, precise darkroom work and applying new procedures with reduced exposure are all very important.

The patients’ radiation protection should avoid somatic and genetic effects. The reduction of radiation exposure is achieved by using long tube technique, a higher kV value and filters. According to the radiation protection regulation, 0.25 mm lead equivalent aprons with thyroid protective collars should be used to protect against primary radiation. X-ray operators should be protected by maintaining a safe distance (minimum 3m), using a shielding wall and measuring exposure continuously. The exposure is measured with dosimeter, and if the maximum dose limit is reached, the operator may be suspended from work temporarily or permanently.
**X-RAY IMAGING TECHNIQUES**

*Intraoral x-ray imaging techniques*

*Periapical images*

This image primarily shows the root tip and the surrounding bone. Using the bisecting angle technique, the tooth and the film are angled so that the length of the tooth is different and distorted compared to that of the original tooth. The central beam is directed at a right angle to a plane that bisects the tooth and film so as to keep the tooth isometric. The image becomes distorted as the root and the crown are projected shorter. When taking images on upper teeth, the tragus-nasal line is horizontal. In case of lower teeth, the tragus oral commissure panel is horizontal.

The long or short bisecting plane of the film should be parallel to the longitudinal axis of the tooth. The film should be placed behind the tooth, 5 mm over the occlusal plane, and the examined tooth should be placed in the centre of the film. The central beam should be at a right angle to the bisecting plane of the film and tooth and should direct to orthoradial and apical directions.
Bisecting angle technique

The orthoradial beam should be parallel to the approximal surface of the teeth. To separate the overlapping structures we take eccentric images, which are distoexcentric in case of a distal beam from the orthorodial direction, and they are mesioexcentric in case of a beam from the mesial direction. The apical direction central beam should be oriented on the tragus-nasal line at the maxillary teeth, and on the parallel line above the mandible base at the lower teeth.

Taking an undistorted image is the main purpose of the parallel technique. In this case the x-ray image is slightly distorted and enlarged, and the exposure is smaller. It is also known as the Fitzgerald technique. The film should be parallel to the long axis of the tooth. The object film distance should be increased. There are film holders to help position the film.
parallel technique

In case of using the right angle technique, the film holder is connected to the tube and x-ray tube so that the central beam should always be oriented to the middle of the film at a right angle.

Structures in the x-ray image:

The crown is visible as a cross section with a sharp outline of the enamel in the proximal, occlusal and incisal surfaces. The pulp is projected mesiodistally and the wider the pulp is, the darker the image. The root canal appears as a dark line in the light projection of the dentine covering the root canal, and it can be blurred at the root tip in the projection of the dentine. On the mesial and distal side of the root, the lamina dura is projected lightly and the periodontium is projected darkly. Identifying and examining the periapical area is essential in diagnostics. The condition of cancellous bone is highly important in the examination of pathological lesions. The lacunas are projected darkly, while the trabeculas and lamellas are projected brightly. Gingiva is not usually visible in the image.
Foreign bodies absorb radiation differently than tissues do. The amalgam fillings absorb more rays than tissues, so they are projected radiopaque. The metal inlays are as radiopaque as the amalgam, but their outlines are smoother. The composite fillings contain a radiopaque material, thus their radiation absorption is greater than that of dentine. The calcium-hydroxide liners absorb radiation similarly to fillings and they cannot be differentiated on radiographs. The temporary filling of zinc oxide sulphate cement and the gutta-percha appear brightly on x-ray imaging. The radiographic image is suitable for examination of fillings, inlays, crown marginal closure, missing contact point, caries and marginal periodontium. The whole porcelain crowns appear more radiolucent than dentine. Prostheses appear according to their material. The glass fibre posts appear lighter than dentine, and their x-ray image depends on their radiation absorption.

The metal implant placed under the periosteum in the bone or through the root canal in the bone appears radiopaque and has sharp outlines in the radiographs. In case of successful osseointegration, there is no radiolucency between the implant and the bone. In case of connective tissue fixed implants, there is radiolucency around the implant.
Indications of periapical images:

It is essential to take radiographs to carry out root canal treatment precisely. Before root canal treatment, the dentist must examine the root canal, the apex, the apical periodontium, the marginal periodontium and relation between the tooth and the surrounding anatomical details. During root canal treatment, the dentist examines mechanical processes (file, poen control), the definitive root filling and the complications (fractured instruments, via falsa). To assess the results of root canal treatment, radiographs should be taken about the root canal treated tooth 6-7 months after treatment, and can be compared with radiographs taken during the treatment.
Radiograph to assessment of root canal treatment

In case of *pectomy* or *amputation*, the radiograph should help to determine whether surgical intervention is indicated or not, to examine the condition of root canal fillings, the outcome of the surgery, healing, and possible obstacles. In case of tooth hemisection, the radiograph helps to examine the scale of root divergency, the apical and marginal periodontium, and the condition of root filling in the remaining root.

In case of *extraction*, the radiograph helps to determine the indication of extraction, the planning of surgical intervention and possible complications.

The **full-mouth intraoral radiographic examination** gives an overview of the condition of teeth and periodontium. In practice, there are 11, 14, 16 and 18 film statuses, which can be periapical and bitewing images.

Localisation in periapical images:

Radiographs show the two-dimensional image of the object. The sharpness of the objects gives information about their positioning. Shorter object-film distances produce sharper and smaller images than longer object-film distances.

Parallax phenomenon: the image of the examined object is shifted if the viewing angle changes. If the central beam is moved in horizontal and vertical directions, all objects that move in an opposite direction to the focal point are buccal to the other tooth, and those that move in the same direction are palatal (lingual) to the other tooth. Nitsche-Vályi method: localisation of upper canine retention.

**Bitewing examination**

Bitewing radiographs delineate the proximal surfaces, and are valuable for detecting interproximal caries, as well as for evaluating the interdental septa and stages of bone decay.

The bitewing image is taken by bitewing film. The head position is normal, the bitewing film is placed behind the maxillary and mandibular teeth parallel with the long axis of the teeth, and the tab of film is bitten on. The film is placed in the standing position at the frontal teeth, and in the lying position it is placed at the posterior teeth. The tube angle should be set at + 5-10 degrees. The central beam is directed to the middle of the film and orthoradial.
Occlusal radiographs

In case of *anterior maxillary* occlusal projection, the head position is normal, the long axis of the film is parallel with the midsagittal plane, and the point of entry at the border of the middle and upper third of the bridge of the nose and the vertical angulation should be +65.

In case of *cross-sectional maxillary* occlusal projection, the short axis of the film is parallel with the midsagittal plane, the point of entry at the root of the nose, and the vertical angulation should be +80.

In case of *lateral maxillary* occlusal projection, the long axis of the film is parallel with the midsagittal plane, and on the side of interest, the point of entry at fossa canina and the vertical angulation should be +65.

In case of *anterior mandibular* occlusal projection, the head is tilted back, the occlusal level is determined with horizontal level +55 degree, the long bisector line of film is parallel with the midsagittal plane and the point of entry is gnathon.

In case of *cross-sectional mandibular* occlusal projection, the head is tilted back, the occlusal level is vertical, the short bisecting line of film is parallel with the midsagittal plane and the point of entry is at the middle of the floor of the mouth.

*Lateral mandibular* occlusal projection: The occlusal level is vertical, the long bisecting line of the film is more lateral from the middle level according to the examining side, the central ray is directed to the axis of the second premolar, and the examination is axial.
Extraoral image techniques

Images are taken on extraoral film closed in cassette.

Tomography

It is a radiographic technique designed to more clearly image objects lying within a plane of interest. This is accomplished by blurring the images of interest through the process of motion “unsharpness”. The plane of interest and its thickness can be selected. The images of objects located outside the focal plane have continuously changing positions on the film; as a result, the images of these objects are blurred beyond recognition by motion unsharpness.

Panoramic tomography is a technique for producing a single tomographic image of the facial structures that includes both the maxillary and mandibular dental arches and their supporting structures.

Two techniques are the panoramic-layer projection and the magnified panoramic projection.

Pantomography is a layer projection procedure in which the mandible and the maxilla are represented on one film. X-rays are orthoradial to compare certain tooth groups, thus the name, orthopantomography. The examined patient’s head is fixed in a head console, the median-sagittal level is vertical and the Frankfurt plane is horizontal.

The patient’s head position is symmetric and bites on the edging bite. The rotation centre moves forward along an arc on a 200-240° angle from right ramus to left ramus, and the film in the cassette move in the opposite direction of the x-ray tube.

Sinus-OPG presents the sinus in the plane of interest. The TMJ can be delineated in opened or closed positions.
Magnifying panoramic radiograph

It is made by an intraoral x-ray tube on an extraoral radiograph. The distance between focus-object is short because of the x-ray tube position and is why the image is magnified.

In case of the maxillary magnifying panoramic radiograph, the cassette is located on the face symmetrically around the tip of the nose, and is fixed by the patient with his hand. The occlusal level of maxillary teeth is horizontal and the patient must bite on the applicator with the incisors. The tip of the the applicator must be in touch with the palate. The angle on the horizontal level is +15°. This radiograph is a good survey of the whole maxilla and particularly the middle part of it.

In case of the mandibular magnifying panoramic radiograph, the film and patient position are the same as with the maxillary magnifying panoramic radiograph. The tip of the applicator must push in the medial-saggital level at the line connecting first or second molars. The angle on the horizontal level is -5°.

CBCT

A 3D cone or pyramidal divergent x-ray beam is directed through a central object onto an area detector, making series of two-dimensional summated images. Software programs incorporating sophisticated algorithms generate the 3D volumetric data. During image acquisition, the x-ray beam rotates 360° or fewer angles. During rotation, many exposures are made at fixed intervals. Thus, the radiation exposure can be reduced by 30-50 times. Voxels are isotropic, therefore the spatial resolution is the same in all directions of space. Thinner image layers result in the elimination of stairs artifacts that allows for homogenic 3D reconstruction. The CBCT is imprecise in radiographic density and soft tissue visualization. There are significantly fewer metal artifacts and the software of CBCT can be easier to use than CT.
CBCT can be used in dysgnath surgery, orthodontics, TMJ diagnostics, implantology, otolaryngology, developmental disorders and dento-maxillofacial traumatology.
We can study the volumetric data with software using different image views. The multiplanar view is the main basic view in case of all CBCT machines. According to the indication, we can select panoramic view, single cross-section, series of cross-sections and 3D reconstruction.

Extraoral radiographic examinations projecting to occlusal film

Mentum projection

The middle plane of the head is vertical; the film is placed under the mentum, the short bisecting line is parallel with the middle level; the mouth is open; the film is fixed by the patient; the central ray is directed to the middle of the lower lip.

Inflated bucca projection

The film is fixed by the patient behind the inflated side of the face in the frontal level, so that the long bisecting line of the film is parallel with the middle level and the central ray is directed towards the bucca. It is suitable for showing the stones in the Stenon duct.

Lateral cephalometric projection

The aim of projection is for the structures on the side near and far from the image receptor to be presented isometrically. It is suitable for orthodontics, maxillofacial surgery and anthropological research. It is a skull projection in which the focus-film distance is 1.5-4 m, the patient’s midsaggital plane is parallel with the film or image receptor, the Frankfurt plane is in the horizontal level and the zygomatic arch and zygoma near the film is in contact with the cassette. We can study the craniofacial morphology, dental position and occlusion analysis.
THE FUNDAMENTS OF RADIOGRAPHY

Radiological diagnosis of dental anomalies

Irregular size or form of teeth

- macrodontia: teeth are larger than normal, may indicate the presence of a haemangioma or can occur in hemihypertrophy of the face.
- microdontia: the involved teeth are smaller than normal.
- dilaceration: a disturbance in tooth formation that produces a sharp bend or curve in the tooth.
- taurodontism: taurodont teeth have longitudinally enlarged pulp chambers.

Irregular number of teeth

- hypodontia spuria: it is an illusory absence of the tooth, while in reality it is retained in the jaw.
- hypodontia vera: it is a true absence of the tooth germ.
- oligodontia: the absence of numerous teeth, the whole group of teeth may be missing.
- anodontia: the failure of all teeth to develop, it is an ectodermal syndrome.
- hyperodontia: development of more teeth than normally.
  1. Supplemental teeth: image of the available tooth.
  2. Supernumerary teeth: atypical teeth with irregular structure, mainly in the upper jaw.

Irregular position

- torsion, rotation: the tooth turns away around its axis.
- extraversion: the tooth leans out from the arch.
- intraversion: the tooth leans into the arch.
- infraocclusion: the tooth occlusal surface is lower than the neighbouring tooth occlusal surface.
supraocclusion: the tooth occlusal surface is higher than the neighbouring tooth occlusal surface.

transposition: it is a condition in which two teeth have exchanged positions. The most frequently transposed teeth are the permanent canine and first premolar.

migration, distalisation: the tooth develops far from its original position.

heterotrophy: the tooth develops a totally different position than normally.

Developing irregularity

Retention: one or more teeth stay behind in the jaw beyond the developing time.

Impaction: the tooth is impacted or cannot develop because of the narrow space.

Purpose of the radiologic examination:

• determination of the retention
• situation of the retained teeth
• dimension of the retention
• number of the retained teeth
• cause of the retention

Retained wisdom-teeth

• vertical: the tooth is vertical.
• mesioangular: the tooth crown leans towards the second molar.
• distoangular: the tooth crown leans towards the ramus of the mandible.
• horizontal: the tooth position is horizontal, the axis is perpendicular to the second molar.
• transversal: the tooth position is horizontal but the crown is in oral or buccal position.
• inverse: the tooth is in the opposite orientation.

Structure irregularity

• Acquired enamel hypoplasia: Dark marks or sharp-bordered horizontal wrinkles are visible on the radiologic image.
• Amelogenesis imperfecta: It is a developmental disturbance, there can be changes in the enamel of all teeth in both dentitions.
• Dentinogenesis imperfecta: A developmental disturbance primarily of dentine. Both deciduous and permanent dentition may show this defect.

Radiographic diagnostic of dental caries

The lesion is seen in the radiograph as a radiolucent (darker) zone in the light contour of the tooth to the pulp chambers.

Proximalis caries

• It is most commonly found in the area between the contact point and free gingival margin.
• Superficial caries is classically a triangular radiolucency with a broad base at the tooth surface.
• The actual depth of penetration of a carious lesion is often deeper than seen radiographically because the lesion may not be evident until approximately 30% to 40% demineralization has occurred.

• Medium caries involves dentine as a triangular radiolucency with its broad base at the enamel surface.

• Profound caries can be well visible.

• Secondary caries developing at the margin of an existing restoration can be well detected.

**Occlusal caries**

• The demineralization process originates in enamel pits and fissures of posterior teeth where bacterial plaque can gather.

• Superficial caries is often obscured by the surrounding enamel.

• Caries in dentine appears as a radiolucent line along the dentino-enamel junction.

• Secondary caries can only be detected by radiograph.

**Buccal and lingual caries**

• It is a well-defined circular radiolucency area with parallel noncarious enamel rods surrounding the buccal and palatal lesion.

• Superficial caries cannot be detected because healthy dentine in front of and behind the lesion may mimic the radiolucency.

**Root surface lesion**

• It involves both cementum and dentine and is associated with gingival recession by periodontitis or other reasons.

**Misinterpretation in radiographic diagnostic**

• Undiagnosed caries – because of teeth superimposition or settings

• Other radiolucency under deep filling which may be diagnosed as caries – burn out effect, dentin pseudotransparency

**The change of the pulp and its consecutive diseases**

• The contour of the pulp chamber and not the pulp can be visible on radiograph.

• We can diagnose the lesions that may result in pulpitis.

• Causes of pulpitis: ocaries oenamel - dentin attrition ocervical root resorption odecaying of alveolar process otrauomatic injuries

**Acute periapical periodontitis**

There are no radiographic changes in very early lesions, 5-10 days are needed to develop typical radiographic signs. The first radiographic signs are the widening of periodontal space and the loss of bone density.

**Acute periostitis**

It cannot be diagnosed on radiograph. Discontinuity of periodontal space can be detected around involved a tooth only when radiographic signs are visible in the case of a chronic periodontitis flare up.

**Chronic apical parodontitis**

It is the chronic inflammation of apical periodontium and typical radiographic signs can be detected. Inflammation develops initially when toxic metabolites from necrotic pulp exit the root apex to incite an inflammatory reaction in the periapical periodontal ligament and surrounding bone. Granulated tissue is formed
because of the inflammation and the bone undergoes resorption, termed ostitis rareficiens. The resorbed bone absorbs less radiation and radiolucency, osteolytic lesion can be visible on radiograph. When most of the lesion consists of increased bone formation, the term periapical sclerosing ostitis is used. The radiograph demonstrates the position and extension of bone destruction in the surrounding periapical osseus structure. The chronic apical periodontitis is chronic inflammation of the periodontal ligament and granulated tissue around the apex. Widening of the periodontal space or thin radiolucency can be detected on radiograph.

**Periapical granuloma**

The periapical granuloma is a formation of granulation tissue mixed with a chronic infiltrate. It is a round shaped osteolytic lesion with a sharp border around the apex. The lesion may appear as a small cyst or granuloma on histological examination but the radiographic signs and treatments are very similar. Lesions larger than 1 cm in diameter are usually radicular cysts.

![Periapical granuloma](image)

**Chronic periapical abscess**

Chronic periapical abscess is long lasting inflammation around the apex; an abscess that is encapsulated by connective tissue. It is a diffuse, irregularly shaped osteolytic lesion of which radiolucency is milder than for granulomas and cysts.

**Chronic periapical progressive**

In case of chronic periapical progressive, the secretion is drained through the fistula. The fistula is rarely visible on radiograph. The fistula’s relation to the involved tooth can be determined by inserting gutta-percha into the fistula and taking a radiograph with it.

**Chronic apical periodontitis ossificans**

In case of chronic apical periodontitis ossificans, the infection is mild. It causes osteoblast hyperactivity in periodontium, which causes hyperossification. Sclerotic bone can be visible around the apex that can be residuum after extraction. It can be misinterpreted as odontoma, osteoma, hypercemetosis or focal sclerotic osteomyelitis.
Diagnostic failures

• we interpret other lesions as periapical periodontitis
• we do not diagnose the periapical pathosis

1. The periphery of osteolytic lesion may be sharp or blurred.


3. Anatomical and radiographic reasons. The negative radiographic findings do not exclude the existing apical periodontitis.

Acute osteomyelitis

Acute osteomyelitis is an acute inflammation of the bone marrow and the most common location is in the posterior body of mandible. No radiographic changes may be identifiable, the density of the involved area is slight decreased with a loss of sharpness of the existing trabecula. Sequestra may be present but usually are more apparent and numerous in chronic forms. It can stimulate either bone resorption or bone formation and the inflammatory exudate can lift the periosteum and stimulate bone formation (onion-skin appearance).

Chronic osteomyelitis

Chronic osteomyelitis is a chronic inflammation of bone marrow, which may be a sequelae of inadequately treated acute osteomyelitis. The periphery may be more well-defined but it is still difficult to determine the exact extent of chronic osteomyelitis. The typical radiographic sign is of sequestra, which appear as a radiopaque island of nonvital bone. The sequestrum is demarcated by a radiolucent zone.

Radiography of periodontitis

It complements the clinical examination. The ideal imaging techniques for diagnosis of the loss of periodontal attachments are the periapical radiographs with parallel technique or bitewing radiograph. The alveolus, periodontal ligament space, lamina dura, roots, migration of teeth, calculus and crown-root ratio can be evaluated on the radiographs.

Evaluation of the alveolar process

The height of the healthy alveolar crest is 1-1.5 mm apical to the cemento-enamel junction of adjacent teeth. The healthy septa among the frontal teeth are triangular and surrounded by cortical bone. The lamina dura of posterior teeth form a sharp, well-defined angle with the alveolar crest. The white line at the top of the alveolar crest is the external line that is the continuity of the lamina dura. The bone decay originates at the alveolar crest and progresses to the apex of the root.

Changes in morphology

• In case of early bone changes the anterior region shows blunting of the alveolar crests and slight loss of alveolar bone height. The posterior regions may also show a loss of the normally sharp angle between the lamina dura and alveolar crest.

• Horizontal bone loss is a term used to describe the radiographic appearance of loss in height of the alveolar bone around multiple teeth; the crest is still horizontal but is positioned apically more than a few millimetres (2mm) from the line of the cemento-enamel junctions. Horizontal bone loss may be mild, moderate, or severe, depending on the extent. Mild bone loss may be defined as approximately 1 mm of attachment loss, and moderate loss is anything greater than 1 mm up to the midpoint of the length of the roots or to the furcation level of the molars. There are equally deep soft tissue pockets next to the neighbouring teeth.

• In case of vertical osseus defect the crest of the remaining alveolar bone typically displays an oblique angulation to the line of cemento-enamel junctions in the area of involved teeth. The infrabony defect is a vertical deformity within bone that extends apically along the root from the alveolar crest. The three-walled infrabony defect has remaining buccal and lingual cortical plates. The two-walled defect has one resorbed
cortical plate and in case of one-walled defects both cortical plates have been lost. The one-walled bone defect is termed a proximal crater.

- The buccal and lingual bone defects are difficult or impossible to recognize on a radiograph because one or both of the cortical bony plates remain superimposed with the defect.

- Visualization of the depth of pockets may be aided by inserting a gutta-percha point of periodontal probe in the periodontal pocket.

Widening of the periodontal ligament space at the apex of the interradicular bony crest of the furcation is strong evidence of furcation involvement in periodontal disease. In case of sufficient bone loss of interradicular septa and buccal and/or lingual plates, the radiolucency of the lesion becomes prominent. The inflammation may result in the loss of density and number of trabeculae or cause deposition of bone on existing trabeculae at the expense of the marrow resulting in a more radiopaque image. If the inflammation spreads on the periodontal ligament, granulation tissue develops with resorption of the lamina dura. Widening of the periodontal space can be detectable on the radiograph.

Periodontal lesions: infrabony pocket and furcation involvement
Calculus deposits can prevent effective cleansing of a sulcus and lead to the progression of periodontal disease. Parts of the calculus on the approximal side of the teeth rise from the contour of the teeth. Subgingival calculus can be narrow like a ring around the neck of the teeth or on the approximal side they can rise from the root’s contour like a prickle, irregular radiolucent area.

**Pericoronitis** (operculitis): the term pericoronitis refers to inflammation of the tissues surrounding the crown of a partially erupted tooth. It is caused by bacteria under the gingiva. The radiographic signs of pericoronitis can range from no changes when the inflammatory lesion is confined to the soft tissues, but if spread to the surrounding bone, it seems to be rarefaction and/or sclerosis. The typical radiologic sign is resorption semilunaris which is a semilunar rarefaction in the distal region.

**Radiographic diagnosis of traumatic injuries**

The radiologic examination is essential when we evaluate the results of injuries. The radiograph provides information regarding the location and extent of the fracture and shows the movement of the fracture’s borderline and how to move off the parts. We can check recovery and long term changes with the follow-up radiologic examination. General signs that may indicate the presence of a fracture: the presence of a radiolucent line within the anatomic boundaries of the structure, a change in the normal anatomic outline or shape of the structure, an increase in the density of the bone, which may be caused by the overlapping of two fragments of bone, a defect in the outer cortical boundary or a step-like defect. We don’t regard the radiolucent dark line on the radiograph as a fracture if it runs over the bones contour and finishes in the soft tissues. As a rule, if we can’t diagnose the fracture after 8-10 days because of the absorbing processes then we should perform the diagnosis with follow-up radiographs.

The fracture may be missed if the plane of the fracture is not in the same direction as the x-ray beam. For this reason multiple films at different angulations should be used.

**Tooth fracture**

Fractures can be demonstrated well in radiographic examinations if the central beam is 15-20° to the occlusion level. The fracture line can be localised more easily 8-10 days after because of physiological processes. When
the fracture line on the film is not visible, it does not mean there are no fractures present. At that time a new radiograph must be taken with another central beam value, and it is necessary to do a control radiograph 8-10 days after.

Fracture of 25 tooth and lesions around fracture

In **luxation**, such teeth are abnormally mobile, angled, displaced, the alveolar bone may be injured or fractured, and the sole radiographic finding may be a widening, narrowing or obliteration of the apical portion of the periodontal ligament space.

**Avulsion** is the term used to describe the complete displacement of a tooth from the alveolar process.
Late consequence of traumatic injuries of 23 tooth: replacement resorption

**Bone hyperplasias**

**Exostosis:** External bone hyperplasia in which the radiographic image is more radiopaque than the surrounding tissue and can be accurately diagnosed with the occlusal radiograph.

**Enostosis:** Internal bone hyperplasia. It is a homogenic radiopaque circular or ovoid lesion. It can be differentiated from cementoma by the blurred periphery to the normal bone. Some enostosis are the consequence of chronic apical periodontitis ossificans.

**Radiographic diagnostic of cysts**

The cyst is a pathologic cavity filled with fluid, lined by epithelium, and surrounded by a well-defined connective tissue wall.

Radiograph determines

- cyst
- type
- size
- relation to the surrounding structure
- growing condition

In the differential diagnosis of cysts, we evaluate the position, periphery, shape, the uni- or multilobular appearance of the lesion, and the periodontal ligament space of the involved tooth or neighbouring tooth. They can be generally evaluated as a radiolucent lesion.
Radicular cyst of 12 tooth. 14 tooth dislocated by cyst.

**Radiographic diagnostic of tumours in jaws**

Tumours in jaws present radiographic findings by conventional imaging techniques. The tumours can be **odontogenic** or **nonodontogenic**.

Odontogenic tumours are generally benign tumours with sharp borders on radiograph.

Nonodontogenic tumours originate from the stomatognatic system, excluding the dental tissue. Extraoral image techniques are needed for tumour diagnosis. The benign tumours are generally radiopaque (osteoma) or radiolucent lesions with sharp borders. The malignant tumours are generally radiolucent and have the typical appearance of ill-defined borders with lack of cortication and absence of encapsulation. Residual islands may appear in the internal structure resulting in a pattern of patchy destruction with some scattered residual islands of bone. The malignant tumours can stimulate the formation of thin straight spicules of bone giving a ‘hair-on-end’ or sunburst appearance. The Codman’s triangle is a very important radiographic sign that is the result of gross disturbance to the overlying periosteum.

**4. Further examination methods (Ákos Nagy DMD)**

Both the development and progression of oral diseases are appreciably influenced by the medical condition of the patient and the oral microbial or the present periodontal pocket flora.

There are microbiological methods to test the oral flora and laboratory tests for testing the patient’s condition. They could be genetic or special tests reflecting on the different pathophysiological processes and the effectivity of certain therapeutic options.

If the results of the clinical examination suggest that the morphology of the oral cavity is abnormal, further investigations are needed. The histological examination may be helpful in establishing the diagnosis and selecting the proper management.

During the extended examination of the dental patients, blood, saliva and crevicular fluid samples can be analysed.
Testing the oral fluid, we can predict the possible risks of the development of periodontal diseases (1,2,3,4). We can also monitor the progression of the disease or the healing process and the outcome of the disease.

Nowadays genetic tests (analysing the blood and mucus samples of patients) have come to the forefront of investigations (5,6). Since periodontitis is a combined consequence of the bacterial infection and the host immune response, each genetic variance can influence the individual response to the biofilm.

4.1. Laboratory tests

The above mentioned laboratory tests are used in the differentiation between oral diseases based on the analysis of blood samples.

HAEMATOLOGICAL TESTS

Typical periodontal alterations can be identified in immunocompromised patients both in acquired immunodeficiency (AIDS) states, and in therapeutic immunosuppression (organ transplantation or tumorous diseases). HIV positive patients are markedly predisposed to developing chronic destructive periodontitis, ulcerative periodontitis (7).

While the administration of medical immunosuppression (Cyclosporin A) is revealed from history taking, an early blood test, ordered because of the non-responding uninflamed ulcers and swollen gums of the patient, may also be helpful in the early diagnosis of leukaemia (8).

ALLERGY TESTS

Several dental materials may cause allergic reactions. Life-threatening anaphylactic shocks develop rarely, but late allergic response or contact allergy for dental materials and auxiliaries are more common (9,10). The most typical form is stomatitis caused by partial or full removable dentures.

About 40 different dental materials (metal, acrylate, and other carriers and restorative materials) can be checked by epicutaneous allergical patch test (11). In case of positive results, we can prevent several unpleasant complications or side effects.

IMMUNOLOGICAL TESTS

Both the bacteria and the immune response of the patient play a role in the development of periodontitis. Some of the patients are not liable to develop periodontitis, and we should also distinguish between patients with active and inactive states of periodontal pockets (12). Inflammatory mediators and substances playing an essential role in immune reaction can be detected in the patients’ blood and crevicular fluid, and they may be helpful in assessing the prognosis of periodontal diseases (12,13).

More than 40 protein molecules have been isolated in the crevicular fluid, out of which the presence of aminotransferase enzymes, alkaline phosphatase, β-glucuronidase and the IgG4 subgroup are strictly correlated with the inflammation of the periodontium. Prostaglandine-E2 and interleukin-1 produced by peripheral monocytes are only detectable in severe periodontitis. The interleukine-1 chair side test provides immediate periodontal risk assessment. Some IL-1 and IL-18 gene polymorphisms are significant predilective factors of severe periodontitis (14,15,16).

BIOCHEMISTRY TESTS

Proteolytic enzymes like elastase are detectable from the crevicular fluid. Its presence is a prognostic sign of periodontitis (21). The CF’s aspartate aminotransferase activity is an additional sign. There are tests available for both enzymes, but their application is difficult and expensive (22,23,24). Nevertheless, further enzymatic actions of the crevicular fluid may also be detected from it, and diagnostic conclusions may also be drawn based on it (25,26).

4.2. Microbiological tests

Dental plaque is the main aetiological factor of both dental caries and periodontal diseases, which are worldwide problems. The different dental and periodontal indices are scored at the first dental visit: plaque index, bleeding
index, calculus index, and CPITN). Patients with high risk are reassessed during the check-up visits to follow the changes in the patients’ condition.

Although the dental plaque in itself usually provokes periodontitis, there are some bacteria which are generally responsible for the serious pathological changes. The following bacteria can almost always be detected in acute, aggressive periodontitis: Aggregatibacter (former: Actinobacillus) actinomycetemcomitans, Porphyromonas gingivalis, Tannerella forsythia, or Prevotella intermedia (17).

Culturing the subgingival plaque is a good option to identify periodontogenic bacteria. The disadvantages are that the technique is very sensitive, special transportation circumstances and laboratory circumstances are needed. Even if they are available, culturing periodontogenic bacteria is very difficult.

If the clinical picture suggests aggressive periodontitis, we can apply special periodontopathogenic chair-side tests. Most of them are based on bacteria specific metabolic pathways (18,19,20).

**MICROBIOLOGICAL ENZYMATIC REACTIONS – BANA TEST**

The periodontopathogenic bacteria Tannerella forsythia, Treponema denticola and Porphyromonas gingivalis are able to catabolise benzoil-DL-arginin-naftilamide (BANA) reagent. By the help of a linked colour reaction, the presence of the upper three bacteria is detectable chair-side. Unfortunately, it can detect only the above three bacteria.

**ANALYSIS OF THE BACTERIAL DNA**

Detection of the bacterial DNA from the plaque via polymerase chain reaction (PCR) is a high specificity method. The disadvantage of this method is the need for high-tech laboratory background.

**BIOCHEMICAL METHODS**

All biofilm (plaques), crevicular fluid (CF) and saliva are potential samples for biochemical analysis. These methods can detect both bacteria and their metabolic products. Periodontogenic P. gingivalis, P. intermedia and T. forsythia catabolise sulphur containing amino acids (cysteine and methionine and produce volatile sulphur compounds (VSCs). The molecules are also responsible for halitosis (bad breath) and are periodontopathogenic.

### 4.3. Histological techniques

Intraoral examination always involves the examination of the teeth, periodontal tissues, oral mucosa and the subepithelial organs like salivary glands. During the so called stomato- oncological screening the dental professional observes the morphological changes of the oral tissues. The most frequently experienced morphological changes are ulcers, white, red and other oral mucosal lesions. Another group is the hyperplasic lesions.

Most of the morphological abnormalities are characteristic and the dentist can easily establish the diagnosis. Some changes are indicative of a biopsy, sampling of cells or tissues for histopathological examination. The result determines or confirms the final diagnosis.

**EXFOLIATIVE CYTOLOGY**

The test itself is non-invasive and relatively cheap. This technique is a microscopic examination of cells desquamated from the oral mucosa surface or lesion (using a sterile tamponade or histology brush) as a means of detecting malignancy and microbiological abnormalities. Such cells are obtained by aspiration, washing, smear, or scraping.

**FINE NEEDLE BIOPSY**

Cells or tissue from subcutaneous or subepithelial lesions are taken by a fine needle. The technique is very sensitive requiring some skills and experience. Well-equipped laboratory and experienced staff are needed at specialty institutes or oral surgical departments. Fine needle biopsy is most frequently used for clarifying the origin of salivary gland or cervical tissue proliferation.

**EXCISION AND INCISION BIOPSY**
If the lesion is superficial, we can use surgical sampling. According to the location, size and relation to the environment, we can remove the whole structure. Excision is carried out if the lesion is not bigger than 2 cm and well separated from the surrounding tissues.

If the lesion is bigger than 2 cm or for any reason it seems to be malignant (anaplastic, non-separated, or invasive) then we carry out an incision. The surgeon does not try for removing the whole structure (27).

5. Dental hygienist examination and treatment planning (Péter Vályi DMD - László Párkányi DMD)

As an independently working member of the dental team, hygienists play an important role in coordinated patient treatment. Their tasks must be performed in an organized way, based on protocols, from the beginning of patient examination till evaluating the efficacy of treatment. Figure 3.5.-1. shows the steps and order of workflow as a dental hygienist:

**Workflow of a dental hygienist**

**DENTAL HYGIENIST DIAGNOSIS**

Based on a thorough patient examination, a dental hygienist has to be able to recognize all risk factors which they learned to detect, have the competence to treat, or those which need referral to a specialist afterwards.

Characteristics of a diagnosis established by a dental hygienist

- has to cover parameters describing patients’ oral and general health, bad habits, health endangering risk factors
- based on the hygienist’s examination and report a dentist can establish the medical diagnosis
- requires interventions within their scope of competence
- it is the basis of treatment planning, procedures and evaluation of its efficacy
Diagnostic models in the dental hygienist treatment plan

Diagnostics based on human needs (The Human Needs Model)

The major difference between the diagnosis established by a dentist and a dental hygienist is that while a dentist diagnoses specific disorders and conditions which are later treated with by the dentist, the dental hygienist recognises human needs, which are then reported to the entire dental team, and treats these needs within his/her own competence.

Darby and Walsh determined 8 diagnostic criteria based on Maslow’s human needs model. Their trigger factors, symptoms, characteristics, as well as health educational, preventive, therapeutic needs have to be well understood by the dental hygienist professional:

1. prevention of health endangering risk factors
2. relief of stress and fear
3. healthy facial appearance
4. functional dentition based on biology – healthy dentition
5. integrity of head and neck soft tissues (skin mucosa)
6. pain relief in the head and neck region
7. explanation of terms, problem solving
8. taking responsibility in preserving oral health

Deficiency in needs and aetiological factors can be identified by observing signs and symptoms. The following figure presents example:

DH diagnostic - sample model
Dental Hygiene Diagnostic Model

According to the diagnostic model, during treatment planning the examination strategy is planned based on a hypothesis following primary examination. After assessing all observed problems, final conclusions are drawn based on a diagnostic decision tree. In this diagram, yes or no questions need to be answered, as seen in the figure below:

Dental Hygiene Diagnostic Model

The Dental Hygiene Process Model

After determining deformities and risk factors, problems are categorised (according to general health, soft tissues, periodontium, oral hygiene, dental lesions), then the ones responsible for the problems are determined. After this, necessary clinical interventions, health educational information and individual oral hygiene instructions will be determined. Expected results and methods of checking them is the next step of treatment planning. The last step in treatment planning is scheduling treatment appointments.

The OHRQoL-model (Oral-Health-Related Quality of Life Model)

According to WHO (1948) definition: “Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.” Health is not only about the physiological integrity of the body, but also about mental, emotional and social health. These factors determine the patient’s life (QL – Quality of Life), which is also influenced by oral health.

According to the OHRQoL concept, we have to evaluate symptoms describing oral health (pain, gum bleeding...etc.), social factors (anxiousness, unhappiness), functional factors (chewing, speech...etc.), environmental factors (school, workplace...etc.), and if the patient is being treated expectations in connection with the therapy (levels of satisfaction). Life quality associated to oral health can be evaluated according to patient groups, social level, age and other factors. It is important to examine the outcome of different treatment methods not only in physiological parameters, but also associated with patient related life quality.

DENTAL HYGIENIC EXAMINATION AND TREATMENT PLANNING

The first step in patient examination is to listen to and evaluate patients’ main complaint. If it indicates important disorders (feeling pain), dental hygienic examination needs to be preceded by dental therapy.

Risk factors

Risk factors influencing oral health can be divided into 4 major groups:

1. risk factors that influence periodontal infections and the efficacy of their treatment (lower tendency to heal)
   • behavioural factors (individual OH, cooperation, nutrition, smoking)
   • systemic conditions and diseases (e.g. diabetes mellitus, osteoporosis, compromised immune status...etc.)
   • hormonal effects (pregnancy, menopause)
   • nourishment
   • iatrogenic factors (overhanging restorations, open contact points, residual calculus) ogenetic factors

2. periodontal diseases influencing general health condition
   • infective endocarditis
   • cardiovascular diseases – atherosclerosis
   • diabetes
   • respiratory diseases
   • complications of pregnancy (early delivery, underweight infant)
   • other diseases

3. risk factors promoting the development of caries
   • behavioural factors (inadequate oral hygiene)
   • nourishment factors (cariogenic foods and drinks)
   • low fluorine intake
   • positional and developmental disorders of teeth (morphologic, hard tissue disorders)
   • dry mouth ofamilial predisposition ogenetic factors

4. risk factors for malignant diseases
   • smoking
   • alcohol consumption
   • sun exposure (lips, skin)

Evaluating the general health of the patient

Physical status and medical effects of smoking need to be evaluated. It is important to determine the patient’s medical, physiological, psychological status, which can all affect treatment outcome. These factors basically determine the methods of treatment (location, patient position), time needed for treatment and possible adverse events of treatment.

The general health of the patient can be determined with the ASA classification (Chapter 3.1.3.), and the OSCAR aid.
Evaluating patients’ knowledge in connection with oral hygiene

Patients’ knowledge regarding oral hygiene maintenance and its effect on oral and general health must be assessed to determine optimal time necessary for the treatment and to achieve the expected results. Planning the individual oral hygiene program and the methods of motivation and instruction are highly dependent on patient education.

Patients ability to function independently (individual oral hygiene)

Age, physical status, certain diseases and conditions influence patients’ independence. The ADL classification (Activities of Daily Living) helps treatment planning and creating an individual oral hygiene program. Levels of ADL classification:

• „0“: can complete tasks without help
• „1“: complete tasks with little help (using instruments)
• „2“: needs a certain amount of help to complete tasks
• „3“: tasks can only be completed with external help, absolutely dependent on help

Periodontal diagnosis

Correct diagnosis can be set up based on periodontal parameters (according to AAP 1999 guidelines), but classification according to case type is even more important: patients can be classified as mild, moderate or severe cases regarding gingivitis and periodontitis. See Chapters 2.15.2, 2.15.3 and 2.15.5.

THERAPEUTIC ASPECTS, PARAMETERS

Maximal result of the treatment can be expected if the treatment plan is based on accurate diagnosis, risk factors and prognostic factors are identified.

Clinical diagnosis, goal and aspects of therapy, evaluation of the results are all listed in AAP (American Academy of Periodontology): Parameters of Care issue, which was written in accordance with modern the classification and treatment of diseases. Therapeutic considerations can be divided in two groups: according to the treatment plan of the dentist and the dental hygienist. While the elimination of aetiological risk factors, identification of microbiological factors and antibiotic treatment are the tasks of the periodontist, the support of mechanical therapy is mostly the task of the dental hygienist under optimal circumstances.

Based on the above mentioned, the diagnosis can precisely determine the role of the dental hygienist in the complex treatment of periodontal diseases.

DENTAL HYGIENIC DIAGNOSIS

Diagnostic models have previously been mentioned. These models determine physiological conditions, behavioural factors, and describe health issues and needs, which dental hygienists can recognise and are competent to treat.

Dental hygienic diagnosis contains all facts which determine treatment methods and expected results, defines parameters which are likely to improve after treatment, and rules out diagnoses which would require a specialist intervention.

The basis of a dental hygienic diagnosis is complex patient examination, which was described in previous chapters.

Dental hygienic prognosis

Prognostic factors need to be evaluated based on three aspects: besides periodontal and cariologic aspects, the risk of developing oral cancer must also be considered.

Dental hygienic prognosis is based on monitoring and re-evaluating oral hygiene parameters during the treatment process as part of a complex therapy:
• complex evaluation of clinical parameters
• patient cooperation
• expected therapeutic result

**Periodontal prognosis**

Prognosis is highly influenced by risk factors and the chosen treatment plan. Prognosis is evaluated at different levels, the same way as risk factors:

• patient level
• dental arch level
• tooth level

Prognosis is considered good at **patient level**, if the risk of developing new diseases and the exacerbation of diseases is low. Prognosis is bad if there is a high risk of new infections or reactivation of treated diseases. Factors influencing the general health of the patient need to be mentioned here:

• untreated or improperly treated systemic diseases (mainly diabetes mellitus, immune-compromising diseases)
• medications modifying physiological gingival reactions, or its response to plaque
• behavioural factors (smoking, alcohol consumption, nutrition habits)
• disorders influencing physiological oral functions (e.g. Sjögren syndrome – salivary production)
• conditions, diseases preventing the application of individual oral hygiene procedures
• genetic factors

At **dental arch level**, mean attachment loss, maintenance of dentition, tooth contacts, anatomical and positional disorders need to be assessed. Factors influencing the physiological integrity of the oral cavity (behavioural, general health...etc.) are also important at dentition level.

Efficacy of individual oral hygiene, clinical attachment level, furcation involvement, mobility, individual anatomical situations need to be examined at tooth level. Based on the above parameters, the prognosis of teeth can be classified into 3 (good, questionable, hopeless) or 5 (good, acceptable, bad, questionable, hopeless) groups. Prognosis is considered questionable if clinical attachment loss is greater than 50%, factors negatively influencing individual oral hygiene are present (class II. or III. furcation, unfavourable root anatomy, crowded teeth), certain factors compromise tooth stability (inadequate crown-root ratio, pathologic mobility: class II.), poor cooperation of the patient. A tooth is considered hopeless if attachment is not sufficient, resulting in class III. mobility.

**Cariologic prognostic factors**

The risk of caries development can be determined by evaluating the balance between its positive and negative prognostic factors, and factors indicative of the disease. (table 3.5.-1.). Risk assessment is inevitable when creating an individual oral hygiene program in order to improve prognosis.
Factors influencing caries risk assessment

Patients can be divided into low-, medium-, and high-risk patients regarding caries. Extreme risk patients are those who suffer from dry mouth besides being in the high-risk category.

**Stomato-oncological risk assessment**

Patients who are considered to be at high-risk for oral cancer development (alcohol, smoking, sun exposure, poor oral hygiene, irritating factors, socio-economic status, specific infections – e.g. HIV) need a more strict follow-up program and treatment to prevent malignant tumours. Extensive screening programs ensure the early detection of tumorous diseases.

**DENTAL HYGIENIC TREATMENT PLAN**

Diagnostic models regulate dental hygienic treatment plans, but its content is basically the same: determines goals (influenced greatly by patients’ expectations), evaluates parameters describing general health, determines necessary interventions. A dental hygienic plan has to fit well in the dentist/specialists complex treatment plan. Steps of preparing:

1. strategy based on the diagnosis
2. order of importance
3. determination of goals – respective to patient motivation
4. selection of evidence based therapy
5. scheduling appointments treatment steps

Dental hygienic treatment plans usually approach therapeutic steps and goals from a human need perspective. The complex dental examination and treatment plan reveals aspects which are essential for success from the perspective of both the patient and the therapy. The first step is to identify patient needs. These are typically periodontitis-related chewing difficulties, bad breath, hyper-mobile teeth. From the patient’s perspective, functional, aesthetic and psychosocial problems are the main issues, which all originate from periodontal infection. At the beginning of the treatment, diagnostic and therapeutic steps which the dental hygienist can perform, and parameters which best describe treatment efficacy need to be determined.
Problems mentioned by the patient need to be solved in order of importance. It is obvious that functional disorders caused by periodontitis are of higher importance to treat than aesthetic issues related to aging (e.g. discoloured teeth).

The next step is to determine the goals. Patient-centred goals can be the following:

- cognitive goals (extend patients knowledge regarding oral health)
- psychomotor goals (educating the patient about preventing measures)
- affective goals (changing patients’ attitude to oral hygiene, their values and beliefs)
- goals associated with oral health (influencing symptoms and parameters of diseases by therapeutic interventions)

The aim of the treatment plan is include the expectations of patients, time needed for treatment, changes needed in patients attitude to oral hygiene, changes intended in individual preventive measures, indicators helpful in evaluating treatment efficacy. Result cannot be achieved without the cooperation of the patient. Knowing that without eliminating plaque retentive factors, surgical therapy is ineffective, so it is not enough to send the patient to a specialist for treatment right away. The patient must understand the significance of the treatment, and needs to be highly motivated to execute the steps of complex therapy.

In the following steps, the choice of treatment methods must follow the treatment plan and literature-based evidence. Steps of the therapy must follow the above-mentioned patient-centred goals and must fit in with the complex dental treatment plan both in priorities and competences.

The last step is to schedule appointments, determine diagnostic and treatment steps and prepare the appropriate documentation of these.

6. Orthodontic diagnostic (Emil Segatto DMD)

The goal of the orthodontic diagnostics is to explore all dentoskeletal positional disorders in the oro-facial region serving the overall orthodontic treatment plan to be compiled with the final aim of the total aesthetic and functional rehabilitation.

To date, orthodontic diagnostics has been entirely restructured. By keeping traditional aspects as well, emphasis has been shifted to aesthetic and functional examination results nowadays at planning orthodontic interventions. Due to long-term effects of treatments, diagnostic examinations cannot be restricted to the main complaint, their number, however, depends on the extension of the deformities. Therefore, the diagnostic examination range is much shorter at a very young age than in case of adult age, when the treatment may require complex orthodontic surgical care. The elements of the documentation compiled for diagnostics are useful not only for treatment planning, but they should be repeated several times in later treatment stages in order to control intermediary outcomes of the treatment. The specialty of childhood orthodontic treatments is that they affect growth direction and pace of dentofacial structures in the growth period. This fact frequently makes it necessary to repeat certain diagnostic steps during the treatment, and to review the treatment plan. Thus orthodontic diagnostics is a dynamic process, which should be constantly balanced with the actual conditions of the structures of intervention areas.

Results necessary to gain the overall diagnosis are served by patient history, clinical examination, functional analysis, cephalometric analysis, photo analysis, X-ray analyses, as well as plaster model analysis, which all have the same weight in the diagnostic process.

**PATIENT HISTORY**

Anamnestic records are divided into two parts: general and orthodontic.

General section collects information on general health condition, focusing on areas, which may have indirect impact on the planned orthodontic interventions, too. These include various diseases of bony structures, use of different medicines, necessity of antibiotic prophylaxis, condition of pharyngeal and palatine tonsils, permeability of nasal tract, and the presence of mouth breathing. Judgment of patient’s mental development is
also important, which is vital in the acceptance of treatment procedures and in the determination of treatment goals. Family history gives help in the exploration of reasons for certain deformities.

In the course of the orthodontic history, attention is focused on personal history directly affecting current conditions. Finding answers to questions on the existence or lack of breast feeding during infancy, sucking a finger or pacifier during childhood, requires parental assistance. Date of the eruption of deciduous teeth plays a role in estimating the duration of future exfoliation, while former facial and dental traumas may cause skeletal and/or dental development disturbance later. When planning a series of orthodontic treatments, all factors should be taken into consideration, which are discovered during history taking. These are, for example, presence of bruxism, as well as practicing on wind instruments. Finally, questions on former orthodontic treatments, as well as on deviations, which have been partly or entirely corrected, are closing the patient history.

CLINICAL EXAMINATION

Practical experience of the specialist performing the examination is a decisive factor in this process. By recording results on a uniform data sheet, they become accessible and also usable by others. Clinical examinations cover three main areas: general (physical) features, extraoral features, as well as intraoral features.

General features

Observations on mental development of the patient are recorded on the datasheet together with physical features. Patient examination is not limited only to sitting in the dental chair. Body and head postural characteristics having very considerable diagnostic and prognostic value can only be diagnosed in a standing position.

Extraoral features

Extraoral features recorded on the datasheet describe facial characteristics and attributes of the individual soft tissue components. Features of temporal or masseter mastication play a decisive role in the vertical development of the dentoskeletal complex.

Detailed analysis of soft tissue profile is done by examination of lateral face photos, however, information gained in the course of clinical examination also has significant added value. Profile features may hide or even unveil sagittal dentoskeletal deviations. The connection between antero-posterior jaw positions and the three profile types has been known for a long time: (Fig. 1).

Soft tissue profile – basic types.

- patients with straight profile is characterised by normal occlusion,
• distoocclusion is more likely associated with a convex profile, together with retrognath mandible or/and prognath maxilla,

• concave profile is usually associated with mesioocclusion, together with retrognath maxilla and/or prognath mandible

At the same time, profile features may indicate vertical dento-skeletal deviations:

• facial vertical overgrowth may lead to frontal open bite, incompetent lip closure, gummy smile and development of increased mandibular angle. Even normal occlusion (Angle I), distoocclusion (Angle II), as well as mesioocclusion (Angle III) may be associated with a profile of long vertical growth (Fig. 2).

• insufficient facial vertical growth may lead to deep overbite, lips leaning over each other and development of decreased mandibular angle. Both forms of distoocclusion (Angle II/1 and Angle II/2) and mesioocclusion may be associated with a profile of short vertical growth (Fig. 3).

Malocclusions associated to high vertical profile types.
Malocclusions associated to low vertical profile types.

While individual profile features may refer to existing malocclusion, there is no such kind of rule in case of combined sagittal vertical deviations.

Notes on the analysis of facial characteristics are completed by the registration of scars/cicatrices and by the examination of existing asymmetries. Traces of previous accidents may refer to the causes of dentoskeletal growth problems. Direction and expansion of deviations measured from the imaginary midline of the face are separately recorded in case of maxillary soft tissue (philtrum) and mandibular soft tissue midlines.

Examination of individual soft tissue components of the face begins with the lips. Length of lips, width of vermillion border of lip, condition of lip epithelium, vertical and sagittal lip closure, all should be examined. In a face with normal vertical proportions, the length of the upper lip is equal with one-third of the lower face height. Width of vermillion border of lip is significantly influenced by lip breadth, dentoskeletal support, tone of m. orbicularis oris, as well as by lip closure. Dryness and crackedness of lip epithelium are pathognomic in patients with mouth breathing, but, without functional deviation, it may indicate adverse anatomic endowments such as disproportionate vertical and/or sagittal lip position. In case of balanced muscle tone, lips are closed at rest – it is called competent lip closure. Lack of balanced muscle tone results in incompetent closure.

Recording of nose characteristics have practical importance mainly in case of treatments requiring (conservative or surgical) orthopaedic interventions. Nose size, run of nose ridge, as well as width of the alae of the nose should be examined. In general, vertical length of the nose equals to one-third of the whole face height. From lateral view, horizontal size of the nose is half of its vertical size. In normal case, width of the alae of the nose equals to 70% of vertical length of the nose. Beside aesthetic aspects, its examination also bears functional importance, since signs of incapacitation of nasal passages can also be often detected in the course of extraoral examination.

**Intraoral characteristics**

Mapping of intraoral characteristics concentrates on three main areas: dental, dentoalveolar as well as soft tissue examinations.

**Dental examinations**

Dental examination consists of two parts.

1. In the first step of the general part, determination of dentition stage occurs, which practically means the following stages: primary dentition, early and late mixed dentition, and permanent dentition. It is followed by the detailed recording of cariologic/endodontic/prosthetic problems/treatments. Further steps include the exploration of shape and numerical deviations of teeth.

2. Orthodontic part focuses on tooth position features and anomalies having a direct diagnostic importance. Beyond the determination of place and position of individual teeth within the dentition, crossbites will be recorded on the clinical examination datasheet together with the indication of relevant area: frontal, lateral – unilateral or bilateral. Recording scissor bite and open bite of lateral area, as well as deep bite and open bite of frontal areas all have similar importance. Examination chain is closed by the analysis of relative position of lower and upper dental arches, in all three spatial planes. Extended examination of the characteristics of sagittal, vertical and transversal relations belongs to plaster model analysis. Certain basic data, however, should be recorded during the clinical examination. These data include the Angle’s relation of molars and canines by sides, overjet (horizontal) and overbite (vertical) as well as midline shifts.

**Dentoalveolar examinations**

Oro-vestibular run of the alveolar process provides definite data on the relation of apical-coronal base. This relationship can be definitely examined by modern medical imaging processes; however, plaster models might also be appropriate for observation of apical-coronal base. Imaginary line connecting root apices – the apical base – defines the dimension of jaw area containing roots. Relationship of apical–coronal base is one of the most important factors affecting the direction of extraction/non-extraction therapy at the treatment of space deficiencies.

**Examination of soft tissues**
Examination of soft tissues in the oral cavity contains mapping of morphologic features and pathological modifications of gum, palate, lips, cheek and tongue:

- treatment aiming at tooth movement can only be initiated when the patient has adequate oral hygiene,

- thorough examination of the oral mucosal membrane and the practical importance of recording pathologic modifications may allow one to discover a connection with mechanical irritation caused by the elements of the used appliance and the incidental allergic reactions,

- shape of the palate is important at designing removable and fixed transpalatal appliances,

- thickening and position of labial frenum and lingual frenum should be taken into account not only at the planned tooth movement (e.g. closure of diastema medianum), but also at installing such appliance elements, which may cause mechanical irritation during use due to their proximity,

- examination of soft tissues is finished by the tongue. Out of abnormal size modifications, increased tongue size has diagnostic importance. Increased size of the tongue should be taken into account at designing lingual or transpalatal appliances.

**FUNCTIONAL ANALYSIS**

Functional analysis consists of three main parts: examination of temporomandibular joint, mapping functional types of malocclusions, as well as examination of functional deviations.

**Examination of temporomandibular joint**

The examination chain aims at judging the presence and severity of pathological symptoms. Its parts are auscultation, palpation, functional examination and X-ray examination.

**Mapping functional types of malocclusions**

This examination chain aims at filtering functional types of individual malocclusions. Their peculiarity is that the mandible is led to any directions owing to the early proximal contact areas during the terminal movement, thus, malfunction characterising occlusal position is created. Analysis of early proximal contact areas and established runs may be carried out by systematic observation of end stage of terminal movements; however, their diagnostic importance is priceless in terms of subsequent treatment design procedure.

**Examination of functional deviations**

The last step of functional examination serves to separate normal and pathological muscle operations of the orofacial region, as well as to map the individual parafunctions. Their practical importance emerges at designing treatment of deviations, where these muscle operations interplay in development and/or maintenance. The examination chain includes analysis of swallowing and respiration, besides lingual, lip and cheek dysfunctions:

- Examination of swallowing. With this analysis normal and pathologic (e.g. tongue thrust) swallow are separated, as well as static and dynamic tongue positions are defined.

- Examination of lip dysfunctions. It is for screening pathologic lip sucking, lip thrust, as well as lip insufficiency. Observation is made while the patient is speaking and swallowing.

- Examination of cheek dysfunctions. Study of the presence of buccal sucking or biting is carried out by observation; it is identified easily by significant horizontal buccal fold, which can be observed via intraoral examination, too.

- Examination of respiration. It is for identifying reasons behind incapacitation of nasal respiration, in case of chronic mouth breathing. Chronic mouth breathing is one of the dysfunctions of orofacial musculature, which may significantly impede proper development of dentition.

**CEPHALOMETRIC ANALYSIS**

In orthodontics, lateral cephalogram and postero-anterior (P-A) images are the routinely used radiograms. Their detailed analysis done by standard methods providing precise information on the size and dimension of skeletal and dental structures, the distance thereof from adjacent structures, as well as growth directions. The most
accepted and most frequent analysing method is the Ricketts analysis, which has separate analysing modules for lateral cephalogram and P-A images, too. Cephalograms shows sagittal and vertical deviations, while P-A images demonstrate transversal and vertical ones, as well as asymmetries.

**PHOTO ANALYSIS**

Recently photo documentation and photo analysis have become the most decisive component of diagnostic examination. Moreover, it is the simplest way to document the continuous change being characteristic of orthodontic treatment. Today, digital reflex camera equipped with macro-objective and ring flash, if possible, is a minimum requirement of an orthodontic consulting room. Baseline requirement of monitoring changes is to take all pictures under standardised conditions, with the same settings. Standard orthodontic settings:

*Extraoral photos:* (Fig. 4).

- frontal view (rest position) - focus on the nasal tip,
- frontal view (smiling) – focus on the nasal tip,
- lateral view (rest position), from both sides – focus on the tragus,
- semilateral view (rest position), from both sides – focus on the alae of the nose
Intraoral photos: (Fig. 5).

- frontal view with lateral lip retractor – focus on the incision superius,
- lateral view with lateral lip retractors from both sides – focus on the upper canine,
- occlusal view with mirror and frontal retractor, lower and upper – centre is along the midline, at the level of canines

Applied enlargements: 1:10 at extraoral settings, while 1:2 at intraoral settings.

**X-RAY EXAMINATIONS**

Beside lateral cephalograms and P-A radiograms used in cephalometric analyses, orthopantomogram (OPG) is the orthodontic radiologic evidence, which is providing the most detailed information (Fig. 6, 7). It is required to take it before starting an orthodontic treatment. Its detailed examination helps screening tooth aplasia, supernumerary teeth, pathologic tooth positions, periapical processes, as well as root resorptions and parodontologic pathologic processes.
In order to determine the position of partially erupted teeth associated with or being independent from cleft palate, a bitewing i.e. occlusal radiogram as well as periapical radiogram with different settings are also used. Analysing of these radiograms taken of the concerned areas are also obligatory before applying skeletal anchorages.

For achieving the mentioned goals, analyses of CBCT images having different fields of view are also appropriate (Fig. 8). However, their use is recommended only in some restricted cases due to the increased administered radiation dose and high costs.
CBCT – cone beam computed tomography

PLASTER MODEL ANALYSIS

Plaster model analysis, performed during clinical examination, enables a more precise evaluation compared to occlusion analysis. Advantages of better observation may only be realised after a proper determination of occlusal relationships. Orthodontic plaster model is one of the basic elements of documentation, but it is also essential at making customised appliances.

Adequate impression is a basic requirement for making a plaster model suitable for further analyses. It has to be performed free from bubbles using alginate impression material by a special high-edge tray. The goal is to model oral formulae in a quick and precise way, also including vestibular area as much as possible. It plays a crucial role not only at previous analyses, but also in subsequent appliance manufacturing later on. Manufacturing of a plaster model is based on defined standards. After pouring, an adequate base makes the finished plaster model enabling for further analyses (Fig. 9). Static examinations are suitable for determining various defects such as Angle’s relationship between molars and canines, position abnormalities of individual teeth, as well as detecting midline shifts, and categorising malocclusions merging different variations of all these.
Angle’s classification

The base of the classification, described by Edward H. Angle in 1899, is the contact relationship of maxillary first molars with mandibular first molars on the same side. Based on this, there are three different sagittal occlusal relationships:

- **Angle class I (normoocclusion)**– the mesiobuccal cusp of the permanent maxillary first molar occludes in the groove between the mesiobuccal and centrobuccal cusps of the permanent mandibular first molar.

- **Angle class II (distoocclusion)**– the mesiobuccal cusp of the permanent maxillary first molar occludes mesial to the groove between the mesiobuccal and centrobuccal cusps of the permanent mandibular first molar. This class has two divisions:
  1. **Angle II/1 division**– it is characterised by protrusion of upper teeth and retrusion of lower teeth in the frontal area, as well as consecutive mouth breathing established due to incompetent lip closure,
  2. **Angle II/2 division**– it is characterised by retrusion of upper incisors, and it is associated with nasal breathing.

- **Angle class III (mesioocclusion)**– the mesiobuccal cusp of the permanent maxillary first molar occludes distal to the groove between the mesiobuccal and centrobuccal cusps of the permanent mandibular first molar. Frontal crossbite is usually associated with this molar relationship.
Occlusion analysis

This 3D analysis determines the intermaxillary relationship of upper and lower dental arches in habitual occlusal position.

- **Transversal deviations:** dental and skeletal midline shift, lateral crossbite (unilateral or bilateral), non-occlusion (vestibular, oral).

- **Sagittal deviations:** increased overjet, frontal crossbite, distoocclusion, mesioocclusion.

- **Vertical deviations:** increased overbite (deep bite), open bite (frontal, lateral, and complete).

Out of the recognisable deviations, overjet and overbite can be numerically determined as follows:

- **Overjet:** it is determined in line with the occlusal plane by measuring a distance between the edge of upper central incisor and the vestibular surface of lower central incisor.

- **Overbite:** it is determined in line with the occlusal plane by measuring a distance between the projection of the edge of upper central incisor to the vestibular surface of lower central incisor and the edge of lower incisor.

Deviation list serving as the basis of treatment plan is compiled with the help of the summary and analyses of various available diagnostic evidence. In an ideal case, treatment plan contains corrective options for all deviations on the list. However, deviations requiring involvement of other specialties into the treatment process are excluded. It is the task of the orthodontist to arrange different treatment options into a rational plan, which provides the most successful treatment for the patient. Elements are prioritised in the deviation list according to the patients’ preferences, in contrary to the previously adopted approach, when restoration of occlusion had been forced by specialties in any way. This is part of that paradigm shift, which represents that the former dentoskeletal focus has been changed to the decisive role of soft tissue parts. Today the primary aspect is a backward therapeutic concept originating in the desired final outcome, which determines all dentoskeletal modifications necessary to establish the visionary aesthetic condition.

Final decision is made together with patient/parents/relatives on the basis of presenting the treatment plans compiled after analysing all available diagnostic documentation. Exact description of the individual therapeutic routes provide significant help in this decision including type, price and wearing period of the used appliance, as well as the means and duration of retention period, moreover, it should also cover dentofacial aesthetic consequences of the planned intervention.

Final treatment plan is selected by taking into consideration treatment difficulties and all known side-effects listed in the informed consent. It is always chosen out of the presented treatment alternatives as the result of a common decision, which the patient and/or his/her relative(s) always had given consent to, on the basis of all this information.

7. Assessment of patient with head and neck cancer (Róbert Paczona MD - József Piffkó MD, DMD)

The surgeon remains paramount in the definitive evaluation of patients with signs or symptoms relating to possible cancer in head and neck region. Depending on the setting that the surgeon may be a general surgeon evaluating an asymptomatic lesion of the neck, an oral surgeon evaluating a lesion of the oral cavity or mandible, or an otolaryngologist or head and neck surgeon being consulted for a primary complaint of otalgia, hoarseness, or dysphagia with or without regional adenopathy. Regardless of the specialty of the initial surgeon, ultimate evaluation, diagnosis, staging and follow-up must be undertaken by a head and neck surgical specialist with sufficient training and expertise to achieve all the above tasks.

**PHYSICAL EXAMINATION**

Primary tumour examination

Obvious tumour masses should be identified along with potential second primary lesions and premalignant regions of leukoplakia or erythroplasia. Suspicious lesions should be defined by primary site location, extension into adjacent areas, and overall size.
• inspection of all visible mucosal surfaces of the head and neck
• including indirect mirror or fiberoptic laryngoscopy,
• as well as palpation of the floor of mouth, tongue and tonsil regions, and neck.

A complete physical examination should include:

**Neck examination**

Associated cervical adenopathy should be assessed, and if significantly enlarged, the regional extent of disease should be quantified for use in clinical staging. Independent of whether surgical resection will ever be deemed appropriate, the potential for resection of the suspected primary site and neck lesions should be determined. Neck lesions fixed to deep tissues are not to be resected.

**IMAGING STUDY**

In addition to physical examination, staging procedures include computed tomography (CT) and magnetic resonance imaging (MRI) of the head and neck to identify the extent of the disease. Patients with a large primary lesion or significant lymph node involvement should also undergo chest CT and perhaps a bone scan to screen for distant metastases.

**PANENDOSCOPY**

The definitive staging procedure is an examination under anaesthesia, which may include laryngoscopy, esophagoscopy, and bronchoscopy. During this procedure, biopsy samples are obtained to pathologically confirm a primary diagnosis, to define the extent of the primary site disease, and to identify additional premalignant lesions or second primary cancers. An accurate description of anatomical involvement is critical to determine the potential morbidity of an initial surgical resection, or to define regions that should be included in a salvage surgical procedure if initial non-surgical treatment failed to control disease.
Chapter 4. PREVENTION

1. Methods of individual oral hygiene (Péter Vályi DMD- Dóra Tihanyi DMD – László Párkányi DMD)

The role of dento-gingival plaque in tooth decay and periodontal disease is well known, as is that of mechanical plaque removal in the prevention of the above mentioned diseases. Complete removal of plaque is not easy, though. It might need several instruments simultaneously, according to the patient’s individual dental condition. Poor oral hygiene can be caused by improper oral hygiene technique, use of non appropriate instruments or insufficient length of the oral hygiene procedure. This is best demonstrated by patients who brush 4-5 times a day, still they have inflamed and bleeding gums.

ORAL HYGIENE INSTRUCTION AND MOTIVATION

The dental team has the most important role in ensuring correct oral hygiene. The most efficient oral hygiene protocol has to be tailored for every patient. The steps of instruction and motivation are individualised according to the patient’s own dental status and dental records. Individual characteristics, such as risk factors, anatomical conditions and the patient’s habits have to be taken into count.

Oral hygiene instructions and motivation have to be integrated in a complex preventive program. (picture).

In order to give our patients useful advice, we have to identify patients’ needs from the information we collected. We have to find which individual preventive measures are lacking and which factors are preventing proper oral hygiene:

• patient awareness of the disease
• brushing technique, duration
• use of improper instruments
• awareness of the significance of oral hygiene
• general medical condition
• social, cultural, economic situation
• bad attitude, inadequate knowledge about oral hygiene

Our answers for the above mentioned questions should help us determine the measures of oral hygiene instruction and motivation. The education is a long process and the results need time to be visible: after we share our knowledge, patients’ interest might lead to their involvement in the program, consciously changing their oral hygiene habits. After realizing that good oral hygiene provides comfort, proper oral hygiene will become daily routine

Implementation of instruction and motivation

First step: explaining the importance and methods of plaque control

Description of the cause of the problem: We explain plaque and calculus formation, their role in the diseases, demonstrate the anatomical properties of the tooth, emphasise critical anatomical areas of plaque accumulation. The interdental gaps and the marginal zone of the teeth (cervical portion of the crown and upper third of the sulcus) are considered predilection areas, therefore the cleaning of these areas is of utmost importance in order to prevent tooth decay and periodontal disease.

Evaluation of oral hygiene with the inclusion of the patient: We show plaque and calculus covered areas to the patient with the help of a probe and a mirror. In case of gum inflammation, point out the possibility of progression. In patients with periodontitis show the tooth surfaces with high probing depth, explain its significance and the need of pocket elimination. For patients highly affected by caries, it is important to give dietary instructions as well.
Demonstration: for demonstration we can use a model or easily understandable images; but the most important thing is to show the right techniques with the specific instruments directly in the patient's mouth, which the patient can observe through a mirror. We help the patient in the learning curve by instructing the hand and instrument movements, correcting holding positions to teach them the correct way of home plaque control. Dying helps us demonstrate the accumulation of plaque and oral hygiene evaluation together with the patient. Dying even helps us determine the areas of plaque-index measurements during clinical examinations.

Instruction: Care needs to be taken to give advice on instrument selection and their use, home plaque dying, and timing and length of tooth brushing. If possible, it is advisable to provide the patient with the written version of this information, for this purpose manufacturer brochures can be useful.

Second step: evaluation of first steps, reinstruction, motivation

Show the patients how the size, colour, bleeding and consistency of the gums has changed. For this purpose, baseline and re-evaluation photos can be helpful. We register bleeding and plaque scores, which can well demonstrate the results of the patient’s and doctor’s effort. Areas which failed to improve after self plaque control need to be identified, and instructions need to be reinforced.

Third step: continuous motivation and instruction. Evaluation of individual oral hygiene, motivation, instruction, necessary corrections in view of general and dental health condition are important parts of the treatment. Oral hygiene education is a lifelong process. Starting from the education of pregnant women to elderly patients (even in the case of complete dentures). Methods should always be chosen according to age (basic knowledge, understanding, capability of application), and carried out through individual or group education.

1.1. Methods of tooth brushing

FREQUENCY OF TOOTH BRUSHING

According to research, frequency and time spent on oral care fail to reach expectations. As an example, research in the USA concluded that people on average spend 37 seconds with tooth brushing, but claim to have spent 2 minutes with it.

According to research, the first subclinical signs of gingivitis appear after 48 hours. In view of this, it would be enough to brush once every 2 days to preventing periodontitis, but these tooth brushings should effectively remove all of the plaque. Obviously, this does not mean that we should advise our patients to brush once every second day only, especially because the efficacy of an average brushing is not more than 60%. It has to be emphasized that several sessions of 15-20-second tooth brushing does not provide sufficient protection. Brushing two times daily, combined with interdental hygiene performed once daily, can prevent gum inflammation and the development of carious lesions. Based on epidemiological studies, brushing more frequently than two times daily, does not improve the condition of the gums significantly.

DURATION OF TOOTH BRUSHING

People brush their teeth on average for one minute with 60% efficacy. Brushing needs can be expressed by the time required or by the movements necessary on the area occupied by the head of the brush. For everyday practice we advice brushing for 3-4 minutes, moreover, correct motion of the brush 5-10 times in every position.

BRUSHING TECHNIQUES

Several brushing techniques can be found in the literature. Being familiar with all of them is very important from the professional perspective. In clinical practice we only use few of them, because the indications and efficiency of most are in doubt. Using either of the techniques, effective cleaning can only be performed when the cleaning is restricted to 2-3 teeth at a time. (Fig.4.1.)
It is advisable to change the order of cleaning the next time to ensure sufficient time for cleaning every tooth. After cleaning proximal tooth surfaces, occlusion cleaning must not be forgotten. Special care needs to be taken at areas where the self-cleaning mechanism of the soft tissues and saliva does not take effect (upper molars mesio- and disto-lingual, and lower molars buccal surfaces), and the predilection areas of calculus formation, which are at the main salivary gland ducts (upper molars buccal and lower incisors lingual surfaces).

**Sulcular brushing technique (Bass 1948) and the modified Bass technique**

This technique focuses on the cleaning of the area right below the gingiva. The head of the toothbrush is aimed towards the apex. The bursts of the brush reach into the sulcus. At the beginning the bursts move back and forth, not leaving the sulcus. On the lingual surface of the front teeth, the toothbrush needs to be held vertically. The Bass-technique is an effective way of removing the plaque not only from the marginal gingiva but also from the sulcus. According to some studies, with this technique the plaque can be removed from the sulcus 1 mm below the marginal gingiva. (Waerhaug 1981)

According to the Bass-technique, the brush draws small circles with a gentle pressure. Care must be taken not to remove the bursts from the tooth surface and the sulcus. This way, 2-4 teeth can be cleaned at a time. The advantage of this technique is that it focuses on the cervical areas of the teeth, which allows interdental cleaning while preventing abrasion of the teeth. The use of this technique can be advised in any age, both in periodontally healthy and in periodontally compromised dentition. This is the only way to clean the area of the sulcus! (Fig.4.2.)
Bass technique: positioning of toothbrush

*Modified Stillman technique*

The original technique, described by Stillman in 1932, is modified by adding the Roll-technique to it, with the head of the brush. The bursts are placed at the mucogingival junction at 45° degree to the vertical plain in the apical direction. The bursts of the brush move back and forth, while the brush moves in a coronal direction, turning coronally around the axis of the brush. The advantage of this technique is that in case of recession, it does not abrade the exposed root surfaces. (Fig. 4.3.)

![Modified Stillman method](image)

*Roll technique (Riethe 1970)*

The head of the brush is held bevelled towards the apex. The bursts are laid partially on the tooth surface and partially on the marginal gingiva. The sides of the bursts gently press the marginal gingiva. The head of the brush needs to be turned coronally from the gingiva, towards the occlusal plane. It is advisable after mucogingival surgery. It can be used on a healthy gingiva in kids, in case the Bass technique is too difficult for them to perform. It can also be used as an addition to vibration techniques (Bass, Charters, Stillmann) to remove loosened plaque.

![Roll technique](image)

*Vibration technique (Charters 1948)*

Originally it was developed to improve the interdental cleaning efficacy and to stimulate the gingiva in this area. The toothbrush is aimed bevelled in an occlusal and incisal direction, contrary to the Stillmann-technique. The bursts are slightly pressed into the interdental spaces and vibrating motion is performed. This method is most effective when the papilla does not fill the proximal areas completely.
Horizontal technique

This method is most commonly used by people not having received oral hygiene instruction. The bursts of the brush are perpendicular to the tooth surface. In this position, the brush moves back and forth. The disadvantage is that it reaches only the self-cleaning surfaces of the teeth (coronal to the highest diameter of the teeth) and it causes severe trauma to the gingiva.

Vertical technique (Leonard 1939)

The brush is held perpendicular to the teeth, the vertical motion goes up and down. The drawback is that similarly to the horizontal technique, it only reaches the self-cleaning surfaces of the teeth (coronal to the highest diameter of the teeth).

Fones-technique (Fones 1934)

The brush is placed in the vestibule with the burst parallel to the teeth. It is performed with rapid circular movements on both jaws with slight pressure. The cleaning of the lingual and palatal surfaces is executed with a back and forth motion. The method is used in children with deciduous and mixed dentition till mastering the modified Bass-technique.

"Scrubbing method"

It is a mixture of horizontal, vertical and circular techniques.

INSTRUMENTS OF ORAL HOME CARE

The most important and most versatile equipment of individual oral hygiene is the toothbrush, which is capable of removing dental plaque, some discolouration and food remnants. It is essential to use a mechanical or electric toothbrush to mechanically remove the plaque, but it always has to be combined with an additional tool for interdental cleaning. Today, the variety of oral hygiene instruments is tremendous. Toothbrushes are available in all sizes and shapes, with a large variety of burst types taking mouth size, tooth alignment and age in consideration.

Manual toothbrushes

The design of toothbrushes are uniform, regardless of their brand. The parts are: handle, neck and head. The bristles are found on the head part. Toothbrushes are mainly made of plastic, they differ from brand to brand in size, shape, burst composition, colour and neck angle. From a functional standpoint, the configuration of both
the handle and the head (bristles) is important. The parameters of the ideal toothbrush were defined at the European Workshop on Mechanical Plaque Control conference in 1998, and these principles are followed by the companies even today.

The main purpose of the handle is to provide a stable grip, but it can have a rough surface inlay for tongue cleaning. The bigger, wider shapes provide a stable grip, which can be important in children and manually challenged people.

The average size of a toothbrush ranges between 15-19 cm, from which the head is 2.5 - 3 cm long and 8-11 mm wide. Bristle length ranges between 9-11 mm, with a diameter of 0.2-0.3 mm. The size and shape of the head determines the number and distribution of bristles. About 5-12 bunches of bristles are distributed in 2-5 rows. One bunch of bristles consists of several hundred individual bristles. Depending on the proximity of bursts they can range from a couple hundred to a few thousand bristles. New configuration brush heads are produced to help clean the areas of limited access. Examples of this are the „double or triple head“ toothbrushes and the so called Pro-Flex shapes, which have individually moving burst groups, for better adaptation to the individual anatomical shapes. (Fig.4.6.)

Brush heads

The bristles are built up of special plastic or polymer, their diameter is not higher than 0.12 micrometer. Natural materials as pig fur are not used anymore because of their hollow structure, which serves as an ideal place for bacterial reproduction. The tip of the bristles must always be rounded. Based on the thickness and strength of the bristle, the brushes are categorised as hard, medium or soft toothbrushes. Today’s modern toothbrushes mostly use tender bristle for effective plaque removal. The hardness of the bristle is directly proportional to the diameter of the bristle and inversely to its length. Recently, bristles have been improved with different accessories like rubber bristles or chalices. These accessories serve to improve the abrasive effect when cleaning the interdental areas, and they provide physiotherapy for the gums.
Bristle surfaces are variable. Uniform length bursts give a „smooth‖ profile, while various length bursts can give a „wavy‖, „bulky‖, „2-staged‖ or „multi-staged‖ appearance. Brushes with angled bursts are called „criss-cross‖ brushes.

Special profiles of heads

Certain heads are developed for specific techniques. For the vertical technique for example the Signal Vertical Expert toothbrush is advised (IMAGE right side). Different surface modifications are produced to increase efficiency. Companies are introducing several innovations to improve interdental cleaning and helping with reaching the most distal areas. A 15-degree inclination of the bristle, crossing them with each other or a 15-degree cut on the tips substantially increase the efficiency of interdental cleaning by penetrating deeper. Nanosilver impregnation can prevent bacterial growth on the bursts for up to 3 month. Efficacy values of mechanical oral hygiene instruments encourage companies to continuously improve their products, although some studies suggest, that these modifications mean only minor improvement in plaque removal; greater improvements can only be achieved by applying the correct brushing technique.

Special tooth brushes

Special toothbrushes are indicated on the entire dentition combined with regular toothbrushes on few, hard to reach areas (wisdom teeth, crooked teeth), on teeth supporting some kind of restoration or on implants.

The orthodontic toothbrush has specially designed bristles that are shorter in the middle to leave space for the brackets and the arch, this way making it more effective in reaching tooth surfaces.

The end tufted brush is based on the most ancient oral hygiene device, the branches of the Salvadora Persica tree (“mishawk”). With this device, teeth can be cleaned one by one, especially at the sulcus area. The point of the technique is to guide the bristles on the side in the sulcus with gentle vibrating movements following the contour of the gums and the curvature of the teeth. Special versions of the end tufted brushes can be used to clean implants and implant supported restorations. The use of special toothbrushes will be discussed in later chapters with images.

Power toothbrushes

The first electric toothbrush was designed by a Swedish watch maker in the 19th century, but modern electric toothbrushes have only been available for little more than half a century. The first electric brushes had a vibrating movement, while newer ones work with rotating movements. Neither of the instruments brought a breakthrough, because studies showed similar efficiency to that of the correctly executed mechanical tooth brushing technique. Electric toothbrushes available today work in 6 different ways. The latest development of Oral-B combines rotating-oscillating movements (8800/min) with ultrasonic pulsation (20000/min and 40000/min) (Fig.4.9.).
Characteristics of power toothbrushes

With modern electric toothbrushes not only regular oral hygiene procedures can be performed, but replacement heads also make it possible to clean special areas. (Ortho-bracket, PowerTip-bridges, crowns, interdental spaces, 3D White-polishing, whitening, removal of discolouration, Tongue Cleaner, Interdental Cleaner). Fig.4.10 and 4.11.

<table>
<thead>
<tr>
<th>Design</th>
<th>Mode of action</th>
<th>Range example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral motion</td>
<td>Bush head action that moves laterally from side to side</td>
<td>Philips Sonicare</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Oral-B TriZone head</td>
</tr>
<tr>
<td>Circular</td>
<td>The whole brush head rotates in one direction</td>
<td>Only the heads in the market</td>
</tr>
<tr>
<td>Rotation oscillation</td>
<td>Alternating rotation (¼ circle)</td>
<td>Oral-B Vitality</td>
</tr>
<tr>
<td>Ultrasonic</td>
<td>The toothbrush filaments vibrate at ultrasonic frequencies (&gt;20 kHz)</td>
<td>Ultrasonex Brush</td>
</tr>
<tr>
<td>Rotation oscillation + ultrasonic pulsating</td>
<td>Combination of 3. and 4.</td>
<td>Oral-B Professional Care 500, 3000, Oral-B Triumph 5000</td>
</tr>
<tr>
<td>Counter oscillation</td>
<td>Adjacent tufts rotate in one direction and then counter-rotate with adjacent tufts moving in opposite direction</td>
<td>Interplak NT-11 (Conair)</td>
</tr>
<tr>
<td>Ionic</td>
<td>An electric current is applied to filaments during toothbrushing that alters the charge polarity of the tooth and results in the attraction of dental plaque towards the filaments and away from the tooth</td>
<td>Hukuba Ionic</td>
</tr>
</tbody>
</table>

Oral-B Triumph 5000 power toothbrush with SmartGuide and special heads
Heads of power toothbrushes (Oral-B)

The devices can be used in several modes. Motion and frequency can be changed, this way even sensitive areas can be cleaned without discomfort. Some even have massage and polishing function. Wireless smart displays can tell us time spent with brushing, time per quadrant and warn us if we apply too much pressure. Bristle wear and too much pressure are shown by marked bristle colour loss. Electric brushes without a display also have a timing function: a pause in the working motion indicates time over for a quadrant every 30 seconds, and a continuous pause in motion indicates the end of the session at 2 minutes. Figure 4.12.

Brand new and used heads

Several studies compared the efficacy of manual and electric toothbrushes, but only about 15% of them can be used for a meta-analysis. One reason for this is contradictory findings. A high number of data support the use electric toothbrushes. People buying electric brushes have much higher motivation: 62% of them use it regularly in the first 3 years after purchase. In developed countries sales doubled every year between 1999-2001.

Regarding efficacy, studies suggested that 1 minute of brushing with an electric device is equivalent to 6 minutes of manual brushing. While 1 minute of manual brushing showed a 60% plaque removal capacity, using the electric brush for the indicated 2 minutes showed 84% efficacy. Using it for a prolonged time, does not significantly increase efficacy (93% after 6 minutes).

Not all of the electric instruments prove to be more effective than manual cleaning: in the short (3 month) and long term (3 years), only rotating-oscillating instruments were proven to be more effective in terms of plaque removal and gingival indices. The individually rotating bristle group equipped devices showed plaque index reduction after 3 month, while ultrasonic vibrating brushes could only improve the plaque score after a longer period. No difference could be detected among the other devices compared to manual cleaning through meta-analysis.
Use of an electric toothbrush is recommended:

- for people with poor dexterity, who cannot perform proper manual oral hygiene
- if individual oral hygiene is temporarily or permanently hampered
- for those who are unwilling to perform proper oral hygiene with manual brushes

Cleaning of the tongue

Cleaning the back of the tongue is vital for proper individual oral hygiene. It is one of the main “ecological gaps” where pathogenic bacteria can live and grow unharmed, and it is also the source of further periodontal infections by enabling biofilm coaggregation and growth. This is the main principle behind the cleaning of the tongue in the full mouth disinfection protocol: it is performed with chlorine-hexidine gel. Patients who underwent this treatment had 40% less microorganisms on the back of the tongue than those, who missed this treatment. Bacterial colonies on the tongue are also important aetiological factors of halitosis.

Microorganisms need to be eliminated from the fissures and papillae of the tongue. For this purpose electric or manual toothbrushes, rubber inlays applied on the handle or the back of the brush (IMAGE) or special tongue cleaning devices (IMAGE) can be used with gentle scraping movements. According to some studies, these special tongue cleaning devices remove microorganisms much more effectively than any kind of brushes.

Textured tongue scarper on toothbrush head

Tongue cleaner

iTOP method

The “individually taught prophylaxis” (iTOP) method was developed based on the principle of individual instruction. This is a complex oral hygiene protocol, which aims to achieve completely plaque free dentition in every individual in a relatively easy way, without harming the dentition. According to the philosophy of Jiri Sedelmayer, the inventor of iTOP, the health of the dentition can be preserved lifelong, as long as perfect
individual oral hygiene is applied. His main achievement was to create an understandable, easily applicable structure for appropriate oral care. It follows 3 main criteria: efficiency, acceptance, atraumatic execution. Both the techniques and the equipment have to fulfill these criteria. Sedelmayer described a new preventive principle, the so called „anti-perio” principle, which means prevention of major periodontal diseases in order to maintain long term oral health. He developed an instruction technique, with which we can teach patients the correct technique like a „guiding hand”.

1.2. Interdental cleaning methods

Cleaning of interdental areas have an increased significance because they act as predilection areas for cavity formation and periodontal diseases. This is why this plays a key role in preserving healthy dentition. Although the efficacy of electric toothbrushes in interdental areas is superior to manual brushing, it cannot replace interdental cleaning procedures.

For effective interdental treatment, it is important to be familiar with the anatomical characteristics. The size and shape of the device should enable us to perform effective, atraumatic plaque elimination. The anatomical conditions and dental status also determine the optimal technique for inserting the dental floss. Methods of interdental cleaning should be determined individually with respect to patient needs and skills.

INSTRUMENTS FOR INTERDENTAL CLEANING

A broad spectrum of instruments are available for different anatomical situations and patient skills. (Fig.4.15.)

Dental floss

They are built up of numerous thin but durable filaments. The surface can be waxed or unwaxed. The latter ones seem to have a better plaque eliminating capacity, but they are also more difficult to use because of their high adhesion tendency. As easy it may seem, flossing is difficult, because it has to be effective and atraumatic at the same time. In order to achieve this, it is important to guide the floss through the contact points with slow, controlled, back and forth movements. After pressing it tightly to the cervical area of the tooth in the sulcus on both sides of the papilla up and down pulling movements will remove the plaque from the upper third of the sulcus.
Insertion of floss

The length of the floss should be about 240 mm. The two ends should be wound around the middle fingers. The index fingers together with the thumbs control the motion to prevent injuries (Fig. 4.17.)

Holding of dental floss

A modified version of the floss is the wider „dental tape”, which has a teflon coating, therefore great gliding ability, which increases the risk of causing injury. The different dental floss types are shown in the image below (Fig. 4.18.):
Types of dental floss

Flosses equipped with a holding frame, and power driven versions of this are also available on the market. (Fig. 4.19.)
Oral-B Birdie with floss

*Interdental brush*

They resemble classic glass cleaning brushes in a much smaller size. The bristles are attached to a flexible central wire. Similarly to toothbrushes, they are also composed of plastic or polymers of different width. The shape can be conical or parallel. Parallel ones are beneficial in space filling.

Necessary features of a good interdental brush: easy insertion in the interdental spaces (which requires flexible, soft bristles) and adequate space filling (which is determined by bristle length). The key to efficient interdental cleaning is proper size selection. Since every dentition is different in size and shape, the brush size needs to be selected individually. This can be carried out by trying the various sizes or by using a colour coded probe developed for this purpose.
Interdental access probing

After determining the interdental gap sizes, the technique is presented to the patients in their own mouth in front of a mirror. It is important to choose the most suitable insertion technique for the individual anatomical situation.

Brushes capable of cleaning interdental spaces entirely occupied by the papilla are the following: GUM SoftPick and TePe Interspace shown in the image below (Fig.4.21., 4.22.).
Interdental brushing with GUM SoftPick
Interdental brushing with TePe Interspace

Open interdental spaces, bridge pontics can be effectively cleaned with the brushes presented in the image below (Fig.4.23.)

![Interdental brushes](image)

**Superfloss - tufted dental floss**

Although the Superfloss is that of a specific manufacturer’s product, it stands for a special dental floss, which consists partly of regular dental floss and partly of a thicker fluffy portion, which has a stiff, line-like ending. It is primarily used for cleaning fixed partial dentures or bridge pontics. (Fig.4.24.)

![Tufted dental floss](image)

**Toothpick**

Despite their popularity caused by their visible efficiency in removing food remnants, their use has been pushed into the background due to low plaque eliminating and traumatising potential. Only soft, moisture absorbing, wax coated balsa toothpicks are suitable for interdental cleaning, as they have a triangular cross-section and blunt ending in order to follow the interdental contours. Handle inserted toothpicks are also available on the market. (Fig.4.25.)
Electric toothbrushes also have toothpick components, moreover there are separate electric devices designed to be used as a toothpick (Oral-B Birdie). (Fig. 4.26.)
Oral-B Birdie with toothpick

Hydrotherapy instruments (oral irrigator)

Electric devices, which use pulsating irrigation to remove food remnants and loosened plaque from the interdental areas and under pontics. Their disadvantage is that they cannot remove matured plaque; therefore they can only be used as additional oral care devices.

1.3. Toothpastes

Toothpastes are the most popular oral health care products in commercials. It is not a surprise, that thanks to strong marketing most people think that toothpastes and mouthwashes are the most important components of oral care. However, this is not true. Although commercials offer a wide variety of toothpastes both in ingredients and in effect for dental diseases, their true role is only to enhance the mechanical cleaning performed by the brush. Some studies have shown that 24 hours after tooth brushing only 27% difference was observed in plaque scores between groups brushing with toothpaste and ones with only tap water. It can be stated as a fact...
that complete removal of the plaque can only be achieved by mechanical biofilm elimination, therefore a well selected toothpaste or mouthwash can never replace thorough mechanical cleaning. It is also known that medium abrasive toothpastes can remove 50% more plaque from the buccal surface than regular ones. This proves that toothpastes play an important role in oral health care, regardless of whether it is regular toothbrushes or interdental brushes they are used with.

Two groups of toothpastes are now available on the market. Ones which have an abrasive component, and ones which do not. Abrasive materials improve the mechanical cleaning effect of the toothbrush, help remove dental pellicle and reduce tooth discolouring. This was recognised by ancient Egyptians 5000-3000 B.C. They made the first toothpaste from ash, egg shell, pumice powder and myrtle. Modern toothpaste was first manufactured in the 19th century. Today several studies precede the market release of every toothpaste in the hope of providing additional beneficial effects.

The main ingredients of toothpastes are identical:

- **Abrasive materials** (calcium-carbonate, calcium-phosphate, aluminium-oxide, silicate) comprise half of their weight. The abrasive effect of the paste depends on particle size and hardness. The abrasive materials also contain oily polishing materials, which even the surface damaged by larger particles.

- **Surface active materials** (fat-alcohol-sulphates, soaps) penetrate the plaque by decreasing the surface tension, this way helping the mechanical removal of the plaque. This is the cause of the foaming effect of toothpastes. Na-lauryl-sulphate is most commonly used for this purpose, because of its anti-microbial and plaque formation preventing effect. In case cation forms are used, it is possible to add chlorine-hexidine (antiseptic effect) and metal salts (reducing cervical sensitivity), which are incompatible with the anion surface active chemicals.

- **Solvents** (water, glycerine, propylene-alcohol) are responsible for the paste-like consistency

- **Sweeteners**

- **Stabilising and bonding agents** (water, glycerine, propylene-alcohol) (oils, gels, alginate) stabilise the consistency of the toothpaste

- **Flavours** (menthol, mint) make tooth brushing pleasant, provide a fresh breath

Most toothpastes on the market today contain some kind of inorganic or organic fluorine (amine-fluoride). Fluorine and strontium are responsible for enamel remineralisation. Fluorine-apatite crystals are more resistant to acids than hydroxyapatites.

Antiseptic ingredients, like chlorine-hexidine are also important chemical anti-plaque agents. There are hardly any regular toothpastes containing CHX, because Na-lauryl-sulphate is incompatible with triclosan and other enzymes.

Special, „tartar control‖ toothpastes containing pyrophosphate or zinc-nitrate are also available on the market, which have the ability to prevent crystallising. Certain products protect against cervical sensitivity by blocking the dentin tubules, this way reducing neuronal transmission. Organic fluorine, strontium-chlorine, potassium-nitrate or hydroxyapatite granules are used for this purpose.

Whitening toothpastes have also spread on the market, but they only serve to fulfil people’s aesthetic demands not to provide clinically beneficial effects.

### 1.4. Chemical plaque control

The most important measure to prevent dento-gingival plaque caused diseases is mechanical plaque elimination. In certain cases, mechanical plaque control is not sufficient or cannot be carried out properly: in these cases chemical agents can help prevent the formation, growth and maturation of the biofilm.

These situations are:

- if mechanical plaque elimination does not guarantee success (cases of aggressive periodontitis, where aetiological factors and severity are not proportionate)
• systemic diseases and risk factors play a role in the development and progression of the disease
• all diseases which do not react well to mechanical therapy
• areas that are hard or impossible to reach during self oral hygiene
• the patient is unable to maintain good oral hygiene due to physical or mental reasons

Goal of chemical plaque control:
• elimination of biofilm and bacteria in the oral cavity
• control the systemic effects of periodontal diseases (bacteraemia)
• infection control — reduces the risk of bacterial contamination during dental treatment

An ideal chemical agent would help re-establish the ecological integrity of the oral cavity by selectively eliminating pathogenic bacteria and supporting the bacterial population of the normal flora at the same time. Regarding prevention, it would be ideal to inhibit bacterial attachment on hard surfaces of the oral cavity. Unfortunately a substance like this does not exist, therefore our goal is to minimise the total bacterial count. Although several agents are effective against planktonic forms of bacteria, none of them can significantly reduce the oral biofilm due to its properties discussed in chapter 1.8.

Requirements for chemical plaque elimination in view of the above mentioned facts are the following:

The chemical agent used:
• persistently prevents bacterial attachment and reproduction or selectively eliminates pathogenic microorganisms
• reaches the areas barely accessible or inaccessible to mechanical plaque control
• is substantive (binds strongly to the surface it is used on and dissolves only slowly in a controlled manner) to provide prolonged effect
• should be biocompatible — not toxic, no local or systemic side effects
• should not disrupt the ecological balance in the oral cavity
• must not accumulate
• must not be irritative

Agents available on the market can be categorised by their biological effect, chemical structure, and the duration of effects. Based on biological effects, we differentiate:

• enzymes
• antiseptic agents (prevent the reproduction and growth of bacteria and fungi)
• antibiotics (not only do they prevent the reproduction and growth of bacteria and fungi, but they also eliminate them)

For chemical plaque control antiseptics are used, which can have short-term effects or long-term effects (substantive). Based on their chemical composition these agents can be divided into groups as follows:

• Phenols (Listerine, Triclozane)
• Quaternary ammonium compounds (CPC-cetylpyridinium-chloride, Benzetonia-chlorine, Domophene-bromide, Octenidine)
• Halogens (iodine – PVP-iodine -, Sn-fluorine, amino-fluorine)
• Metal salts (silver, zinc)
• Herbal alkaloids (sanguinarin)
• Bis-biguanide derivatives (chlorhexidine-digluconate)

**Applied agents**

_Triclosan (2,4,4’-trichlorine-2’-hydroxi-phenilether)_ and _CPC (cetylpyridinium-chloride)_ are agents of many toothpastes and mouthwashes. Because of their low substantivity, they have moderate antibacterial effect. Triclosan combined with zinc salts or copolymers (methoxymethylenne-malonic acid) have an increased antibacterial effect. Their extensive use is due to the lack of side-effects.

_Fluoride compounds_ play a major role in caries prevention. Their bacteriostatic property takes effect even in a low concentration, and it inhibits bacterial attachment on both tooth surfaces and acquired pellicle. Recently, tin- and amino-fluorine combinations have been used to prevent periodontal diseases in some toothpastes and mouthwashes, but their effect does not reach that of the „gold standard‖ chlorine-hexidine.

Another halogen compound, _PVP-iodine_ is not as well utilised in periodontal therapy as it should be. Its immediate antibacterial effect is comparable with chlorine-hexidine, but it has much higher biocompatibility. Contrary to elemental iodine, allergic reactions are also much rarer.

_Chlorine-hexidne (1,6-bi-4-chlorine-phenil-diguanide-hexane)_.

Chlorine-hexidine was first used after World War 2 (still used in the United Kingdom) for skin disinfection, and from the 1970s it has been used as a chemoprophylactic agent against dentogingival plaques. The former communist countries, like the Check Republic had substantial literature on its use originating from that time.

It inhibits both Gram positive and Gram negative bacterial reproduction, and also has anti-fungal effect. After it adheres to the cell surface, it penetrates the cytoplasm and destroys it, this way causing cell disintegration (bactericide effect). Attached to the pellicle and salivary mucin, it prevents bacterial adhesion. Substantivity is excellent (8-12 hours), but used in combination with Na-Lauryl-Sulphate care needs to be taken to wait 30 minutes between their use, otherwise the interaction inactivates chlorine-hexidine molecules. Its effect gets weaker with maturation and growth of the plaque.

Its effect depends on the quantity used not the concentration. This means that a greater amount of 0.12% solution can be as effective as somewhat less solution of 0.2% concentration. Concentration higher than 0.2% should not be used, because the efficacy does not increase but side-effects do. In the gel form 1% concentration is used. For long term use 0.06% solution is advisory. Besides the above mentioned, it is available in spray form as well.

Side-effects can be tooth and restoration discouloration, taste dysfunction, increased supra-gingival calculus formation, allergies in rare occasions. Some patients claim to suffer from irritation, burning sensation when using its alcohol-based solution. To minimize side-effects, only the water based solution is still available on the market.

_Essential oils (Listerine)_

In the last couple of years there was a strong campaign to promote Listerine. The active agents are several essential oils: thimol, menthol, methyl-salicylate, eucalyptol. Methyl-salicylate is a non-steroidal anti-inflammatory natural salicylate derivative, which has been used for treatment since the ancient Greeks as a painkiller and anti-fever medicine. It can absorb very well through the skin (it is the active compound of Ben Gay ointment, which is used for muscle and joint pain) and can even be secreted into breast milk.

Listerine belongs to the group of phenols. It destroys bacteria by damaging their protective layer, inhibits enzymatic activity and dissolves lipopolysaccharide components. It has a great penetration potential into the biofilm. It proves to be effective even against matured biofilm. In experimental settings, it showed 60% of chlorine-hexidine efficacy, but clinical settings showed an equivalent effect when combined with mechanical plaque removal. The water-based solution is preferable to alcohol solutions in this case as well. Listerine, besides CHD, was approved by the American Dental Association (ADA) as having anti-plaque potentials.

_Octenidine_
The active compound belongs to quaternary ammonium compounds. It has great efficacy against Gram negative and Gram positive bacteria, its biocompatibility is far superior to chlorine-hexidine. However, its effectiveness in dentogingival plaque elimination has not been proved clinically. It is used to irrigate root canals during endodontic treatment as an alternative for chlorine-hexidine and to disinfect and treat superficial skin wounds.

When using mouthwashes, one must keep in mind that they are effective max. at a 3-mm depth in the sulcus, this way treating the sub-gingival area can only be done by irrigation, or with a hard carrier in case of chlorine-hexidine (PerioChip)

1.5. Oral hygiene of patients with orthodontic appliances

The duration of orthodontic treatment is usually years. Whether the appliance is removable or fixed, it increases the risk of dentogingival plaque accumulation. It is of utmost importance to have good communication among the members of the treating dental team (dentist, paediatric dentist, orthodontist, dental hygienist). Regular and proper oral hygiene recalls, instructions and motivation are especially important to maintain periodontally healthy conditions throughout this increased risk period of wearing the appliance.

RISK FACTORS RELATED TO ORTHODONTIC TREATMENT

Age

The most affected age-group is that of teenagers. In this age-group, the incidence of gingivitis is relatively high and it shows an increasing tendency. In girls, this age comes with intense hormonal changes, which means another modifying factor.

Changes related to dentogingival plaque

The plaque retentive property of appliances can lead to caries and gingivitis. Gingivitis can be moderate or severe, often accompanied by gum overgrowth. As a result of inadequate oral hygiene cavities can form in a relatively short time (Fig.4.27.)
Gingivitis and gingival enlargement due to poor individual oral hygiene

**Tooth malalignment**

One of the indications of orthodontic treatment is malalignment of the teeth within the arch. This is already a risk factor for plaque accumulation. If severe malalignment inhibits the occlusion of opposing teeth, natural cleaning of the teeth cannot occur. If this is combined with inadequate oral hygiene, occlusal and even smooth surfaces of the teeth can develop calculus. (Fig. 4.29.).

Crowding and poor oral hygiene

**PROBLEMS ASSOCIATED WITH THE APPLIANCE**

Appliances increase the risk of plaque accumulation regardless of whether they are removable or fixed. On the other hand, orthodontic components themselves can cause injuries (rings, arches, brackets).
Individual oral hygiene cannot be performed

Regular oral hygiene is not sufficient when wearing fixed appliances. A special toothbrush is necessary to clean all components of the appliance. In case of a removable appliance, daily cleaning is also important, because biofilm formation also occurs on their surface.

Periodontal patients are also an important group, because correction of orthodontic disorders with a periodontal background is getting more and more common. These patients have to be very well controlled, because in this case patients with a disease which might have been caused by poor oral hygiene get an appliance that is highly plaque retentive.

IMPLEMENTATION OF PREVENTIVE MEASURES DURING ORTHODONTIC TREATMENT

During orthodontic treatment, a strict individual oral hygiene program must be applied. The methods of cleaning are determined by type and position of the appliance, and the anatomic conditions of the teeth and gingiva. Before handing over the appliance, steps of individual oral hygiene must be discussed and demonstrated in front of a mirror. At every follow-up appointment, plaque scores must be evaluated, and instruction and motivation performed if necessary. Patients with fixed appliances have compromised chewing and self-cleaning mechanism of their teeth. Chemical plaque control is always recommended in these cases. Interdental spaces are more difficult to clean, plaque accumulates in the brackets. To avoid the development of caries, fluorine containing toothpaste or even local fluorine application (medium to high caries risk) can be recommended.

Oral hygiene instruments

When having a removable appliance, care needs to be taken to clean the device besides regular oral hygiene procedures. Besides denture cleaning brushes, electric ultrasonic devices can be used for this purpose. The tank
of the device is filled with water. A special tablet is used in the water, which provides physical and chemical cleaning at the same time. (Fig. 4.33.)

Ultrasonic device with cleaning tablets

Cleaning of fixed appliances should be carried out with electric and manual toothbrushes. Besides these, special instruments need to be used (end tufted brush, interdental brush, Superfloss).

Steps of individual oral hygiene procedure for patients wearing fixed appliances

Sulcus cleaning method to clean areas in the proximity of the gums.

Regular toothbrush or a rotating-oscillating electric toothbrush with an Ortho-head can be used for this. Recent research suggests that electric toothbrushes are more effective than regular brushes in patients with fixed appliances in terms of plaque and bleeding scores. Using an Ortho-head with the electric brush provides better plaque removal than the regular head.

Cleaning the locks and arches of the appliance is best performed with electric or special toothbrushes. An oral irrigator can be used to remove the debris in advance. Steps of using the special ortho toothbrush, are demonstrated in the following images (Fig. 4.34-36.).
Interdental brush or Superfloss can be used to clean the appliance (Fig. 4.37., 4.38)
Cleaning the orthodontic appliance with interdental brush

Cleaning the orthodontic appliance with tufted dental floss

The next step is cleaning the interdental areas. Interdental brushes, Ultrafloss or Superfloss can be used for this (Fig.4.39.).

Cleaning of interdental area

Neglecting interdental cleaning can lead to carious lesions or gum inflammation, leading to enlargement of the papillae (see images above)

Maintenance therapy – retentive phase

In the retentive phase, extra-coronal splints are often used, which also support plaque accumulation and make oral hygiene procedures difficult. On the other hand, patient visits become more seldom. Just like in the case of active devices, oral hygiene instructions for retentive devices have to be shown to the patient. Follow-ups should be scheduled at least every two months. Plaque scores should be measured and professional oral hygiene performed at these visits.
1.6. Oral hygiene of patient with dentures

Dentures are devices with artificial hard surfaces to replace one or more teeth. As on natural hard surfaces (teeth), biofilm formation can also occur on dentures. This can cause damage to the abutment teeth, their periodontium or even to the oral mucosa. Oral hygiene aspects of denture design is discussed in chapter 5.7.

CLEANING OF FIXED PARTIAL DENTURES (FDP)

Cleaning of FDPs may start with removing the debris with an oral irrigator. Removal of the biofilm must be the next step.

The sulcus cleaning technique is the most appropriate in this case. High abrasive toothpastes must be avoided because they can damage the denture and even the exposed root surfaces. Both exposed root surfaces and the marginal areas are at risk of developing carious lesions. Fluorine containing toothpaste should be selected for these patients. For the proximal surfaces, interdental brush or Superfloss may be used. The area under the pontic can also be effectively cleaned with these instruments. (Fig. 4.40, 4.41.)

Cleaning of bridge with interdental brush
Cleaning of pontic by Superfloss

ORAL HYGIENE METHODS FOR REMOVABLE PARTIAL DENTURES

The margins of primary telescopic crowns are the biggest issue. The area between the gums and the restoration placed on the remaining dentition is hardly accessible to oral hygiene instruments. These restorations are made of different materials, which is another cause for concern. When choosing the correct toothpaste, abrasive and corroding effect must be considered.

The removable part must be cleaned in the morning, in the evening and after every meal. It is recommended to keep it in a disinfecting solution during the night. The steps of cleaning are the following:

Rinsing – Removes the debris but not the attached biofilm

Mechanical biofilm removal – Denture cleaning brushes are most suitable for this: the side with many bristles is good for cleaning the denture base, while the back side of the brush can be used for cleaning the mechanic anchorages (secondary telescope, matrix, etc.) (Fig. 4.42, 4.43.)

Denture base cleaning
Telescopic (secondary) crown cleaning

Electric toothbrushes should not be used for cleaning removable dentures, because some of their components can possibly damage the device by the bristles getting caught in them. A regular toothbrush is not recommended either, because denture components can destroy the bristles, making it ineffective for cleaning.

Professional oral hygiene treatments and home treatment (ultrasonic cleaning device, denture cleaning tablet) are both suitable for the antimicrobial therapy of removable parts. For home cleaning, dentures are put in a plastic bag filled with water, sealed, then put in the ultrasonic bath. (Fig.4.44.)
Denture cleaning in ultrasonic bath

For cleaning the remaining dentition, sulcus cleaning technique is recommended. Telescopic crowns and micro anchorage components need special brushes to be cleaned (interdental brush, end tufted brush). (Fig. 4.45.)

Primary telescopic crown cleaning with TePe Compact Tuft

The risk of caries can be reduced with appropriate diet, local fluorine treatment (Elmex fluid) and by following the right oral hygiene protocol.

**ORAL HYGIENE WITH COMPLETE DENTURES**

During the wear of a complete denture, pellicle and biofilm form on its surface. The biofilm contains mainly Gram positive (cocci, rods) and filamentous bacteria. Candida strains can also be found to some extent. If oral hygiene is inadequate, the biofilm on the denture gets mineralised and forms calculus. It can most often be seen near the main salivary gland excretions (upper molars buccal surface, lower incisors lingual surface). Improper cleaning can be a cause of halitosis. Candida albicans infection of the denture base can lead to the development of thrush.

**Professional cleaning**

Ultrasound cleaning mentioned in connection with partial removable dentures is recommended at every follow-up visit (6 month or 1 year). Heatable devices help to eliminate temperature sensitive microorganisms. Regular relining of the dentures does not only protect the edentulous ridge and create retention, but the process, which is done under high pressure also eliminates fungal strains persisting in the pores of the denture.

**Methods of individual oral hygiene**

Principles of partial removable denture cleaning apply for complete dentures, too. It is always recommended to add rinsing and soaking to scrubbing. Denture cleaning brush or regular nail brush and soap is suitable for removing the biofilm. In the angles and at the rugae area, end tufted brushes are the best choice. Abrasive pastes should not be used, because they damage the denture surface responsible for the vacuum effect.

Soaking can be added to this procedure: tablets release oxygen and create a foam effect, which removes the debris from the surface. Approximately 1 tablespoon of 5% sodium-hypo chlorine diluted in 100 ml water and 2 teaspoons Calgon can be used to make an antimicrobial and discolouration removing solution. Denture bases containing metal are not recommended because of their corroding properties. Hypo chlorine and peroxide based disinfection agents are also on the market, but they are not popular in Hungary yet. Requirements for denture cleaning agents: fungicide, bactericide, ease of use, effective in removing depositions, non-toxic, no damage to dentures, affordable price.
Patients wearing a complete denture have to clean the underlying mucosa as well after removing the denture. After rinsing with warm salty water at least once a day, the mucosa should be cleaned using a soft toothbrush. The cleaning of the tongue is also of major importance. Once a day the gums should be massaged with a regular or electric toothbrush, which improves circulation and improves mechanical resistance of the gums by facilitating keratinisation.

Denture glue is being used more and more frequently to stabilise dentures. Glue remnants have to be removed during every cleaning procedure from the denture and also from the gums. It is important to know that some denture glues might cause allergic reactions.

During dental examinations special attention must be directed to the most common complications of denture wearing: traumatic lesions on the mucosa, fungal infection (most common on the palate), angular cheilitis (inflammation of the corner of the mouth), epulis, gingival fibromatosis.

1.7. Oral hygiene of implant patients

The key to long term implant success is to preserve the health of peri-implant tissues, which have a much lower resistance compared to periodontal tissues. This is determined by two factors: the absence of inflammation around the periodontal structures of the remaining dentitions, and the composition and amount of biofilm surrounding implants, and its regular elimination. Pockets around natural dentition are reservoirs for periodontal pathogenic bacteria. Peri-implant tissues can get infected through these bacterial deposits. Prior to implant placement, successful treatment of periodontal diseases is required for the overall success of the surgery. After restoring the implants, a successful maintenance therapy can prevent the infection of peri-implant tissues.

METHODS OF INDIVIDUAL ORAL HYGIENE

The methods of individual oral hygiene are basically determined by the implant supported restoration. Based on the type of the restoration, we can classify 3 groups: cemented, screw-retained (crowns, bridges) and overdentures. In case of crowns and bridges, the cleaning protocol is basically the same as with conventional tooth retained crowns and bridges. Sulcus cleaning is recommended with an electric or manual toothbrush. Studying the peri-implant tissues in the aesthetic zone revealed that rotating-oscillating instruments did not cause any kind of damage.

However, at the peri-implant connection (both the proximal and buccal/lingual sides) special instruments are recommended. Interdental brush (with only plastic components!), toothpick, end tufted brush, and Superfloss can clean the proximal surfaces. Rounded, end tufted brushes and ovoid shaped special implant cleaning brushes are suitable for the buccal/lingual cleaning. (Fig.4.46.)
Special considerations are needed when selecting the right toothpaste. Highly abrasive toothpastes can damage the implant and fluorine can also cause unfavourable alterations on the titanium surface.

**PROFESSIONAL ORAL HYGIENE**

Strict maintenance therapy is essential for implant patients. In the first year following treatment, a 6-8 week follow-up is necessary to evaluate motivation, individual susceptibility to calculus formation and the condition of remaining dentition. After the first year, a minimum of 3-6 month follow-up is recommended, depending on patient.

Besides visual inspection of the peri-implant mucosa, probing depth, mobility, bleeding and plaque values are recorded, calculus formation and individual oral hygiene is evaluated. Radiographs are taken on a yearly basis to evaluate and compare bone levels and bone defects. From this point regular periodontal maintenance therapy is applied. Based on the recorded measurements, therapy is executed according to the CIST (Cumulative Interceptive Supportive Therapy) protocol. The principle of the CIST protocol is to introduce additional treatment steps if symptoms get more severe. (Fig.4.48.)

<table>
<thead>
<tr>
<th>PPD mm</th>
<th>PL</th>
<th>BOP</th>
<th>Bone loss</th>
<th>CIST</th>
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<tbody>
<tr>
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<td>PL &lt; 1</td>
<td>-</td>
<td>-</td>
<td>A</td>
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<tr>
<td></td>
<td>PL ≥ 1</td>
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<td>4-5 mm</td>
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<td>Notable cratering ≤ 2 mm</td>
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<td></td>
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<td>Bone loss &gt; 2 mm</td>
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<table>
<thead>
<tr>
<th>CIST</th>
<th>Procedures</th>
</tr>
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<tbody>
<tr>
<td>A</td>
<td>Mechanical debridement – scaling and polishing</td>
</tr>
<tr>
<td>B</td>
<td>Antiseptic cleansing 0.1 % CHX gel (2x daily for 3-4 weeks)</td>
</tr>
<tr>
<td>C</td>
<td>Systemic or local antibiotic therapy</td>
</tr>
<tr>
<td>D</td>
<td>Resective or regenerative surgery</td>
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</table>
PREVENTION

CIST protocol

The special instruments of diagnostic steps and therapeutic procedures are mentioned in chapter 5.2.2.

1.8. Oral hygiene of smokers

Treating smokers creates a great challenge for medical and especially dental professionals. The medical aspects of smoking are discussed in chapter 6.2. Considering that a large segment of the population visits dental practices regularly, dental professionals and dental hygienists are responsible for counselling in connection with smoking cessation. Smoking puts patients at a high risk for precancerous conditions and malignant cancer, therefore the examination and treatment of these patients need special precautions.

During examination, it is important to thoroughly discuss smoking habits and parameters. During history taking and extra-oral examination signs of smoking have to be discovered (halitosis, smell, yellowish finger tips, aging, lesions on the skin and lips). It is important to notice changes in vital signs (blood pressure, breathing, pulse, body temperature).

During intra-oral examination mucosal lesions must be noticed and shown to the patients. They have to be warned about their significance. Detailed documentation is important. If symptoms do not resolve after 2 weeks, patients need to be referred to a specialist, which needs to be documented on the patient chart. Self-examination of the oral cavity needs to be taught to patients. Any kind of lesion can lead to patients’ motivation to cessation.

SPECIAL CONSIDERATIONS IN INDIVIDUAL ORAL HYGIENE

Prevention of caries and periodontal diseases in smokers can be achieved with an increased level of individual oral hygiene compared to non-smokers. The technique is really important. Sulcus cleaning methods can stimulate the circulation in the anaemic marginal gingiva caused by smoking. Smoking supports calculus formation, together with stains from food, and it can lead to pronounced discoloration. To fight this highly abrasive toothpastes are beneficial.

SPECIAL ASPECTS OF PROFESSIONAL ORAL HYGIENE

Treating smokers takes a longer time due to the greater amount of deposition and more pronounced discolouration. It is important to inform the patient that healing after periodontal therapy takes a longer time for them and it is less effective. Special care needs to be taken for appropriate suction during treatment, since smokers are susceptible to respiratory diseases. Smokers like to use alcohol based mouthwashes against bad breath, but the long term use of these can increase the carcinogenic effect of smoking. A smoker’s lifestyle has to be well assessed. They have to be warned of the synergistic effect of smoking and alcohol consumption. The antioxidant capacity of smokers’ blood and their serum level of vitamin C is lower than that of others, therefore they have to be informed about the importance of antioxidant consumption in the form of fruit and vegetables.

2. The role of nutrition in prevention (Orsolya Rigó DMD)

IMPORTANCE OF NUTRITION IN ORAL HEALTH

Nutrition is an essential part of the human health, one of the modifiable factors that have an impact on the integrity of the hard and soft tissues and the host immune response. Dietary components play a major role in the pathogenesis of dental caries, but diet plays primarily a modifying role in the progression of periodontal disease. Periodontal health depends on a balance between the host, environmental and bacterial factors. Periodontal diseases are bacterial/inflammatory disorders that can be treated and prevented by the elimination of dental plaque in the adequately nourished individuals. Periodontal lesion is essentially a wound, and sufficient host resources must be available for optimal healing. Nutrition can influence the speed of plaque formation, and its composition as well.

Prospective studies that have been conducted suggest that there may be a link between periodontal disease and nutritional factors. Nutritional deficiencies can also weaken the resistance of oral tissues to plaque bacteria, which can result in more inflammation. Periodontal diseases are among the most common chronic inflammatory conditions seen in adults. Systemic inflammation alters the utilisation of fats, carbohydrates and protein and accelerates the metabolic consumption of key antioxidant vitamins and minerals. Some nutrients play a direct
role in the resolution of inflammation. Because of the role that key nutrients play in both the modulation of inflammation and the promotion of wound healing, oral health clinicians would do well to focus more attention on the interface between nutrition and periodontal diseases.

**DIETARY RECOMMENDATIONS**

A well balanced diet is important for the body and subsequently for the health of the mouth. There is an emerging need for clinical guidance on what nutritional advice should be offered to dental patients. The previous nutritional status of the patient and dietary intake during the recovery are all important aspects of nutrition that must be considered to improve the outcomes of periodontal treatments as well as other dental procedures. Food contains major or minor nutrients. Major nutrients are consumed in gram quantities and include protein, carbohydrates, lipids and water. Minor nutrients are required in milligram to microgram quantities and include vitamins and minerals.

**Major nutrients**

** Protein**

Protein has a large number of important functions in the human body, and in fact, the human body is made up of about 45% protein. It is an essential macromolecule without which our body would be unable to repair, regulate, or protect itself. Some proteins, for example collagen, which is a major organic component of bone, teeth, periodontal ligament and muscle, provide the structure of the body. Proteins are needed to provide adequate host defence. Mild protein malnutrition has been shown to reduce the effectiveness of the inflammatory response to invading pathogens.

**Recommended Dietary Allowance (RDA):** 0.8 g protein per kg of body weight; 50–75 g for adults (10–15% of total calories). The main food sources are meat, dairy products, legumes, soy protein, and fish.

**Carbohydrate**

Carbohydrate is basically sugar and starch. It is broken down into glucose molecules in the body during digestion. Carbohydrates serve as a source of energy, needed for connective tissue synthesis and are essential in fat metabolism, in erythrocyte and brain functions. Besides providing energy, sugars also produce the sensation of sweetness. Cellulose and some related substances are not used by our body as a significant source of energy, but these components are very important as they constitute dietary fibre. Dietary fibre is a complex carbohydrate with both soluble and insoluble form. High fibre diets have a cholesterol lowering and beneficial effect on intestinal disorders as well. The oral health status can affect the ability to ingest adequate levels of high fibre foods.

**RDA for carbohydrates:** 130 g per day for adults. RDA for fibre: 25–38 g/day. The main sugars in food are sucrose, glucose, fructose, maltose and lactose. Sucrose is obtained from sugar cane and is usually called 'sugar'. In addition, sucrose (as well as glucose and fructose) is found in fruits, fruit juices and honey. The main fibre sources: whole grains, vegetables, legumes, beans, potatoes, and nuts.

**Lipid**

Lipids include saturated and unsaturated fats, which have 9 calories per gram. While eating too much fat can lead to weight gain and a high risk for heart disease, getting a moderate amount of some kinds of lipids can improve human health. Lipids help provide energy, energy storage and thermal insulation and are needed for the body to absorb the lipid soluble vitamins A, D, E and K. Saturated fats are one of the main dietary factors that can raise the cholesterol level while unsaturated fat can lower the cholesterol when taken instead of saturated fat. Unsaturated fats are “good” fats: 30% of daily calorie intake should be unsaturated fat, like plant oils or fish oil as the Omega 3 fatty acid. A central hypothesis of periodontal medicine states that periodontal infection presents a chronic inflammatory burden at the systemic level. Omega 3 fatty acids have anti-inflammatory properties, inhibit systemic inflammation and their addition to the diet may help reduce the inflammation associated with periodontal diseases.

**RDA:** 44 to 78 g total fat per day (20 to 35 percent of total calories). Lipid food sources: saturated fats are found in foods that come from animals, like meat, and whole-milk dairy products. Monounsaturated fats are found in nuts, olives, dark chocolate, legumes, peanuts, and avocados. Polyunsaturated fats are found in nuts and seeds, soy, margarines, sunflower-seed oils and fishes.
Minor nutrients

Vitamins

Vitamins are organic substances utilised in metabolic reactions in the body. They are present in food in small (in milligram to microgram) quantities.

Vitamin A - a fat soluble vitamin, which is needed for maturation of epithelial tissues and required for vision. Excessive vitamin A originating from nutritional supplements or tablets can accumulate in the body and can lead to pathological conditions, it results in an increase in collagen catabolism and resorption of bone.

RDA: 700–900 µg/day. Food sources: carrots, pumpkin, spinach, collards, kale, offals like liver.

Vitamin C - ascorbic acid is a water soluble vitamin, which is an antioxidant and it is involved in many cellular functions. Vitamin C is needed for collagen production; proline hydroxylation requires ascorbic acid. The most obvious, first effects (gum and hair problems) of absence of ascorbic acid in humans come from the resulting defect in hydroxylation of proline residues of collagen, with reduced stability of the collagen molecule causing scurvy. Recent epidemiological data suggest the odds of having periodontal disease are 1.2 times greater in those with low dietary vitamin C intakes. Smokers and former smokers with low vitamin C intake are at 1.6 times greater risk of having a periodontal disease.

RDA 75–90 mg/day, for smokers a higher dosage +35 mg is recommended since oxidants from cigarette smoking lower vitamin C levels in the blood. Food sources: citrus fruits, dark green leafy vegetables, pepper, potato, kiwi, parsley.

Vitamin D is a fat soluble vitamin, which besides being obtained from the diet can be synthesised in the body as a result of adequate exposure to sunlight. Vitamin D functions to maintain blood calcium levels and the metabolism of osseous tissues. Vitamin D also enhances the absorption of calcium from the intestines. Low serum levels of vitamin D have been linked with a loss of periodontal attachment. Both collagen synthesis and accumulation of mineralised bone are dependent on adequate levels of vitamin D and calcium.

RDA for vitamin D: 400–600 IU/day (for adults under 51 years of age is 5 µg, for 51–70 years of age is 10 µg, for older than 70 years is 15 µg). Major food sources: fish, milk, eggs, liver and cereals.

B complex vitamins (B1, B2, B6, B12) water soluble vitamins, which are involved in energy production, red blood cell formation, protein synthesis and amino acid metabolism. Inadequate intake leads to breakdown of mucosal barrier to pathogens.

RDA: B1(thiamin) 1,2 mg, B2 (riboflavin) 1,3, Niacin (16 mg), B6 (piridoxin) 1,7mg, ,Folic acid 400µg, B12 (cyanocobalamin) 2,4 µg. Food sources: wholegrain cereals, potatoes, brown rice, bananas, lentils and beans.

Vitamin K

Vitamin K is fat soluble and required for blood clotting. Vitamin K can be obtained from our diet, but a significant amount of vitamin K is also produced by intestinal bacteria. There is an association between vitamin K insufficiency and reductions in bone density and possibly bone strength. This may be a concern among people taking K antagonists such as oral anticoagulants as a significant decrease in bone mineral density has been observed.

RDA for vitamin K: 90–120 µg/day. Food sources: green leafy vegetables, liver.

Vitamin E is fat soluble, and its primary role is to act as an antioxidant. Vitamin E is incorporated into the lipid membrane of cells, helping improve the cellular immune function.

RDA: 15 mg/day. Food sources: nuts and seeds, polyunsaturated vegetable oils, whole grains.

Minerals

Minerals make up about 4% of body weight and are found mainly in the skeleton, enzymes, hormones and vitamins. Minerals involved in the calcified tissues include boron, calcium, sodium, potassium, copper and magnesium. As dental professionals, we should focus on the connection between oral health and dietary calcium intake.
Calcium

Calcium is the most abundant mineral in the body. Calcium is required for vascular contraction and vasodilation, muscle function, nerve transmission and hormonal secretion, though less than 1% of total body calcium is needed to maintain these critical metabolic functions. Serum calcium is very tightly regulated and does not fluctuate with changes in dietary intake; the body uses bone tissue as a reservoir and source of calcium, to maintain constant concentrations of calcium in blood, muscle, and intercellular fluids. The remaining 99% of the body’s calcium supply is stored in the bones and teeth, where it supports their structure and function. Bone itself undergoes continuous remodelling, with constant resorption and deposition of calcium into new bone. The balance between bone resorption and deposition changes with age. Bone formation exceeds resorption in periods of growth in children and adolescents, whereas in early and middle adulthood both processes are relatively equal. In aging adults, particularly among postmenopausal women, the breakdown of bone exceeds its formation resulting in bone loss, which increases the risk of osteoporosis over time.

Calcium deficiency can lead to a decrease in serum calcium, resulting in mobilisation from host tissues. Low dietary intake of calcium is a risk factor for periodontal disease. Regular consumption of milk products appears to have beneficial effect on inhibiting the development of periodontal disease. The role of calcium supplementation is unclear and controversial.

RDA: women 14–18 years 1300 mg/day; 19–50 yrs 1000 mg/day, above 50 yrs 1300–1500 mg/day. Food sources: milk and dairy products, cheese, other foods containing lactic acid (yogurt), legumes, and cereals.

Nutritional supplements

Probiotics

By definition probiotics are “live microorganisms that, when administered in adequate amounts, confer health benefits upon the host”. Probiotics are specific bacteria, fungi and yeasts that are natural and necessary components of the digestive tract. They are beneficial to our body and an integral part of our immune system. There is an increasing interest in bacteria that can inhibit pro-inflammatory pathways. These microorganisms form a living colony (a biofilm) that lives within the lining of our digestive tract, in a symbiotic (mutually beneficial) relationship with us. "Bacteriotherapy” means administration of naturally occurring bacteria of human origin as therapeutic manipulation. Mechanism of action of probiotics: reinforcing the mucosal barrier; normalising an increased permeability; activating and modulating the immune system, so preventing infections at peripheral mucosal surfaces. Proven effects of probiotic treatment in medicine: beneficial for intestinal immunity; inhibition of bacterial colonisation; treatment of acute rotavirus diarrhoea; prophylaxis against fungal diseases; benefit on antibiotic associated diarrhoea; and benefit against inflammatory bowel disease.

According to recent clinical experience, probiotic intervention helps reduce the plaque and gingivitis, and accordingly it could be a useful tool in the treatment of inflammation and the clinical symptoms of periodontitis. In gingival sulcus probiotics inhibit the level of inflammatory mediator molecules (PGE2, IL 6-8). "Replacement therapy “– adhesion of probiotics instead of pathogens – can be a new treatment approach for periodontitis. A newly developed oral probiotic chewing gum, which includes approximately 500 million active Streptococcus salivarius bacteria, help fight infection and bad breath. It can help prevent tooth decay since it stops the ‘bad’ bacteria from sticking to the teeth. According to literature sources, the gum seems to be an effective pathway to introduce beneficial organisms to the oral cavity.

Polyphenols

Polyphenols are a group of chemical substances most commonly found in plants. Many polyphenols are the most abundant powerful dietary antioxidants, their antithrombotic effect is similar to that of the aspirin. Antioxidants are basically chemicals that reduce oxidative damage to cells. Medical researchers have found high correlation between oxidative damage and the occurrence of disease. Research suggests that consumption of food rich in antioxidant reduces the damage to cells from free radicals. Polyphenols have proven anti-inflammatory, antithrombotic and chemo-preventive (cancer preventive) effects.

Foods containing polyphenols are cranberries, black raspberries, strawberries, cocoa, dark chocolate, coffee, tea, red wine, tomato, citrus fruits, peach, soy, etc. Cranberry containing products have been shown to have beneficial effects on oral diseases. Studies have shown that cranberry juice concentrate can prevent the attachment of human pathogens to host tissues, and it is a potent inhibitor of biofilm formation and adherence properties of Porphyromonas gingivalis. Black raspberries have been investigated in relation to the treatment
and/or prevention of colon cancer, oesophageal cancer, and skin cancer. A topical black-raspberry gel has shown promising results in the prevention of the progression of oral precancerous lesions to oral cancer in a clinical trial.

**DIETARY COUNSELLING IN ORAL CARE**

In a healthy person who is not malnourished, these nutrient needs can be met through a balanced diet. However, alterations in diet to include food high in vitamins and minerals, and food rich in omega 3 may have positive effects on periodontal health. In addition, oral health clinicians have an important role in advocating healthful diets to their patients, to improve both oral and systemic health.

Healthy diet recommendations are reducing dietary saturated acids; minimising consumption of food and beverages with added sugar, eating a diet rich in vegetables, fruits, and whole grain foods; avoiding the use of and exposure to tobacco products; and achieving and maintaining healthy cholesterol, blood pressure and blood glucose levels, emphasising the importance of physical activity and weight control. Numerous epidemiological studies suggest that obesity is significantly associated with periodontal diseases, probably through the secretion of pro-inflammatory cytokines by adipocytes.

The role of antioxidant supplements, such as vitamins in preventing diseases is controversial and supplements are not recommended, but plant derived foods rich in antioxidant nutrients, such as fruits, vegetables, whole grains and vegetable oils, are recommended.

They are called “poly”-phenols because they have more than one phenol group per molecule. A phenol is any chemical compound which contains a six-member aromatic ring, bound directly to a hydroxyl group (-OH).

**3. Fluoridation (Ildikó Pinke DMD)**

The two important dental prevention principles are good oral hygiene and eating a balanced diet with moderate frequency of carbohydrate intake (especially sugar contained snacks), and in addition we can describe a third element of preventive dentistry: fluoride. Fluorides play an important role in the prevention and control of dental caries.

**SOURCES OF FLUORIDE**

Fluoride is one of the physiologically essential elements for normal growth and development, and its content varies in different types of food.

- The principle source is water (concentrations generally range from 0.01 to 0.3 ppm)
- Plants like tea also contain fluoride (an average of 97 ppm)
- Fish have a high content of fluoride (seawater contains more fluoride than fresh water with concentrations ranging from 1.2 to 1.5 ppm).

In plasma, fluorides exist in two forms (ionic and non-ionic): the normal plasma fluoride concentration (0.006 – 0.054 ppm) and the concentrations of breast milk between 0.003 and 0.011 ppm.

Fluoride in placenta and foetus: the placenta act as the barrier between the maternal blood and the foetus. Foetal blood contains 75% of maternal blood fluoride concentration.

Fluoride in hard tissues: 99% of all fluorides in the human body are found in calcified tissues such as bone and teeth.

Fluoride in plaque: the concentration of fluoride in plaque is 10% to 20% higher than in plasma.

**FLUORIDE INTAKE**

Over the past decades, due to the use of fluoride, the incidence and prevalence of caries have decreased significantly. The optimal fluoride intake is between 0.05 mg and 0.07 mg/kg (generally regarded as optimum).

1. The methods of fluoride delivery are systemic and topical
2. Other classification of the methods (according to mode of application)

- Community methods: on population basis – water, milk, salt
- Self-applied methods: used at home – toothpaste, mouth rinses, tablets, lozenges, drops, chewing gums
- Professional methods: used by health care professional -gels, foams, varnishes -slow-release fluoride devices

**SYSTEMIC FLUORIDATION**

- **Water fluoridation**: the best-known systemic fluoride method and the most effective way to deliver fluoride. (Fluoride concentrations: 0.7 – 1.2 ppm and fluoride compounds: hydrofluorsilicate, sodium fluorosilicate, sodium fluoride)

- **Salt fluoridation**: an alternative fluoridation method to drinking water. (Concentration: 250 – 300 mg/kg; fluoride compounds: sodium fluoride, potassium fluoride) The first epidemiological studies to evaluate the effectiveness of salt in reducing the caries prevalence were performed in Switzerland and Hungary.

- **Milk fluoridation**: (Concentrations: 2.5 – 5 mg/L; fluoride compounds: sodium fluoride, disodium monofluorphosphate)

- **Fluoride tablets and fluoride drops**: the individual way for children at high risk of caries. (Fig.4.49.) Tablets are chewed before swallowing (for a topical effect) and best taken at bedtime.

<table>
<thead>
<tr>
<th>Age</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 – 24 months</td>
<td>none</td>
</tr>
<tr>
<td>2 – 6 years</td>
<td>0.25 mg F/day</td>
</tr>
<tr>
<td>7 – 18 years</td>
<td>0.50 mg F/day</td>
</tr>
</tbody>
</table>

*Individual fluorid intake of children with high risk of caries*

**TOPICAL USE OF FLUORIDES**

Topical application of fluoride is one of the total preventive programmes that can be both professionally applied and self-applied by the patient.

- **Solutions**: the first professionally used fluoride (NaF, SnF2, APF, AmF, NH4F, TiF4) •Gels and foams (5000–12,500 ppm): they are more viscous than the solutions; and extensively used professionally and they may be self-applied as well. The are use for caries prevention in permanent teeth. Gels can be used with brushes or cotton pellets, but we can also use trays (minimising the risk of excessive fluoride intake).

- **Varnishes**: (1000–56,300 ppm): should be used in the prevention of caries in both primary and permanent teeth (professional use 2–4 times a year). The main advantages are the prolonged contact time between fluoride and tooth surfaces.

- **Rinses**: mouth rinsing is effective means for self-application home use of fluoride (daily: 0.05% NaF; weekly: 0.2% NaF)

- **Fluoride dentifrices**: The fluoride containing dentifrices are used for brushing two or three times daily.

They provide a frequent source of fluoride in low concentration (500–1500 ppm) that can inhibit demineralisation and enhance remineralisation. (Fig.4.50.)
Effect of fluoridation: teeth exposed to optimum level of F appear white, shining and opaque.

**TOXICITY**

Acute toxicity: rapid intake of an excess dose over a short period of time.

Chronic toxicity: excessive fluoride ingestion results in dental fluorosis.

The mechanisms affected by long-term chronic exposure to low levels of fluoride. When the level of fluoride is higher than 1.5 ppm in drinking water, dental fluorosis may occur.

**DENTAL FLUOROSIS**

The most important risk factor is the total amount of fluoride consumed during the critical period of tooth development. Fluorosis may occur when teeth are developing. All forms of fluoride intake comprise the daily consumption which includes water, foods and swallowed toothpaste (especially in young children). Children under 2 years swallow 50% of toothpaste during tooth brushing, and 5-year old children swallow 25% of the toothpaste.

An index with 6 categories according to the clinical manifestations of fluorosis (including normal teeth) was set up by H.T. Dean (Fig. 4.51.)

<table>
<thead>
<tr>
<th>Score</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normál (0)</td>
<td>Smooth enamel surface, glossy and creamy white color</td>
</tr>
<tr>
<td>Questionable (0.5)</td>
<td>Few white flecks, white spots</td>
</tr>
<tr>
<td>Very mild (1)</td>
<td>Small opaque paper white areas, not involving as much as 25% of tooth surface</td>
</tr>
<tr>
<td>Mild (2)</td>
<td>More extensive white opaque areas, not involving as much as 50% of tooth surface</td>
</tr>
<tr>
<td>Moderate (3)</td>
<td>All enamel surfaces are affected. Brown stain is frequently a disfiguring feature</td>
</tr>
<tr>
<td>Severe (4)</td>
<td>Includes teeth classified as moderately severe and severe. All enamel surfaces are affected, and hypoplasia is marked the general form of tooth. The major diagnostic sign is confluent pitting and teeth often present a corroded-like appearance.</td>
</tr>
</tbody>
</table>
Dental caries is a common childhood condition; it is about five times more prevalent than asthma. It is preventable by child oral health professionals, who can play a significant role in reducing the burden of the disease. Oral disease is diet- and behaviour-related and one of the most costly diseases.

ROLE OF ORAL HEALTH TEAM – FROM BIRTH TO SIX YEARS – encouraged to take actions:

1. Assess the risk of oral diseases (first at six month of age)
   - Inadequate fluoride exposure;
   - Caries in siblings and parents (identify women at high risk for dental caries as early as possible)
   - Age-appropriate oral hygiene of parents;
   - Frequent and prolonged exposure to sugary substances or use of night time bottle or sippy cup containing anything other than water;
   - Medications that contain sugar;
   - Clinical finding of maxillary biofilm or any signs of decalcification/ white spot lesions;
   - Special health care needs.

2. Counselling mothers to reduce the risk of caries in children
   - Wipe the infant’s teeth after feeding with a soft cloth or soft bristles toothbrush (especially the gum line);
   - Supervise brushing (small amount of fluoride toothpaste, because children have not fully developed the swallowing reflex);
   - Avoid putting the baby in bed with a bottle containing anything other than water (weaning children from the bottle and sippy-cup by nine to ten months of age);
   - Feed sugary foods at mealtimes only and limit the amount. Oral health professionals should focus on reducing the exposure to sugars;
   - Children should be protected from adult transmission of Streptococcus mutans that can be spread by mother, caregivers, siblings and other children: avoid saliva-sharing behaviours (sharing a spoon when tasting food, cleaning pacifier by mouth or wiping the baby’s mouth with saliva and between children via their toys, pacifier);
   - Visit a dentist with the child between six and 12 months of age.

3. Health promotion: educated pregnant women (and parents) aim to improve their oral health
   - Brush teeth twice daily with a fluoride toothpaste and floss daily;
   - Eat foods containing sugar at mealtimes only, in limited amounts;
   - Avoid carbonated beverages during pregnancy – choose water as a beverage;
   - Necessary dental treatment before delivery.

4. Facilitate appropriate referral for children assessed to be at increased risk for oral disease (carious lesions or white spot lesions)

ACTIVITIES FOR DENTAL HEALTH:
Periodic examination, preventive dental services and treatment for children

1. Birth to 12 months:
   - Perform oral assessment and diagnostic tests – to assess oral development or pathology;
   - Counselling for parents – emphasise oral hygiene, including the implications of oral hygiene of the caregiver and assess the parents’ risk for oral disease;
   - Remove biofilm and supra-gingival stains;
   - Assess appropriateness of feeding practices – bottle and breast-feeding (provide dietary counselling related to oral health as indicated);
   - Assess the child’s systemic and topical fluoride status – following assessment of total intake from drinking water, diet and oral hygiene products;
   - Provide age-appropriate injury prevention counselling for oro-facial trauma;
   - Provide counselling for bad habits (non-nutritive oral habits: pacifiers, thumb sucking);
   - Consult with the child’s physician (as needed);
   - Arrange the next appointment.

2. 12 to 24 months:
   - Repeat procedures listed in No I. every six months;
   - Review patient’s fluoride status, and provide topical fluoride treatment every six months (or more frequently if indicated).

3. 2 to 6 years:
   - Repeat procedures listed in No II. every six months (or more frequently if indicated);
   - Complete a radiographic assessment – pathology and abnormal development;
   - Professional cleaning;
   - Provide: age-appropriate oral hygiene instructions, topical fluoride treatments, pit and fissure sealants for primary and permanent teeth, assessment of developing malocclusion, treatment of developing malocclusion, or referral for developing malocclusion, diagnosis and treatment or referral for any oral diseases or habits.
   - Assess speech and language development

4. 6 to 12 years:
   - Repeat procedures listed in No III. every six months (or more frequently if indicated);

5. 12 to 18 years:
   - Repeat procedures listed in No IV. every six months (or more frequently if indicated);
   - Provide substance abuse counselling – e.g. smoking

PREVENTIVE ORAL HEALTH PROGRAMS

Programs in kindergarten: (Fig.4.52.)

- improve oral health behaviour, attitudes (especially for children from ethnic minorities and for children from a poor socio-economic background);
• develop their abilities through playing and exercise: the topic of discussion can be presented in half an hour with activities and discussion to serve a full session on dental care – questions, explanations and activities;

• enhance their knowledge – children will develop in three important ways: physically, emotionally and academically;

• focus on supervised tooth brushing with fluoride containing toothpaste (usually started by dental professionals);

**Presentations and activities:**

• Talking about teeth – why are teeth important?

• Talking about food – what is your favourite food?

• Keeping teeth clean – why do we need to clean our teeth?

• Dentist and dental helper (dental hygienists and assistants) – why is important to visit the dentist?

*The most important concepts to get across are:*

1. our teeth are important and we need to take good care of them;

2. the dentist is a friendly doctor who helps you take good care of your teeth.

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**School-based program:**

School years are an influential part of people’s life for the development of lifelong oral health related behaviour and attitudes

• introduced by oral health professionals in close cooperation with the teaching stuff;

• oral hygiene (daily supervised tooth brushing with fluoride containing toothpaste) and appropriate dietary practices;
• control the fluoride use;

*Presentations (grades 1-4) Fig. 2.:*

• Why is it important to have healthy teeth?

• What can we do to stop the decay?

*Conclusion:*

• Brush your teeth twice a day and floss your teeth daily;

• Eat nutritious foods every day from the five main food groups – grains, fruits, vegetables, meat/poultry and dairy products, and limit snacks;

• Visit your dentist.

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Preventive program for young school children

*Presentations (grades 5-6) Fig.3.:*

• Why is it important to have healthy teeth?

• What can we do to fight plaque and keep our teeth healthy?

*Conclusion:*

• Brush your teeth twice a day and floss your teeth daily;

• Eat nutritious foods every day from the five main food groups, and limit snacks;

• Wear a mouth guard (learn about what to do in case of dental injuries);

• Get sealants;

• Do not chew pencils or other things;
• Visit your dentist.

Presentations (grades 7-12):
• Home care: tooth brushing twice a day with fluoride toothpaste and flossing;
• Professional care: regular check-up and professional cleaning;
• Sport protection: mouth guards should be used by everyone who plays contact sports, such as football, boxing, soccer, ice hockey, basketball,
• Orthodontic therapy: orthodontic care for children with developing malocclusions
• Prevent bad habits:
  1. Tobacco
  2. Oral piercing

5. Fissure sealants (Ildikó Pinke DMD - Zsuzsanna Tóth DMD)

The occlusal surfaces of posterior teeth are known to be the most susceptible location for dental caries. Natural cleaning mechanisms such as saliva, tongue, other oral muscles, as well as the mechanical cleaning methods such as the toothbrush bristles cannot reach to the base of the pit and fissure.

Approximately 3 years after the eruption of teeth, occlusal caries reaches its peak incidence in newly erupted molars, with high prevalence and rapid onset.

Pits and fissures:
• occlusal surface of the lower molar – with vestibular surface
• occlusal surface of the upper molar – with palatinal surface
• occlusal surface of premolar
• palatinal surface of upper lateral incisor

The high caries incidence of these areas is related to the morphology of the pit and fissures. Occlusal pits and fissures vary in shape but are generally narrow, tortuous with invaginations and irregularities, where bacteria and food are mechanically retained.

**DIAGNOSIS**

Tooth selection for sealant placement: evaluate pit and fissure surfaces

1. Caries – free:
   - SEAL (Fig.4.55.): if a patient is at risk for caries based on evaluation (pit and fissure morphology, eruption status, caries history, risk factors for future caries)
   - Do not seal: monitor – if the patient and teeth are not at risk (re-call every 3 month)

2. Questionable:
   - SEAL

3. Enamel caries:
   - SEAL, extended seal

4. Dentin caries:
   - Restorative procedures

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**FISSURE SEALANT**

**Definition:**

Fissure sealant is a material that is placed in the pits and fissures of teeth in order to prevent the development of dental caries.

• Preventive procedure: part of a complete prevention program
• Occlusal pits and fissures, buccal pits
• Newly erupted teeth (between 6 and 12 month)
**Indication of sealant placement**

- Newly erupted primary molars and permanent molars with pronounced pits and fissures
- Upper lateral incisor (cingulum pits)
- Patients with previous caries experience (high caries susceptibility)
- Level of caries activity

**Contraindication of sealant placement**

- Caries free teeth with shallow wide grooves
- Presence of occlusal caries
- Approximal surface

**Clinical Procedures**

**Patient preparation**

Explain the steps and the procedure to be performed because we have to use chemicals of etching and sealant and light curing lamp. The patient must wear safety eyewear for protection.

**Rules of treatment**

- Treat each quadrant separately
- Success and effectiveness of treatment depends on the method with the assistant (use four-handed method)
- Follow manufacturers’ direction
- Ensure wet control

**Technique of sealant application**

1. Prepare the teeth:

- Clean the pit and fissure surfaces
- Utilise a dry toothbrush, prophy cup with pumice or prophy paste, or air abrasion
- Use an explorer (probe) to remove any debris in the pit or fissure
- Rinse for 20–30 seconds
- Re-evaluate surface for residual or loose debris

2. Isolate the teeth:

   The purpose of the isolation is to keep the tooth clean and dry for optimal action, bonding and sealant.
   - Use cotton rolls with saliva ejector and/or rubber dam

3. Dry the surfaces:

   - dry teeth with air for 20–30 seconds

4. Etch the surfaces:

   Purpose: provide retention for sealant; remove contamination from enamel; provide antibacterial action
• Apply etching gel as directed by manufacturer (low viscosity of gel or semi-gel to ensure good penetration (Figure 4.56.))

Duration: depends on the manufacturer and the tooth development
• Usually between 30–60 seconds
• Newly erupted molars – half time
• Fluoridated tooth, and primary teeth – double time

5. Rinsing and drying the teeth:
• Rinse surfaces for 60 seconds
• Dry teeth with air for 20–30 seconds
• Check for effectiveness of etchant by drying with air; surface should appear “chalk white” (if does not, repeat etching process)

6. Application of sealant material and apply curing light to material:
Apply the material according to manufacturer (Figure 4.57.)

- Apply the minimum amount of sealant – prevent air bubbles,
- Cover all pits and fissures (don’t overfill to a high-flat surface),
- Optimum penetration – leave it on for 10 seconds.
- Curing: 20–30 seconds (according to instructions); longer time increases retention
- Polishing, finishing

Application of fissure sealant

7. Occlusal evaluation:
- Evaluate sealant for any marginal discrepancy or retention problem
- Check occlusion with articulating paper
- Local fluoridation

8. Re-call:
- Sealants can be retained for many years
- Check the sealed teeth every six months for reapplication if needed
- Re-examination and replacement when indicated
Before and after fissure sealing

EXTENDED FISSURE SEALING

In case of caries-free dentin, the discoloured but healthy fissure is not to be prepared. (Black, brown and grey discoloration can originate from pigments of foods and beverages.) After careful preparation of demineralised but cavity-less fissure with narrow taper fissure bur, the extension of carious lesion will be seen. This is the moment to decide whether fissure sealing, composite or amalgam filling should be prepared.

The adhesive technique is used also for extended fissure sealing, that is why only the carious hard tissue should be removed (minimally invasive preparation).

Steps of extended fissure sealing:

1. Isolation
2. Cleaning of the fissure
3. Exploration of fissure with narrow taper fissure bur: fissurotomy
4. Minimally invasive removal of small-size caries
5. Drying of the tooth surface
6. Etching – rinsing – drying
7. Composite filling – photopolymerisation
8. Fissure sealing of the remaining part of the fissure system – photopolymerisation
9. Checking the occlusion – finishing, polishing if necessary
10. Dental recall
Chapter 5. THERAPY

1. General and local anaesthesia (Judit Méray MD - Csaba Berkovics DMD)

GENERAL ANAESTHESIA

The aim of every form of anaesthesia is to eliminate pain sensation. Local (and regional) anaesthesia reversibly blocks the ability of nerve endings, peripheral nerves, nerve bundles and spinal roots to conduct stimuli, whereas in general anaesthesia the block of the perception is attained by the temporary blockade of the corresponding structures of the central nervous system.

The main characteristics of general anaesthesia are the following: analgesia (freedom from pain), narcosis/hypnosis (loss of consciousness), anti-nociception (blockade of undesirable vegetative reflexes) and – if necessary – muscle relaxation (blockade of the striated musculature).

Earlier it was attempted to achieve all the above effects through the administration of a single anaesthetic agent – the classic mononarcosis – though this involved the danger of overdosing the drug in question. A classic example was ether anaesthesia. Monoanaesthesia is rarely applied nowadays; usually only for brief interventions.

In combined general anaesthesia we try to meet the above mentioned requirements through the combined use of a number of appropriately selected drugs (e.g. hypnotic, analgesic medication, muscle relaxants). In this way lower doses of each drug can be given, so that there is a lower risk of overdosing and the danger of undesirable side-effects can be reduced as well.

For the purposes of general anaesthesia (disregarding unusual, not routinely applied methods like electro-anaesthesia or psychological hypnosis) so called anaesthetics are applied. In order to elicit their effect they have to get to the appropriate structures of the central nervous system through the blood stream. The blood stream can be entered in several ways, but only inhalational or intravenous application is really suitable for the purposes of anaesthesia. In the former case anaesthetic gases and vapours are added to the inhaled mixture in a given concentration and they are taken up by the lung capillaries all over the big surface represented by the alveoli, whereas in the latter case a precisely calculated amount of the anaesthetic is injected into the venous system. The methods can be combined; this combination is called balanced anaesthesia.

Groups of anaesthetic drugs most often used for general anaesthesia

- Inhalational anaesthetics: gases (e.g. nitrous oxide, xenon) or vapours – volatile anaesthetics (e.g. sevoflurane, desflurane, isoflurane)

- Intravenous anaesthetics: drugs with narcotic/hypnotic properties (e.g. propofol, thiopental, etomidate, ketamine), analgesics (pain killers) and muscle relaxants (e.g. rocuronium, pancuronium, atracurium, etc.)

For the purposes of surgical anaesthesia we use almost exclusively synthetic opioids - narcotic analgesics (morphine, fentanyl, sufentanil, remifentanil, etc.). Small doses of some sedato-hypnotic agents have anxiolytic and sedative effects if given in a low dose, whereas higher doses make the patient sleep. Certain properties of anaesthetic agents show some kind of overlapping: opioids have sedative effects, inhalational anaesthetics (and benzodiazipines as well) may enhance the effect of muscle relaxants.

Implementation of general anaesthesia

The performance of general anaesthesia is the task of specially trained doctors, anaesthetists. During anaesthesia the anaesthetist monitors the general state of the patient and controls the vital functions with appropriate methods (drugs, fluid substitution, transfusion, etc.) in order to preserve a balance, so the surgeon/dentist can concentrate on the operation. The anaesthetist is assisted in his/her work by the nurse anaesthetist.

The anaesthetist is also responsible for the maintenance of free airways and ventilation over the course of the operation. Since during head and neck surgery (ENT-, maxillofacial surgery, dentistry) the working areas of the two disciplines overlap, the cooperation between them is extremely important.
This cooperation should prevail in the establishment of the indication and the choice of the method of anaesthesia (local or general) as well. The decision is influenced by the general status of the patient and the entire risk of anaesthesia.

**Preanaesthetic procedures**

Anaesthesia always carries a certain level of risk – depending among other things on the state of the patient, the knowledge and skill of the participants, the equipment and the type of the intervention, but under optimal circumstances, in patients who are in good general state, the danger is minimal. It is important that planned (elective) interventions should only be performed in the best possible condition of the patient.

The **preanaesthetic examination** of the patient is necessary, even if he/she claims to be healthy. The extent of this examination depends on the age, general condition of the patient and the type of the intervention. A short assessment of the general status of the patient has to be done even before urgent interventions. The usual steps of the examination are the following:

- **History**(previous anaesthesia, accompanying diseases, bleeding disorders, medication, allergies, infectious states, etc.)
- **Physical examination**(blood pressure, pulse, airways, auscultation, etc.)
- **ECG, laboratory tests, cross matching, chest X-ray… etc.**

The necessity of these is decided by the anaesthetist on the basis of the general condition, age of the patient, planned surgery etc.)

The most important steps of preparing the patient:

- **Achievement of an ideal condition**(control of blood pressure, diabetes, etc.)
- **Empty stomach**: patients are not allowed to have solid food starting from 5-6 hours before anaesthesia, but they can drink clear fluids (water, tea, juice without pulp) until 2 hours before the intervention, except for those with slowed gastric emptying (chronic alcohol abuse, diabetic neuropathies, pregnancy, etc.)
- **Empty urinary bladder**
- **Premedication**: Usually anxiolytic medication is ordered (e.g. benzodiazepines, like midazolam). Oral premedication should be taken 1 hour before the start of anaesthesia followed by a small mouthful of water. (IM or IV premedication is rare nowadays.) Sometimes it may be necessary to order other drugs, like antacids, antihypertensive medication, asthma-aerosols, etc.

Somewhere between interventions performed in local anaesthesia on totally alert patients and those under general anaesthesia with loss of consciousness (plus commonly of the ability of spontaneous ventilation as well), there is surgery using local anaesthesia but under various depth of sedation of the patient. Figure 5.1. shows the degrees of sedation.
The degree of sedation

It is recommended to order oral sedatives (e.g. midazolam) for all anxious, worried patients, especially for those with hypertension or ischaemic heart disease. A typical example of „awake sedation‖ is the inhalation of nitrous oxide through a nasal mask: the patient is inhaling a mixture of oxygen and nitrous oxide, the sedative and analgesic properties of which may alleviate the dental treatment of hard to manage patients.

All sorts of intravenous sedation and the use of volatile inhalational anaesthetics fall under the term of deep sedation. As there is a continuous transition from deep sedation to general anaesthesia, and the individual reaction of the patients to the agents used for this purpose may be very different, in respect of the necessary conditions deep sedation should be considered general anaesthesia.

The necessary requirements of general anaesthesia

General anaesthesia can only be performed if all the necessary preconditions are fulfilled.

Subjective (personal) conditions

• Anaesthetist - a physician with appropriate professional knowledge and experience. (The surgeon carrying out the intervention must not administer anaesthesia at the same time, even if he is specialised in anaesthesia as well.)

• Nurse anaesthetist

• Adult person attending the patient after discharge

Objective (material) conditions

• Readily accessible operating table or dental chair that can safely be brought into a lying position;

• All the necessary means of airway management: Guedel airways, face masks, endotracheal tubes, laryngeal masks with appropriate connectors in all the necessary sizes, laryngoscope and tools for the fixation of the tube.

• Equipment for artificial ventilation with accessories and connectors (tested, ready to use), oxygen cylinders, reductor (or central oxygen supply), tubes, anaesthesia machine or respirator;
- High performance, perfectly functioning complete suctioning system with oral suction piece and tracheal suction tube;

- Equipment for intravenous cannulation and maintenance of the venous access – IV cannulas in all sizes, infusion sets, syringes, needles in the necessary number and size.

- Anaesthetic drugs and emergency medication

- Monitoring system. The minimal monitoring: ECG, automatic blood pressure, pulsoxymetry, temperature measurement, stethoscope, capnography. (For patients in critical state or for more serious interventions many other tools may be necessary!)

- Phone connection, availability of emergency medical help (ambulance service, defibrillator, intensive care unit);

- recovery room with bed and monitoring devices

In addition, the patient should get satisfactory information about the planned anaesthesia, its preparatory steps and the post-anaesthesia period, as well as about the risks of the procedure. They must be able to comprehend the information provided and after consideration make a balanced decision and sign a so called „informed consent”.

The indications of general anaesthesia in dentistry, dentoalveolar and maxillofacial surgery are the following:

- short, painful interventions where no satisfactory local anaesthesia can be applied (e.g. drainage of abscesses)

- long interventions including the facial bones, often with serious blood loss (e.g. resection of the maxilla, excision of intraoral malignancies with aesthetic reconstruction, etc.)

- dental, dentoalveolar interventions in difficult to treat, uncooperative patients (especially if previously attempted local anaesthesia and sedation did not work)

- allergy to all possible local anaesthetics (extremely rare situation!)

- long, complex dental rehabilitation – at the special request of the patient or for other reasons (childhood) - only if the risk of general anaesthesia does not exceed its benefits.

The necessary intervention should be carefully planned beforehand and if possible, everything has to be done in „one session”.

The usual phases of general anaesthesia:

1. **Induction.** It lasts from the start of administering the anaesthetic medication to the time when the patient is ready for the surgical intervention. In order to achieve this we use hypnotic/narcotic agents (e.g. propofol, ketamine or a volatile agent like sevoflurane), secure the airways (e.g. through endotracheal intubation or laryngeal mask), give opioids and may use muscle relaxants. Before interventions with significant bleeding in the oral cavity it is recommended to apply a throat pack.

2. **Maintenance:** During surgery or dental treatment a satisfactory depth of anaesthesia and analgesia has to be maintained (using intravenous or inhalational anaesthetics and opioids). The vital functions of the patient are continuously monitored and adjusted as necessary, using drugs, fluid replacement etc.

3. **Awakening period:** Near the end of the intervention we reduce the concentration of the anaesthetics in order to achieve a spontaneous termination of their effect. Naloxone for the antagonisation of the effect of opioids or anexate - the antidote for benzodiazepines - are rarely used, in exceptional circumstances, whereas antagonisation of muscle relaxants is often necessary. The classic antidote is neostigmine, a cholinesterase inhibitor, where the unwanted effects of the accumulated acetylcholine can be prevented by giving atropine. (After the use of rocuronium the most effective antidote is sugammadex - the only obstacle in the way of its extensive use is the high price of the drug.)

4. **In the post-anaesthesia period** patients should be kept under close supervision, their vital functions have to be continuously monitored. Besides regular measurement of blood pressure and pulse rate the monitoring of breathing (e.g. oxygen saturation monitoring) is also very important, as the residual effects of both opioids
and muscle relaxants may cause respiratory depression, hypoxia and hypercapnia. It is also important to provide satisfactory analgesia: for strong pain opioids and non steroidal anti-inflammatory drugs (NSAIDs) are ordered, for medium and small pain weak opioids (e.g. tramadol, nalbuphine) and NSAIDs (e.g. diclofenac) or only the latter alone may be enough.

**LOCAL ANAESTHESIA**

Local anaesthetics are chemical substances that reversibly suspend the conduction of the neural tissue. Strength of a given local anaesthetic is characterised by the minimal concentration at which the substance temporarily suspends neural conduction (Minimum Effective Concentration, MEC). Strength and toxicity defines the therapeutic value of the given anaesthetic as dependent on efficacy and tissue tolerance. Diffusibility is the ability of the given substance to reach tissues (relatively) distant from the site of application. Compounds of good diffusibility may also anaesthetise the mucous membrane. Duration of action is the length of time the anaesthetic has an effect (anaesthesia).

Anaesthetics used in the daily practice contain vasoconstrictor compounds (tonogenes), which delay the absorption of the compound, thus protracting the duration of action. Adrenalin is the most frequently used tonogene. Anaesthetics free of tonogen are also available, with limited indications (e.g. in severe cardiac decompensation). It must be taken into consideration that tonogene-free anaesthetics are less potent.

Currently, articaine and lidocaine are used in oral surgical and dental practice. For allergic individuals, compounds like bupivacaine are available.

**Lidocaine**

As it has an excellent diffusibility, it can be used for terminal, conduction and mucous membrane anaesthesia, too. A solution of 2% cc. is used, of which, together with adrenaline (1:100,000) maximum 20 mL may be given (10 mL without adrenaline). It is also available in spray (10%) and ointment (5%) forms.

**Articaine (Ultrapain, Septanest, Ubistesin, Scandonest-sine tonogeno)**

It has an outstanding diffusibility, and it is also effective in the anaesthesia of inflamed tissues. It is available in a concentration of 4% with adrenaline (1:100,000). Its maximal dose is 12.5 mL (500 mg).

**Types of local anaesthesia**

In **terminal (infiltrative)** anaesthesia, nerve conduction is blocked directly at the level of the sensory endings. This can be done in three ways:

1. **Mucosal anaesthesia**

Superficial anaesthesia, mostly used for the extraction of milk teeth, smaller mucous membrane corrections or the pre-anaesthesia of injection sites.

2. **Submucous infiltration**

The most frequently used terminal method for mucous membranes with submucosa. The anaesthetic solution is injected over the periosteum and near the apex. This way a depot is formed from which the anaesthetic diffuses to its destination through the periosteum and bones.

3. **Intramucous infiltration**

The anaesthetic solution is injected into the tight alveolar gum with a thin needle, but it may be painful, thus, preliminary submucous infiltration is recommended. It is a useful anaesthetic method when a periapical soft tissue abscess is present.

4. **Intraligamental anaesthesia**

This method is used for the individual anaesthesia of single teeth. A thin needle and a high pressure syringe are used.

In **conductive anaesthesia**, conduction in the peripheral nerve trunk is blocked. A depot is created around the nerve trunk from which the anaesthetic diffuses to the nerve fiber through the peri- and endoneurium. Precise
anatomical knowledge is vital. If the anaesthetic is deposited too far from the nerve, it may be diluted beyond 
the minimum effective concentration.

When performing submucous and conduction anaesthesia, delicate movements of the syringe and needle prevent 
the anaesthetic from accidentally getting into a blood vessel.

2. Periodontal therapy (Péter Vályi DMD - László 
Párkányi DMD - István Varga DMD)

2.1. Treatment planning in periodontology

Periodontal treatment planning involves a complex set of tasks involving the entire dental team (the general 
practitioner, the periodontologist, and the dental hygienist) and of course the patient himself/herself. The dental 
hygienist needs to be able to understand the entire periodontal treatment process, not because of having to 
perform them all, but to understand the goal, to have an overview of the order of steps, and to be able to evaluate 
the efficacy of the treatment.

Treatment planning is based on proper patient examination and diagnosis, and evaluation of risk and prognostic 
factors (on patient, dentition, and defect level). The following basic questions need to be answered:

• Have periodontal tissues suffered damage?
• Is the damage reversible (gingivitis) or irreversible (periodontitis)?
• What type of periodontal inflammation is present?
• Is it localized or generalized?
• Is there any systemic condition/disease in the background?
• Is there any bad habit in the background?
• Does any local natural or iatrogenic factor influence the disease?
• Does any alteration related to periodontal destruction aggravate the condition?
• How well does the patient cooperate?
• What are the patient’s needs?
• Costs, cost-effectiveness.

APPROACHING THE THERAPY

When treating periodontal diseases, multiple approaches can be used considering the above mentioned issues:

• conservative
• radical
• palliative.

The treatment method depends on the nature of the disease, its severity, and the cooperation of the patient. A 
mild to moderate chronic periodontal infection (where aetiological factors correlate to the disease severity) can 
be well managed with conservative (non-surgical) methods, since pockets are moderately deep, supra-osseal 
defects within the reach of professional oral hygiene devices. In the aggressive form (where aetiological factors 
do not correlate to the severity of the disease) however, the same depth of a periodontal pocket can indicate 
much higher risk of progression because of the defect in anatomy; therefore, radical (surgical) therapy is 
recommended to provide an access for individual and professional oral hygiene.
If the patient is incapable of maintaining individual oral hygiene for some reason, even after multiple instruction and motivation sessions, the goal would be to slow down the progression of the disease by means of regular professional oral hygiene treatment, until a complete edentulous stage. This is called palliative therapy taken over from oncological terminology.

**PERIODONTAL TREATMENT PROCESS**

Periodontal treatment plan is described in the image below:
"0" preliminary phase

Some precautions are required related to the management of systemic diseases (antibiotic prophylaxis, prevention of bleeding, adjustment or change in medications if necessary) beside the treatment of symptoms of acute pain.

Initial therapy

Initial therapy has many synonyms: primary, causal, non-surgical phase. The goal of the therapy is to eliminate aetiological factors by means of non-surgical methods: removing dentogingival plaque, correction of plaque retentive factors, correcting major occlusal discrepancies in a conservative way. The aim of this phase is to motivate patients, and to teach them individual forms of oral hygiene.

Periodontal health is also influenced by endodontic diseases; therefore, endodontic treatment should be performed at least partially in this phase. It involves chemo-mechanical treatment of the canals, and at least a provisional root canal filling to stop the infection.

To achieve a better healing outcome, mobile teeth may be splinted for the duration of the treatment.

Re-evaluation

Re-evaluation is performed by measuring periodontal parameters 6–8 weeks after treatment. This timeframe is necessary to evaluate regenerative potential of the tissues, motivation of the patient, and efficiency of non-surgical treatment. If the inflammation could not be reversed, and it is not possible to provide individual and professional oral hygiene, corrective phase is the next step. Patient motivation and cooperation is a prerequisite for this phase. If the recorded parameters are not acceptable, the hygienic phase may be repeated to increase patient motivation. If it is still not successful, palliative treatment is to be performed with the appropriate restorative therapy.

The corrective phase

The most important part of the corrective phase is periodontal surgery. During periodontal surgery, aetiological factors remaining form non-surgical therapy can be eliminated to provide the circumstances for reparation or regeneration by establishing ideal conditions for individual and professional oral hygiene. If regenerative treatment is performed, some of the damaged tissues go through complete healing, and thus improving tooth prognosis.

Periodontal plastic surgery deals with soft tissue correction to improve oral hygiene, and satisfy patients in terms of function, phonation, and aesthetics.

An important part of corrective therapy is occlusal therapy. The goal of occlusal therapy is not only to achieve ideal contact of the teeth, but a long-term stabilisation of the teeth, until the determination of the final prognosis (maybe month or years later) with semi-permanent splints. Orthodontic treatment can be a part of the correction phase. Its goal is not only to establish ideal contact between the teeth and to correct defects compromising individual oral hygiene, but it may also influence neighbouring bone volumes, and this way correcting some defects. With orthodontic extrusion, a less traumatic crown lengthening, with an extraction, vertical bone gain can be achieved for optimal implant placement.

Final step of the corrective phase is a restorations. Restorative treatment consists of procedures (direct or indirect restorations, prosthetic treatment, insertion of dental implant, etc.) which reconstruct the dental arch according to the requirements, such as loading, aesthetics, phonetics and functions.

Maintenance therapy

The described procedures provide a stable dental condition with which teeth can be preserved in their place for a long time. Maintaining the results is as important as the active therapeutic phase. To maintain healthy conditions and prevent progression, regular check-ups are needed to be done, and treatment performed if necessary. The frequency of check-ups varies depending on the patient’s motivation, nature of the disease, and presence of aetiological factors.

Besides regular professional oral hygiene treatment, re-motivation, and re-instruction, some corrective treatments may also have to be performed again, or in case of recognising development of a new disease.
2.2. Periodontal instruments

Periodontal instruments can be classified in two ways: as diagnostics and therapy-related instruments, or as manual and machined instruments.

**DIAGNOSTIC INSTRUMENTS**

*Instruments to detect periodontal tissue destruction*

**Periodontal probes**

Periodontal probes are used to evaluate apical shifting of the attachment level (attachment loss), distance between epithelial attachment and the gingival margin (probing depth) and distance between the gingival margin and the cemento-enamel junction (recession).

Periodontal probing must be performed with a standard force (equivalent to 0.2 grams, or 50N/cm²). Some probes are pressure-sensitive: to ensure this standard force the flexibly attached tip mark gets in line with a calibrated mark.

![Pressure sensitive periodontal probe](image)

Periodontal probes are calibrated in millimetres, or its multiples (2-3 mm). The tip is rounded or it ends in a 0.5-mm sphere. Some special probes are not scaled in millimetres; the lines, which are often colour-coded, only indicate the severity of periodontal tissue destruction (e.g.: WHO probe). For probing implant sites special plastic probes are used.
Some probes work attached to a computer, this way giving precisely calculated data instead of subjective figures. (E.g. Florida probe, Inter-Probe, Toronto Automated Probe System)

*Furcation probes*

Probes for measuring furcation involvement are special curved probes with similar markings, but the curvature enables their insertion in deep pockets with furcation involvement.

*Nabers probe tip*

*Mobility measuring instrument*

*Periotest*
Oro-vestibular movement of the teeth is measured bimanually; the severity is expressed in millimetres. A device called Periotest gives a much more accurate figure by using oscillating movements and calculating mobility based on tooth movement response.

Other diagnostic devices

Photo-spectroscopic instruments, like Periotron measures the amount of sulcular fluid. Some devices can measure blood circulation in the gingiva using the Doppler-effect (acoustic, photo-acoustic), others can provide a visual image of the sub-gingival root surfaces by using endoscopic methods.

THERAPEUTIC INSTRUMENTS

Manual instruments for removing bacterial deposits

Manual instruments are built up of three parts: handle, neck and blade. These instruments can be classified and distinguished from each other according to blade characteristics and its relation to the terminal neck.

Manual instruments for plaque and calculus removal are divided into two main groups: scalers and curettes.

Scalers

Nowadays the most commonly used scalers are sickle scalers and hoe scalers. Files and chisels are not used anymore, and they are not even produced by manufacturers. Sickle scalers can be distinguished from curettes on the basis of cross-section (triangle) and ending (tip). Similarly to universal curettes, the facial surface and the terminal neck close a 90° angle and they have two working edges.

Sickle scalers are used supra-gingivally for scaling and for removing soft tissue remnants during gingivectomy. Their sharp, narrow blade, especially that of the mini version, can be inserted in the narrowest interdental spaces.
Curettes are divided into universal and Gracey types. Their cross-section is a half-elliptical semicircle and they end in a rounded tip. Manufacturers keep developing their products, producing different blade sizes: normal, micro and macro blades are available for different indications. The neck of the instrument is either rigid or flexible.

Plastic and titanium curettes are produced to treat implant surfaces.

Universal curettes

Several types of universal curettes exist: some consist of one single instrument, while others combine a set of instruments. They all have two working edges and end in a rounded tip. Their cross section is a half-elliptical semicircle, the terminal neck and the facial surface close a 90° angle, and the blade runs straight from the facial aspect. Instrument sets are based on tooth types (Columbia set), or jaw and tooth types (Langer set).
Universal curettes are capable of removing large deposits, but they also remove more tooth substance than Gracey curettes. They can be used for supra- and sub-gingival instrumentation and for removing granulation tissue and debriding root surface during surgery.

**Gracey curettes**

Gracey curettes are also called surface-specific instruments because certain instruments work well on one tooth type only or on the tooth surface. Due to blade angulation, it removes less tooth structure and leaves a smooth surface behind. This is why they are also called finishing curettes. The original set consisted of 7 instruments, to which 2 additional instruments have been added, to provide better access to the mesial and distal surfaces of second and third molars. The first 5 instruments are angled two dimensionally at the neck, the other 4 instruments are angled three dimensionally.

They only have one working edge, which is faced down. The facial surface and the neck are angled at 70°. The blade is curved and the edge is convex with a rounded tip.

The figure below summarises the indications for the use of Gracey curettes

<table>
<thead>
<tr>
<th>Gracey instruments</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gracey 1-2</td>
<td>incisors</td>
</tr>
<tr>
<td>Gracey 3-4</td>
<td>front teeth</td>
</tr>
<tr>
<td>Gracey 5-6</td>
<td>front teeth</td>
</tr>
<tr>
<td>Gracey 7-8</td>
<td>buccal and oral surfaces of premolars and molars</td>
</tr>
<tr>
<td>Gracey 9-10</td>
<td>buccal and oral surfaces of premolars and a molars</td>
</tr>
<tr>
<td>Gracey 11-12</td>
<td>molars, mesial surfaces, incisors buccal surfaces</td>
</tr>
<tr>
<td>Gracey 13-14</td>
<td>distal surfaces of molars, oral surfaces of incisors</td>
</tr>
<tr>
<td>Gracey 15-16</td>
<td>mesial surfaces of 2. and 3. molars</td>
</tr>
<tr>
<td>Gracey 17-18</td>
<td>distal surfaces of 2. and 3. molars</td>
</tr>
</tbody>
</table>

The indications of Gracey curettes

The Gracey minimum set contains 4 instruments (Gracey 5-6, 7-8, 11-12, 13-14). This is sufficient to treat all tooth surfaces.

Gracey instruments are indicated for root instrumentation, removing smaller deposits of calculus and root planing in both conservative and surgical therapy.
Properties of manual instruments

Comparison of universal and Gracey curettes

**Special curettes**

The fine, tooth structure preserving properties of Gracey curettes have been combined with the effectiveness of two working edges: Double Gracey (American Eagle), Syntette (LM Dental). The latter one is capable of instrumenting all surfaces of every tooth due to the angulation of the neck.

Syntette™ (LM Dental)
Interproximal curettes for approaching concave surfaces of mesial and distal surfaces: Svardström 1-3, 2-4 (LM Dental). Instruments for furcation instrumentation in areas which are inaccessible even to universal and Gracey curettes: diamond-coated files, Svardström 5-6, 7-8, Furcation diamond file, Furcator, Diamond file mesio-distal (LM Dental).

**Maintenance of manual instruments**

Scalers and curettes need sharpening prior to every use. This can be done manually or with the help of a machine. The point of sharpening is to remove an even layer from one of the surfaces of the working edge. To perform correct sharpening, one must know the angles of the instruments well to be able to reproduce it. Colouring the edges with a marker can be helpful. Curvature of the blades must also be recognized and followed when sharpening, especially with Gracey instruments.

**Principles of sharpening**

Moisturising is very important while sharpening to reduce heat production. Paraffinic oil can be used with natural stones (Arkansas) and water with synthetic ones. Sharpening machines do not need moisturising. The movement of the stone should always be aimed at the edge to prevent burr forming.
Sharpening must always be performed after disinfecting, which is followed again by disinfecting and sterilisation. Sterilisation is recommended at 121º Celsius in an autoclave, which ensures longevity and sharpness of the instruments.

**Power driven instruments**

Power driven instruments can be classified in the following way:

- rotary instruments
- sonic instruments
- ultrasonic instruments
- “reciprocating” instruments
- abrasive powder instruments

**Rotary instruments**

Hand piece scalers are not used anymore, but special ceramic gingiva trimmers are still available on the market. They are used for sulcus spreading and removing granulation tissue without cooling.

The most common rotary instruments are polishing devices, brushes, rubber cups used with slow motion contra-angle hand pieces, perpendicular to the tooth surface. Special prophylactic hand pieces are also available, in which screwed devices can be inserted. Polishing is done with polishing pastes. Abrasive granules polish the surface, while a carrier substance is used to prevent overheating of the tooth. Granule size determines the field of application: (170-250 micron) removal of discolouration, surface irregularities, (120 micron) removal of plaque and smaller particles, (40 micron) final polishing of composite surfaces.
Polishing brushes and rubber cups

Polishing pastes

*Sonic scalers*
Sonic scalers are connected to the turbine hose. They are air driven instruments with eccentric movement. Frequency ranges between 4-7 kHz, far below that of ultrasonic devices. The head makes a circular movement. Besides removing calculus, diamond-coated endings can be used for root planing or even preparing a cavity. Heat production is compensated by water irrigation.

Sonic scaler

_Ultrasonic scalers_

The device works with electrically induced vibration. The vibrating movement can be generated by an electromagnetic field (magnetostrictive) or piezo crystal (piezoelectric). Magnetostrictive scalers vibrate around an elliptic field at a frequency of max. 25-30 kHz. It is contraindicated in patients with pacemakers. The piezo instrument works at a frequency of 35-42 kHz with linear movement and can be used to treat patients with pacemakers, too. The application of the same force will result in a smoother surface with this instrument than with the magnetostrictive scaler. Both instruments require water-cooling because of heat production. Piezo instruments can be used in endodontic treatment with the use of special tips, and even for root planing with diamond coated tips. Some models are portable with integrated water tanks. Ultrasonic instruments are getting popular in bone surgery: they facilitate smooth cutting of the bones. Some special tips even enable soft tissue manipulation.

Ultrasonic scalers - magnetostrictive
Ultrasonic scalers - piezo electric (integrated in dental unit)

A special ultrasonic device developed in 1990, called Vector, induces vibrations parallel to the tip, but cleaning is not direct but done by transmitting energy into saline solution. The instrument was developed for sub-gingival instrumentation and cleaning of implants.

Reciprocating movement hand pieces (EVA hand pieces, PER-IO-TOR®, Profin system®)

Reciprocating instruments (EVA, Profin) have diamond coated tips, which are suitable for the correction of restorative margins, treating surface irregularities and polishing with hard plastic tips. http://www.dentatususa.com/products/finishing-polishing/profin.html


Air-abrasive systems

Some of them are attached to the turbine hose, some are separate devices, sometimes combined with built-in ultrasonic scalers. Applied pressure ranges from 40 (turbine hose attachment) to 140 psi (separate device); in some instruments the pressure can be adjusted. Aluminium-oxide, baking soda, glycine base, hydroxycarbonate-apatite, sodium-phosphosilicate or calcium-carbonate powder (27-50 micron) does the cleaning.

They can be applied supra-gingivally but some new instruments can be used sub-gingivally as well. (Air-FlowPerio®).

Abrasive material needs to be sprayed at a 55 (palatal surface of incisors)-80° angle. Proper suction is important to prevent the inhalation of the powder and any damage to soft tissues.
NON-SURGICAL THERAPY

Non-surgical therapy has various names in the literature: initial therapy, hygienic phase, cause-related therapy, soft tissue management. In most cases, authors do not mention occlusal therapy, which is obviously not surgical. In fact, it continues in the corrective phase of the treatment, does not end in the initial phase. If every step is included, non-surgical therapy includes the following components:

- mechanical therapy, or professional oral hygiene treatment
- medical treatment (antiseptics, antibiotics, and drug modified host response)
- occlusal therapy

AIM OF NON-SURGICAL THERAPY
The basic goal of non-surgical therapy is to eliminate aetiological factors: mechanical plaque and calculus removal, correction of plaque retentive natural and iatrogenic factors, treatment of carious and endodontal lesions. Because complete mechanical removal of the biofilm and bacteria is not possible, the use of antibiotics and antiseptics is needed. In the background major tissue destructions, often immune deficiencies are present: leukocyte functional disorders, hyper-reactive monocyte-macrophage phenotype (overproduction of pro-inflammatory cytokines: host response should be modified).

**PROFESSIONAL ORAL HYGIENE TREATMENT**

Professional oral hygiene treatment is the key of complex periodontal therapy: this phase eliminates disease causing factors.

Professional oral hygiene treatment includes the following procedures:

1. oral hygiene motivation, instruction
2. supra-gingival scaling
3. sub-gingival instrumentation
   - subgingival scaling
   - root planing
   - subgingival (closed) curette
4. elimination of plaque retentive factors
   - correction of overcontoured fillings
   - correction of overcontoured crowns
   - provisional treatment of carious lesions
   - provisional endodontic treatment
   - correction of anatomic deficiencies
   - closure of open contact points

Hypermobile teeth with a poor prognosis are considered plaque retentive factors, because appropriate individual oral hygiene cannot be performed in the adjacent area without extracting the tooth.

Hypermobile but sustainable teeth are also plaque retentive, therefore they need to be temporarily splinted for the duration of treatment. Splinting does not only support adequate oral hygiene, but it also promotes stable occlusion, equal distribution of masticatory forces, and even helps healing by stabilising the blood clot around the root in the postoperative phase.

Malpositioned tooth can be corrected with orthodontic treatment. Orthodontic forces can only be tolerated by healthy periodontal tissues, therefore periodontal patients can only undergo orthodontic treatment after necessary surgical interventions.

Oral hygiene instruction and motivation have been mentioned earlier, in the chapter for Prevention, which can be found here.

**Mechanical removal of dental deposits**

Scaling means the elimination of dental plaque and its mineralised form (calculus). It can be done manually or with machined instruments as well. Usually highly effective ultrasonic devices are used first: the cooling spray helps to wash off removed deposits and blood, this way keeping the working area clean and visible. For larger supra-gingival calculus removal sickle shaped scalers or universal curettes are recommended. For the instrumentation of sub-gingival areas, Vector or Gracey curettes are favoured.
Root planing: Its goal is to prepare denuded, hyper-mineralised, intoxicated cementum for reparation and regeneration. Only the superficial layer of the porous cementum gets infected, therefore extensive removal of the cementum is unacceptable. Excessive instrumentation of the cementum can result in cervical sensitivity, possibly even pulp necrosis.

Root instrumentation can be carried out with machined instruments (ultrasonic or sonic device, Vector), but the final step must always be the checking of surface irregularity. Since there is a lack of visual control, palpation with a hand-instrument (Gracey curettes) is always recommended.

Instruments for scaling and root planing and their use was discussed in the previous chapter, which can be found here.

Removal of plaque retentive sites

It is well documented in literature that a significantly greater amount of bone resorption can be found around overdimensioned, deep marginal closure restorations than around the contra-lateral unrestored tooth. This problem is not the result of irritative substances in the restoration, but it is due to bacteria trapped in the pores and irregularities of the restoration.

Several possibilities are available for the correction of the above mentioned, plaque retentive, predilection spots. To deal with overdimensioned crown contours the best solution is to remove the old restoration and prepare the teeth with a chamfer. For the time of the periodontal therapy, precise marginal closure provisionals need to be prepared, since the level of marginal ginviva will change after the treatment. Permanent restorations should be prepared after finishing surgical therapy, in a stable periodontal condition, in the restorative phase. In some instances, the removal of the restoration is not an option. The correction of restoration margins with a rotary instrument can be a solution, but this is mostly associated with aesthetic compromises. In case the preparation of the crown was convex (the equator of the tooth is wider than the cervical portion), marginal correction is not applicable, the restoration has to be removed.

Overcontoured fillings can be corrected manually (finishing tape) or with machined instruments (EVA system). If the fillings are undercontoured or there is a gap at the margin, the restoration has to be changed. During the initial therapy, long-term provisional fillings are recommended (e.g.: glass-ionomer). The final restorations should be delivered in the restorative phase, after determining tooth prognosis. This cannot only save the patient from useless (expensive) interventions, but stable, inflammation-free tissues are much easier to work with.

Professional oral hygiene procedure

Supra-gingival scaling and plaque elimination can be done in one session, but sub-gingival instrumentation needs more sessions to be effective. Removal of sub-gingival deposits and root planing can be done quadrant-wise, or as „full mouth disinfection“ (Marc Quirynen et al, 1995). This means mechanical instrumentation in the entire mouth within 24 hours, combined with the use of antiseptics against persisting bacteria in the „ecological gaps“. The literature describes several interventions under the same definition, some with solely mechanical therapy (Full-mouth Therapy, Full-mouth scaling and root planing) and some combined with antiseptics (Full-mouth disinfection).

Full mouth disinfection

Non-surgical mechanical plaque and calculus removal cannot eliminate the entire biofilm and it has no effect on bacteria in the „ecological gaps“. Bacteria persisting in surface irregularities, cementum lacunae, fissures along the CEJ, on the epithelium and papillae of the tongue predispose to reinfection. Non-instrumented pockets are also possible sources of infection.

The concept of „full mouth disinfection” is based on the concept above, combining observations and theories of several authors:

- scaling and root planing in 2 sessions, within 24 hours
- rinsing with chlorine-hexidine-digluconate (CHX) 0.2% solution (including the throat) before mechanical treatment
- rinsing pockets with 1% CHX gel after instrumentation
• scrubbing the tongue for 1 minute with 1% CHX gel

• oral rinsing at home, twice a day for half a minute with 0.2% CHX solution for 2 month to prevent reinfection

The benefits of this method are still not obvious. From an aetiological aspect, aggressive and severe, advanced chronic cases can benefit from the procedure compared to the standard mechanical, quadrant-wise procedure. According to data in the literature, patients with poor oral hygiene and advanced periodontal infection benefit from the procedure. It significantly reduces the number of microorganisms in the periodontium and in the whole mouth, reduces the chance of reinfection and the risk of additional surfaces getting infected.

**Photodynamic therapy**

The goal of photodynamic therapy is to eliminate bacteria from limited access areas, where mechanical therapy would be insufficient or not even possible. The principle is that a tissue friendly material is exposed to light, as a result it releases nascent oxygen, which can cause substantial chemical damage to microorganisms. Photodynamic therapy is successfully used in *dermatology* to treat acne vulgaris, but it has also been efficient in treating macula degeneration in elderly people, and is some cases even in tumour patients.

From the *oral perspective*, it is used to treat premalignant lesions as Lichen planus, erythroplakia and leukoplakia.

As a *light source* both visible light and laser light sources are used. Devices used in the dental practice are special instruments or diode lasers, either in the visible or in the laser light spectrum.

*Photoinitiators* can be pigments (e.g.: metilene-blue, toluidine-blue), chlorine compounds, porphyrin, xanten, monoterpen, depending on the light source.

To begin the treatment, a photoinitiator fluid (gel) needs to be injected in the pocket through a cannula, which has to be lit with a light transmitting fibre inserted in the pocket. Side-effects can be thermal damage or cytotoxic effect of the photoinitiator, but to our best knowledge, temporary discoloration of the gums is the only problem expected.

Photodynamic therapy does not replace mechanical therapy, but can be an efficient supplement. In vitro studies showed substantial antibacterial effect, but in vivo experiments are not sufficient enough to draw positive conclusions clinically; an adequate number of clinical studies and meta-analysis still needs to be conducted.

**Use of lasers in non-surgical therapy**

Lasers are special light sources, which use stimulated emission to produce a light beam. Laser beam is coherent (in time and space), almost parallel, mono-coloured light, which can achieve high density of energy, even at a long distance. As the energy of lasers concentrates in a narrow space, the power density of a laser light can be multiple compared to normal light. Laser beam is an electro-magnetic wave, which consists of a one wavelength component.

Laser light is amplified by stimulated emission, this way more photons are produced in the laser beam than absorbed. Applying the energy level needed for stimulated emission on the laser is called pumping. Different energy types are suitable for pumping: light energy (flashing light, another laser), electric energy (gas discharge) or chemical energy (chemical reaction). Classification of lasers, regarding laser agent:

• solid body-laser or polluted ion crystal-laser (Neodinium-YAG, Erbium-YAG)

• semi-conductive laser (Diode laser)

• gas-laser (e.g.: CO2 laser)

• dye laser

Lasers work in a continuous or pulsating manner. Conduction of the laser light generally goes through glass fibre. In case of the highly available diode laser, the working end must be activated by a coloured material (occlusion paper, cork) when cutting or vaporising, but decontamination of tooth or implant surfaces can be performed without activation. CO2 lasers and certain Er:YAG lasers conduct light through fixed supports. The summarised properties of dental lasers can be seen in the figure below:
Properties of lasers used in dentistry

Effects of lasers on the biofilm have been studied both in vitro and in vivo. Obvious advantages of Er:YAG lasers have been discovered. This type is even capable of removing solid, mineralised substances. Disadvantages are its size and retail price. The effects of small-sized diode lasers on the biofilm are questionable. Their disadvantage is that working at an 810-nm wavelength, they produce much heat. An interesting field of application of these lasers is experimentally discovered biostimulation effect: it stimulates the proliferation of ligamental and gingival fibroblasts.

HEALING AFTER NON-SURGICAL TREATMENT

After non-surgical treatment, in optimal conditions, the periodontal pocket is filled with a blood clot. On the soft tissue side a 0.2 mm wide necrotic zone develops, where leukocytes accumulate. Their task is to clean the wound, protect it against bacterial forces and stimulate healing process. Healing starts within 8-24 hours after intervention: secretion of inflammatory mediators, beginning of cell proliferation, transformation of the blood clot. Epithelisation starts from the marginal gingiva, under the protection of the leukocyte infiltrated necrotic zone, covering the healthy portion of gingival connective tissue. At day 7, most of the connective tissue is covered by new epithelium and a connection is built between the epithelium and the root surface. Considering that complete removal of the biofilm is technically impossible and bacteria will persist in surface irregularities and dentine tubules to a certain extent, a constant inflammatory reaction is present. If there is a persistent large supply of bacteria (e.g.: failing restorations), the healing process cannot proceed and pocket depth will remain the same. The rest of the process includes maturation of the tissues.

Clinical attachment level will be disposed 1-2 mm coronally from the baseline situation, while pocket probing depth can decrease by 2-4 mm on average, owing to the long junctional epithelium and regression of the inflammatory swelling. Marginal gingiva will shift 1-2 mm apically causing recession. Connection between the gingiva and the root surface is not regenerated attachment (new attachment) but long junctional epithelium formed by epithelial cells of the gingival margin.

PHARMACOTHERAPY

Having become familiar with the aetiology of periodontitis, we will now focus on the possible ways of intervention to influence inflammatory and immune processes. This is shown in the image below:
Etiopathogenesis of periodontitis and possibilities of pharmacotherapy

The following possibilities are at our disposal to influence reactions of the human body through medications:

**Antibiotics and antiseptics**

Antiseptics have been discussed in the previous chapter. Antibiotics might be required to eliminate bacteria from limited access areas. In particular, bacteria of the aggressive form (Aggregatibacter Actinomycetemcomitans and Prevotella intermedia, nigrescens) tend to infiltrate periodontal pockets and persist in them. They cannot be eliminated by scaling and root planing, therefore the risk of reinfection can be reduced by the use of systemic antibiotics. In moderate and mild chronic periodontitis (1-2 medium depth pockets/quadrant, 4-5 pockets/mouth), local antibiotics can be effective.

Systemic antibiotic treatment cannot replace mechanical plaque-control, it is only additional treatment. The key to antibiotic treatment is to prevent selection and reproduction of resistant strains. The criteria of antibiotic application must be observed. Antibiotics may only be used to supplement oral hygiene procedures after the diagnosis of an active, progressing periodontal infection, possibly after identifying periodontal-pathogenic strains. Considering the characteristics of typical mixed infections, combined therapy is applied in most cases to achieve a synergistic effect of the medications: Metronidazol+Amoxicillin, or in case of Penicillin allergy, Metronidazol may be combined with Ciprofloxacin or Cefuroxam. For monodrug therapy, the first choice is Metronidazol, if no A.a. strains can be isolated, then Clindamycin, for non-specific infection, Doxycyclin.

For local use, tetracycline compounds are the most common, but Metronidazol based local antibiotics are also available. CHX based local medications are also on the market. The formula can be gel, microspheres or some resorbable means of delivery. The main requirements are continuous release of the active compound (7-14 days) and total resorption of the carrier.

**Influencing inflammatory response**

Immuno-modulating cytokines have been mentioned earlier. Pro-inflammatory and anabolic process modulating anti-inflammatory cytokines can both be regulated.

**Non-steroid anti-inflammatory drugs (NSAID)**

PGE2 is the product of arachidonic-acid metabolism, regulated by cyclooxygenase-2 (COX-2) enzyme. It plays a major role in periodontal inflammatory processes and the destruction of the alveolar bone. NSAID drugs
decrease prostaglandin release by blocking cyclooxygenase enzymes. Prostaglandins are responsible for conducting pain sensation, inflammatory reactions, and acts as febrifuges by blocking the heat centre of the central nervous system. Unfortunately, protective cyclooxygenase-1 (ventricle mucosa protection, blood-clotting) is also affected by the blocking effect of drugs used in periodontal treatment. Selective COX-2 inhibitors can cause severe damage to the liver, therefore their use should be avoided in periodontal therapy.

Though promising results have been obtained in regulating inflammatory responses, the use of NSAIDs is not recommended because of their side-effects. However, in post-operative care specific PGE2 inhibitor vinegar-acid substitutes (Indometacin, Diclofanac) play an important role, together with Propion-derivatives (Naproxen, Ibuprofen, Flurbiprophen).

**Tetracycline group**

It was used successfully against the localised form of aggressive periodontitis to inhibit the reproduction of Aggregatibacter actinomycetemcomitans (50-100 mg/day), due to its high excretion concentration in the sulcular fluid. It has been observed that even in a low dose (40 mg/day), it is still effective in blocking collagenase enzymes. It takes it effect through three mechanisms:

- Extracellular mechanism: MMP inhibition,
- Cell regulation: pro-inflammatory cytokine, NO-synthetase, phospholypase A2, Prostaglandin synthesis inhibition, effect on protein kinase C enzyme, calmodulin,
- Pro-anabolic regulation (stimulating collagen production and osteoblast activity: de novo bone formation).

**Bisphophonate**

Bisphosphonates were originally used to treat osteoporosis and prevent tumour metastasis through their potential to prevent bone reposition by affecting osteoclasts. They were also introduced in periodontal therapy and could slow down the progress of fast exacerbating aggressive periodontal diseases. Because of the recently discovered severe side-effects, they are not being used any more for this indication.

**Other medications – biologic therapy**

Biologic therapy refers to medications which target a specific immunological process or induce an immune response to achieve the desired effect.

Anti-cytokine and other biological compounds are still in the trial phase. Pro-inflammatory cytokines IL-1, IL-6 and TNF-alpha are the main targets of recent trials. Research activity is focusing on B cell lymphoma drug, CD20 antibody, which causes B-cell depletion, and T-cell activity inhibitor CD80 and CD86 molecule binding biologic substances.

These compounds are not yet applicable in periodontal therapy.

**OCCLUSAL THERAPY**

Changes in occlusal antagonist contact points can cause dysfunctions in the masticatory system (TMJ disorders, see chapter 2.2.) or in the dentition (traumatic occlusion). Changes in occlusion can lead to damage of the periodontal apparatus, influence on its inflammatory processes, this way causing irreversible periodontal damage, or malpositioning of the tooth. It is important to mention that occlusal trauma does not cause periodontal damage directly. These processes eventually end up in pathologic tooth mobility after a while.

There are 3 basic procedures suitable for occlusal correction:

- removal of hard tissue (occlusal correction)
- build-up of tooth surface
- orthodontic treatment

To create a stable occlusion of hypermobile teeth they have to be stabilised by splinting.
Occlusal correction of the teeth is always the task of a dentist, but recognising alterations of occlusion is the duty of the dental hygienist as well.

**Splinting of teeth**

Devices intended to stabilise periodontally compromised teeth are called splints. The biological background is that splinting hypermobile teeth with different planes of mobility, or splinting hypermobile teeth to physiologically mobile teeth in the same plane eliminates pathological movement.

Requirements for splints: they have to be durable, made with no or minimal damage to healthy tooth structure, and they may not prevent the application of proper individual oral hygiene. They also have to provide for necessary masticatory function and phonation, and their aesthetic appearance should also be acceptable. Splints can be classified regarding fixation, method of preparation and therapeutic duration.

Regarding fixation they can be **removable** or **fixed**, sometimes the combination of these two. As for preparation, they can be divided into **extra-** and **intra-coronal splints**. In the aspect of therapeutic duration they can be classified as: **temporary** (for the duration of the treatment), **semi-permanent** (till deciding the final prognosis of the tooth – months or even years), **permanent** (as part of a final restorative therapy plan).

**2.4. Periodontal surgery and the instruments used in surgical therapy**

Most instances of periodontal diseases are due to accumulation of dentogingival plaque. The most important method of prevention and treatment at the same time is individual and professional oral hygiene. To achieve this, proper access is required for the instruments to reach the tooth surfaces. Both individual and professional oral hygiene will be hampered if pockets develop as the result of inflammation, or irregularities of the bone or soft tissues occur in destructive processes. This results in further plaque and calculus accumulation on the hard to reach surfaces, making the disease more severe.

The goal of periodontal surgery is to facilitate professional oral hygiene at areas, where they are difficult to perform, and to achieve a maintainable condition by correcting soft and hard tissue deformities.

Periodontal surgeries are divided into two main groups: **periodontal pocket surgery** and **periodontal plastic surgery**. In some cases, the two cannot be separated: the same surgery eliminates the pockets, and corrects tissue deformities.

**PERIODONTAL POCKET SURGERY**

The methods and steps of periodontal surgery are the following:

- surgical access enables root debridement under visual control
- periodontal pocket is corrected: pocket eliminated or reduced to an acceptable level that can be maintained
- alveolar bone and soft tissues are modified to recreate their original morphology

The role of periodontal surgery in the complex therapy, indications and contraindications of periodontal pocket surgery

The need for periodontal surgery can be determined on re-evaluation following initial therapy. Indications of surgery depend on local and systemic factors as well. Regenerative capacity of periodontal tissues needs to be evaluated. It is also an important factor how healing processes affect cleaning of the surfaces, as well as other general factors such as systemic diseases and behavioural habits (smoking, cooperation). Surgical treatment is the most important part of the corrective phase. It may only be performed after successful initial therapy with the following requirements:

- plaque and calculus free supra- and sub-gingival surfaces,
- there are no active, bleeding pockets, and
- no natural or iatrogenic plaque retentive factors.
Surgical therapy is **indicated** if:

- access for professional oral care is inadequate,
- deep vertical pockets, bony craters, or irregular bony contour exist, or
- individual oral hygiene is inaccessible.

**Contraindications** can be divided into relative and absolute ones. If the patient cannot maintain inflammation free condition besides supportive therapy every 2–3 month, the contraindication is **absolute** because the patient is not cooperating. This is expressed in the form of the plaque index: according to some authors, this rate should be under 15–25% of tooth surfaces. Some haematological diseases are also absolute contraindications. Some systemic diseases and smoking are considered to be **relative** contraindications.

Influence of systemic diseases on planning of periodontal surgery:

- cardiovascular diseases (hypertension, angina pectoris, infarction, anticoagulant treatment: consultation with physician, and antibiotic prophylaxis might be necessary)
- haematological diseases (leukaemia and agranulocytosis are absolute, whereas coagulopathy and thrombocytopenia are relative contraindications)
- endocrine diseases (untreated diabetes is a risk factor)
- neurological diseases (severe epilepsy and psychopathy are relative contraindications)
- immunosuppression (antibiotic prophylaxis is required).

**Healing after pocket surgery**

Periodontal pocket surgeries can be classified according to the outcome of the treatment:

- **resective** procedures: removal of pocket walls (bone, soft tissue)
- **reparative** procedures: re-uniting of periodontal tissues result in tissues different from the original ones
- **regenerative** procedures: new fibre attachment develops on the root surface, besides new cement and alveolar bone: regeneration of the entire periodontium

Healing always depends on which cells of the periodontium the root surfaces are populated by (Melcher 1976)

- If epithelial cells populate the root (this is most common due to fast regenerative potential of these cells), long junctional epithelium develops with the help of hemidesmosomes: the former pocket length will have epithelial attachment to the root on its whole length.
- If connective tissue attaches to it, parallel fibre attachment will develop on the root surface.
- If the root is surrounded by bone, it becomes ankylotic.
- If periodontal ligaments attach to the surface, new attachment develops, which is considered actual regeneration. This is because ligaments contain undifferentiated stem cells, which are able to differentiate into any periodontal cell type with the modulation of mediators.
Healing after periodontal surgery

Long junctional epithelium is maintainable by excellent oral hygiene. Ankylosis and tight adaptation of the connective tissue results in external root resorption. Ideally the gingival margin shifts to the level of fibre attachment, creating a physiological pocket depth. This can be achieved in two ways: either by removing the wall of the pocket, and contouring the bone (ostectomy or osteoplasty) with concomitant apical disposition of the flap, or by using regenerative techniques to gain new periodontal tissues (cementum, fibres, and alveolar bone) to shift the marginal bone coronally.

Types of periodontal pocket surgery

Resective techniques:

- soft tissue surgery (gingivectomy, inner oblique gingivectomy, gingivoplasty)
- bone surgery (osteoplasty, osteotomy)

Gingivectomy refers to the surgical resection and elimination of the pocket or pseudopocket. Gingiva is removed till the alveolar bone, the root surface is instrumented. Another variation of this is inner oblique gingivectomy, which leaves 1–2 mm of soft tissue to cover the bone. Conventional gingivectomy is indicated in the treatment of gingival overgrowth and consequential pseudopockets, whereas inner oblique gingivectomy is
used to treat gingival hyperplasia and consequential pockets, pseudopockets, when sufficient keratinized mucosa remains after eliminating pocket walls. Today the latter technique is preferred.

**Gingivoplasty** aims to correct irregular gingival deformities to correct aesthetics with no aim of eliminating pockets. Gingivoplasty is performed around newly erupted teeth, in case of soft tissue excess.

Today resective bone surgery is used to recontour the marginal bone. Indications are shallow, wide, single wall bony defects, or if regenerative treatment is unsuccessful or not applicable due to financial reasons. Osteoplasty corrects irregular bone contours without removing supporting bone. It is most commonly used around molar teeth.

The advantage of resective surgery is that it results in major pocket reduction, thus providing a predictable result. The disadvantage is that soft tissue contour can often have unfavourable morphology causing aesthetic and phonetic inconvenience, and it results in more postoperative complications.

**Repairative techniques:**

- open flap debridement

Open flap debridement can be applied at any site where periodontal pocket surgery is indicated. The advantage of the technique is a direct visual view for complete mechanical debridement, while preserving soft tissues for primary healing (less post-operative pain). Depending on the surgical technique used, flaps can be apically positioned to the bone level (apically positioned flap), or coronally maintained (Modified Widman Flap). Nowadays elimination of soft tissue pockets is done by a Modified Widman Flap. It is applicable in any region with preservation of the tissues, and it gives a repairative result sometimes with slight signs of regeneration.
Regenerative techniques

During regeneration, the tissues lost due pathologic reasons are replaced by histologically and functionally identical tissues. Complete periodontal regeneration results in new cementum, new periodontal fibres, and new alveolar bone formation.

**Indications** for regenerative surgeries involves periodontitis related **intraosseal** and **furcation** defects. The narrower a defect is, and the more walls it has (3 or 4), the better the chances are for regeneration. The **goal** of regenerative treatment is to re-establish supporting apparatus, and minimise gingival recession.

The **principle** behind regenerative techniques is to prevent the rapid apical migration of the epithelium, to enable regeneration of periodontal ligaments originated from mesenchymal cells of the periodontium. Basically it is space maintaining for regenerative processes. The key to successful regeneration is stabilisation of the blood clot and its protection from mechanical and bacterial trauma.

**Separating of the epithelium** form the regeneration area is possible with the use of a barrier membrane or with a reinforced connection of the coagulum and the root surface (biomediators: enamel matrix protein: Emdogain®).

**Space maintaining** function can be achieved by a membrane or a bone substitute material, which fills the defect, and provides a scaffold for bone forming cells, which are responsible for new bone formation. Ideally the bone filler resorbs after a while, and it is replaced by new bone.

Currently these techniques are more often **used in combination** therapy, unlike the originally described technique using only barrier membranes (GTR: Guided Tissue Regeneration).

Bone substitutes may be natural or artificial. They can be classified as **autografts** (harvested from the patient’s bone intraorally or extraorally), **allografts** (same species, from a different individual), **xenografts** (derived from different species), and **alloplastic materials** (natural minerals or artificially produced substances, e.g. bioglass).
From a regenerative aspect, bone fillers can be **osteogenic** (contains active bone precursor cells). Bone marrow containing active osteoblasts induces ankylosis and consequential root resorption in direct contact with the root surface. Successful periodontal regeneration has been achieved with xenografts (Bio-Oss), and membranes in cases of 3-wall defects. A minimally invasive technique may be successful even without the use of a membrane.

Augmentation of the edentulous alveolar ridge is possible by means of **Guided Bone Regeneration (GBR)**, which has a different biologic principle to GTR. In this case, the membrane and bone filler only serve as a scaffold to stabilise the blood clot until bone forming cells populate the area. No ligamental mesenchymal cells are present during the process.

Modern regenerative surgical techniques aim for perfect wound closure, which can be achieved by maintaining the soft tissue volume: these are called papilla preservation techniques. They require microsurgical instruments and materials, which increase the success rate of regenerative interventions.
THERAPY

Open flap debridement (OFD)

Chemoprophylaxis – rinsing with Chlorine-Hexidine for 30 seconds

1. Anesthesia
2. Flap incision outline
3. Raising of the flap
4. Debridement of granulation tissue
5. Removal of deposits on root surface, root planing

Resective procedure
6. Correction of bony defect - osteotomy
7. Flap closure - sutures

Regenerative procedure
6. Biomodification of root surface
7. Insertion of regenerative materials
8. Flap closure - sutures

Open flap debridement

Regenerative surgical procedure

a: 9 mm deep intraosseal pocket, b: flap elevation with papilla preservation, c: defect, d: application of bone filler, e: positioning of the membrane, f: closed flap margins, maintained papilla

PERIODONTAL PLASTIC SURGERY
Ideal mucogingival morphology is not only important to maintain periodontal health but also for aesthetics. Although anatomic situation is mainly determined by the biotype (chapter 3.2.), the resistant thick biotype can also be damaged as the result of plaque accumulation.

Interventions intended to improve soft tissue aesthetics were formerly called mucogingival surgeries. Their goal is to achieve aesthetic morphology, volume, and position of the soft tissues. This definition was extended in the late 1990s, and it is called periodontal plastic surgery, which includes elimination or correction of inherited, trauma induced deformities on the gingiva or the edentulous ridge.

The goal of periodontal plastic surgery is to create the harmony of white and pink aesthetics, which is a balance in aesthetic soft and hard tissue appearance. All restorative treatments aiming to achieve white aesthetic improvement are only applicable in healthy periodontal conditions in a correct way.

Aesthetics should not have selfish goals: they should not jeopardise the structure of the periodontium, or the maintenance of its health. Plaque and inflammation free condition is a prerequisite for these kinds of interventions.

**Indications and methods for periodontal plastic surgery**

**Narrow band of attached gingiva**

According to our knowledge today, appropriate individual oral hygiene can maintain periodontal health even in the complete lack of attached gingiva. However, this has its limits: restored teeth with a thin biotype cannot be maintained in the long term. If the crown margins are anchored subgingivally, and the collagen content of the keratinized gingiva is low, 4–5 mm of attached gingiva is necessary to maintain periodontal health. Sometimes this can only be obtained by augmentation procedures.

![Image of teeth with attached gingiva](image)

Thin biotype, thick, uncleansible crown margin, inadequate keratinized gingiva, pull of frenulum: recession development

**Highly attached overdeveloped frenula**
Frenula can also hamper adequate oral hygiene, and its spreading into the papilla may cause a pulling effect. They can be removed by routine surgery or even with the help of laser technique.

![Highly attached frenula](image)

**Gingival recession**

Gingival recessions can have several aetiological factors, but the most common is alveolar bone dehiscence or fenestration (chapter 1.6.). The gingiva is more vulnerable to mechanical or bacterial irritation without underlying supporting bone, especially in a thin biotype. These irritating factors can lead to recessions of large extent.

Alveolar bone dehiscence is not uncommon in itself (around 20%), but it can also develop after orthodontic treatment. Beside aesthetic compromises, gingival recession can lead to tooth sensitivity, root caries, chemical (acids) or mechanical (abrasion, occlusal force) damage, or wearing. Although most patients visit their dentist with the fear of losing their teeth, this can not happen under proper individual oral hygiene, and healthy, inflammation-free periodontal conditions.

The first attempts for root coverage were made by split thickness pedicled (connection, and mutual blood supply with neighbouring tissues) grafts transpositioned to the recession site (Fig.5.31.).

![Recession due to trauma (toothbrushing)](image)

Later on, palatal free grafts were used (no blood supply), which gained their nutrition supply through diffusion from the neighbouring tissues, until it revascularised.
Laterally positioned flap method

Nowadays, two basic techniques are used. In one of them a flap is raised by cutting through the papilla, and then it is moved coronally. The other possibility is to preserve the papilla, and mobilise it towards the occlusal plane.

Tunnel technique: mobilisation of the soft tissues without cutting the papilla (photo by Dr. Dávid Hangyási)

In both situations, if thin biotype is present, thickness of the tissues can be boosted by inserting a subepithelial connective tissue graft under the flap taken from the palate.
Subepithelial connective tissue graft

_Gingival excess – Gummy smile_

Gingival excess can be caused by inflammation, mouth breathing, medication, or genetic disorders. However, a short clinical crown, or huge lip line (see also chapter 3.2) can also cause aesthetic disadvantages. These conditions can be managed alone or in combination with osteoplasty.

_Gingival enlargement due to Calcium channel blocker medication - before and after surgery_

_Asymmetric gums_

True asymmetry is symmetrical change compared to the bipupillar plane, relative asymmetry is compared to the lips or the incisal edges. Treatment of true asymmetry consists of apical displacement of the gingival margin (crown lengthening), or coronal advancement of the gingival margin.
Destruction of the edentulous ridge

Aesthetically pleasing pontic, or functionally and aesthetically satisfactory implant restoration can only be accomplished by the correction of the resorbed edentulous ridge. Resorption may be horizontal (Seibert class I), vertical (class II) in dimension, or the combination of the two (class III). Deformities are classified into mild (<3mm), moderate (3–6 mm) and severe (>6 mm) defects. Correction can be performed by inserting a subepithelial connective tissue graft, or by bone augmentation, if a dental implant is the planned solution.

Reconstruction of edentulous ridge with connective tissue graft

Insufficient sized clinical crown

After severe damage in the hard dental structures, anchorage of restorations cannot be adequate without violating the biological width. If crown–root ratio is appropriate (<60–40%), and the neighbouring periodontium would not suffer harm, surgical crown lengthening can be performed. If these criteria are not met, orthodontic treatment is the choice to move the tooth into a coronal position.

Crown lengthening

INSTRUMENTS OF PERIODONTAL SURGERY

Some of these instruments are adapted form oral surgery, whereas others are specially designed for these procedures. The required equipment may change according to the type of the surgery to be performed.

Instruments for gingival incisions:

- disposable scalpel/scalpel blade (15C,12, 12D,11) /
- microsurgical blade
- periodontal knives (Kirkland-, Waerhaug-, Orbán-)
- electro-, laser and radiocauter (improper use can cause bone resorption!)
Scalpels, blades and Orban periodontal knife

*Instruments for flap elevation and retraction*

- elevator (Freer, Prichard, Molt, etc.)
- tunnelling knives (straight, bended)
- surgical retractors

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Elevators
Tunelling instruments

**Instruments for scaling, root planning, and removal of granulation tissue** (see chapter 5.2.2.)

**Bone correction instruments**

- chisel (Ochsenbein)
- bone scraper (Kirkland, Rhodes)
- bone plier - bone files - power driven rotary instrument (steel, diamond burs) - piezo-surgical instrument

**Instruments for bone correction and grafting**

**Instruments for regenerative techniques**

- membrane positioner
- bone graft material spoon
- bone condenser

**Scissors** - gingival scissors (straight, bent, multi-bent)
Tweezers

- dental tweezers
- surgical tweezers
- periodontal tweezers (pocket marking-, gingival-)

Instruments for flap closure

- suture (monofil; resorbable/non-resorbable; 4-0, 5-0, 6-0, 7-0)
- surgical needle (G, B; triangular cross-section)
- surgical needle holder (Mathieu, Hegar-Mayo, Crile-Wood, Castroviejo)
2.5. Therapy of acute conditions

Acute diseases of the periodontium have been discussed in a previous chapter. In acute diseases our first task is symptom management, and only after that can periodontal therapy be performed.

GINGIVOSTOMATITIS HERPETICA

The disease is triggered by Herpes Simplex Virus 1 and only symptomatic therapy can be applied against it. The primary disease is most frequent in childhood, and it can have severe systemic symptoms (high fever) and painful lesions in the oral cavity, which hinder eating and drinking leading to dehydration and upset the salt and water balance. To prevent this, reduction of pain and fever, and adequate water intake are the most important measures to take.

Fever can be depressed with NSAIDs, but before the administration of drugs pain needs to be managed. The same is true for food and water consumption. Local pain management can be performed with cotton pellet soaked in anaesthetic solution (SuspensioanaestheticaFoNo, or anaesthetic gel) Painful lesions may be treated with these solutions.

After the settling of acute symptoms (after day 7), Oxycort spray (corticosteroid and oxy-tetracycline) may be beneficial to prevent bacterial superinfection and stimulate healing processes. Antiviral agents should only be used in case of severe general symptoms or systemic diseases. The disease heals spontaneously in 10-14 days. If indicated, initial periodontal therapy can be performed right after this.

NECROTIZING GINGIVITIS

Acute symptoms are really painful, therefore the reestablishment of adequate oral hygiene can only be managed carefully, step by step. The first session should be limited to careful supra-gingival debridement, supplemented with 3% hydrogen-peroxide irrigation, which helps remove necrotic tissues. As hydrogen-peroxide releases oxygen during its decomposition, it is effective in eliminating dominant Fusobacterium species. Since individual oral hygiene is difficult to perform when in pain, hydrogen-peroxide or CHX mouthwashes are recommended for home use. The medication prescribed should be Metronidazol (3x250 mg for 7 days). The next session should be scheduled in two days to start actual cause-related therapy. After this, the patient is checked on day 7.
and every week thereafter, until oral hygiene is re-established. If the alveolar bone is involved, surgical correction might be necessary.

**PERIODONTAL ABSCESSES**

*Periodontal abscess*

The basic principle of treating periodontal abscesses is to enable pus drainage. This can be achieved in several ways: closed curettage, vertical incision, circular dissection of periodontal fibres, inserting a gauze strip, open flap surgery or extraction of the affected tooth.

Nowadays sub-gingival curette is preferred as standard care, as it ensures fast healing, rapid pain relief, and prevents incision related major recessions. Regenerative therapy can correct the defect caused by the infection at a later stage. It is important to irrigate the pocket after the drainage of pus with CHX, but after sub-gingival instrumentation only saline solution may be used. After the intervention, careful adaption and compression of the pocket is important to create a thin blood clot, which can facilitate the healing processes. Splinting of mobile teeth and correction of major occlusal deficiencies is required before the treatment.

Recession due to vertical incision

*Gingival abscess*

Surgical opening and drainage is recommended

*Pericoronal abscess*

It is recommended to prevent its development by extracting wisdom teeth. In case serous inflammation develops, elimination of aetiological factors through instrumentation and CHX irrigation of the pseudo-pocket is recommended. If purulent inflammation is present, drainage of the inflammatory process is recommended, which might make an incision necessary. If systemic symptoms develop, antibiotic (systemic) treatment is inevitable (Penicillin derivatives).

**COCCAL GINGIVITIS**

It usually develops on the ground of withering systemic diseases. Treatment of underlying diseases is of primary importance in this case. Besides this, application of antiseptics and systemic antibiotic treatment can be effective.
2.6. Dentinal Hypersensitivity

**DEFINITION**

Dentinal hypersensitivity is characterized by short, sharp pain sensations arising from exposed dentine in response to stimuli which are typically thermal, evaporative, tactile, osmotic or chemical, and which cannot be ascribed to any other form of dental defect or pathology.

**AETIOLOGY**

The most common cited reason for exposed dentinal tubules is gingival recession (Fig. 1). Chronic exposure to bacterial plaque, toothbrush abrasion, gingival laceration from oral habits such as toothpick use, excessive flossing, crown preparation, inadequately attached gingiva and gingival loss secondary to disease or surgery are some of the causes of gingival recession. Gingival recession is the reduction of the height of the marginal gingiva to a location apical to the cemento-enamel junction. Recessed areas may become sensitive due to the loss of cementum, ultimately exposing dentine. Probing depths, recessed areas and sensitivity reported by the patient must be accurately recorded and monitored to provide a reference for the patient’s disease activity over time.

Patients undergoing periodontal treatment are particularly susceptible to this condition because of the recession following periodontal surgery or loss of cementum following non-surgical periodontal therapy. In addition, periodontal disease and improper brushing habits can also result in gingival recession accompanied by sensitive teeth.

Recession and abrasion (from Prof I. Gera)

**CHARACTERISTICS OF DENTINAL HYPERSENSITIVITY**
It is manifested in a manner that is physically and psychologically uncomfortable for the patient and it may be defined as acute pain of short duration caused by the presence of open dentinal tubules on an exposed dentinal surface. The stimulus that triggers the onset of pain can be of thermal, chemical or mechanical origin. The most common complaint is caused by cold stimuli. Pain may also occur as a result of chemical stimuli, such as acidic foods (mainly fruit), sweets and rarely salty foods. Mechanical stimulus frequently occurs when the patient rubs the sensitive area with a finger nail or toothbrush bristles during brushing, setting off pain. The atmospheric cold air during mouth breathing, particularly in winter also causes pain.

**PREVALENCE**

The prevalence of dentinal hypersensitivity has been reported in a variety of ways over the years: as greater than 40 million people in the U.S. annually, 14.3% of all dental patients, between 8% and 57% of adult dentate population, and up to 30% of adults at some time during their lifetime. Dentinal hypersensitivity has been shown to peak in 20 to 30 year olds and then rise again when in their 50’s. The condition generally involves the facial surfaces of teeth near the cervical aspect and is very common in premolars and canines.

**THEORIES**

Several theories have been cited to explain the mechanism involved in dentinal hypersensitivity. From the dentinal receptor mechanism theory to the hydrodynamic theory all have all been presented and discussed throughout the years.

*The dentinal receptor mechanism theory*

It was an early hypothesis which suggests that dentinal hypersensitivity is caused by the direct stimulation of sensory nerve endings in dentine. On the basis of microscopic and experimental data, it seems unlikely that neural cells exist in the sensory portion of the outer dentine so this theory is not widely accepted.

*The odontoblast transducer mechanism*

It was suggested that odontoblasts act as receptor cells, mediating changes in the membrane potential of the odontoblasts via synaptic junctions with nerves. This could result in the sensation of pain from the nerve ending located in the pulpodentinal border; however, evidence for the odontoblast transducer mechanism theory is generally lacking and inconclusive.

*The “hydrodynamic theory”*

It was developed in the 1960’s and based upon two decades of research, is widely accepted as the cause of tooth sensitivity. Assumptions of the hydrodynamic theory conclude that when fluids within the dentinal tubules are subjected to temperature changes or physical osmotic changes, the movement stimulates a nerve receptor sensitive to pressure, which leads to the transmission of the stimuli (Fig 2. and Fig 3.). The various stimuli that are reported to cause this transmission of sensation are cold, hot, osmotic, electrical, dehydration, and chemical. Scientists described this phenomenon as the following: The coefficient of thermal expansion of the tubule fluid is about ten times that of the tubule wall. Therefore, heat applied to dentine will result in the expansion of the fluid and cold will result in the contraction of the fluid, both creating an excitation of the mechano-receptor. According to the hydrodynamic theory, dentinal hypersensitivity is a transient tooth pain. The symptoms include a short, sharp pain arising from exposed dentine in response to a stimulus that cannot be ascribed to any other form of dental defect or pathology. Therefore, in order to exhibit a response to the stimuli, the tubules would have to be open at the dentine surface as well as the pulpal surface of the tooth. The most important variable affecting the fluid flow in dentine is the radius of the tubuli. If the radius is reduced by one-half, the fluid flow within the tubuli falls to one-sixteenth of its original rate. Thus, the creation of a smear layer or obliteration of the tubule can greatly increase the effectiveness of the treatment of dentinal hypersensitivity.

**DIAGNOSIS**

A detailed history associated with a careful clinical and radiographic examination allows dentinal hypersensitivity to be differentiated from other pathologies that affect the teeth. It is extremely important since the history may be clinically confounded with incipient caries, restorations in a poor state of conservation or performed recently, cracks or dental fractures and teeth with reversible or irreversible inflammatory processes of the pulp.
The reasons for tubules to be exposed or open should be assessed during a visual examination of the teeth as well as a detailed dietary history (frequency of food and acidic beverage intake). Useful diagnostic tools are the air/water syringe (thermal), dental explorer (touch), percussion testing, bite stress tests and other thermal tests such as an ice cube and assessment of occlusion. The degree of pain severity can be quantified by means of a descriptive scale: slight, moderate or intense pain.

**TREATMENTS**

Treating dentinal hypersensitivity can be challenging for the dental professional because of the difficulty related to measuring the pain response since the response varies from patient to patient. In addition, if the dentine exposure is due to personal habits, it may be difficult for patients to change their behaviour. If the diagnosis confirms dentinal hypersensitivity in the absence of underlying diseases or structural problems, the following steps can be taken into account:

- remove the risk factors by educating the patient about dietary acids and other oral care habits;
- recommend different tooth brushing methods, if appropriate
- initiate treatment by recommending a desensitising agent for home use
- applying topical desensitising agents professionally

Treatment can be invasive in nature or non-invasive.

**Invasive procedures**

They may include gingival surgery, application of resins or pulpectomy. In addition, four kinds of lasers have been used for the treatment of dentinal hypersensitivity and the effectiveness ranged from 5.2 to 100%.

**Non-invasive treatment options**

They are topical agents and dentifrices that contain a desensitising active ingredient. These are considered to be the simplest, most cost-effective and efficacious first line of treatment for most patients.

**Dentifrices**

Dentifrices are the most common vehicles for desensitising agents and they must be applied with a toothbrush. They contain a combination of a desensitising agent, fluoride source, anticalculus and/or whitening ingredients to provide multiple therapeutic and cosmetic benefits. The mechanism of action is based on the obliteration of dentinal tubules (Fig. 4. and Fig. 5). Many dentifrices contain abrasives which may also cause obliteration.

According to the literature, the most widely available desensitising toothpaste ingredient is potassium nitrate. Potassium ions are thought to block the synapse between nerve cells, reducing nerve excitation and the associated pain.

Another active ingredient that exhibits a similar mechanism is potassium chloride. Other treatments are designed to reduce flow into the dentine tubules by occluding or sclerosing the tubules. Active ingredients include stannous fluoride, strontium chloride hexahydrate, and aluminium, potassium or ferric oxalates and fluorides.

**Adhesive materials**

Adhesive restorative materials and dentinal adhesives are considered dentinal tubule sealers. When there is no loss of dental structure, dentinal adhesives in the form of bonding agents and varnishes can be indicated. They produce an immediate effect, but they are easily removed.

**A few fluoride desensitizing products**

There are several products available over-the-counter for consumers that also claim to reduce tooth sensitivity. Furthermore, several scientific studies mention fluoride varnishes and sealants as a possible treatment for dentinal hypersensitivity.

**CONCLUSIONS**
Dentifrices are commonly used as a delivery system for therapeutic agents such as antimicrobials and anti-sensitivity agents. Therapeutic oral care products are available to help the patient in the control of dental caries, calculus formation and dentinal hypersensitivity. The dental practitioner makes recommendations regarding selection of the appropriate therapeutic dentifrice based on the diagnosis of the disease or condition. These recommendations are based on extensive knowledge of the aetiology of the disease/condition, the mechanism of action of the various active agents in the dentifrice and mouth rinse and the host’s needs and response to treatment.

2.7. Coronal Polishing of the teeth

**DEFINITION**

Tooth polishing is the smoothing of all exposed tooth surfaces with a rubber cup, a brush, or by an air polisher driven by a slow-speed hand piece or water unit. Traditionally, it follows scaling and root planning.

**OBJECTIVE AND RATIONALE OF POLISHING**

The primary objective of polishing is to remove extrinsic stain and supragingival plaque. The rationale for this procedure includes improving the appearance of dentition, demonstration of standard of oral cleanliness for the patient to attain on a daily basis, and motivating the patient to improve plaque control, as well as the belief that the outcome of high-quality periodontal service should be a plaque free mouth. However, the therapeutic advantages of polishing are uncertain, because plaque is reformed within 24 hours.

**INSTRUMENTATIONS**

**Different forms of polishing brushes and rubber cups**

Rubber cups and polishing brushes are used in the hand-piece (slow speed, without water spray). The use of the brush should be confined to the crown to avoid injury to the gingiva and cementum.

**Polishing pastes**

An ideal prophylactic paste should combine good cleaning ability with simultaneous polishing (morphological smoothing of dentin and enamel surfaces). In addition, the agent should cause minimal abrasion and surface roughness of dental hard tissues.

Many types of polishing pastes containing varying degrees of abrasiveness are commercially available. The abrasives are of various particle sizes and are categorised as fine (1–45 μm), medium (74–105 μm) or coarse-grade (74–177 μm) prophylaxis pastes. They are normally colour-coded for convenience. These abrasives can increase the roughness of the dental hard tissues as well as the restoration surfaces but are necessary for effective plaque and stain removal. Some pastes also contain sodium fluoride or stannous fluoride as a desensitising agent.

**Tooth Polishing Strips**

They are used for polishing the proximal surfaces of teeth that are inaccessible to other polishing instruments.

- thin flexible backing with aluminium oxide coating
- centre-gapped strip with two abrasive grades per strip which allows interproximal access
- different types (different colours mark different size of granules)

**TECHNIQUE**

The use of the appropriate methods neutralises some of the disadvantages of polishing. Operating the slow speed handpiece at lower than 20,000 revolutions per minute decreases abrasion. Lower speed also prevents excessive heat caused by friction that can damage the pulp. To decrease heat production and the rate of abrasion, apply only mild, intermittent pressure during polishing, just enough to flare the rubber cup to adapt to the tooth surface. Be cautious to keep the rotating cup away from the gingival margin, where it can abrade and burn the tissue.
SIDE EFFECTS

A number of side effects associated with polishing have been demonstrated. Because polishing involves the use of an abrasive, a microscopic amount of tooth structure is lost during the process. For this reason, polishing may not be appropriate for the patient at risk of caries because the outer tooth surface is rich in fluoride. Such a loss of tooth structure may accumulate over a lifetime from repeated polishing. Abrasion of tooth surfaces occurs at a much faster rate when it involves carious or demineralised enamel or root structure, which is about as calcified as enamel. Therefore, routine polishing is contraindicated on decalcified enamel and exposed root surfaces.

After polishing, tooth surfaces actually might be rougher because the abrasive used in prophylaxis pastes are harder than those in dentifrices used daily by the patient. Indeed, scratches made during polishing are visible with magnification. Absence of plaque and stain then should exclude the possibility of polishing. Likewise, fixed dentures covered by plastic may also be easily damaged by abrasion; therefore they should not be polished.

The abrasive particles in the prophylaxis pastes can enter the gingival tissue without intact epithelial barrier, which is a frequent result of periodontal instrumentation. It may result in delayed healing or a foreign body reaction. For this reason, polishing is often delayed after non-surgical periodontal procedures until gingival healing occurred. Finally, aerosol and splatter formed during polishing procedures pose a challenge to infection control. The clinician should consider it when planning treatment for a patient with compromised health or for a patient at high risk for infectious diseases.

Selective polishing is a treatment approach that prevents such complications and side effects. Using this philosophy, polishing is reserved for only those teeth with obvious stain remaining after scaling and is limited to intact enamel surfaces. In other words, polishing is not automatically implemented but is provided for those patients with specific needs. If necessary, stain can be removed with manual instrumentation, and plaque can be removed with tooth brushing.

AIR POLISHING DEVICES

An automated polishing method, sometimes called air-powered polishing or jet polishing has been available for the past three decades. This method uses finely powdered sodium bicarbonate as an abrasive, which is delivered under pressure through a narrow nozzle of a specialised handpiece surrounded by a mist of warm water. The resulting aerosol is propelled against the tooth surface to remove plaque and extrinsic stain by mechanical abrasion. The advantage is a remarkably thorough process of deposit removal accomplished in a short time, although the long-term effect of it on oral health status has not been researched yet. The abrasive is quite fine and despite the pressure of application, minimal loss of enamel is resulted when used according to manufacturer directions. However, significant loss of root structure may occur even with careful use. For patients with gingival recession who elect stain removal for aesthetic reasons, air-powder polishing removes less tooth structure than manual instruments.

CONTRAINICATION

Air-powder polishing is contraindicated for patients with respiratory diseases such as chronic obstructive pulmonary disease, asthma and emphysema, for patients who wear contact lenses or are on a low sodium diet, on composite restorations, around the margin of cast restorations, and on demineralised enamel.

How to use it?

Using the proper technique with this polishing method minimises the side effects and prevents patient injury. The handpiece should be held with a modified pen grasp but no fulcrum is needed because no pressure is
required during a stroke. The tip is positioned 4 to 5 mm away from the tooth surface and is kept in constant motion using circular, brush-like strokes directed at the middle third of the anatomical crown. Avoid long, continuous use, and allow the patient to rinse the mouth at appropriate times. The spray is directed at an 80 degree angle to facial and lingual surfaces of the posterior teeth and at a 60 degree angle for the anterior teeth. A 90 degree angle is used only on occlusal surfaces.

- rub vaseline onto the patient’s lips
- use cotton rolls for soft tissue retraction
- use suction tube/ exhaustor
- use the powder spray only indirectly in the area of: composite fillings root cement demineralised enamel

After the treatment, inform the patient to avoid smoking and consuming tooth colouring foods in the first 2–3 hours.

*Infection control during polishing*

- You should not forget to ask about infectious diseases when taking patient history
- Treat infectious patients at the end of surgery time
- Use personal protective barriers: -mask, gloves -protective glasses or shield -laboratory coat
- Use an antiseptic mouthwash before scaling -it signifi cantly reduces the amount of viable bacteria in aerosol

**CONCLUSIONS**

Polishing has limited benefits for periodontal health and it has potential adverse consequences. The clinician and hygienist should explain the effect of this practice to the patient and discuss its use instead of reflexively proceeding with it. Promoting oral self-care may be a better investment for clinician time and effort.

**2.8. Periodontal maintenance therapy**

Periodontal maintenance therapy is continuous professional oral care provided by the dental team (general practitioner, periodontologist, and oral hygienist), following active therapy (surgical and non-surgical). Periodontal maintenance therapy needs to be performed regularly (typically every 3–6 months) on natural teeth as well as on implants.

**SIGNIFICANCE OF MAINTENANCE THERAPY**

To our best knowledge, plaque related inflammatory diseases end up in periodontal tissue destruction. The main goal of our medical/dental professional work, the prevention of diseases is related to plaque control in every way. Effective plaque removal can prevent the development of gingivitis (primary prevention), the progression of gum diseases into inflammatory processes with periodontal attachment loss (secondary prevention), and the spreading of inflammations and developing of accompanying diseases (tertiary prevention).

Research related to the aetiology of periodontal infections has proven that patient groups included in maintenance therapy programs are at a significantly lower risk for the progression and recurrence of periodontal as well as carious lesions than patients not receiving maintenance treatment. In a study by Axelsson and Lindhe, patient groups under regular therapy done by dental hygienist have been compared with patients with no regular recall visits. The mean attachment loss after 6 years was 0.2 mm in treated groups, while this value was 1.8 mm in patients without maintenance. The biggest difference was observed in interproximal plaque scores between the two groups: patients not in the maintenance programme had a mean of 50% surfaces covered by plaque. As a result, test groups could maintain 0–3 mm pocket probing depth in the interproximal spaces, while one third of the control patients with no therapy developed pockets of 4–6 mm within the study period.

Early studies results have been supported by meta-analyses. Non-surgical therapy is efficient and economical both in initial and maintenance therapy. The efficacy of maintenance therapy depends on the motivation of the patient, know-how of the dental team (dental hygienist, periodontist), and the cooperation of the patient.
The success of surgical therapy is highly dependent on maintenance. Only regular maintenance therapy can achieve long-term success of periodontal surgeries. This applies especially to the aggressive form, where minimal amount of plaque can lead to serious damage. Missing maintenance therapy can result in up to 1 mm attachment loss a year.

**AIM OF MAINTENANCE THERAPY**

The primary goal of maintenance therapy is to preserve oral health. Further goals are to preserve masticatory function, prevent the development of infections, carious lesions, and re-infection of residual pockets. Periodontal maintenance is effective if:

- inflammatory reactions of the periodontium are controlled
- attachment levels are stabilised
- alveolar bone is preserved

We cannot aim at keeping all tooth surfaces plaque-free at all times. Development of the biofilm is much faster than cleaning of the teeth is possible and recommended. According to a study by Lang and co-workers, perfect cleaning of the teeth once every two days prevents the development of periodontal inflammations. If the surface covered by plaque does not exceed 25% proximally, and 5% marginally, no progression of the inflammatory process will occur. If the above mentioned individual’s oral hygiene is accompanied by professional oral health care, composition of the plaque will not reach the level needed to damage the periodontium for a long time. Regular professional oral hygiene can compensate for poor individual oral hygiene for some extent, and shift the composition if the biofilm in a way that it reduces the pathogenic potential.

The above mentioned argumentation proves that with maintenance therapy:

- professional measures can reduce "bacterial load", and influence the composition of the plaque that reduces its "pathogenic effect"
- the patient’s motivation and instruction is increased, and this way, the individual oral hygiene can reach the desired level of efficiency
- new pathological processes can be diagnosed at an early stage, thus preventing their progression
- adverse effects related to therapy can be prevented

**METHODS OF INDIVIDUAL ORAL HYGIENE**

**Diagnostic measures**

**History update**

During patient history taking, special attention needs to be given to new systemic diseases, changes in existing ones, and changes in taking medications. Unfavourable habits, like smoking, need to be carefully monitored.

**Clinical examination**

At every recall, stomato-oncological screening and evaluation of all oral lesions need to be performed. In case of removable dentures, the effect of the denture base on the soft tissues needs to be evaluated with special attention to cleaning. Plaque and bleeding scores need to be measured at every recall visit. Every 6 months (in low-risk patients), pocket probing depth, attachment level, mobility furcation involvement, activity of residual pockets, occlusal changes, carious lesions, and restorations need to be analysed. Every 3–4 years, a full radiographic status has to be assessed, and sensibility testing of all natural teeth performed.

**Risk analysis**

The frequency of recall visits cannot be determined in general. Several factors, like nature of the disease, patient motivation, general health, bad habits can all influence the progression of the disease; therefore, recall appointments need to be individualised. In determining the time frame between recall visits and tertiary preventive measures, risk analysis can be of help. This means determining the factors which need to be considered in controlling of the disease, on a patient, dentition, and disease location level.
A full risk assessment of the patient can be done through a special chart, the "Bern spider", based on the idea of Lang and Tonetti. It assesses six factors:

- gingival condition (bleeding),
- number and ratio of residual pockets,
- ratio of missing dentition,
- attachment level in relation to age,
- medical condition (primarily diabetes mellitus), and
- smoking.

Based on the covered surface, patients can be categorised as low, medium, and high risk patients.

Risk assessment by "Bernese spider" (Lang et al)

Attachment level loss, furcation involvement, mobility, malformation, malposition, and iatrogenic factors (failing restoration margins or occlusal discrepancy) need to be evaluated on a dentition level.

At the defect site, pocket activity, gingival bleeding, probing depth, attachment level, and morphological deformities need to be evaluated.

An individual hygiene programme can be created based on the information above. Recalls are scheduled for every 3–4 months in aggressive forms, and every 6 months in slow progression diseases, one year in highly motivated, periodontally stable patients. In high-risk patients, who cannot reach the required plaque scores, these visits can be increased to 2-month intervals. Recalls should not be performed more frequently because the periodontal tissues would not have a chance to regenerate; therefore, periodontal parameters would worsen.

**Therapeutic methods in maintenance therapy**
Patients are reassured in their motivation and instruction after every visit based on their actual status. Professional oral hygiene is being performed, active pockets treated, if any is present. If disease progression is noticed, active treatment is performed again. Failing restorations are corrected and carious lesions are being treated. Fluorine treatment is also important to prevent root caries and hypersensitivity of the root.

About one hour of time is needed for the prophylactic treatment of the dental hygienist. It is divided into four parts:

- examination, re-evaluation
- motivation, instruction, professional oral hygiene
- treatment of active pockets
- polishing, fluorine treatment,
- supervision by a periodontist, and
- scheduling of a new appointment

"Prophylactic hour" – by HF Wolf and KH Rateitschak

THE ADVERSE EFFECTS OF MAINTENANCE THERAPY

Mechanical periodontal treatment – besides its beneficial effects – comes with drawbacks, too: hard tissue loss on of the roots can become critical, which can make individual oral hygiene difficult, and can cause further complaints. Root caries is more frequent, and tooth sensitivity is also quite common in these situations.

To prevent the above mentioned adverse effects, the following can be applied:

- fluorine treatment: professional (Elmex gel) as well as home treatment, in the form of fluorine containing toothpastes, mineral water, and salt
• use of antiseptics: chlorine-hexidine-digluconate, which are also effective in caries prevention by eliminating streptococcus mutans bacteria

• application of liners on the root surface

• casein-phosphopeptide calcium-phosphate containing toothpastes in patients with extremely high caries frequency

• xilit based chewing gum or candy can also be used to prevent caries

3. Surgical aspects of dental implantation (Árpád Joób-Fancsaly DMD - József Barabás MD)

Dental implantation is one of the most recent and up-to-date possibilities for the replacement of dental deficiencies, this procedure achieving excellent long-term functionality and effectivity. The aims are the complex prosthetic rehabilitation of the patient with the aid of implants, and the attainment of aesthetically satisfactory dental substitution without influencing the other teeth. This implantation is often referred to incorrectly as tooth implantation, although it in no way involves the implantation of a real tooth.

What are actually implanted are artificial roots, made from biomaterials, which are embedded in the jaws in order to support the dental replacement. The implants are most often screw-shaped and are fixed in the bone by means of the thread on their outer surface, in conjunction with their special surface morphology. There is also a thread in the interior of the implant, into which a healing screw is inserted for the duration of healing, after which it is exchanged for a prosthetic head suitable for the anchoring of the dental replacement. The replacements are very similar in appearance to the natural teeth, the difference being virtually imperceptible. The structures built onto the implants are of practically the same types as in the case of traditional dental replacements (e.g. covering crowns, bridges and removable full plates).

The implantation procedure demands considerable attention, careful planning, precise work and a high level of professional understanding. As a number of techniques may be available for the solution of a given dental situation, it is important from the aspect of the achievement of the best result that there should always be consultations with the treating doctor regarding the treatment plan, with careful consideration of the individual characteristics of the oral cavity in the given patient. Besides the implantation solution, the patient should always be offered an alternative solution involving a traditional technique.

MATERIALS AND DESIGN CONSIDERATIONS

The materials currently utilized for purposes of implantation are the practical results of biological and materials research. These materials are referred to overall as biomaterials: they do not harm the organism and they do not cause allergies, inflammation or tumours. They ensure the connection between the implant and the adjacent tissues (primarily bone tissue), they maintain their shape, they give an X-ray shadow, they display favourable strength, they do not undergo corrosion and they are resistant to tissue reactions. The material of the implant must meet the fundamental and most important requirement that it is tissue-friendly (biocompatible). At present, these properties are best demonstrated by pure, unalloyed titanium. Titanium alloys are preferably used for the preparation of prosthetic elements. Titanium is fully accepted by the human organism and there is therefore no danger of rejection. Naturally, this does not mean that a titanium implant will necessarily remain in place throughout life, but, in consequence of the natural or artificially produced stable oxide layer on its surface and additionally the specially created surface morphology, it will be accepted by the organism for a period of even several decades. Titanium has long been used in other areas of surgery, for instance as the material from which hip prostheses are made. Thanks to the oxide layer that forms spontaneously on the surface of titanium, the bone cells grow onto this surface, and the implant is ossified and becomes part of the organism (Fig. 1).
As concerns tissue-friendly ceramics, the use of implants made of zirconium oxide has recently come into the foreground, mainly because of its aesthetic advantages. Zirconium oxide had been employed for years for purposes of metal-free tooth replacement, and the use of implants produced from zirconium oxide was officially accepted in 2004. In addition to its aesthetic and mechanical features, this material is characterised by the fact that fur is not likely to be deposited on the neck of the implant in the oral cavity. As a result, there is a reduced risk of the development of gingivitis around the implant. Zirconium oxide has the drawback that the possibilities for the preparation of prostheses to serve as implants are limited.

The differences between the individual implantation systems often lie primarily not in the nature of the material used, but rather in the procedures applied for the surface treatment of these materials. Countless surface-forming
processes are known, including turning, polishing, etching with acid, sand-blasting, laser surface treatment, roughening with titanium oxide, and so on. In the interest of attaining the best result, these procedures are nowadays generally combined. Since the long-term success of implantation depends considerably on this surface treatment, extensive research is currently being carried out on this topic.

The basic materials applied for the production of implants must satisfy the strictest specifications and health regulations. Moreover, the implantation systems in use at present are governed by international quality assurance specifications.

AIM, INDICATIONS, CONTRAINDICATIONS

As is the case for all medical activities, the indications for implantological interventions are influenced largely by two factors: the risk factors accompanying the intervention and the expected result from the aspect of the patient. The demands of the patients as regards the result of the implantological activity are generally high, while the risk factors too are relatively significant. The patients expect a dental product that is aesthetically impeccable, functions perfectly and ensures appropriate phonation, but at the same time both the risk involved in the surgical intervention and the material risk are high. A thorough knowledge of the complications that may possibly arise during such surgical interventions is indispensable for the practising doctors dealing with implantology. Only in possession of this knowledge can they make a correct and responsible decision as to whether to place an implant into the jaw of a given patient, or whether to refrain from this solution. In reaching this decision, they must take into consideration the individual physical characteristics and general state of health of the patient, and fully assess both the indications and the contraindications.

It is generally accepted in the literature that, in the event of an appropriate general state of health, virtually anyone may receive a dental implant once the growth of the jawbone has been completed (which is the case on average at the age of 18). There is no upper age limit for the intervention, but it is clear that, with the advance of age, the patients may have an ever increasing number of health problems which prevent implantation. Implantation is justified in cases involving a dental deficiency where dental replacement through the traditional procedures would lead to aesthetic and/or functional disadvantages.

Dental implantation is recommended in the following cases:

- a deficiency of one tooth (in the event of sufficient space)
- a unilateral or bilateral deficiency of an end-tooth
- a too long intermediate tooth deficiency
- an edentulous jaw
- a confirmed allergy to plastic
- for the fixation of a defect prosthesis after the removal of a tumour from the oral cavity
- in certain diseases where a removable dental replacement would endanger or hamper the masticatory and respiratory processes of the patient (diseases such as asthma and epilepsy)
- certain professions (e.g. actors or TV presenters)

However, there are also situations where the state of health of the patient may influence the expected success of the implantation in a negative manner, or where the intervention may be disadvantageous as concerns the patient’s organism. The likelihood of the “organisation” (ossification) of the implant may decrease. In such cases it is necessary to consider carefully whether this solution should be attempted at all to replace the deficiency. These factors must be assessed exclusively on a personal basis. Two groups may be distinguished: general and local contraindications.

General contraindications:

- tumorous diseases undergoing treatment
- severe cardiovascular diseases, and haematopoietic and blood clotting disturbances
- severe immunological disease entities (e.g. systemic lupus erythematosus)
• severe diabetes mellitus (however, if the blood sugar level is increasingly checked during the period of treatment, even diabetics may receive implants)
• severe osteoporosis (secondary form) and the medication taken to treat this
• major malocclusion
• certain drugs
• strong smoking (20 or more cigarettes a day)
• alcohol, narcotic or medication dependence

Local contraindications:
• pathological lesions on the mucosa or in the bone (leukoplakia, residual roots or cysts)
• anatomical obstacles which can not be corrected by surgical means (e.g. the close vicinity of a nerve to the operating area, or a limited ability to open the mouth)
• neglected oral hygiene, where it is impossible to motivate or instruct the patient Certain conditions do not definitively exclude the possibility of dental implantation:
• pregnancy (the use of X-rays to prepare a radiogram may be harmful for the foetus, or hormonal changes may occur)
• a significant bone deficiency at the site of the intervention (this problem may be overcome in some cases through the use of bone supplementation)

PATIENT ASSESSMENT, TREATMENT PLANNING

One very important question that arises concerning the site of the deficiency is the time at which the tooth was removed. The implantation may be performed at different points of time relative to this:

1. **Immediate implantation:** when the removal of the tooth is followed at once by the insertion of the implant. The main advantage of this method is the shortening of the treatment. In such cases the bony healing of the extraction wound proceeds simultaneously with the development of the connection between the implant and the bone (bone integration). This procedure has the drawback that the shape and size of the root of the extracted tooth are not the same as those of the implant. Moreover, it can not be applied in the event of acute or chronic inflammation, and the adaptation of the gingiva is difficult. One condition for success is the existence of an appropriately thick, healthy buccal bone plate.

2. **Delayed implantation:** in such cases the implantation is carried out 4-6 weeks after the tooth extraction. The duration of treatment will therefore clearly be longer. On the other hand, the implantation can then be performed into an area that is already covered by healthy mucosa and free from inflammation. If necessary, the bone substitution is possible simultaneously with the implantation.

3. **Early implantation:** the implantation takes place 3-4 months following the extraction of the tooth. This has the advantage that the mucosa and the bone conditions are now mature, though degradation of the bone may already have begun by this time. A further disadvantage is the loss of time.

4. **Late implantation:** the implantation is performed at an unplanned point of time at least 6 months following the extraction. In spite of the fact that this is the most frequent surgical procedure, it has the great disadvantage that at that stage the degree of bone absorption may already be advanced. In numerous cases the implantation of the artificial root is then possible only after bone supplementation has been performed, or the implantation may be ruled out completely.

Before every intervention, the first, extremely important steps are to establish the general case history and to carry out a dental examination. Implantation may be carried out only in a mouth that has been adequately pretreated and displays a satisfactory level of oral hygiene. The implantation procedure must therefore be preceded by professional dental cleaning and conservational and prosthetic rehabilitation of the teeth. An indispensable step in the first examination, and a precondition for the preparation of the treatment plan, is the recording of a panoramic radiogram, or in certain cases a cone beam (3-dimensional) computer tomogram.
allows the treating physician, with the agreement of the patient, to formulate the most appropriate treatment plan, covering such points as the need or not for a bone replacement procedure (prior to or simultaneously with the implantation), in what position the implantation should be made, the number of implants required, their type, their size, and what prosthetic solutions are feasible in the given situation.

**SURGICAL PROCEDURES**

Dental implantation is a surgical intervention that (like all others) demands a high level of expertise from the operator. It is usually carried out under local anaesthesia, which in a given case may be supplemented with intravenous sedation, but it is often performed under general anaesthesia.

An incision is made in the mucosa at the site of the implantation, care being taken not to damage the anatomical structures in its environment (papillae and neurovascular structures). The mucoperiosteal flap created in this way is carefully separated from the surface of the bone with the aid of a blunt instrument (a xyster) (Fig. 2). In certain cases the implantation is achieved without this flap formation, but the routine use of this latter procedure has not become widespread.

The “bony bed” of the implant is prepared through the use of various drills, care naturally being taken that the bone should be continuously cooled: overheating of the bone leads to its necrosis and to loss of the implant. For this reason, the manual device of the surgical motors used for the implantation injects a uniform stream of physiological saline solution onto the surgical area. The site for the implant is first indicated with a marking drill. This may be either a globular drill or a lance-shaped drill (Fig. 3). Figure 3 A surgical template is often used for the marking (pilot, initial) drilling. This template is a drill-guide prepared by the dentist and the technician.
Marker drilling

In the next stage, the bed of the bone is predrilled with spiral drills (Fig. . The speed of drilling in these phases lies in the range 1200-1500 rpm. Following the predrilling, the accuracy of the drilling is checked with a depth-meter and an indicator of parallelity (Fig. ).
Predrilling

Verifying of drilling depth

After this, expansion drills with various diameters are used at lower drilling speeds (600-800 rpm) to form the basic dimensions of the implant (Fig. 5). Figure 5 Profile drills and thread cutters are then applied to create the appropriate final site in the bone to accommodate the exact shape and dimensions of the implant. Thread cutting must be carried out if the cortical substance of the bone is too hard.
Expansion drilling

As mentioned previously, the implant is primarily fixed into the bone with the aid of the thread on its external surface. The insertion and screwing of the implant into the bone may be carried out either manually or mechanically. The commercially available implants are supplied with an insertion instrument in sterile packaging. This instrument facilitates precise insertion of the implant into the bone (Fig. 6). In modern systems, the stress involved in the insertion can be checked with a stress key. This is important, for if the magnitude of the stress is not appropriate, the implant will not be sufficiently stable, and the healing and the later immobility will become uncertain. Nor is it satisfactory if the stress is too great, for the implant will then exert harmful pressure on the bone in its environment, which will result in rapid, pathological absorption of the bone.
Insertion of an implant, and the supporting structure used for this

The wound is closed with knotted sutures; atraumatic thread with a thickness of 4/0-5/0 is generally used. The sutures are removed one week postoperatively. (Fig.)
The wound is closed with knotted sutures

The implant may be inserted in one phase or in two phases. In the former case, healing proceeds transgingivally (in communication with the oral cavity), while in the latter case it proceeds subgingivally. In the one-phase technique, with the aim of closing the cavity, for the duration of the healing a healing screw is placed into the internal thread in the implant, which is inserted to the required depth, and the gingiva is then closed around it with the aid of sutures. In this single-phase intervention, there is no need for a second operation to free the implant (Fig.).
One-phase surgical technique

Gingival closure demonstrating mature, tight morphology develops together with the bone integration. A closing screw is likewise placed in the implant in the two-phase technique, but both this and the implant itself are covered by mucosa, completely enclosing the surgical area. (Fig.)

Two-phase surgical technique
Control radiograms are recorded in every case following the intervention. An antibiotic is recommended after the operation and, in the event of necessity, an analgetic may be administered. In order to ensure that a satisfactory connection develops between the bone and the implant, and that the load-bearing ability of the implant is perfect, the implantation must be followed by a 2–3-month healing stage.

If the implant heals transgingivally, the final dental replacement may be made immediately after the bony healing. If healing proceeds subgingivally, the implant must be freed before the dental replacement and provided with a gingiva-forming healing head. The aim of this is the development of a massive, tight mucosal profile directly around the implant. A one-week healing period is generally sufficient for the development of appropriate epithelial adhesion at the neck of the implant.

In many cases, the implantation is preceded by various supplementary surgical interventions (such as bone substitution), or these may be performed post-implantation (e.g. in the case of gingivoplasty).

**LONG-TERM SUCCESS OF IMPLANT, MAINTENANCE (SUPPORTING) THERAPY**

The implantation may be stated to be successful if there is no foreign body sensation, no pain, no inflammation around the implant, the implant is stable, and there are no radiographic signs of osteoporosis around the implant. The permitted level of bone absorption adjacent to the implant is 0.2 mm per year. The success of the implantation depends not only on the individual characteristics of the patient, but also to a large extent on the planning and on whether the patient turns to a specialist with sufficient experience and expertise.

Following the implantation, the healing process requires about 3 months. The rate of healing depends appreciably on the surface properties of the implant and on the nature of the bone into which the artificial roots are implanted. During the healing period, the bone cells grow directly into the micropores created on the surface of the implant. This integration with the bone is responsible for the stable fixation of the implant, which will enable the implant to transfer the masticatory forces acting on it (pressure, tensile forces and shearing forces) to the bone tissue, lastingly and without the development of any damage.

The contact between the patient and the treating physician does not come to an end when the dental implant has been inserted into the mouth, as constant cleaning, continuous maintenance of a good state of oral hygiene and regular dental control are among the most important conditions of the long-term success of dental implantation. Besides the accurate diagnosis, the detailed preparation and the careful insertion of the implant, its functioning and its expected lifetime are influenced decisively by the level of oral hygiene.

The critical point following the implantation procedure is the meeting point of the mucosa (the gingiva) and the neck of the implant. In an unfavourable case, this is the site at which such a degree of inflammation may occur that it can lead to the loss of the implant (mucositis or peri-implantitis). With a view to preventing this, patients who have received such an implant must be recalled for regular control examinations and for professional oral hygiene treatment. If the treating physician discovers signs of incipient inflammation at the prescribed control visit, and subjects this to appropriate high-level treatment, there is generally no risk of later complications.

The long-term success of dental implantation also depends appreciably on the patient, for thorough oral hygiene achieved with the most suitable equipment is indispensable. In addition to the normal toothbrush, it is essential to use special brushes for cleaning between the teeth, various forms of dental floss are also important, and after the implantation has been performed the patient must receive adequate advice concerning the correct tooth-cleaning techniques, the use of the various special equipment, and the importance of all this, as it is the patient who can do most to maintain a satisfactory level of oral hygiene and hence promote the long-term success of the implant (see the Chapter 4.1.7.)

**4. Caries therapy (Zsuzsanna Tóth DMD)**

When chewing, food is grasped and cut up by the teeth, but the teeth play an important role in speaking and sound-formation as well. Healthy and nice teeth are significant factors of attractive appearance. Only sound or restored teeth fulfil these functions. Because of caries incomplete, broken, discoloured teeth have poor aesthetics, they can be sensitive, and their cleaning is more difficult.

Prevention is the most effective therapy, keeping in mind restoration as well. Treatment of a carious tooth will prevent consequent pathological changes, for example inflammation of the dental pulp. The shape of a well restored tooth is similar to the original, it is connected with the adjacent teeth by contact points or contact surfaces, fits in the occlusion and has proper aesthetics.
After taking medical and dental history, clinical investigation will be carried out starting with stomatological screening. Based on the diagnosis, treatment planning will followed. Treatment starts with the solution of emergency conditions (severe pain, swelling, bleeding), followed by full restoration of the masticatory organ. An important part of dental care is to maintain the restored status by regular check-ups. Caries therapy is part of a complex therapy aimed to get the patient healthy in every respect. That means oral health from a periodontological and cariological point of view as well.

**Caries Risk Patients**

Special attention should be paid to the treatment and dental care of caries risk patients. A person with at least two of the following four criteria should be considered as caries risk patient:

- two or more active caries,
- more existing restorations in the oral cavity (age related high DMF value, in adults higher than 8),
- inappropriate nutritional habits (snacking, high carbohydrate intake etc.),
- decreased salivary flow

The determination of caries risk is particularly relevant before orthodontic treatment, in case of possible occupational diseases or dry mouth (due to medication). Conclusions about caries activity can be drawn based on saliva secretion, buffer capacity, acid production of plaque bacteria and the quantity of pathogenic microorganisms in the dental plaque (Streptococcus mutans, Lactobacillus and Candida albicans). If we detect caries activity in the initial stage and introduce prophylactic measures, the macroscopic damage to tooth structure and cavity formation can be prevented.

**Treatment Planning**

The aim is always to restore and maintain complete oral health. The sequence of interventions depends on urgency and on the general condition of the patient. Every treatment plan should be individualised and scheduled in agreement with the patient.

At first we should get the oral mucosa inflammation free and healthy by scaling and other periodontal procedures. For a successful therapy the co-operation of a motivated and instructed patient is needed. The necessary periodontal and dental surgical measures are followed by conservative, endodontic, orthodontic and prosthetic treatments. The course of the treatment together with all related data, medical reports and records have to be administered in a detailed and accurate way.

**The Sequence of Caries Therapy**

The goal of our therapy is complete rehabilitation of the oral cavity. Both from patient’s and from tooth’s point of view the most favourable condition is aimed to be created and maintained. A sound tooth is more valuable compared with a restored one, and a vital tooth is more valuable in comparison with a root canal filled one. Therefore, deep and active carious lesions of vital teeth should be treated first to save their vitality. Starting with the most severe case, all decayed teeth have to be filled. Parallel to these procedures, demineralisation has to be hindered by remineralising incipient carious lesions to avoid further cavity formation. Endodontic treatments of non-vital teeth and revision (retreatment) can be the next step. The maintenance of the restored status needs preventive measures, which are necessary at this stage as well (see Chapter IV.).

**Treatment of Caries**

Caries incipient

Caries incipient is a reversible lesion. The correct therapy in this case is non-invasive remineralisation. Mineral precipitation occurs and the changes of crystal structures advance in the direction of crystal growth. Saliva is a solution supersaturated by calcium and phosphates, and the crystal formation is accelerated by small quantities of fluorides that catalyse remineralisation and inhibit demineralisation. The constant presence of low fluoride concentration is more effective than rarely used high concentrations. That is why the use of topically applied low doses of fluoride is recommended (toothpaste, mouthwash, gels).
Concerning the dosage, see chapters about Prevention, Toothpaste and Fluoride. Nowadays, the ion balance can be influenced by newer products as well, for example by milk-derived peptides, casein, CPP-ACP (casein phosphopeptide-amorphous calcium phosphate) or by bioactive glass, for example NovaMin. There are antibacterial preparations available for professional use, for example chlorhexidine, which can promote caries control.

Caries control in deciduous teeth can be very effective with silver diamine fluoride containing preparations, but they cause permanent discolouration. Also xylitol, a sugar substitute can facilitate remineralisation by increasing saliva secretion. The beneficial effect of xylitol-containing chewing gum is also proven. The remineralised surface is more resistant than the original enamel, but colour change can occur. If this colour change does not cause a cosmetic problem, no treatment is required.

Arrested caries is a stagnating process without cavity formation. It requires treatment only if the brown or dark discoloured spots on the surface are aesthetically disturbing for the patient.

**Caries with breakdown of the surface**

In case of an advanced process, the integrity of the tooth surface is damaged, the porous enamel breaks, the process becomes irreversible and a cavity is formed. From that moment on, the appropriate treatment of the carious lesion is discovery and removal of the decayed parts followed by filling or the application of an inlay. In case of secondary caries, the removal of the carious tissue and tooth restoration can only be performed after the removal of the inadequate restoration. By cleansing the softened and bacterium- rich area, the oral cariogen flora will be reduced in number and the most protected plaque retention sites will be eliminated.

Nowadays a number of improvements have been made to replace the traditional round bur in the debridement of carious cavities. The goal of these new mechanical methods (sono-abrasive, air-abrasive, air- polish technique) or chemo- mechanical excavation, (Carisolv - Na hypochlorite gel, Mediteam, Sweden) is less invasive treatment for the patient. Ozone gas reduces the pathogenic flora of carious hard tissues within seconds (HealOzone, KaVo , Germany). Experiments are under way to remove carious dentine through enzymatic digestion. To save patients from stress, investigations are being performed disinfect the infected tissue with a suitable laser device or with antibacterial therapy (ZnO , Ca(OH) 2, fluorides, chlorhexidine, antibiotics) without any caries removal. Maintenance of a healthy state for a long time is possible after thorough motivation and instruction about diet, oral hygiene and regular dental check up, provided that the advice will be taken.

There are more options to restore carious teeth: filling, inlay-onlay, veneer or a crown (crowns will be further discussed in Prosthodontics).

**RESTORATION OF CARIOUS CAVITIES**

**Classification of filling materials**

- Temporary filling materials
- Base or liners
- Restorative materials

Temporary filling materials Temporary fillings are used when a treatment cannot be completed due to lack of time, excessive bleeding, root canal treatment, pulp healing, separation, orthodontics, making inlay or prosthetic intervention. In this case the cavity should be filled with gutta-percha or with paste type products, which cure fast under moist circumstances (for example: Cavit). Sometimes; however, a long-term temporary filling is required, in these cases glass ionomer cement, zinc oxide eugenol cement (healing) or composite fillings may be recommended.

**Expectations temporary filling materials have to fulfil**

- Biocompatibility
- Easy to apply - easy to remove
- Fast curing
• Loading resistance
• Impermeability
• Tooth coloured aesthetics without discolouration
• Good adhesion
• Isolation
• Resistance to dissolution
• Radio-opacity
• Reasonable cost

Base, liner materials Some authors argue that there is no need for lining under fillings with a hermetic seal. However, if the filling material conducts heat or electricity, lining has to be used for the protection of the pulp.

If healing of the dental pulp or prevention of its pathological changes are indicated, a special base is needed to reduce inflammation, stimulate odontoblast activity and perform antisepic or even remineralising activity. Strongly alkaline materials containing Ca(OH) 2 have antibacterial activity and reduce inflammation while stimulating the formation of tertiary dentine. Because of the lower mechanical resistance of some products, the placement of an extra cement layer is necessary.

Zinc oxide - eugenol cement has analgesic and antibacterial effects due to its eugenol content, it is reasonably wear-resistant and seals properly, therefore it is appropriate to be used as a temporary filling for a longer period (Fig. 1). Eugenol can be toxic near the pulp, so it is indicated to cover the deepest part of the cavity with Ca(OH) 2. Notice that eugenol interferes with the polymerisation of resins.

Zinc oxide eugenol cement intermediate restoration for a longer period

When replacing amalgam with inlay, undercuts can be completed with liner materials as well. Zinc oxyphosphate, polycarboxylate and ionomer cements self-or light cured are the most commonly used liner materials beside the above-mentioned Ca(OH) 2 - , and eugenol-containing cements.

Final restorations
Expectations dental restorative materials have to fulfill

- Biocompatibility
- Adequate mechanical properties:
  - Stress and tensile strength
  - Flexibility
  - Hardness similar to enamel
  - Wear rate similar to enamel
  - Form and shape stability
  - Thermal expansion similar to dental tissues
  - Water resistance, insolubility, adhesion
  - Thermal and electrical isolation
  - Antiseptic and caries prophylactic properties
  - Radio-opacit
  - Optical properties similar to tooth
  - Easy application and finishing, polishing
  - Easy removal
  - Reasonable cost

**DIRECT RESTORATIONS**

Direct restorative materials - plastic filling materials - are placed into the prepared cavity in a plastic, soft, pliable state and then set hard. In the case of direct restorations the tooth is prepared and the filling material is applied during the same visit.

**Amalgam**
For a long time the most commonly used filling material was amalgam, but recently its popularity has been strongly reduced due to its disadvantageous colour and because of the wide variety of more and more improved composites.

The properties of amalgams (unfavourable properties in bold)

- Volume stability
- Insolubility
- Mechanical resistance
- Ductility
- Thermal and electrical conductivity
- Corrosion
  - Non-toxic!!! - Allergy, but very rarely
- Radio-opacity
- Silver coloured
- Fluorid-containing is also available
- Favourable price

The steps of amalgam filling preparation

- **Cavity preparation:** in case of class I., II. and V. caries affecting the occlusal, proximal or buccal surfaces of premolar and molar teeth, a conventional cavity with macro retention has to be prepared. Vital teeth may need local anaesthesia.

- **Isolation:** relative: with cotton rolls and saliva ejector, absolute: with rubber dam and saliva ejector, exhauster

- **Cavity cleaning and drying**

- **Adaptation of matrix and wedges**

- **Restoration:** the tooth is isolated, matrix band and wedge is adapted, the cavity is clean and dry (in case of deep caries liner is used), layer by layer amalgam has to be applied and condensed. The cavity will be overfilled, occlusal surface has to be carved, contoured, and after removing the wedge, matrix retainer and band occlusion check up is the next step. In case of bonded amalgam restorations the bond has to be adapted before inserting (in accordance with the manufacturer's instructions). *Precautions: the patient should not eat until the effect of anaesthesia has worn off. New amalgam fillings are susceptible to be damaged by chewing for approx. 2 hours!*

- **Finishing, polishing:** during the next visit re-contouring, finishing and polishing should be performed. A perfectly smooth surface is easy to clean, it is not susceptible to plaque accumulation and the likelihood of secondary caries is reduced. The finishing and polishing procedures should not be initiated on an amalgam restoration until the amalgam has reached its final set, at least 24 hours after it has been placed and carved.

A well-made amalgam filling lasts for many years without any side effects; however, nowadays it is not preferred because of its colour (Figure 2)

**Instruments required (armamentarium):** mouth mirror with handle, dental explorer (probe), cotton pliers, rubber dam kit with rubber dam sheets, rubber dam punch, frame, retainer forceps template, clamps, dental floss, circular, semi-circular matrix and bands, wedges, amalgam carrier, Williams plugger, round condenser, Vajna protector, Heidemann (Flaggs) spatulas, Black-excavator, amalgam carver, Thomas, Westcott instrument, Hollenback carver, dental articulating paper and stones for the next visit, stainless steel or tungsten carbide finishing, polishing rotary burs, amalgam polishers, polishing paste, various discs.
Amalgam filling is not preferred because of its colour

**Cements**

Cements are used as base and for fixing crowns and bridges. Silicate cement is translucent and porcelain-like when set, formerly it was used for aesthetic restoration of the anterior teeth. This material is worth mentioning only for its historical significance, as an aesthetic solution prior to resin fillings.

**Glass ionomer cements**

Self-cured and light-cured glass ionomer cements are used in dentistry. They bind to the tooth chemically and do not shrink. Aesthetically they are less favourable than composite materials. Due to fluoride release from glass ionomer filling materials, the formation of secondary caries is inhibited. It is an intelligent filling material. Its clinical application is relatively large, it is a suitable material for bases, build-up, fixing, but it does not work very well as a fissure sealer. It is also used for filling, but due to its inadequate mechanical properties it is not suitable for filling teeth exposed to extensive loading. However, it can be recommended for long-term temporary fillings and restorations of deciduous teeth. Glass ionomer root canal obturation paste is also available, but it cannot be removed from the root canal. Cermet cement is a self-cured ionomer mixed with silver powder, offering neither mechanical nor aesthetical advantage.

**Composite resin fillings**

Patients will have an aesthetical and permanent solution, so the majority of restorations are made from composite filling materials nowadays.

The properties of composite resins (unfavourable properties in italics)

- Physical, chemical and mechanical properties are improved by increasing the filler content
- It wears due to food, occlusion, slipping, interproximal rubbing effect and tooth brushing
- Allergy is more common
- Water absorption
- Shrinkage - significant polymerisation shrinkage
- It can be polished
- Colour does not change
- Radio-opacity
• Tooth-coloured, translucent

• It is more expensive than amalgam, but much cheaper than inlay

• Technology – sensitive

Besides restoring carious cavities, composite filling materials are also suitable for other aesthetic solutions for example management of diasthema, discolouration and enamel defects, replacement of a veneer, fixing orthodontic brackets or splinting loose teeth. The application is done with adhesive technique (total etch, wet bonding).

The steps of applying composite restorations

• Cavity preparation: this filling material is suitable for nearly all types of cavity restoration, and the preparation is limited to minimal invasive preparation (removal of the carious tissue). The cavity is bevelled at 45 °, 0.5-1 mm wide everywhere except for the occlusal surface. Vital teeth may need local anaesthesia.

• Selecting the appropriate shade matching the colour of the tooth

• Isolation: possibly absolute isolation with rubber dam, saliva ejector and exhauster - Cavity cleaning and drying

• Adaptation of matrix systems and wedges (can also be performed later, after bonding)

• Etching: enamel should be etched for 30 seconds, the entire surface of the dentine for 15 seconds with 35-37 % phosphoric acid etching gel

• Washing: with water for 30-60 seconds

• Drying: with gentle air, but the dentine should not be dried completely in order to avoid the collapse of collagen fibres

• Adaptation of bonding materials: according to the manufacturer's instructions, sprayed with syringe in a thin layer

• Polymerisation: for 10-20 seconds * Note: when using self-etch systems, etching and bonding is performed in one step without washing and with polymerisation.

• Restoration: with composite: max. 1-2 mm thick layers

• Polymerisation: 20-40 seconds per layer

• Finishing, polishing: after the removal of the wedge and matrix the filling should be checked, occlusion assessed, surfaces contoured, finished and polished. Precautions: the patient should not eat until the effect of anaesthesia has worn off. After the new composite filling has set, it is not compromised by chewing.

Instruments required (armamentarium): mouth mirror with handle, dental explorer (probe), cotton pliers, rubber dam kit with rubber dam sheets, rubber dam punch, frame, retainer forceps template, clamps, dental floss, polymerisation lamp, applicators, various matrix systems and bands, wedges, Williams plugger, round condenser, Heidemann (Flags) spatulas, Black- excavator, Thomas, Westcott instrument, Hollenback carver, dental articulating paper, finishing and polishing stones (diamond, Arkansas), rotary burs, polishers, brushes, strips, various discs, polishing paste, shade guide.

Compomers

Compomers are composite filling materials containing reactive glass filler, their name combines composite and glass ionomer. Self-curing and light-curing types of Compomers have not fulfilled the expectations to perform restorations more easily and more quickly but of the same quality as composite fillings.

INDIRECT RESTORATIONS - SOLID RESTORATIONS

Inlays-onlays
Inlays and onlays are restorations made in a dental laboratory on models made from impressions of the tooth prepared by the dentist and they are placed and fixed in a specially designed cavity in a solid form. Inlays have occlusal and proximal surface(s) of a posterior tooth and maybe one or more, but not all of the cusps. If the whole occlusal surface, all the cusps are completely covered, it is called an onlay. These restorations generally require multiple visits and the placement of temporary restorations in the prepared teeth between appointments. The use of inlays is a long-term solution of high quality. It insures an optimal occlusal surface design and contact point conditions preserving the remaining tooth structure as well. It is an expensive restoration because dental laboratory background or sophisticated computer (CAD -CAM = Computer Aided Design - Computer Aided Manufacturing) technology and equipment is needed.

Steps for inlay preparation:

- **Cavity preparation**: The preparation should be carried out regarding the properties of the planned inlay (e.g. cast metal, ceramics and composite). When preparing the cavity there are important factors not to forget: the protection of the remaining tooth material, the potentiality of insertion and the requirements of material thickness for the proper mechanical properties. Vital teeth may need local anaesthesia.

- **Impression**: A precision-situation impression should be taken from the entire dental arch with antagonistic impression, bite registration and selection of the appropriate colour. In CAD -CAM technique, optical digital print gives the required information for designing and manufacturing the restoration, which will be ready soon and may be fixed during the same visit.

- **Temporisation**: with an intermediate restoration removable in one block.

- **Check up**: the finished indirect restoration should be checked on the model and in the prepared cavity after the removal of the temporary filling. It occurs during the next visit, but in case of CAD -CAM technology at the same time. The occlusion of an aesthetic indirect restoration can only be controlled after placement in the mouth in order to avoid breaking. Occasionally you may need to anaesthetise the patient.

- **Placement**: the technique of the fixation depends on the type of indirect restoration. In case of:

  1. *cast metal and metal ceramic restoration* with zinc oxyphosphate or glass ionomer cement. The placement of cast metal and metal ceramic restoration is a less technique-sensitive procedure. After the insertion of inlays or onlays in the properly prepared and cleaned cavity the removal of excess cement is simple. For some hours chewing should be avoided.

  2. *aesthetic indirect restoration*: with dual-curing resin cements in order that the resin sets beneath the restoration, at the bottom of the cavity as well.

  3. **Arrangements before the placement of indirect restorations**:

      a. *ceramic inlays*: etching with hydrofluoric acid (HF) for one minute, washing with water for one minute, drying, application of silane

      b. *composite inlays*: etching with phosphoric acid, washing, drying

  4. **Isolation**: absolute: rubber dam isolation, saliva ejector, exhauster

  5. **Cavity cleaning and drying**

  6. **Adaptation of matrix systems and wedges**

  7. **Etching**: enamel should be etched for 30 seconds, the entire surface of the dentine for 15 seconds with 35-37 % phosphoric acid etching gel

  8. **Washing** with water for 30-60 seconds

  9. **Drying** with air but the dentine should not be completely dry in order to avoid the collapse of collagen fibres

  10. **Application of dual bonding resin cement** in accordance with manufacturer's instructions. The mixed dual bond material covers the cavity or/and the restoration, excess should be removed.
11. **Polymerisation** (starts by light curing) only after the insertion of the inlay for 20-40 seconds from each direction.

12. **Finishing, polishing:** after removal of the wedge and matrix the restoration should be checked, occlusion assessed, finished and polished. *Precaution: the patient should not eat until the effect of anaesthesia has worn off. The new aesthetic inlay is not compromised by chewing.*

Instruments required (armamentarium): mouth mirror with handle, dental explorer (probe), cotton pliers, rubber dam kit with rubber dam sheets, rubber dam punch, frame, retainer forceps template, clamps, dental floss, polymerisation lamp, applicators, brush, various matrix systems and bands, wedges, round condenser, Heidemann (Flaggs) spatulas, dental articulating paper, finishing and polishing stones (diamond, Arkansas, stainless steel, tungsten carbide), rotary burs, polishers, polishing brushes, strips, various discs, polishing paste, shade guide.

**Veneers**

The veneer is a direct or indirect restoration covering the buccal - exceptionally oral - surface of the teeth made from composite or ceramic. Direct restoration is made by the dentist, the indirect method also needs the work of a technician based on impression. Veneers can be considered to restore carious teeth and to correct aesthetically disturbing enamel defects, abrasion or discolouration as well.

**Steps for veneer preparation:**

- **Cavity preparation:** The preparation should be limited to the enamel to ensure the safest adhesion. Occasionally anaesthesia should be considered.

- **Impression:** A precision-situation impression should be taken from the entire dental arch with antagonistic impression, bite registration, selecting the appropriate colour and determining character.

- **Temporisation:** Is not required if the preparation involved only the enamel. Possibly it can be made from provisional composite removable in one block.
• **Check up:** the finished veneer should be checked on the model and on the tooth after the removal of the temporary filling during the next visit. Anaesthesia is not likely to be needed.

• **Arrangements** before the placement of veneers:

  1. ceramic veneer: etching with hydrofluoric acid (HF) for one minute, washing with water for one minute, drying, application of silane

  2. composite inlays: etching with phosphoric acid, washing, drying - Isolation: absolute: rubber dam isolation, saliva ejector, exhauster

• **Cavity cleaning and drying**

• **Adaptation of matrix band and wedges**

• **Etching:** enamel should be etched for 30 seconds, the entire surface of the dentine for 15 seconds with 35-37% phosphoric acid etching gel

• **Washing** with water for 30-60 seconds

• **Drying** with air but the dentine should not be completely dry in order to avoid the collapse of collagen fibres

• **Placement:** thin ceramic veneers can also be fixed with light-cured bonding resin cement, because it can be transilluminated with the polymerisation lamp

• **Application of dual bonding resin cement** in accordance with the manufacturer's instructions. The mixed dual bond material covers the cavity or/and the restoration, excess should be removed

• **Polymerisation** (starts with light curing) only after the insertion of the veneer for 20-40 seconds from each direction

• **Finishing, polishing:** after the removal of the wedge and matrix band the restoration should be checked, occlusion assessed, finished and polished regarding the margins

*Precaution: the patient should not eat until the effects of anaesthesia wear off. The new aesthetic veneer should not be compromised by chewing.*

**Instruments required (armamentarium):** mouth mirror with handle, dental explorer (probe), cotton pliers, rubber dam kit with rubber dam sheets, rubber dam punch, frame, retainer forceps template, clamps, dental floss, polymerisation lamp, applicators, brush, matrix bands, wedges, round condenser, Heidemann (Flaggs) spatulas, dental articulating paper, finishing and polishing stones (diamond, Arkansas), rotary burs, polishers, polishing brushes, strips, various discs, polishing paste, shade guide, test paste on colours of bonding materials

5. **Bleaching (Zsuzsanna Tóth DMD)**

Nowadays it is more frequent that the conditions of a pleasing look and of feeling comfortable are not only caring for one’s self, but also having whiter teeth (sometimes blinding white). At the same time bleaching has medical (professional) indications as well.

*REASONS OF DISCOLOURATION (Fig.)*
The reason of acquired endogenous pre-eruptive discoloration can be a disease (e.g. neonatal hepatitis) or the side effect of taking certain medication, for example in the case of tetracyclin. After eruption the tooth surface changes physiologically with aging, but often because of a pathological condition too, for example necrosis, trauma, or bleeding (Fig.2). The shade of the tooth differing from normal can be chalk-white (fluorosis), bluish-grey (e.g. dentinogenesis imperfecta), light yellow (e.g. with aging), dark yellow (e.g. necrosis), brown (e.g. tetracyclin), black (e.g. because of amalgam), or pink (e.g. internal resorption).

METHODS OF ELIMINATING DISCOLOURATION

The start point of treating a discoloured tooth is to find the cause. This is the basis of treatment planning and prognosis. Changing the shade of a tooth can be simple cleaning and polishing, changing discoloured restorations, bleaching, but sometimes only aesthetic restorations (crown, veneer) will mean a solution.
Bleaching can be carried out externally in case of vital teeth and internally in case of root canal treated teeth.

**Treatment planning for bleaching**

- diagnosis following medical history, clinical examination and evaluating the X-ray
- treatment planning, choosing the appropriate bleaching method, timing
- photo documentation - professional cleaning and polishing.

Prosthetic restorations cannot be whitened. Bleaching can only be performed on restored teeth, because bleaching material will penetrate into cracks, caries, leaking fillings and cause resorption.

**Mechanism of bleaching**

The effective chemical in bleaching is free active oxygen or oxygen released from chemical bond, which causes the fading or discolouration of colouring pigment materials by the oxidation of a double bond.

Oxidising agents:

- urea peroxide (CH4N2O-H2O2),
- sodium perborate (NaBO3.4H2O),
- sodium percarbonate (2NaCO3.3H2O2)

Hydrogen peroxide (H2O2) is released during the decomposition of every chemical compound, but hydrogen peroxide is also used for bleaching on its own. The packing can be a gel, a liquid or a powder. According to the modified decree 40/2001 (IX.23.) of the Hungarian regulations, since November of 2012, hydrogen peroxide with a concentration higher than 6% cannot be sold or used! The other bleaching agents can only be sold in a concentration that will contain no more than 6% hydrogen peroxide after decomposing. Only a dentist can buy the bleaching material, and in the interest of professional control, only he/she can apply it initially and in the case of adult patients above the age of eighteen.

Bleaching is contraindicated in the case of allergy, severe systemic disease, pregnancy, breastfeeding, hypersensitive teeth and teeth in very poor condition, in case of extreme discoloration and heavy smokers. Bleaching is not suggested to be done at home by patients complaining about TMJ.

The long-lasting success of bleaching depends mostly on the diagnosis, on the method of bleaching and on the individual habits. There are factors influencing the result, e.g. dietary habits, smoking and oral hygiene. Retreatment might be needed in 1–2 years.

**Bleaching vital teeth: external or enamel surface method**

1. **microabrasive method**: a demineralised thin layer is slowly removed by a rotating disc in rubber dam isolation paying attention to the eye protection of the patient and the staff. Demineralisation is performed using a mixture of hydrochloric acid, water and polishing paste

2. **in-office bleaching (chair-side)**: the oxidising agents are applied on the outer surface of teeth isolated by liquid dental dam for gum protection. The effect of it can be intensified by light (LED, halogen lamp, plasma lamp, or laser) (Fig.3), by heat, although there are various opinions present in the literature. Its effect can be increased by addition of home bleaching.

3. **home bleaching**: an individual or prefabricated plastic tray is fitted on the treated arch, which will contain the bleaching agent, and which will be worn under specialist control until a desired shade is reached.

As a side-effect, gingival irritation, hypersensitivity of the tooth or the cervical part of the tooth, unsuccessful treatment may occur, or arthropathy of the temporomandibular joint because of wearing a splint.
Light-activated bleaching in the dental office

**Bleaching of devitalised teeth: internal or dentin bleaching method**

Prior to bleaching, a preoperative X-ray must be taken of a root canal treated tooth to check the quality of the root canal filling. An incomplete root canal filling must be revised. The root canal filling is then prepared back on the coronal end and isolated (by glass ionomer cement) from the bleaching agent placed in the pulp chamber. Documenting and determining the initial shade are important even in the case of a single tooth!

1. **thermocatalytic method:** a bleaching agent placed in the pulp chamber is more effective when heated.

2. **light-activated bleaching:** the oxidising chemical is more effective when light-cured by LED, halogen lamp, plasma lamp, or by laser.

3. **walking bleach:** the oxidising agent placed in the pulp chamber of an appropriately root canal filled and sealed tooth is hermetically closed by glass ionomer cement and a week later if necessary the oxidising agent is changed. Final restoration may only be done two weeks after the bleaching in order to avoid the disturbance of polymerisation caused by the oxidising agent (Fig. 5).

Fragility, internal resorption or unsuccessfulness may occur as a side-effect.

Walking bleach: periapical X-ray, pre-treatment and post-treatment photo of tooth 43

### 6. Therapy of pulpal diseases (Zsuzsanna Tóth DMD)

**THERAPY OF VITAL TEETH**

The teeth are exposed to numerous irritants every day following eruption. Depending on its quality and intensity, some of these irritant factors do not reach the pulp; on the other hand, some have an effect on the pulp through the enamel and the dentin. The response is a symptomless (silent) reaction of the pulp that means
histological structural change, which cannot be differentiated from the physiological pulpal changes. Physiological changes occur with age by the decrease in the number of cellular elements and the increase of fibrous elements. The consequence is similar, the physiological response of the pulp deteriorates. This phenomenon has no symptoms and does not need therapy.

When healthy hard tissue is damaged, for example by caries, all irritant stimuli reaching the tooth are felt more intensely, in addition, the carious lesion in itself is also a pathologic stimulus. Incipient caries is the visible demineralisation of the tooth surface without cavity formation. The correct therapy is early remineralisation. Due to the odontoblastic processes reaching into the dentin tubules, an early carious lesion has an effect on the odontoblasts enhancing their dentin production to a certain degree. The tooth is unable to repair the cavity, the intervention of a dentist is needed, and a filling must be prepared. There can be alterations in therapy depending on the depth of the cavity.

In case of a deep carious lesion (see Lining materials) a liner might be needed on the base of the cleaned cavity, whereas usually fillings are done without lining. During treatment of a deep carious lesion, if there is a risk of opening the pulp chamber, indirect pulp capping can be performed. After removing the infected, soft dentin, the demineralised dentin usually also removed is left on the bottom of the cavity, which is then covered by Ca(OH)2, a bactericide pulp capping agent, which stimulates dentin formation of the odontoblasts. This is then covered by glass ionomer liner than restored by amalgam or composite filling.

If the pulp is exposed because of trauma or during preparation, we apply Ca(OH)2 pulp capping following blood-clotting, and this is called direct pulp capping. A good result can be achieved by hermetic sealing of the pulp wound by the expensive MTA (mineral trioxide aggregate) or simply by bond application. To finish the restoration, we apply glass ionomer liner followed by amalgam or composite filling. It is needed to check the vitality of the tooth on a regular basis. The number of successful therapy is set back by the fact that we cannot determine the depth and intensity of the infection. On immature teeth, the infected pulp is removed by pulp amputation (pulpotomy) to achieve further development of the apex by maintaining the vitality of the tooth, so later a properly condensed root canal filling can be done following the full development of the apex. In case of a necrotic immature tooth, we try to promote further development, the closing of the apex by apexification, that is by removing the necrotic tissue and changing Ca(OH)2 orderly in the root canal. The approximately one and a half or two year long treatment is then finished by lege artis root canal filling. If due to a carious lesion reversible pulpitis develops, in order to maintain vitality of the tooth, the progress must be reversed. By eliminating the irritant factors, namely the carious tissue, normal circulation may be restored even though the degenerative changes of the pulp cannot be reversed. To functionally restore the tooth, a filling is prepared.

ROOT CANAL THERAPY

If the pulp undergoes irreversible damage due to caries, its circulation collapses and will end in tooth necrosis. Pulp necrosis, like the other periapical diseases, acute apical periodontitis, chronic apical periodontitis, condensing ostitis, acute apical abscess, chronic apical abscess, is treated similarly as to its therapy. The pulpal cavity of a necrotic tooth is isolated from the blood circulation; therefore from the defence system of the body, while necrotic tissue becomes culture medium of anaerob bacteria, which may be the cause of focal infection. Focal infection affects the whole body; it is accompanied by subfebrility, elevated sedimentation rate, listlessness and severe organic disorders (e.g. infective endocarditis, glomerulonephritis, polyarthritis, iridocyclitis, etc.). It can be eliminated by extraction or root canal therapy under antibiotic prophylaxis. If a tooth is worth saving, our first choice is root canal treatment, as it is in the interest of the patient. In some cases, the root canal therapy is extended with a surgical endodontic treatment (e.g. incision, resection, cystectomy) (Fig. 1). The loss of a tooth is a functional loss and will need a more expensive solution.
Aim of root canal therapy

By endodontic treatment, we aim to maintain or restore the health of the periapical space and the neighbouring tissues of the tooth. By preparing the root canal system of a tooth, we eliminate the irritant factors and impede the bacteria from infecting and invading the root canal system. This is achieved by hermetic obturation of the root canal system in three dimensions with a properly prepared root canal filling.

Steps of root canal therapy

After establishing the exact diagnosis, a treatment plan is made in accordance with the patient’s needs. The precision of the treatment is enhanced if it is done magnified with the aid of a loupe or an operating microscope (Fig. 2).

The use of operating microscope during root canal treatment

Following a preoperative periapical X-ray, the previous restoration is removed under anaesthesia if needed, the carious lesion is cleaned and under absolute isolation (rubber dam) the pulp chamber is unroofed, and the access cavity is prepared. In case of caries spreading subgingivally if perfect isolation cannot be obtained, a crown lengthening is performed. The endodontic treatment is continued following healing. By preparing an
access cavity with as little loss of tooth structure as possible, we are able to reach the root canal the straightest way and remove the pulp tissue fully (Fig. 3).

Access cavity and extirpation

Correct determination of the working length is done by initially estimated working length (by diagnostic periapical X-ray, not by OP!), by electronic (Fig. 4) and radiological working length determination.

Cleaning and shaping

The vital, not infected root canal is also to be prepared so a proper shape and size can be reached for completing a correct root canal filling. The shape of a root canal appropriate for obturation is tapered widening towards the crown and having a constriction at the apex, which prevents the root canal filling material from penetrating the periapical area. The organic debris, the microorganisms, their end product of metabolism and endotoxins are also to be eliminated from the infected root canal. Because certain areas of the root canal system are inaccessible to the instruments (e.g. lateral canals, ramifications, intercanal communications) (Fig. 5), mechanical cleaning is completed by chemical cleaning, so root canal preparation is a chemo-mechanical preparation.
Anatomy of the root canal system

**Instruments of mechanical root canal preparation**

Shaping can be performed by hand and rotary instruments, or by the combination of these methods. Vital pulp tissue is removed by hand instrument, Donaldson-needle. Preparation of the dentin wall by hand instrument is performed with successfully increasing size of stainless steel, nowadays done by nickel-titanium Kerr- and Hedström files.

When using rotary files, nickel–titanium alloy needles are used locked into a handpiece or a suitable endomotor. The motion of rotary instruments can be a simple rotary movement and can be reciprocating movement that is a 360° rotation back and forth. A wide range of instruments from different systems are offered; they differ in size, taper and cutting edge.

Using hand and rotary instruments demand routine, the previous one is more time consuming and more tiring, the latter one is faster, but is a more expensive method.

**Materials used for chemical root canal preparation**

Debris produced during root canal preparation is removed physically by rinsing (Fig. 6). Areas inaccessible to endodontic instruments and sequestered regions where the dentin tubules open into the root canal are focuses of bacterial invasion; therefore, to reduce the number of microorganisms, chemical disinfectants are used as irrigants.

What is an ideal irrigant like? It is non-toxic, but adequately disinfects, at the same time, it dissolves organic tissue and removes debris from the dentin wall; furthermore, due to its low surface tension, it penetrates the dentin tubules. Unfortunately none of the solutions meet all the criteria, so the literature suggests a combination.

Sodium hypochlorite (NaOCl) diluted by distilled water to 0.5–1–2.5% is less toxic, but is a good disinfectant and dissolves organic tissue. For dissolving hard tissue chelators 17% EDTA (ethylenediaminetetraacetic acid) or 10% citric acid is available. Their water diluted solution is an irrigant, their viscous form is a lubricant aiding the passage of the instruments in the root canal. Cleaning the dentin wall and opening the tubules upgrades its disinfectant effect. At the end of the preparation, the root canal should finally be irrigated by distilled water.
Irrigation

In case of a resistant bacterial flora, a 5 and 10% Lugol-solution (potassium iodine and iodine solution) can be used. The evaporating iodine penetrates the dentin tubules, which is effective, but it inactivates rapidly. That is why the completely prepared and cleaned root canal system is filled with it for ten minutes. Solumium is a licensed Hungarian solution, which by the volatility of hyper pure chlorine dioxide insures the bactericidal effect in areas inaccessible to instruments.

*Materials used for temporary sealing of the root canal system*

If a final obturation cannot be done, the root canal system is temporarily sealed until the next visit. Nowadays instead of the toxic phenol and aldehyde derivatives used before, we fill the root canal with a paste gained by mixing Ca(OH)₂ powder and distilled water. Prior to sealing the root canal is dried, and then it should be filled 1–2 mm up to the apex with the paste. It is inserted into the canal by Lentulo spiral drill. This paste is a very strong alkaline (pH 12.5). It is bactericidal, denaturing the cell membrane. It is appropriate for long-term temporary sealing, because it dissolves very slowly. The hermetic sealing of the temporary filling is very important to prevent the tooth from re-infection.

*Hermetic obturation of the root canal system: root canal filling*

A tooth, the root canal system of which was prepared (Fig. 7), must be treated with a root canal filling. A proper root canal filling prevents microorganisms and materials, serving as a culture medium for bacteria, from penetrating into the root canal system. At the same time it prevents bacteria and their toxins from reaching the periapical area. So the root canal filling must seal hermetically apically, at the tip of the root, laterally, towards the lateral canals, and coronally, that is towards the crown of the tooth, to prevent leakage of microorganisms and their toxins in any direction.

The main material of root canal obturation is gutta-percha and some sealer (Fig. 8). The root canal filling can be done with cold and warm gutta-percha techniques. In both cases the hermetic sealing and compacted homogeneity are the main criteria. If the root canal filling is completed, the coronal end is protected from microorganism invasion by glass ionomer liner (Fig. 9).
Completed root canal obturation (tooth 27)

Glass ionomer liner over the root canal filling (tooth 27)

The root canal filling is checked by X-ray. With a final restoration, the tooth fulfils its role in mastication, in phonation and meets aesthetic needs of the patient (Fig. 10).
X-ray taken after root canal filling of tooth 35 with internal resorption restored with prefabricated post (Radix Anker) and composite resin build-up

**Coronal restoration of a root canal treated tooth**

Due to root canal treatment, the tooth becomes weak and susceptible to fracture. This is not due to desiccation because of the lack of circulation. There is no significant difference in the amount of water in the tissues of a tooth root canal treated years ago or a vital tooth. Susceptibility to fracture is because of the loss of hard tissue due to caries and root canal therapy; just think of taking away the whole roof the pulp chamber, for example. With the loss of the pulpal tissue, a protective function is lost, which prevented the tooth from overload (sensory role of the pulp).

The final restoration planned to bind to remaining hard tissue should meet functional and aesthetic criteria, and must protect the weakened tooth from fracture.

Final restoration covering all cusps may be made of amalgam or composite resins.

Onlays, crowns are prepared using indirect method with impressions. Preparation of gold restorations sacrifice less tooth material, but if needed, the restoration should meet aesthetic criteria. By covering the whole occlusal surface, the possibility of fracture is reduced as masticatory forces are evenly dispersed. To reduce the possibility of fracture, the crown restoration must grasp the remaining tooth structure at the cervix in a minimum 2 mm wide band (ferrule effect).

These are criteria even if the tooth is so destructed that it can only be restored by a post (Fig. 11). A post can be made of cast metal alloy as post/core foundation following impression, or of prefabricated carbon fibres, glass fibres, or nowadays zirconium post/core, made of aluminium-oxide. The post is protected by a crown as a final restoration with which the functional, phonetic and aesthetic needs of the patient are fulfilled.

Post restorations, crown

**FOLLOW-UP OF A RESTORED TOOTH**

We recall the patient for regular follow-up in case of any restoration done on vital tooth or on a root canal treated tooth. The clinical examination, the symptoms and the observations include the sensitivity check of the vital tooth and the examination of the periapical area of the root canal treated tooth (Fig. 12). The root canal treatment is successful if the tooth shows no symptoms, and there are no complaints for four years.
7. Prosthetic Dentistry (Anette Stájer DMD)

Prosthetic dentistry has been part of dentistry which deals with the treating of patients whose teeth are deficient or totally missing.

CONSEQUENCE OF TOOTH LOSS

One, two or more missing teeth inside the mouth are harmful for the functions which are ensured by the teeth. Mainly the masticatory system and the chewing capability will be disturbed. At the site of the missing teeth, the alveolus is involved because after extraction, the involution will be worsened when the edentulous part remains for a long time, since both the lost teeth are missing and the remained ones suffer negative alterations.

In case of lack of the neighbouring teeth, the remained ones are able to change their position, and the axis of the teeth can alter either by moving from the original place by tilting towards the vacant place, or by rotation around their axis.

In case the antagonist teeth are missing, they can elongately come out from the alveolus. Once the radicular part of the elongated teeth gets into the mouth cavity above the gingiva, caries can be developed easily on the root surface.

The dislodgement of the teeth force remain no equable when the direction of the force will be no parallel with the axis of the remained teeth, in consequence, some of the teeth or teeth-groups will be overloaded. Abrasion and attrition may appear on the remained teeth, which can be observed easily.

The tight contact between the neighbouring teeth will disintegrate, patients can feel changes: food retention, interdental papillary injury, and increased caries development. In consequence, they can cause gingivitis and periodontal destructions, which evolve on this area. These changes may cause both loosening of the teeth together with the final result of loss off further teeth. The contact points of the teeth also alter when early contact can be observed in them. In addition, traumatic occlusions appear, because contact points get to incorrect places on the teeth, which overloads the remained ones.

The missing teeth may lead to the reduction of occlusal vertical dimension, which promotes the increase in the development of incorrect contact surfaces, and in consequence, the situation becomes more severe, e.g. it may cause difficulties in chewing time, and it is harmful also for the temporomandibular joint system.

The alterations result in further changes, too. Patients cannot bite and chew well, they have to swallow bigger mouthfuls. In consequence, gastric and digestion problems, as well as other additional diseases may appear, and malnutrition can develop. Alterations in the temporomandibular joint occur because of an inappropriate loading, pain, clicking and restricted mouth-opening and functional disorders may evolve, too.
The earliest complaints, with which patients arrive to the surgery, are aesthetic problems (the tooth-loss is in the visible part of the mouth). Even the lack of a single tooth (mainly in the frontal part of the mouth) may interfere with the patients’ daily routine and social activity. When more teeth are missing from the front-part, however, the patient looks older. The distance between the nose and the chin shortens, in the total edentulous cases the muscles around the mouth lose their tonicity, and wrinkles would be deeper. All of these changes can lead to the development of psychic problems and deterioration in the quality of life.

Last but not least, missing teeth may hinder phonation, thus, the speech will be slurred or incomprehensible. Patients can form certain phonemes only at out of their ordinary site leading to problems in phonation. It may result in social isolation and introverted behaviour. The consequence of tooth loss is shown in Figures 5.82. and 5.83.

Consequence of tooth loss - deepening of the bite
Consequence of tooth loss - deepening of the bite

CLASSIFICATION OF THE DENTURES

Dentures can be divided in two groups: fixed and removable dentures.

Fixed dentures

The crown

The complete crown is a fixed appliance which covers the suitably prepared surface of the teeth, cast and posts or implants, and it does not take up more space in the mouth than the natural teeth.

To make different functional crowns, the original form of the teeth needs to be changed to be suitable for it. This so-called tooth preparation is the first important step in building crowns. In case of sensitive teeth, the intervention is painful, thus, the dentist should give anaesthesia for the patient. The form of the natural teeth may be barrel-like, when the biggest outline of the clinical crown, which is the most prominent part of the axial walls, is situated between the half of the tooth and the marginal gingiva. This is the anatomical equator. During the preparation, the largest diameter of the tooth will be where the edge of the crown is in the future. From this part the convergence to the occlusal surface is about 5–8 ° compared to the axis. The retention of the crown would be worse if the axial walls are tapered even more. To prepare a crown, we need to make at least a 3 mm tooth in height, and from all of the surface, we has to remove at least 0.5–1.5 mm depending on the type of the crown. It is essential that we form the wall of the crowns to minimal thickness.

The margin of the crown (that is the margin of the gingival part of the crown prepared) may end supragingivally, paragingivally or subgingivally. Different methods of preparation are possible, and in every case, the dentist decides about which preparation to apply, and it is the dentist who has to decide on basis of the different criteria in each patient, which crown and preparation method would be applied.

Classifications of crowns:

1. according to the odontotechnological preparation methods
2. according to the materials used
3. according to their application
1. The oldest used crown-types were the swaged (strained) crowns prepared from sheets by cold working (made from stainless steel mainly, sometimes from gold-alloys).

In its preparation, only a small part of the tooth would be removed. Pulpal lesions occurred rarely because only a little part of the enamel was removed. Sometimes, we can still find this crown in the patients’ mouth, but it is considered only a historical curiosity nowadays.

The other sort of crowns which is not used any more nowadays is the two-part crown. This form was more advantageous, and its contact area was more precise than the swaged crowns. The axial wall and the top of the crown were prepared from two parts, and then they were soldered together. Unfortunately, corrosion might develop between the two parts.

For our patients, nowadays, we use widely the third type of crown: the cast crown. The details about the cast crown is reviewed in the next section.

2. Crowns sorted by materials: cast metal crowns, metal-free crowns (full ceramic crown, full acrylic resin crown, fibre reinforced crown, zirconium-(di)oxide crown, aluminum oxide crown) or combined crowns: metal–ceramic or metal–acrylic resin crowns.

Cast metal crowns are very rare nowadays, usually the patients require it at non-visible sites in the mouth. The possible material is precious or non-precious metal. The earlier crowns have been made from platinum–gold alloy or sometimes from silver–palladium alloy. From the metals which contain no precious metal, the nickel–chromium alloy was applied a few years ago, but currently, because of nickel allergy, it is not widely used. From the full metal cast crowns and metal basic crowns, the cobalt–chromium alloy is very popular nowadays. The combined crowns are also wide-spread because they can provide good aesthetic view in the visible parts. The application of resin veneer restorations had been popular before the metal–ceramic technique was fully developed. Due to the problems with wear and discoloration of the polymethyl methacrylate, the field of indication of the veneer is limited. To cover the occlusal surface during the tenure, the acrylic part will be worn-out. Another problem is with the transparency, as it is not possible to reach the same effect as with the natural teeth.

In brief, metal fused ceramic crowns should give perfect aesthetic appearance, and there is no change in the long-term look (Fig. 5.84.).

![Metal used ceramic crown (from P. Vályi)](image)
only temporarily because they can break easily, and they are worn quickly. In consequence, the material of the acrylic crowns is only a little bit elastic, which results in that the cement used for fixing it is fractured after the cementation. In addition, it can be dissolved from the gingival part, and plaque accumulation appears at these sites, which will cause gingivitis and periodontitis.

The other metal-free ceramic crowns provide natural appearance and more aesthetic effect than the metal fused ceramics because they have no metallic parts, so the metal framework will not be visible even at the gingival part. There will develop no galvanic effect in the metal-free crowns, which can be observed when different metals are in conducive fluid (so in saliva) and electricity is present between the metals. The first group of the metal-free ceramic crowns was prepared by individually baked platinum foil method, when the platinum foil was removed at the end of the procedure. The second group of the hardcore system of metal-free ceramic crowns are prepared on aluminum oxide or zirconium-oxide frameworks (Fig. 5.85.). The aesthetic feeling by this group is excellent, and patients may use them life long.

Metal-free porcelain crown - with zirconium-oxid framework

The third group is glass ceramic, which includes casted and pressed types of crowns. Glass ceramics are moulded as glass, and then treated by heat to produce crystals inside the glass structure. The crystalline particles protect the crowns from cracks in the material and increase the strength and hardness at the same time. The technicians paint and burn them to the suitable colour. Finally, let’s have a look at the newest technique, the so-called milling technique method. This procedure is known as the Computer Assisted Design/Computer Assisted Manufacturing (CAD-CAM) method. The crown is designed on a monitor screen with computer assistance, and a block of machinable ceramic is selected by shade. The restoration is milled from a ceramic block. This technology is known in several versions, but the innovation costs are very high.

3. When sorting the crowns by use, there are more functional possibilities: the single crown is not to connect to any other denture. The second group is the retainer crown, which assists for other dentures in keeping the denture at the right place. These are retainers for bridge or retainers for removable partial dentures which clasp to retained crowns, telescopic crowns, maybe they provide the fix parts of the precise attachment to partial dentures.

The use of minimally invasive techniques increases nowadays all over the world, one of them is the laminate veneer, which improves aesthetic appearance of the teeth by modifying their shape, colour or position.
Only the vestibular part is prepared; the dentist lutes the ceramic or acrylic laminate with dual cement or composite on the tooth surface. The depth of the preparation is only 0.5 mm, inside the enamel only. Indications are discoloured teeth, when the shade will not change after tooth whitening, modification of the shape, or position of the teeth (great space between teeth, short clinical crowns, developmental problem, or tooth-injury), caries, or damage of the clinical crown. In these cases, the filling does not solve aesthetic problems, but the dentist does not want make a crown yet.

The main steps of making crowns

At the first attendance, the dentist asks for the patient’s chief complaints and acute problems or the reason of the visit. Detailed history taking is very important including the general medical history, dental history and previous dental treatments. Extraoral and intraoral clinical examination and radiological findings are necessary to establish the diagnosis.

Every treatment can be started when the patient reports and gives information about the option of interventions, the costs of the denture, and finally, the dentist needs to ask for the patient’s agreement to the new treatment. First, if a study model is essential, the dentist takes study impressions from both dental arches. Besides, he/she needs to make wax-bitting, which can help register the position of the upper and lower arches (and inside the teeth). From the alginate impressions, the assistant can make casts, in several cases they mount the casts into the articulator, and this will help make the definitive plan for the dentist.

After all above mentioned things are performed, the treatment can begin (usually on the next occasion), when the first step will be the administration of anaesthesia. The dentist has to make previous impression about the intact teeth, which he will prepare later, after the preparation he needs to fabricate temporary crowns during the consulting hours. (The other procedure is that prefabricated celluloid or acrylic crowns will be adapted on the teeth prepared.) After that the dentist can begin the preparation of teeth according to the regulations (Fig. 5.86.).

Illustration of a prepared molar tooth

When no impression has been taken in the same sit, a provisional crown will be made for the prepared teeth based on the previous impression. The provisional cemented temporary crown gives protection against chemical, mechanical, thermal and biological effects, which act harmfully to the pulp. There are other respects which could be important to make a temporary crown: it helps prevent the movement of the prepared tooth, restore the occlusion, assure good aesthetic appearance, take part in the phonation, it may also help the regeneration of the periodontium, and may preserve the relation of the arches. After the provisional fixing of the temporary crowns, the treatment of the patient is finished until the next session.
On the next occasion, before taking the impression, the dentist displaces the marginal gingiva. This is necessary when the margin of the crown is prepared subgingivally because it indicates for the technicians to make the margin of crown precisely. After that a precision impression will be made from the prepared teeth, and an antagonist impression from the antagonist teeth as well (Fig. 5.87.), and the dentist also sends a new bite-registration to the laboratory.

**Precision impression**

In the laboratory, the technician can make the crown by means of the impression, cast and biting. When after trying the framework, the denture will be ready, and the dentist can fix it on the tooth, first temporarily, and then, after 1 to 2 weeks, the perfect crown can be permanently fixed (Fig. 5.88.).
Permanent cementation of the metal–ceramic crown

Dental posts

When the crown of the tooth cannot be restored because the part of the tooth does not reach the minimum height of 3 mm, it is not suitable for preparing the previous crowns. For this case, when we have a sufficiently strong root which has a correct root canal, or root canal treatment is available, the crown can be restored by using the pin superstructure. The root together with the root canal is prepared and lege artis, pin is inserted.

We can use different size of drills, and appropriate size of these pins can be used; the dentist can choose the right size taking into account the thickness of the root. Part of the finished pins is anchored to the root, while the other half extends into the mouth; this external shape and size is the same as the tooth’s shape and size prepared. The pin has to be cemented, and it can be prepared for the prosthesis.

The pins can be categorised in several ways, one is prepared directly in the dentist’s office or sent out to the technical lab, where the technician prepares and then sends it back to the office for cementing. Multiple types of materials can be used for posts, the metal types have been successfully used for several decades by dentists.
Nowadays, there is a growing use of metal-free post systems. Carbon-fibre pins are used with good results, but glass-fibre reinforced pin systems meet the most strict requirements and can be used for metal-free aesthetic restorations.

**Bridges**

In cases when one or more teeth are missing but the number and location of the remained teeth allows for preparing fixed restoration, the so-called bridge is prepared. A bridge is a fixed restoration used to replace a single missing tooth or several teeth by joining either an artificial tooth permanently or to adjacent teeth or dental implants and does not take up more space in the mouth than the original teeth. Bridges are permanently cemented in the mouth; hence, they can not be removed without damage to the fixed prostheses. The components of the bridge have the following parts: the prepared tooth or the dental implant called abutment, the retainer, which covered the prepared surfaces of the tooth and the part which replaces the missing teeth, the name of which is pontic bridge (Fig. 5.90.).
Bridges can be classified variously: according to the function of abutments, the location of the abutments, according to the solubility of the anchor, anchor types according to their extent, and according to their material. The abutment’s number and size and location within the jaw is very important. In accordance with Ante’s Law, the dimensions of the bridge are defined by the root surface area of the abutment teeth, which has to be equal or surpass that of the teeth being replaced with pontics.

The abutment teeth can be divided into different groups based on the extent which holds the bridges.

- primary abutments are the upper and lower canines and molars (except wisdom teeth) and the upper central incisors;
- secondary abutments are the lower and upper premolars, and
- third abutments are the lower incisors.

The lateral upper incisor can belong to the 2nd or 3rd type abutment depending on its development (well- or undeveloped).

Some factors modifies the choice of abutments: the periodontal status, tooth alignment, shape of the crown, size of the crown, arch size, upper or lower, the dimension and shape of the edentulous ridge, occlusal disorders, parafunctions, patient’s gender, age general health status and occupation.

The bridges require special procedures for cleaning, about which you can read in the chapter on “Prevention” (Chapter 4.1.6.).

Sometimes, there is no need to reshape the tooth, because there are prepared bridges in which the teeth should be minimally altered or not at all, so the treatment time in the next sit will be shortened. Several types of systems are also used, one of the oldest is the so-called Maryland-bridge (Fig. 5.91.), which is also used as an adhesive-bridge. In this case, we do not prepare the oral surface or if necessary, apply only minimal preparation and affix arms to the abutments, which will hold the artificial teeth (the material of the artificial tooth can be plastic or ceramic). This option is recommended if the pulp chamber is relatively wide, or we make teeth for
temporary purpose (such as in the recovery period after implantation because of aesthetic reason or as a placeholder after orthodontic treatment).

Fiber-reinforced composite Maryland bridge (from P. Vályi)

**Removable dentures**

Before permanent dentures, some words should be mentioned about the temporary removable dentures. Sometimes the dentist extracts one or two teeth, and it is impossible to wait for the healing with the lack of teeth in visible places for 3 months. During this time, the dentist and the technician can make clips from acrylate. The retainers are prepared from wire or acrylic clasps, and they have no good effect on the periodontium of the teeth, the appliance is recommended to be used for a short period (few months) only. The dentist has to change for permanent RPD (removable partial dentures) as soon as possible. The dentist takes impression all of teeth including the possibly removable teeth. The technician will prepare a cast about them, and he/she will remove the teeth from the cast which will be extracted later. Then he/she will make the temporary dentures. The dentist will put it inside the mouth in the surgery, where the first step is that the removable teeth will be extracted, and after that hemostasis is achieved, finally, the dentist tries and frames the denture into the mouth. This is advantageous for the patients to solve the aesthetic and functional problems; however, after a few months the dentures have to be change for a permanent one.

**Removable partial dentures (RPD)**

RPD is a dental device which restores one or more, but not all the natural teeth and associated structures (Fig. 5.92.) in the mouth. Its retention and support are provided by natural teeth (dental implants) and/or mucosa (and the bone under it). Its preparation requires more space in the mouth than the natural teeth, as the mucosa and the edentulous ridge are also covered. It is connected to the natural teeth, and the patient him-/herself can remove and place it back.

By making an RPD, our aim is to plan and construct a prosthesis that can resist dislodging forces. The prosthesis should not be moved away from its base either in rest or during function (stability in rest and in function). Our task is to compensate for the dislodging forces. Forces in vertical direction could be loading force – chewing, lifting force – weight of the upper denture, and sticky food. Forces in horizontal plane involve chewing.

Retention of the RPD means the fixation of the RPD against the forces that would provoke dislodgement or lifting the prosthesis from its place. The retention is characteristic for a denture which resists the outward displacement of the denture (away from the tissues).
It is very important that RPD supports the prosthesis, which comprises the transmission of vertical components of chewing force to the tissues of the mouth and the resistance of these tissues to this force. It involves the hard and soft tissues that bear the loads of mastication and clenching exerted on the denture.

The method of support is dental, mucosal, dentomucosal or mucodental. The number of the remained teeth, position of the remained teeth in the dental arch and bearing of the mucosa will determine which type of support dominates. Indications for RPD are long bounded saddle, free-end saddle; bad periodontal or endodontic status of teeth, if splinting is necessary, great atrophy/defect of the edentulous ridge, patient’s request, and young age (under 18 years), when later fixed denture or implant will be possible; general physical or mental status of the patient (tooth preparation procedure cannot be carried out); and temporary treatment before or after implantation.

Parts of the RPD are the base plate, which contains the saddle, major, minor connectors and occlusal rest; artificial teeth and retainers (clasp, precision attachment, bar attachment, telescopic/double crown, ball retention – any type of device used for the stabilization or retention of a prosthesis). These dentures are usually made from cobalt–chromium alloys or sometimes from gold alloy.

**Clasp retained removable partial denture**

In contrast of the wire and acrylic clasps which are not proper for teeth because they load the retainer teeth also in rest position, thus being harmful to the periodontium, and they may be the cause of tooth loosing; these cast clasps are rigid retainers. Many types of clasps are used by dentists, all of the clasps bodily encircle the abutment tooth in about 270°.

The Ney-system clasps are frequently used, and the Bonwill, Roach and ring clasps are also well-known. In Ney-systems we can distinguish between six types of clasps, five basic and one modified clasp. When the teeth are suitable for wearing a clasp (shape, size and position are suitable), it is not need to make a clasp with retained crown. The ideal holding tooth is free from caries or restorations, has favourably contoured crown, is undercut buccally, and it is not undercut (flat) lingually; the crown in adequate in length; the periodontal status is normal; there is a long root with large surface area; the vertical and horizontal position within the arch is good, and the opposing occlusion is stable.

The clasps retained RPD has also disadvantages: aesthetically it is not suitable for patients who have clasps on visible teeth (Fig.12).
The clasps retained RPD has also disadvantages

**Precision attached retained RPD**

The retention means higher stability, good function and more aesthetic appearance. This prosthesis always consists of a fixed and a removable part (Fig. 5.94.). The dentist can usually make this precision attachment in pairs inside one arch. Every precision attachment needs two prepared teeth with crowns, and the two crowns are connected to each other. The requirements for the preparation of precision attachments are healthy periodontal status and almost caries resistant teeth. In case of elongated teeth, the turning-moment will be higher, which may lead to easier tooth loss. You should always check the possibility for preparing parallel axis of the involved teeth! Another precise retainer can be the bar-retention, which requires at least two teeth for stabilising the bar. This part of RPD will be luted with two crowns. These precision dentures ensure aesthetic appearance in which, despite the removable part, the metal part is not visible during speaking and smiling.

![Fixed and removable parts of the Precision attachment](image)

**RPD retained by telescopic (double) crown system** (Fig. 5.95)

Telescopic crown is a tool for dentally supported and retained RPD. This is an individually constructed double crown which fits accurately. It consists of two parts: primary and secondary telescope crowns. The primary crown is luted on the surface of a prepared tooth, the secondary crown can be found inside the RPD.
The mechanism of the retention is assured by friction of the primary and secondary crowns. By very precise technical work, the friction force has to be 5–10 Newton between these two crowns. The technician constructs the primary crown by fraser-technology, finally, the wall of the primary and secondary crowns become parallel. The well adapted telescopic retained RPD can be fixed to the base of the denture when the alveolar bone does not move during mastication; however, by removal from the mouth, the connection (friction force) can be solved easily.

The advantage of RPD is that the axial load direction is central, so the periodontium is protected. At the same time, it is disadvantageous, however, to remove a large amount from the surface of tooth because the double crown and the artificial teeth will be bulky which is not aesthetic. When the friction force is higher than 10 N, the periodontium of the remained teeth has to be impaired or the primary crown luting on the tooth will be removed from the right place. On other occasions, when the friction force is not strong enough, the retention will not be sufficient to keep the RPD in the right place.

In subtotal cases (e.g. the patient has 1 or 2 remained teeth), a type of the telescopic RPD, the so-called overdenture is recommended (Fig. 5.96.). This has the advantage over the total denture that the functional stability is higher, and the patient can chew better with this RPD, and the load on the mucosa–alveolar bone is smaller. The patient adapts RPD more easily than the totally denture, and later this RPD prosthesis helps patients in getting used to the total denture more easily.
Lower overdenture

**Removable complete denture**

Complete denture is an artificial device which restores and maintains the oral function by replacement of missing teeth and other structures (mainly the atrophied alveolar bones). The denture space is part of the edentulous oral cavity, which is bordered by the internal part of the cheeks, tongue and residual ridge, formerly filled with teeth and supporting structures. This portion of the oral cavity is available for dentures.

The denture can stay in the right place of the mouth by **adhesion**, which is a binding forces exerted by molecules of unlike substances prove (surfaces) contact in a humid medium in the mouth (for instance saliva), where the adhesion is larger. **Gravity** is favourable only for the mandibular denture. **Vacuum** is created between the denture and tissues in the mouth allowing negative atmospheric pressure to squeeze the denture to the oral mucosa. The surrounding soft tissues, muscles and the proper occlusion which can correct smaller movements of the denture are also important factors in the maintenance of stability.

**Parts** of a complete denture are the base plate, artificial gingiva and artificial teeth.

Forms of the maxillary arch are round square off, most commonly has shape of the letter U, half ellipsoidal or V shaped (referred to as gothic) roof of the mouth.

The height of the alveolar ridge can be either in a good retained state, or it can be also in a resorbed state. The reason of deviation can be that resorption between the upper and lower jaws is different, and it can be also different in case of the same jaw. The surface is covered with tightly attached mucosa. The resorption in the upper jaw starts from the outer surface, and it continues to the inner surfaces, it is the opposite in the mandible so it starts from the internal part and becomes continuous to the external parts. The posterior border of the maxillary denture extends between the nose-blowing line and the vibrating line. The first (nose-blowing) means the blowing from the patient nose while we hold it closed, we can observe the soft palate moving down and forward simultaneously. In the other case, we can detect when the patient says "Ah!," when the soft palate moves up and backward. In this way, the dentist must know the exact border lines of the upper and lower dentures because it is the key for preparing good and well functioning devices.

Nowadays these dentures can be left in the patient’s mouth up to 24 hours daily. For these dentures the patient sometimes applies adhesive creams for the feeling of comfort and confidence.
On the removal of the denture, it needs cleaning, it is important to brush the denture both outside and the internal surfaces with tooth- or nailbrush. The cleaning of the oral cavity is also important, hence the patient should be well instructed and motivated about the oral hygiene. Considering the mandibular and maxillary bone loss, the dentist has to underline the dentures to supply the correct stability and replace the missing bone structure. The patient should be asked to return annually, and these changes should be corrected at the dentist’s responsibility (Fig. 5.97.).

![Upper and lower total denture](image)

**Implant prosthetics**

Implant prosthodontics started to appear in the 1980s in Hungary, and since that time, it has widely spread and become a commonly used method. The essence of the procedure is that a Ti screw (resembling a tooth root) is placed into the jawbone, and it supports crowns, bridges and generally other structures.

The main indications for the application of prosthetic implants are one or more missing teeth, when the patient wants to avoid the preparation of adjacent teeth (Fig. 5.99.); a posterior edentulous ridge without implants only when removable partial dentures can be prepared; a completely edentulous jaw: an implant-retained overdenture for a better stability (Fig. 5.98.) (retention) of the denture, or in patients who are not confident with the stability of their denture, and they want to improve it with dental implants.
In every case, it requires a detailed plan of the prosthodontic treatment, which includes the placement of the implants in the bone, for the achievement of an aesthetic (end) result, which will function for a long period of time. For this purpose, the abutment prepared of titanium or zirkonium will be attached to the upper part of artificial titanium root. These abutments support the crowns, bridges (the structures that are visible) placed into the oral cavity. The so-called cervix, which is the upper part of the implant, is a transmucosal part, and it is located at the level of the gums. The surface of the upper portion of the root portion needs to be smooth, having a polished surface because gingival attachment can develop only on a surface like that. For further information on the professional cleaning of dental implants, see Chapter 4.1.7.
8. Treatment of inflammation and cysts of head and neck (Csaba Berkovics DMD)

TREATMENT OF THE INFLAMMATIONS OF THE HEAD AND NECK REGION

The ultimate aim of the treatment is to eliminate the cause. As the causative agent maintains the inflammation, healing is only possible when it is not present anymore. Should the inflammatory process (as a reaction to the causative agent) pose a threat to the body in itself, elimination of the inflammation must be first priority followed by the causative therapy.

Symptomatic treatment

Strong acute or hyperacute inflammations endanger the body regardless of the cause. For instance, an anaphylactic reaction with circulatory collapse can cost the patient’s life. In such a scenario, the first thing to do is to call the ambulance, so that the hospital can admit the patient as soon as possible. It is not a rule that the anaphylactic reaction reaches this stage, therefore, its treatment is graded. The mildest manifestation is urticaria (hives), which is treated orally with calcium-containing solutions, in the form of an effervescent tablet (in a glass of water), or in intramuscular injection (Calcimusc injection). If more severe symptoms occur, the venous introduction of antihistamines and steroids may become necessary. If the process develops rapidly, and/or the aforementioned medications cannot exert their effect, one must expect circulatory collapse and give adrenalin either in the form of an intravenous or intramuscular injection. Respiratory failure due to laryngeal oedema indicates conicotomy on the spot. Apart from these emergency cases, the symptoms of acute inflammation are routinely alleviated by NSAIDs, mostly diclofenac and ibuprofen (brand names may vary by country).

TREATMENT OF ODONTOGENIC INFLAMMATIONS

In the case of physical and chemical irritants, the removal/neutralisation of the irritant usually solves the problem, and healing can happen per vias naturales. In infections, it is the site and spread of the infection that determines the treatment plan.

As it has been mentioned before, in acute periapical periodontitis, the microbes break out into the periapical area through the foramen apicale. As long as they are confined here, endodontic treatment is possible, as the periapical tissues are anatomically intact, and the immune system reaches the infection site. Debridement and a root filling cut the way of further microbes. If the tooth is decayed to a considerable degree, extraction is advisable. If the infection has reached the bone or bone marrow, the cure of these structures must come first, and the tooth itself is dealt with only later. Infiltration of the surrounding soft tissues may heal spontaneously, but it can grow or lead to abscess formation. The outcome depends on the strength of the immune system and the severity of the infection.

In case of submucous and subperiosteal abscess formation, the abscess must be opened and its contents drained. It is done by making an incision in the oral mucosa or the skin of the face, depending on the location of the abscess. The cavity of the abscess is lavaged with desinfectant solution (H2O2, Betadine, etc.), and the incision opening is drained (with iodoform gauze in the oral cavity, and with a rubber drain from the face). Until healing, the cavity must be lavaged every day! As the abscess is usually caused by anaerobic bacterium species, they are most often killed simply by contact with air. If these bacteria do not form an abscess, the problem is more serious, as in such cases they usually spread in a phlegmonous manner, which is a life-threatening situation that requires hospital treatment. In these cases, the affected spatium (intermuscular space) is opened, sometimes with several incisions, lavaged, drained, and specific antibiotic treatment is started (after culturing).

Acute osteomyelitis also indicates hospital treatment. Until the results of culturing arrive, broad-spectrum antibiotics are applied, which, with the result in hand, can be changed to specific ones. Pus drainage is promoted by the extraction of the affected tooth. Extraction of the neighbouring teeth may become necessary. When the crown is decayed or (in teeth with more roots), there is a large osteolytic process involving the furcation, extraction is indicated.

In an optimal case, however, endodontic treatment leads to healing ad integrum, and the bone of the periapical area is regenerated. In most of the cases, even the fistulae disappear.

Should any microorganisms persist (for instance, in the small canals of the apical ramification), healing cannot take place in spite of the root canal treatment. Such a scenario indicates apical resection. Apical resection is
done as follows: after incision, the mucous membrane and the periosteum are lifted off the bone together. Periapical bone is removed with a drill, and then 3–4 mm of the apex is excised. Curettage is performed to remove inflamed tissues and debris, and, if necessary, retrograde root canal filling is done. The operative wound is closed with sutures. The measure of success is ultimately the lack of symptoms half a year following the intervention, regardless the bone or connective tissue occupies the periapical area or not.

Pericoronitis

It may develop in the dental sac of erupting teeth (mostly impacted wisdom teeth), or in the deep pockets around semi-erupted teeth. Disinfection is done with disinfectant wash (Hydrogen-peroxide, Betadine, Chlorhexidine). Iodoform gauze is also applied. The infection can spread in multiple directions but mostly toward the spatiun pterygomandibulare, where it causes a perimandibular abscess. In this case the patient complains of difficulty swallowing. The abscess must be opened, and antibiotic therapy should be started. Once the inflammation is ceased, it should be considered whether the affected tooth will ever erupt into normal occlusion (by which it is not an inflammatory focus anymore). If not, either the dental sac must be surgically narrowed or the tooth must be extracted.

TREATMENT OF CYSTS

A main principle is that cysts must be removed and sent for histological examination. It is only the histological results on the basis of which a lesion can be diagnosed as a cyst.

Given the different sizes and localisations of cysts, different techniques are used to treat them.

Oral cystostomy (Partsch I.)

The aim of the intervention is to transform the cavity of the cyst into an additional sinus of the oral cavity or sometimes the maxillary or paranasal sinuses. This can be done with the total or partial excochleation of the cyst epithelium. This intervention stops the growth of the cyst; however, the regeneration of the bony cavity takes longer than in case of total cystectomy. This technique does not restore the original anatomical relations, but the result may be satisfactory both functionally and morphologically. Although it is easy to perform, and recurrences are rare, long post-treatment is necessary to facilitate the epithelialisation of the newly formed cavity. If the cyst is large, sometimes the application of an obturator (in itself or combined with a tooth prosthesis) is necessary to keep the stoma open.

Decompression is a preparatory operation of cystostomy. The cyst is opened and an obturator is inserted. This way, the internal pressure drops, bone regeneration starts and the cavity shrinks. Finally, the shrunk cyst can be removed entirely (the process is monitored with X-ray).

Cystectomy (Partsch II.)

The aim is the total removal of the cyst.

This may be contraindicated if:

• the cyst is larger than 2 cm in diameter, -
• there is a risk of injury to the surrounding structures (vessels, nerves, the mucous membrane of the sinuses, or permanent teeth in development),
• the cyst is inflamed. Radicular cysts often get infected through the roots, causing periapical inflammation.
• the cyst is a follicular one, around a tooth delayed in eruption (provided the goal is the eruption of the given tooth, e.g. an upper canine).

Healing of the cavity can be fostered by the insertion of tissue-friendly materials and also by bone augmentation.

Antral cystostomy

Large cysts can be united with the maxillary sinus.
9. Maxillo-facialis traumatology (Dr. Raskó Zoltán)

It is essential to restore the appropriate preinjury occlusion – so called mandibulo-maxillary fixation (MMF) - in both upper and lower jaw fractures. Two methods can be used: one of them is when a dental splint is fixed with a thin wire running between the teeth of the jaws, then these two splints are fixed to each other with wire loops or gummies after setting the occlusion precisely. The other option to perform MMF is with special screws that are put in the bony area between the canine and first premolar, then fixed to each other with wire loops after setting the occlusion. Sometimes pre-drilling is necessary, but modern screws have self-drilling and self-tapping features, so a single screw driver is enough for their implantation.

TREATMENT OF MANDIBULAR FRACTURES

Basic criteria have to meet for the physiological healing of maxillofacial bones, just like everywhere else in the human body. One of these basic conditions is biological: blood supply. Maxillofacial bones have periosteal and intraosseal blood supply. The other criterion is stability secured by adequate osteosynthesis. Last but not least, micromovement between the fragments is important; because this way osteoclasts and osteoblasts are stimulated to build new bony substance.

It is an interesting historical fact that a few decades ago mandibular fractures were treated exclusively with the extraoral approach, since the intraoral exploration was unimaginable considering appropriate visual field and access. Besides, stout and strong compression plates were used for stabilisation. The intraoral approach was introduced in the 1970s. The basics of maxillofacial biomechanics were laid by discovering the tension and compression forces generated by sublingual and masticatory muscles surrounding the mandible. It has been proven that miniplates affixed in the appropriate position on the outer bony surface of the mandible fixed with monocortical screws can assure enough stability for the fragments, and can create self compression that can help bone healing.

Incision has to be made in the area of the vestibular mucosal layer; the bony surface is reached by cutting through the periosteum. Haematoma and occasionally entrapped soft tissues have to be removed from the fracture gap. After adjusting precise occlusion, fractured ends of the mandible have to be restored in the anatomical position. Sometimes not all fragments can be put back in their normal anatomical situation caused by a difficult fracture pattern, so the prime directive is precise adjustment of the occlusion. In this case you can rely on the regeneration ability of the body – so called remodelling in case of bones. This means that bony substance is formed to restore the anatomical situation even in case of non-anatomical unification if the fracture is stable enough.

The next step is stabilisation; for this purpose miniplates are used. Application of two miniplates is necessary in the frontal and premolar region of the mandible, in accordance with biomechanical principles. Since tension and compression forces generated in the bone running from backward to forward turn into a twisting force in the premolar areas, if you used only one miniplate, it would become loose because of the twisting forces, and would lose its function, so it could not stabilise the fracture. The incision has to be made at the area behind the third molar tooth above the oblique line. Occlusion has to be adjusted first. Application of only one miniplate of the appropriate length is enough in this region, because compression is generated by the forces at the base of the angular zone facilitating bone healing. It is important to mention the teeth in the fracture line. According to earlier approaches, these teeth had to be removed. Fortunately this theory has been disproved by now. Nowadays the goal is to save every living, healthy and stable teeth if they do not interfere with the anatomical reduction of the fracture. Every teeth in the fracture line is in contact with the oral cavity, so every fracture with a tooth in the fracture line has to be considered open fracture, therefore primarily intravenous antibiotics have to be administered to the patient.

Treatment of condylar fractures is subject of serious professional discussions even today. It has been stated that every condylar process fracture can be treated conservatively. Fractures can heal with the application of mandibulo-maxillary fixation. However, it does matter whether the patient is going to have an appropriate mouth opening, deviation of the chin or pain persisting in the region of the injury.

The question is when a condylar process fracture should be operated. Absolutely conservative treatment is accepted in childhood. If the fracture has occurred inside the joint capsule, conservative treatment is to be chosen.

There are countless criteria for operative conditions. A few essential conditions have to be emphasised. A fracture has to be treated surgically, if conservative methods have not been successful. An operation has to be
performed if mandibulo-maxillary fixation is contraindicated; like in epilepsy aspiration can occur in case of an attack. Other problem worth mentioning is anorexia and bulimia, when appropriate nutrition of the patient would be the aim, but it cannot be realised through a closed mouth.

Position of the fractured ends determines the indication of surgery. Operative reduction is necessary when more than 5 millimetres of compression and/or more than 40 degree of angular dislocation is observed. If the fragment is seriously dislocated laterally or above the middle scale, operation is needed.

Surgical exploration is performed in the retro/subangular region. Miniplate osteosynthesis is performed after anatomical reduction of the fragments besides controlling occlusion. If the patient has a high condylar process fracture, exploration can be done via a preauricular incision. Bone fragments can be reached directly under the skin. Stabilisation is done with miniplates after anatomical reduction.

Damage to the facial nerve always has to be considered in case of condylar process fracture treatment, so a high level of experience is required for the treatment of these injuries.

Intravenous antibiotic administration is not necessary in case of condylar fracture treatment, as these operations are performed from the sterile extraoral approach.

A new method has to be mentioned: intraorally performed endoscopically assisted reconstruction; the anatomical restoration is made through the utilisation of a mini camera. Danger of facial nerve injury is minimal, but high-cost instrumental background and greater practice or experience is required.

**TREATMENT OF THE MIDFACE FRACTURES**

The technique of the treatment is determined by the type of the fracture. The main task is the adjustment and stabilisation of the occlusion in case of fractures affecting the occlusion. After a mucosal incision is made in the upper vestibular area, anatomical reduction of the fragments is performed, and fractures can be stabilised with miniplates.

In case of fractures not affecting the occlusion – zygomatic fracture, injuries of the orbit, fractures of the frontal bone – the closing of the jaws is not necessary.

Dislocated zygomatic fractures can be reduced with a bone hook inserted through the skin – at the point two lines cross: a horizontal line at the base of the nasal base and a vertical line at the lateral canthus. After inserting the hook at this point, it has to be pulled with a continuous force for the reduction of the fragment while checking the state of the infraorbital rim. Arrival of the fragment at its anatomical position is indicated by a clicking sound.

Another method is the Gillis manoeuvre. Incision at the temporal area is made, then a superficial layer of the temporal fascia is cut through. Since the superficial fascia layer is attached to the zygomatic arch, if we penetrate with a special elevator under this layer, the anatomical reduction of the zygomatic complex can be performed.

If the reduced fragment is stable in its anatomical position, the operation is successful.

Reduction of unstable zygomatic fractures can be performed by several approaches.

Intraoral approach: mucosal incision at the area of the upper first and second molars is made, after anatomical reduction the fracture is stabilised with the help of miniplates applied on the zygomatico-maxillary pillar.

Extraoral approach: incision is made at the lateral edge of the eyebrow. After exploration of the fragments, a bone hook is used to pull them into the anatomical position. Miniplate osteosynthesis is performed after anatomical reduction.

In case of the reduction of orbital injuries infraorbital, subciliar or transconjunctival incisions can be performed for exploration. The aim of the intervention is to release entrapped soft tissues from the fracture line and reconstruct the thin bony wall. Success of the operation is justified by checking full and free eye movements.

Restoration of fractures of the upper third or the frontal bone is performed through a bicoronal incision. The frontal bone, the upper rim of the orbit, the nasal bone and the zygomatic arch can be visualised by this exploration, fractures can be stabilised safely, without the danger of nerve injury.
POSTOPERATIVE CARE AND FOLLOW-UP

Radiological examination has to be performed postoperatively to ascertain the position of the fragments and stabilising materials. Repeated CT scan can be necessary to check difficult fracture patterns.

Maintaining good intraoral hygiene is of high importance in case of fractures affecting occlusion; precise instructions have to be given to the patient.

Although appropriate adaptation as well as movement and loading stability is ensured by miniplate osteosynthesis, full loading is not available, therefore a liquid-pulpy or soft diet has to be prescribed for the patient. The solidity of the food does not affect the stability of condylar process fractures, so the patient can choose the solidity of the food according to the degree of pain.

If mandibulo-maxillary fixation is applied for a longer period, we have to keep in mind that the limitation of joint movement will reduce over as much time as it was fixed. Appropriate movements, calisthenics have to be shown to the patient, and if necessary, the guidance of a physiotherapist or physiotherapeutic management has to be offered.

Intravenous antibiotic administration can be changed to oral preparations on the 3rd or 4th postoperative day, and it can be finished at the time of stitch removal – 7-10 days – if signs of inflammation are not observed. Regular follow-up is very important.

Occlusion and wound healing always have to be checked at the time of these examinations, so that accidental complications can be avoided or treated in time.

10. Head and neck cancer (Róbert Paczona MD - József Piffkó MD, DMD)

The worldwide incidence of head and neck cancer exceeds half a million cases annually, ranking it as the fifth most common cancer worldwide. Head and neck cancer describes a range of tumours that arise in the head and neck region, which includes the oral cavity, pharynx, larynx, nasal cavity, paranasal sinuses, thyroid, and salivary glands. Most commonly “head and neck cancer” refers to epithelial squamous cell carcinomas that arise from the mucosal surfaces of the upper aero-digestive tract. However, the impact of this disease on society is not measured solely by its relatively low absolute mortality, but also by the acute and chronic cosmetic, functional, and psychological morbidities experienced by all patients. Head and neck cancer thus remains a feared disease associated with significant rates of both morbidity and mortality.

PRINCIPLES OF TREATMENT

Once a patient’s primary diagnosis is established and initial staging studies are completed, a tumour board decision should be obtained. The application of modern multidisciplinary treatments requires early input for all potential care providers. A team approach to care is clearly superior, that team commonly includes a head and neck surgeon, medical and radiological oncologist, a nutritionist, a social worker, a psychiatrist, and maxillofacial prosthodontist, among others.

Primary tumour treatment

Head and neck cancers differ greatly in local–regional stage and primary site. In general, all patients with untreated local–regional head and neck cancer are considered potentially curable if there is no evidence for distant metastatic disease.

Resectable tumours

Treatment decisions begin with an understanding of primary site extent and a decision as to whether the lesion is resectable (removable with a high probability of negative surgical margins). If a lesion is resectable, careful consideration must be given to the specific morbidity that would attend curative surgical resection. With few exceptions, primary lesions that are both resectable and operable (resectable with acceptable morbidity) benefit from primary surgical resection.
Primary site preservation is now a well-established goal in multidisciplinary treatment programs designed to avoid relatively morbid procedures such as orbital exenterations in case of paranasal sinus tumours or total laryngectomy in laryngeal or hypopharyngeal cancers.

When surgery is necessary, procedures that preserve function are often possible. An experience with transoral laser and robotic surgery of pharyngeal and laryngeal carcinomas is evolving with decreased functional morbidity. When surgery is ablative of significant function or cosmetics, modern reconstructive techniques can be restorative.

The use of microvascular free tissue transfer has allowed significant strides in functional reconstruction with acceptable cosmetic appearance. These techniques have allowed for highly successful reconstruction of mandible and pharyngo-oesophageal defects, as well as the management of wounds previously felt to be irreparable.

Unresectable tumours

Lesions that are classically unresectable, or in which primary surgery would have high-associated morbidity are offered primary chemotherapy/radiation for cure.

**Neck lymph node metastasis treatment**

Regional nodal disease from head and neck cancers is strategically approached as a separate entity. With rare exception, all patients with defined nodal disease should receive radiation therapy either before or after surgical resection of the involved nodes. Traditionally, patients underwent surgical resections of the neck when a nodal disease was advanced and not fixed to underlying structures.

**LIP CANCERS**

Malignant tumours of the lips are almost invariably squamous cell carcinomas and most commonly affect the lower lip (approximately 90% of cases). They occur predominantly in pipe smokers. Prolonged, intense sun exposure is considered as a co-factor.

**Symptoms**

Early tumours often appear clinically as “intractable” ulcerations in the vermilion border of the lip but may also consist of large, exophytic lesions. Whenever a tumour is suspected, a biopsy should be taken to confirm the diagnosis. Differential diagnosis: Differentiation is mainly required from keratoacanthoma and a primary syphilis chancre. Basal cell carcinoma involves the vermilion border of the lip only by secondary spread.

**Treatment**

The treatment of choice is almost always surgical excision followed by a local primary closure or plastic repair of the defect using various reconstructive techniques. As a rule, even extensive tissue defects can be repaired using regional flap techniques. Carcinomas of the lip have an inherently low rate of metastasis to regional lymph nodes, but a neck dissection should be performed in patients with more advanced tumours.

**ORAL CAVITY TUMOURS**

Squamous cell carcinomas also predominate in the oral mucosa and are variable in their clinical appearance. Approximately 90% of patients have a long history of nicotine and alcohol abuse.

**Symptoms**

Symptoms vary with the location and extent of the tumour, and may consist of painful swallowing, blood-tinged saliva, and a fetid breath odour. Some tumours are completely asymptomatic, however.

**Diagnosis**

Visual inspection can raise the suspicion of a malignant neoplasm. This should be followed by bimanual palpation, since many tumours infiltrate deeper tissues and the visual impression of superficial findings can be misleading. The clinical examination also includes palpation of the regional cervical lymph nodes to exclude metastases. Imaging procedures (ultrasound, computed tomography, magnetic resonance imaging) are generally necessary only for extensive masses, as many tumours can be adequately evaluated clinically owing to their...
exposed location. But with more advanced lesions, imaging is valuable for defining the depth of the tumour infiltration and assessing the involvement of adjacent structures (bone). It is also an important tool for excluding regional cervical lymph node metastases.

**Treatment**

The treatment of choice in most cases is surgical removal of the primary tumour. The resulting defect is either closed primarily or reconstructed using pedicled flaps or microvascular free transfers (e.g. a radial forearm flap). A unilateral or bilateral neck dissection may be necessary depending on the location and size of the primary tumour. Radiation to the tumour site and lymph areas is frequently indicated following surgery. Primary radiotherapy or combined radio/chemotherapy may be considered as alternatives for more advanced tumours.

**Prognosis**

The prognosis of oral malignancies depends on the location and stage of the disease. The five-year survival rate varies accordingly, ranging from 0% to 80%.

**PHARYNGEAL CANCERS**

**Epsypharyngeal cancers**

Carcinomas of squamous cell origin account for the great majority of malignant nasopharyngeal tumours. A basic distinction is drawn between squamous cell carcinomas and lymphoepithelial carcinomas. Much less common tumours of this region are adenocarcinomas, adenoid cystic carcinomas, malignant melanomas, sarcomas, lymphomas, and plasmacytomas. The Epstein–Barr virus (EBV) appears to have a key role in the aetiology of undifferentiated lymphoepithelial carcinoma.

**Symptoms**

Early symptoms of nasopharyngeal malignancies are unilateral conductive hearing loss with middle ear effusion. Any persistent middle ear effusion of long duration in an adult patient with no prior history of middle ear disease is suspicious for being a tumour and should be investigated accordingly. Cervical lymph node metastasis, usually involving the nodes at the mandibular angle, is another common initial finding. Features of advanced disease include nasal airway obstruction, recurrent epistaxis, headaches, and cranial nerve palsies.

**Diagnosis**

The primary study is endoscopy of the nasopharynx, nasopharyngeal malignancies can have a variety of appearances ranging from a smooth, well-circumscribed tumour surface to mucosal ulcerations. Some of these tumours are initially submucosal and are easily missed at endoscopy. Otomicroscopy reveals unilateral tympanic membrane retraction and a middle ear effusion as a result of impaired Eustachian tube ventilation. Given the EBV association of many nasopharyngeal cancers, the EBV antibody titre should be determined (this shows an elevated IgA, contrasting with the elevated IgM/ IgG that is found in infectious mononucleosis). MRI or CT is useful for defining the tumour extent.

**Treatment**

The treatment of choice for most nasopharyngeal carcinomas is primary high-voltage radiotherapy, because most of these tumours are very radiosensitive and the unfavourable tumour location and rapid invasion of the skull base preclude curative surgery in many cases.

**Oropharyngeal cancers**

The overwhelming majority of malignant tumours of the oropharynx are squamous cell carcinomas, and approximately 80% are located in the palatine tonsils or tongue base. Less common sites are the soft palate and posterior wall of the pharynx.

**Aetiology**

In most patients, chronic nicotine and alcohol abuse have a major etiologic role in the development of oropharyngeal cancers.

**Symptoms**
Cancers at some sites in the oropharynx may remain clinically silent for some time. Otherwise the symptoms depend on the location and extent of the tumour. Besides dysphagia and odynophagia, the symptoms may include blood tinged saliva and a fetid breath odour. Advanced stages often produce trismus, signifying that the tumour has invaded the surrounding musculature (pterygoid muscles).

**Diagnosis**

Tonsillar carcinomas may appear as exophytic lesions or may show an ulcerating, infiltrating type of growth. Occasionally they are not grossly visible (microcarcinomas of the tonsils), and the first presenting symptom of the disease is cervical lymph node metastasis. CT and MRI are useful in defining the extent of tumour growth and detecting the invasion of surrounding structures.

**Treatment**

The treatment of choice for most cases is surgical tumour removal. The resulting tissue defect may be closed primarily with local pedicled flaps or by using microvascular free tissue transfers, depending on the size and location of the defect. A neck dissection may be necessary on one or both sides, depending on the location and stage of the primary tumour. Postoperatively, radiation should usually be delivered to the tumour site and lymphatic pathways. Alternatives for the treatment of advanced tumours are primary radiotherapy or combined radiation and chemotherapy.

**Hypopharyngeal cancers**

Histologically, almost all of these tumours are squamous cell carcinomas. As with oral and oropharyngeal carcinomas, there is an aetiologic link to chronic alcohol and nicotine abuse.

**Symptoms**

Most malignant tumours of the hypopharynx are diagnosed at an advanced stage because earlier lesions do not produce symptoms. Initial complaints tend to be nonspecific, depending on tumour size and location, and consist of dysphagia and a fetid breath odour. Later there may be pain radiating to the ear. Hoarseness and possible dyspnoea signify tumour extension to the larynx. In many cases, cervical lymph node metastasis is noted as the earliest sign of disease.

**Diagnosis**

Besides the mirror examination or indirect laryngoscopy, the diagnostic workup should include endoscopic examination under general endotracheal anaesthesia, as this is the best way to evaluate the tumour extent. A biopsy can also be taken in the same sitting for histological confirmation. Additionally, sectional imaging modalities can help to define the tumour size and check for involvement of adjacent structures while also evaluating the cervical lymph node status.

**Treatment**

Treatment depends on tumour size but usually consists of local surgical excision with a concomitant neck dissection. Many malignant tumours of the hypopharynx have already spread to the larynx, making it necessary to perform a laryngectomy in the same sitting. The tissue defect is closed primarily whenever possible. This cannot be done with extensive hypopharyngeal resections due to the high risk of stricture formation, and larger defects should be reconstructed by means of a free jejunum transfer with microvascular anastomosis. Surgery should be followed by radiation to the tumour site and lymphatics. Alternative treatments for advanced hypopharyngeal cancers are primary radiotherapy and combined radiation and chemotherapy.

**PARANASAL SINUS TUMOURS**

Malignant tumours of the nasal cavity and paranasal sinuses are far more common than benign masses. Histologically, the great majority (> 80%) are tumours of the epithelial series (e.g. squamous cell carcinoma, adenocarcinoma, adenoid cystic carcinoma). Neoplasms of mesenchymal origin such as osteosarcomas and chondrosarcomas, as well as malignant lymphomas are much less common. Metastases from other malignancies are occasionally found, with the primary tumour residing in the kidneys, lungs, breasts, testes, or thyroid gland. The main sites of predilection are the nasal cavity and maxillary sinus, followed by the ethmoid cells, frontal sinus, and sphenoid sinus.
Symptoms

Because many tumours originate in the paranasal sinuses themselves, they often do not produce clinical manifestations until they have reached an advanced stage. Symptoms that are suspicious for malignancy include sudden onset of obstructed nasal breathing combined with bloody rhinorrhoea and a fetid nasal odour, especially in patients over 50 years of age. A malignant tumour should also be considered in the differential diagnosis of unilateral sinusitis that is refractory to treatment. Advanced tumour stages may be marked by swelling of the buccal soft tissues, swelling at the medial canthus of the eye, headache, facial pain, and hypoesthesia or numbness of the cheek due to infraorbital nerve involvement. Orbital infiltration can lead to displacement of the orbital contents, diplopia, or proptosis.

Diagnosis

The clinical examination includes endoscopic inspection of the nasal cavity and a search for regional lymph node metastases by bimanual palpation of the cervical soft tissues. Since sinus tumours are apt to invade the nasal cavity secondarily, endoscopy alone may provide little information on the extent of the mass. For this reason, computed tomography and/or magnetic resonance imaging should always be performed and should cover the cervical soft tissues to check for nodal metastases. The disease can be staged based on the results of the examination.

Treatment

Treatment is individualised according to the histology and extent of the malignant tumour, and the treatment plan should be coordinated with the radiotherapist and medical oncologist. Since the great majority of lesions are squamous cell carcinomas, however, the treatment of choice will usually consist of surgery and postoperative radiation.

The goal of radical tumour removal may require a very extensive procedure with partial or complete removal of the maxilla or partial resection of the anterior skull base. As a result of close interdisciplinary cooperation with neurosurgeons, maxillofacial surgeons and ophthalmologists, as well as modern intensive care options, even very extensive sinonasal malignancies can now be managed by surgical treatment.

Since only about 20% of sinonasal malignancies metastasise to regional lymph nodes, a neck dissection is necessary only in patients who have clinically positive cervical nodes. Many of these cases will require postoperative radiotherapy.

LARYNGEAL CANCER

The incidence of laryngeal carcinoma is relatively low in comparison to that of carcinomas of all organs. There are areas where the incidence is higher (greater than 10 per 10,000) including Spain, Italy, France, Brazil, and India. Worldwide, the peak incidence of laryngeal cancer is highest in men aged between 55 to 65 years. The male-to-female ratio varies from 5 to 20 in 1, however, in the last decades, there has been a decrease in this ratio because of an increase of laryngeal cancer in women. There is a notable social class difference, in that laryngeal cancer is twice as common in men with low socioeconomic status.

Symptoms

The natural history and clinical picture of laryngeal cancer is dictated by its site of origin and its tendency to disseminate to the regional lymphatics. Progressive continuous hoarseness is the cardinal symptom of laryngeal carcinoma, especially the glottic one. Dyspnoea and stridor, pain, dysphagia, cough, swelling in the neck, hemoptysis, halitosis, reflective otalgia, tenderness of the larynx and weight loss can also occur during the course of the disease.

Neck metastases are more common in supraglottic cancers due to rich lymphatic network leading to early dissemination of primary tumours to the regional lymph nodes. On the contrary, glottic tumours because of the poor lymphatic network of the true vocal cords rarely present with regional neck metastases at the time of the establishment of the diagnosis.

Treatment

The standard options for treatment of laryngeal cancer are surgery, radiotherapy, chemotherapy or a combination of them. It is widely accepted that early-stage can be adequately treated with single modality
therapy, either surgery or radiotherapy, with a 5-year local control of 85–95%. For more advanced diseases, a multimodality treatment based upon a combination of surgery and irradiation is the most common approach. Small glottic cancers have better prognosis than supraglottic ones. The overall 5-year survival following treatment is 80% for glottic and 50% for supraglottic tumours, mainly because the latter presents an increased incidence of nodal metastases.

**SALIVARY GLAND TUMOURS**

Cancers affecting the salivary glands are rare. They can occur at any age, but are more common in people over 50. There are different types of salivary gland cancer depending on the type of cell that has become cancerous.

**Symptoms**

*Parotid Glands:* Painless, slowly increasing swelling in the region of a salivary gland. Facial palsy – most probably incomplete or of only a few peripheral nerve branches – is present in only one of four cases of parotid gland malignoma. Pain and skin infiltration are negative prognostic factors, indicating an advanced disease. Advanced tumours present with cervical adenopathy.

*Submandibular Glands:* Both benign and malignant tumours usually present as a painless, mobile mass in the submandibular triangle. Pain, skin infiltration and fixation to the mandible are signs of local extension. Weakness or numbness of the tongue indicates spreading along the hypoglossal nerve or the lingual nerve, respectively.

*Minor Salivary Glands:* Presentation depends on the site of the tumour and does not differ from other malignant tumours such as squamous cell carcinoma. The palate is the most common site, and the tumour usually manifests as a submucosal mass or ulceration. The second most common site is the sinonasal tract. Symptoms are nasal obstruction, epistaxis or a nasal mass.

**Diagnosis**

Inspection, palpation and fine-needle aspiration cytology (when necessary, ultrasound guided). MRI of the neck: evaluation of the extent of the diseases, especially for deep-lobe parotid tumours or those with parapharyngeal extension, also useful in the early detection of perineural spread. CT of the neck: in case of possible bone infiltration.

**Surgical Treatment**

The main goal is the complete resection of the tumour and prevention of unnecessary morbidity. The minimal approach to small parotid tumours of the superficial lobe is a lateral parotidectomy. Larger tumours and all deep-lobe tumours are treated by total parotidectomy. A tumour extending beyond the parotid gland may need an extended parotidectomy including skin, soft tissue, masseter muscle resection, infratemporal fossa dissection, mastoidectomy or even petrosectomy.

Facial nerve resection in cases without facial nerve infiltration (radical parotidectomy) does not result in increased tumour control. In cases of facial nerve infiltration, the involved branches of the nerve are resected.

Small submandibular tumours are treated by resection of the gland, while advanced tumours need wide en bloc resection of the submandibular triangle, and may need resection of the floor of the mouth, mylohyoid and digastric muscles, or marginal/segmental mandibulectomy.

Infiltration and consecutive thickening of the lingual, hypoglossal, mylohyoid or marginal mandibular nerve indicate resection of the involved nerves. In the oral cavity, minor salivary gland tumours are treated by wide local excision; advanced tumours require radical excision with segmental/marginal mandibulectomy, or partial/total maxillectomy.

**Treatment of the neck:** in cases of clinical evidence of a neck metastasis, a neck dissection is performed as selective, radical–modified or radical neck dissection according to the extent of neck infiltration.

**Conservative treatment**

Post-operative radiation is often advised to reduce the risk of local–regional relapse with indications being: close surgical margins (a common occurrence with facial nerve preservation), an intermediate- to high-grade cancer,
or nodal disease at presentation. Adenoid cystic cancer of salivary gland origin is an unusual cancer with a distinct natural history characterised by a high incidence of perineural spread retrograde along nerve tracts, more distant than regional relapses, and late recurrences up to 10–20 years after initial diagnosis. Post-operative treatment of this lesion often requires radiation targeted not only to the primary site, but also along nerve tracts between the primary tumour and the base of the skull whenever perineural invasion is demonstrated.

Patients with unresectable salivary gland tumours are treated by definitive radiation often with poor results. Selected patients in this setting may benefit from the use of concurrent chemotherapy with radiation. For metastatic lesions or refractory local–regional disease, therapy is given with palliative intent, usually chemotherapy.

**ACUTE AND CHRONIC COMPLICATIONS OF THE TREATMENTS**

The complications of surgery are principally in the categories of pain, function (speech and swallowing), and cosmesis. In general, pain is transient and well managed. Dramatic improvements in post-treatment speech, swallowing, and cosmesis have been achieved by two means: the avoidance or limiting of surgical intervention in selected patients through the use of concurrent chemotherapy and radiation, and the use of novel reconstructive techniques. Palatal prosthetics and dental implantation into revascularised bone grafts after mandibular reconstruction can similarly augment functional oral rehabilitation.

Complications of radiation therapy are divided into acute and chronic (late) toxicities. Acute toxicities principally relate to radiation dermatitis and mucositis with consequent issues of wound care, dysphagia, excess oral secretions, and aspiration. Chronic or late radiation toxicities can vary from mildly disabling (abnormal taste, xerostomia, and accelerated dental disease), to severely disabling (soft tissue fibrosis with neck stiffness or tongue restriction, second primary cancers, accelerated carotid artery atherosclerosis).

Complications of chemotherapy vary depending on the specific agents used and the setting. Full dose chemotherapy brings expected risks of myelosuppression, mucositis, diarrhoea, nausea and vomiting, alopecia, and nephrotoxicity. When chemotherapy is used with radiation, chemotherapy specific toxicities are often minimal as the dose of chemotherapy is often less than maximal. However, all forms of chemotherapy with radiation significantly increase the risk of radiation-induced dermatitis and mucositis.

**FOLLOW-UP**

A regular oncological follow-up is mandatory for patients with head and neck cancers, as the peak incidence of local and loco-regional recurrence is between 18–36 months after the first treatment. Indeed, following the curative treatment of a primary head and neck cancer, the annual risk for developing a second unrelated squamous cancer of the upper aerodigestive tract is approximately 2%.

**11. Orthodontic treatment (Emil Segatto DMD)**

The goal of orthodontic treatment is to eliminate malocclusion by setting a balanced occlusion, improving aesthetics for a better smile and facial appearance. All therapy efforts for eliminating malocclusion aim at functional rehabilitation. Facial aesthetics is also an important consideration for treatment goals; out of the available alternatives, it is always the most aesthetically one that is the preferable option to choose. Orthodontic treatments have a wide-range spectrum including incentive therapies characteristic of primary dentition stage, treatments with decisively removable appliances of mixed dentition stage, as well as the complex orthodontic-surgical treatments of permanent dentition stage. Initial mild anomalies worsen from childhood and they reach full manifestation in the permanent dentition stage. This is true not only for the deviations in tooth position but also for location and size features of jawbones. Therefore early screenings and well-founded treatments started in time can significantly shorten and simplify orthodontic interventions that are usually long and complex.

No later than age seven each child should go through his/her first mandatory orthodontic examination. Both paediatric dentists and dental hygienists have great responsibility for this as part of their patient management obligations.

The importance of orthodontic screenings performed at age seven is:

• posterior occlusion is established when the first molars erupt. At that time, the clinician can evaluate the sagittal and transverse relationships of the occlusion, as well as discover any functional shifts or crossbites.
with the eruption of incisors, problems can already be detected, such as crowding, habits, overjets, open bites and some jaw discrepancies.

for some, a timely evaluation will lead to significant treatment benefits; while for others, the principal immediate benefit is parents’ peace of mind.

At this age there are no complex orthodontic treatments performed; interventions are usually aimed at eliminating those perceived deformations, which would cause adverse consequences later. Such an early intervention is the correction of crossbites – if they remain untreated, they can result in severe dentoskeletal discrepancies. After these targeted local treatments it is usually sufficient to monitor growth for planning further complex treatments.

Lateral crossbites usually inhibit normal transversal growth of the affected half of the maxilla. The condition is more severe in case of bilateral deviations. The resulting maxillary narrowness leads to the development of further lack of space and consecutive tooth crowding. In this period, treatment of lateral crossbites can be performed with a removable active plate appliance, or, in case of fully erupted molars, it is corrected with a transpalatal arch appliance.

It is characteristic of frontal crossbites that lower incisors bite in front of upper incisors. The consequence of this disorder is that the sagittal growth of the mandible remains uncontrolled, and maxillary growth is prohibited. In case of normal growth, potentially significant lack of space may develop in the frontal maxillary zone, which manifests as tooth crowding. A more severe consequence is the negative influence on the development of the alveolar ridge, which may lead to severe skeletal discrepancy.

Certain bad habits can lead to further changes requiring early treatment, which can have adverse effects through affecting the function of the perioral musculature in the long run. Some of these bad habits are thumb-sucking, tongue thrust swallow, lip biting, mouth breathing as well as sucking and chewing different objects (e.g. a pacifier). Parents and close relatives have a principal role in breaking these bad habits; however, explanatory work is the task of skilled medical staff. Since these bad habits help children to calm down and fall asleep, they usually show serious opposition to giving them up. Parents usually have strong inclination to avoid these conflicts and, as they are unaware of the severity of adverse effects, they may even take it to extremes. Therefore providing information has primary importance in breaking bad habits, since the limited number of available orthodontic appliances is still not well received by children. Beyond direct adverse impacts, it is very important to make it clear that even the simplest orthodontic appliances would certainly fail as long as the bad habits persist.

Besides early patient care and patient and parent information activities, staff performing regular oral hygiene should have some basic orthodontic diagnostic skills. With this knowledge, it is possible to send children both at mixed and permanent dentition stages and adults to orthodontic consultation in cases when already developed malocclusion could indicate further adverse effect. It is well known that the dental care of crowded dentition is more limited; therefore the risk of plaque accumulation is much higher. If periodontal tissues are continuously exposed to inflammation, it can lead to significant mobility enhancement over time, indeed, in an extreme case, it can even endanger conservation of a particular tooth. On the other hand, decreased autocleansing surface of crowded teeth and enlarged contact surfaces can significantly increase the danger of caries development. Although – thanks to modern endodontic treatments – tooth loss deriving from caries is very rare, expansive restoration treatments are disadvantageous both in aesthetic and functional terms. Nowadays the functional consequences of malocclusion are also in focus; out of them, the temporomandibular joint has particular attention. Beyond the adaptation limit, both asymmetric and significant sagittal burden may initiate temporomandibular dysfunctions. It starts by clicking, then pain is felt and malfunctions (hyper- or hypomobility) occur, imposing tremendous psychological burden on patients.

Basic diagnostic examinations performed by dental hygienists that can contribute to the success of secondary orthodontic prevention are the following:

Begin by examining each arch separately and evaluate the following categories:

1. Arch width (molar-to-molar transpalatal width of 36 mm is average)
2. Excessive spacing or crowding present
3. Missing or ankylosed teeth.
• Next, note the relationship between the upper and lower teeth in occlusion, and evaluate the following:

1. Angle’s classification
2. The amount of overbite and overjet present
3. Any openbite and/or crossbite

• Facial observation should cover the following: present

1. Frontal view – asymmetries (facial; dental; dentofacial), horizontal and vertical disproportionalities
2. Lateral view – profile evaluation (determining the position of the jaws relative to each other and the cranial base - straight, convex or concave profile), relation and location of lips, vertical facial proportions, degree of mandibular angle.

ORTHODONTIC APPLIANCES

Instruments of orthodontic treatment cover the therapeutic tools of secondary and tertiary prevention. They can be categorised in several ways. The simplest classification method is categorisation by fixation: there are removable, fixed and auxiliary (so-called anchorage) appliances.

Removable orthodontic appliances

The majority of removable appliances are made on individual plaster models in a dental laboratory. Their preparation is quick and relatively cheap, and they are mainly recommended in the mixed and early permanent dentition stages.

Active plate appliances are suitable for directly moving the teeth, which is performed by special built-in screws and springs. Their main advantage is that they are quite comfortable to wear due to their size. There are some cons as well, namely that they demand continuous wearing (at least 14 hours per day), and they can only incline teeth, they cannot perform controlled root movements.

Another group of tailored removable appliances is the group of functional appliances, which are suitable for affecting jaw growth direction and pace in the growth period. Their disadvantages are the uncomfortable, big size and the fact that they are required to be worn most of the time (minimum 16 hours daily). Their advantage is that if they are worn for the appropriate time and in the appropriate way, they are suitable for the conservative correction of the majority of skeletal deviations, thus preventing patients from subsequent surgical corrections.

A special group of removable appliances is the preassembled myofunctional trainers (available in different sizes), which perform their effect on the muscles and soft tissues influencing the development of the dentoskeletal complex. Their advantages are the low price and they do not require impression and plaster model for production. Therefore, their main disadvantage is that their function cannot be set either to person or to dysfunction.

Different positioners are the most frequent type of removable appliances used in adults. These appliances are manufactured with the help of preset plaster model series and they develop the planned arch form and teeth position movements for precisely defined periods and sequence. Pros for these appliances are their minimum visibility, but they are expensive and there are several cons as well, e.g. their required daily wearing time period, which is minimum 20 hours per day, and the fact that they have only limited applicability.

The last group of removable appliances is the group of removable retainers, which ensure holding and stabilisation of teeth after the active treatment period. Combined with fixed retainers, they only have to be worn at night in the first and second year after the active treatment. Traditional retention appliances were passive variants of active plate appliances with an acrylate base plate. Their appearance is similar to that of active appliances, but they do not contain any active elements (like expansion screw or spring). More recent variants of removable retainers are appliances similar to positioners manufactured with the vacuum foil method. Wearing itself is more comfortable but extra care is needed for maintenance, as they are more vulnerable.

Fixed orthodontic appliances
The biggest advantage of fixed appliances is that no cooperation is required from the patient to reach their goal. They are fixed on the surface of the teeth with special bonding materials. Classification is based on the materials and the achieved effects.

A group of simpler fixed appliances consists of appliances used mainly in the mixed dentition stage and auxiliary (secondary) appliances used in the permanent dentition stage such as:

- **Inclined plane** – it is made of acrylic and serves to correct frontal crossbites. It is fixed on lower frontal teeth and should be removed after 3 weeks.

- **Goshgarian transpalatal arch** – it is connected to a palatal lock of bands bonded to upper molars. It is appropriate for rotation and transversal expansion of molars.

- **Quad helix** – it is connected to a palatal lock of bands fixed to upper molars. Beside rotation and transversal expansion of molars, it is suitable for the expansion of premolars, too.

- **Rapid palatal expander with Hyrax screw** – it is connected to bands fixed to upper premolars and molars. It serves to disrupt suture between palatal plates and to abduct palatal plates from each other with a view to achieving transversal expansion of the maxilla, too.

- **Lingual arch** – it is connected to a lingual lock of bands fixed to lower molars. It is suitable for protruding lower frontal teeth.

- **Lip bumper** – it is connected to a vestibular lock of lower molar bands. As a result of its functional effect, it facilitates the protrusion of lower frontal teeth, as well as tilting back and uprighting the molars by keeping away the lower lip.

The best known and best quality group of fixed appliances is the multibracket systems. Their price is higher than the cost of traditional removable appliances. Most of them are prefabricated and they contain general information necessary to move the individual tooth. A minority of them are tailored to the patient’s need, and they contain prefabricated arches having all the information for the subsequent work stages in a preset way. The manufacturing cost of these appliances is much higher.

The use of multibracket systems requires permanent teeth. The prerequisite of bonding partial appliances is the presence of erupted first molars and all four incisors. Orthodontic arch wires responsible for teeth movements (made of different materials and in different diameters) are connected to brackets firmly attached to the surface of the teeth, thereby delivering power and information necessary to teeth movements. Based on the way of fixation in the brackets, there are two types distinguished: conventional ligating and self-ligating systems. In the former case, the fixation of the arch in the bracket slot is performed by elastic or stainless steel ligatures, while in case of self-ligating systems a clip built in the bracket is responsible for that. Based on the fixing features of the clip there are passive and active self-ligating systems available.

*They are classified by:*

- **ligating mode**
  1. conventional systems
  2. self-ligating systems (passive and active)

- **material of the bracket**
  1. metal
  2. aesthetic (plastic, ceramics, sapphire crystal)

- **location**
  1. labial (outer)
  2. lingual (inner).

*Anchorages*
Anchorages are used to neutralise unwanted side-effects, as well as to save dental groups not involved in the movements. Most well-known anchorage forms are:

- dental anchorage – teeth, dental groups are used for anchorage
- dento-mucosal anchorage – besides the teeth, soft tissue surfaces covered by mucous membrane are also involved
- muscular anchorage – different muscular effects act as anchorage source
- extraoral anchorage – different areas of the skull are used for fixing, e.g. mentum, forehead, calvarium, nape
- skeletal anchorage – it requires special auxiliary elements anchored on bone surface or in the bone itself, like microimplants, miniplates, palatal implants.

**ORTHODONTIC TREATMENTS**

Since the majority of orthodontic appliances are tailored, it is necessary to make an adequate impression and plaster model for their manufacturing. Plaster models done during the diagnostic process have documentary importance, therefore new impressions are necessary for manufacturing the appliances. In order to manufacture the majority of removable appliances, a precise modelling of the shape of the alveolar ridge is necessary; which cannot be achieved unless applying a special orthodontic impression tray with a high edge. In general (except for planning mechanics for microimplant), the adequate impression material is alginate. Due to continuous changes (growth, changes induced by the treatment), new plaster models are done for recording the individual stages again and again. Plaster models of a given patient should be stored separately in an adequate box. It facilitates the review of prolonged treatment procedures, determining the actual status on the treatment timeline, as well as planning necessary changes.

**Treatment with removable appliances**

**Active plate appliances**

Frequently, treatment with removable appliances is not performed as a separate treatment, but as the first step of a two-phase treatment. The name “night appliance” usually used by patients is incorrect and misleading, since night-time wearing itself is insufficient for the desired results. Plate appliances may be used on the upper, lower or both jaws at the same time. Active plates are appliances stabilised on the dento-mucosal area, and their impact is visible on the dento-alveolar system immediately after their insertion. Either as part of multi-stage treatments or in separate use, plate appliances usually contain an expansions crew built in the base plate (Fig.5.100.).
Removable active plate appliance

Expansion of plate halves is established by its activation. Activation is carried out once a week by the patient (parent, relative). The degree of expansion is checked by the specialist every 1-1.5 months on average. Activation of springs, as well as checking the fixation base plate (holding elements, labial arch) is executed during follow-ups. Correction/reduction of areas preventing the stability of the base plate may be necessary, which is performed by the specialist using a suitable straight handpiece and bur (Fig. 5.101.).
Straight piece and bur

Setting and activation of holding clasps and wire springs is done with special pliers (Fig. 5.102.).

Wire bending pliers

Taking into account that active plate appliances can perform tooth movements only by tilting, they are used in the following cases:

• to correct deep bite – using the special features of the base plate;
• to procline upper frontal teeth – in case of anterior crossbite;
• for labial, lingual, mesial or distal tilting of teeth;
• to establish disocclusion – to accelerate the correction of malocclusions;
• to correct maxillary narrowness – to eliminate uni- or bilateral crossbites.

**Elements of standard active plate appliances:**

• **base plate** (it can be made of autopolymerising acrylic or vacuum foil), which is responsible for the anchorage of the appliance, as well as for the anchorage of holding and active elements

• **holding elements or clasps** (the most widely known ones are Adams, semicircular, round, sagittal or delta clasps), which ensure the stability of the base plate

• **active elements** (expansion screw, finger springs, “S” springs, “8” springs), which are responsible for performing teeth movements.

Besides the above mentioned parts, special plate appliances also contain special elements, like straight or inclined bite pad, occlusal surface coverage, guide bars, blocks, etc., which help them to fulfil special challenges.

**Advantages of the use of active plate appliances:**

• they can be easily manufactured by an experienced dental technician,
• they are easy to wear (in case of children),
• they are suitable for the correction of deep bite,
• they are able to tilt teeth,
• their adaptation requires very little chair time,
• they promote better oral hygiene (since they are removable),
• they are less or hardly visible.

Disadvantages of the use of active plate appliances:
• they are not suitable for eliminating rotations,
• they are not suitable for bodily tooth movement (only for tilting),
• in case of lack of cooperation the set aim cannot be reached due to its frequent removal.

So one of the largest disadvantages of active plate appliances that uncooperative patients can remove it arbitrarily at any time, thereby shortening the wearing period, which definitely results in treatment failure. For dental specialist and dental hygienist it is a must to inform and motivate patients to achieve cooperation and treatment results.

Information activity should cover:
• introduction of the insertion and removal of the appliance (not only in theory but in practice, too!),
• explanation of inconveniences typically experienced in the first period,
• giving advice on wearing the appliance during eating and physical activities,
• the necessity of immediate and compulsory visit to the attending specialist when instability occurs in the appliance after a longer period of disuse,
• providing information on the rules of cleaning and storing the appliance (Fig.5.103.),
• time and mode of activating the expansion screws (not only in theory but in practice, too!)

Box for removable appliances

Functional appliances
Removable functional appliances are basically used for the correction of Angle class II deviations (disto-occlusion). It is expected that treatment will move the mandible forward, slow down the growth of the maxilla, settle frontal teeth conditions, as well as eliminate the class II deviation of molars. Treatment is efficient only in the growth period; therefore its application is indicated in the late mixed dentition period, as well as in the adolescent phase of permanent dentition stage. The secret of successful treatment is continuous wearing (minimum 16 hours per day). Missed periods, as well as shortened wearing time will cause deterioration of the achieved condition and relapse. The special design of the appliance ensures the desired outcome. A common feature of many different functional appliances is that after inserting them in the mouth, the future position of the two tooth arches relative to each other is established according to a position determined previously through a preliminary construction bite-taking. Functional appliances cannot produce significant tooth movement. Their impact on jaws is usually achieved by stimulating the surrounding muscles.

The most frequently used removable functional appliances are the following:

• **Andresen activator**– it is mainly suitable for the correction of Angle II/1 deviations

• **Fränkel functional regulator**– four types are used to correct different deviations (Fig.5.104.):
  1. used in case of Angle II deviations
  2. used in case of Angle II and overjet
  3. used in case of Angle III
  4. used in case of skeletal open bites

• **Twin Block appliance according to Clark**– to correct mandible with backward position

• **Hansa appliance according to Hasund**– to treat Angle II/2 (Fig.5.105.)

• **Double plate appliance according to Sander**– to correct mandible with backward position (Fig.5.106.)
The key elements of success when using removable functional appliances is adequate information, motivation of children, as well as telling them how to wear, maintain and store the appliance.

There are also fixed type functional appliances available, which can be connected to multibracket systems or separate appliances as well. Out of the latter types, the most frequently used is the Herbst hinge appliance, which is basically a separate appliance (Fig.5.107.), but it can also be used as a supplemental unit of multibracket system treatments. Fixed functional appliances form a different subgroup of fixed appliances, since they require adequate cooperation of the patient, too. Their design and significant size demand enormous tolerance from patients. It is counterbalanced by skeletal changes reached within a relatively short period of time, and these changes are much less likely to relapse.
At regular follow-ups during the treatment with removable appliances, there is usually a need for adaptation and reduction of the acrylic parts (base plate, shields, pelottas), as well as for activating and bending wire elements. When manufacturing acrylic components, burs are held into a straight handpiece, while the bending and cutting of wire elements is performed using special pliers and cutters.

During dental hygienic care, regular clinical cleaning of the appliances is also necessary, as they may be inappropriately cleaned by the patient. In case personal appliances have been cleaned and stored inappropriately, patients should be informed of the general and detailed rules of maintaining their appliance again. Due to its smell and look, an untidy and neglected appliance will be rejected by its owner, provoking the failure of the treatment.

**Treatment with fixed appliances**

*Simple treatment with fixed appliances*

Simple fixed appliances can be used separately or as a supplement to standard multibracket systems. Most fixed appliances (transpalatal arch, lingual arch, quad helix, etc.) (Fig.5.108.) are attached to bands bonded to premolars and/or molars. Connection between arches and bands is usually established in a dental laboratory by soldering or via tubes fixed to the bands serving as a socket for arch ends.
Transpalatal bar according to Goshgarian

In case of adjacent teeth, the insertion of bands bonded to the teeth requires the creation of an interdental gap. It is mostly done by inserting separating elastic rings (Fig.5.109.) between the teeth, which are to be removed before bonding the metal bands. Testing the selected bands is followed by isolation of the area, then preparation and drying of the teeth. Bonding is carried out by glass ionomer cement, which is also available in light-cured variant nowadays. For the sake of adequate cleaning, spilt bond should be cleaned up immediately before it gets fully fixed!

Elastic separator

At the end of the treatment, bands should be removed together with arches, as they are not necessary anymore. Removal is performed by special pliers, followed by cleaning the surface of the teeth from the remaining bonding material. This process is done by using rotating tools (special carbide burs), and subsequently all affected surfaces need to be polished (Fig.5.110.).

Contra-angle piece with bur
Rapid maxillary expansion treatments

The most efficient treatment of maxillary narrowness can be performed with a rapid expansion appliance with Hyrax screw. The essence of the method is the transversal expansion of palatal plates mobilised by ripping the median palatine suture. Before the ossification of sutures (around the age of 14-15) this can be done without any surgical interventions; after this age it is necessary to weaken the bony area of the planned movement. Hyrax screw requiring two activations per day can be fixed either to the acrylic splints covering the teeth of the upper lateral zone; or to the bands bonded to the first premolars and molars and at the same time to microimplants fixed to palate (Fig. 5.111.). The application of different alternatives is determined by the presence or stability of teeth necessary for the support. Activation performed twice a day at home results in the desired expansion within 2-3 weeks; after that the screw will be blocked in order to prevent twisting back to the original position. Ossification of areas between hard palate shelves separated from each other is carried out within 3-4 months, therefore the rapid expansion appliance cannot be removed before this time. After the removal, cleaning off the bonding agent and polishing the teeth take place as it has been described earlier.
Owing to its special configuration and expansion protocol, painful symptoms (tension, more difficult tongue functions) are more frequent during rapid palatal expansion, and chewing is also uncomfortable due to rapid teeth movements. Due to large retentive surfaces and sensibility, it is much harder to clean the appliance itself and maintain oral hygiene, therefore increased care is required. The role of dental hygienists is extremely important in providing exact instructions (activating screws twice a day, coaching of parents together with practicing) at weekly follow-ups (Fig.5.112.).

![Activator key for Hyrax screw](image)

**Treatment performed with multibracket systems**

The characteristics of multibracket systems used mainly in permanent dentition stage:

- teeth are moved very precisely and under control,
- they can move numerous teeth simultaneously,
- they have a much more complex structure than removable appliances,
- they are suitable for performing bodily tooth movements (simultaneous movement of crown and root),
- they are suitable for treatments involving tooth extraction,
- in many cases they follow previous treatment with active plate appliances or functional appliances,
- they are also used for preparing orthognathic surgical interventions,
- since they are fixed, patients cannot remove them.

**Stages of treatment** with multibracket system:

- **Alignment** – aligning irregular teeth (rotations, height problems, correction of tooth positions outside and inside dental arches, etc.),
- **Working** – resolving abnormal overbites and/or overjets and space closure after extractions,
- **Finishing** – torquing incisors and mainly fine aesthetic detailing,
- **Retention** – maintenance of treatment results with retainers.
The appliance is fixed with a special bonding material. Former two-component bonding materials have been fully replaced by light-cured forms. Removal of the spilt bond is necessary not only for oral hygienic but also for aesthetic reasons. Fixing of the appliances should be performed by a specialist. They are fitted to the teeth either by:

- **direct bonding** – the specialist fixes each bracket one by one,
- **indirect bonding** – fitting takes place simultaneously, by transposing all appliance elements from the plaster model at the same time with the help of a special tray, where elements have been temporarily placed previously.

Removal of the appliance is performed with special debonding pliers. Removal of bonding agent from the tooth surface is done with a special carbide bur, which is always followed by polishing the tooth surface.

Main components of multibracket systems:

- **bracket** – elements fixed to the labial or buccal surfaces of the teeth, which are responsible for the transmission of forces provided by the arch wires moving the teeth. They can be made of metal (Ni, Ti, Au alloys), plastic, ceramics or sapphire crystal (Fig.5.13.).

There are slots to house arch wires; the shape of each slot is individual, according to the movement features of the tooth. For fitting the arch wire in the slot, either a ligating auxiliary element (elastic or steel ligature) or a special built-in clip is used, which can be opened accordingly. Brackets with external ligature considered to be conventional, while brackets with a clip are part of self-ligating systems. Modern self-ligating bracket systems can be divided into two groups:

- active self-ligating systems – self-locking clips are made of elastic material, teeth movement is more controlled (Fig.5.14.),

Active self-ligating multibracket system
• Passive self-ligating systems – self-locking clips are usually rigid, treatment process is quicker at the beginning, but teeth movement is less controlled (Fig. 5.115.).

Passive self-ligating multibracket system

Arch wire – modern straightwire technique uses prefabricated arch wires, which are made of elastic material with round cross section in the first treatment stage. In subsequent stages the cross section of the arches increases, its form becomes rectangular; the elasticity of the materials decreases, so their force transmission is increasing step by step. In the last stage (finishing) arch wires with the largest cross section (rectangle form) and with maximum rigidity are used. Aesthetic systems contain white arch wires (Fig. 5.116.).
Metalic and esthetic archwires

Patient with special treatment needs

The number of arch wire changes depends on the complexity of the treatment. In the course of arch changes, the old arch is replaced by a new one in the subsequent stage. In case of conventional systems, this action is performed together with the replacement of old ligating systems. In case of self-ligating systems, arch wire change lasts for a much shorter time. After opening the clips a new arch is inserted in the slot and it is fixed by closing the clips. Rigid arch wires used in certain treatment stages are also suitable for performing individual bendings, which enable an individual tooth to be moved to a special direction and extent. Performing these bendings requires comprehensive knowledge and considerable practice by using adequate instruments (special arch bending pliers i.e. How/Weingart orthodontic pliers). Beside this, the order of the applied arches should also be determined by a specialist, but removal of the used arches as well as insertion of the selected new ones could also be performed by an experienced dental hygienist. However, the danger of inadequate insertion should always be borne in mind, since adequate teeth movement can only be ensured by properly selected and installed arch wires.

-band – earlier widely used metal bands provided adequate fixation for brackets soldered to the surface of the metal bands on the molars (Fig.5.117.). In case of modern appliances – mainly due to their easy cleaning and strong bonding materials applied – metal bands are replaced by molar brackets or tubes, which can be fixed directly to the teeth. By this plenty of previously known side effects can be eliminated, such as separation by fitting bands resulting in an interdental gap between teeth, as well as frequent gingivitis and caries owing to difficulties in cleaning.
Molar bands and direct bondable molar brackets

Patient with special treatment needs

 supplementary elements for well-defined tasks such as:

• eyelets
• Kobayashi ligatures
• long steel ligatures
• elastic chains
• intermaxillary elastics pushing and pulling springs
• arch retrakctors,
• cross tubes, etc. (Fig. 5.118.).
Stainless steel and elastic ligatures

Each step of treatment with multibracket systems has its own instrument. Due to the short follow-up periods, adequate sterilisation of special instruments is vital. With regard to their high price, adverse impacts to fine edges as a result of frequent sterilisation require careful maintenance – this way they lifetime can be lengthened. Important instruments of major working stages are the following (Fig. 5.119.):

<table>
<thead>
<tr>
<th>Bonding</th>
<th>Archwire insertion, changing:</th>
<th>Bracket removal:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Retractors for the isolation of the working area</td>
<td>• Special opening instrument (for the clip of the self-ligating bracket)</td>
<td>• Bracket removal plier</td>
</tr>
<tr>
<td>• Bracket holding pliers (Fig. 5.120.)</td>
<td>• Distal cutter</td>
<td>• Adhesive removal freezer</td>
</tr>
<tr>
<td>• Bracket positioning instrument (Fig. 5.121.)</td>
<td>• How- or Weingart plier</td>
<td>• Polishing disc</td>
</tr>
<tr>
<td></td>
<td>• Mathieu needle holder (for the ligature of the conventional bracket) (Fig. 5.122.)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Ligature cutter (Fig. 5.123.)</td>
<td></td>
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</tbody>
</table>
Bracket tweezer

Bracket placement marker
Although fixed appliances require less cooperation from the patients, total ignorance of instructions leads to treatment failure in most cases, due to the complexity of multibracket systems. **Rules** of wearing appliances are divided into two groups (Fig.5.124):
### Rules of wearing appliances

Dental hygiene and appliance maintenance instructions after bonding should be completed by a practical demonstration on a model; dental hygienists have a significant role in the implementation and in subsequent regular follow-up. In the initial period of wearing the appliance, tooth hypersensitivity and pain develop frequently; patients should be prepared for it. Protruding appliance elements can irritate or – in more severe cases – even hurt the oral mucous membrane. In such cases it is recommended to use relief wax, and the patient should be taught how to use it as soon as the appliance is bonded (Fig.5.125.). Similarly, practical training is necessary to make patients learn the use of intermaxillary elastics, which is often characteristic of the latter treatment stage. It is also the task of the dental hygienist to make patients practise it using a special tool in front of a mirror.

<table>
<thead>
<tr>
<th>Preservation of the appliance integrity</th>
<th>Cleaning of the appliance</th>
</tr>
</thead>
</table>
| • The fiddling of the appliance by hands and foreign objects is forbidden  
 • The bite and chewing of the hard foods is forbidden  
 • In case of nocturnal bruxism and sport activities with body contact the wearing of protective splints is strongly recommended | • Toothbrushing after main meals is recommended  
 • Thorough rinsing is necessary after every meal  
 • Areas between the brackets and the archwire need to be cleaned with interdental brushes (recommended after main meals, compulsory before sleeping)  
 • The increase of the time and the solidity of the rinsing after toothbrushing is recommended |
Orthodontic relief wax

**Treatment using anchorage appliances**

Out of the anchorage appliances listed earlier, extraoral and skeletal anchorages should be mentioned due to their special features.

**Extraoral anchorages** – are auxiliary anchorage instruments, which rest on extraoral areas (chin, forehead, calvarium, nape). Via their connection to intraoral elements, these appliances are appropriate for moving teeth or teeth groups, or even for altering growth direction and pace of jaws. They are used at home, during silent occasions, as well as night-time. There are two well-known representatives in the group: head gear and facemask (Delaire and Petit mask).

- **Support of head gear** can be placed either on the calvarium or on the nape or on both sites. Support itself also determines the pulling direction. The intraoral system connected to it is usually a multibracket system, rarely a removable functional appliance. It has a skeletal effect mainly on the maxilla – slows down its growth and modifies its growth direction.

- **The support of facemask** is placed on the chin and forehead. Its effect is opposite of that of the head gear; it is used for enhancing the growth of the maxilla and for influencing the direction of growth through the intermediary multibracket system.

**Skeletal anchorages** – are auxiliary anchorage instruments, which have intraoral support on jaw surfaces adequate to perform their impact. Their insertion is considered as a dental surgical intervention, but it is recommended to be executed by the orthodontist who is planning its further use. They have two large subgroups:

- **Miniplates** – they are fixed by microscrews to the jaw area previously made free by gingival flap formation. Their specially formed ends are left uncovered in the oral cavity and they can be directly connected to brackets and bracket systems fixed on the teeth. Bony fixation provides solid support for teeth and teeth groups to be moved.

- **Microimplants** – penetrating through mucous membrane, they are fixed in the cortical bone of the edentulous region of the jaw. The head of non-osseointegrating microimplants makes them suitable for supporting appliance elements that move teeth and teeth groups, as well as for establishing indirect anchorage similar to miniplates.

Significant development of orthodontic treatment techniques experienced in recent years makes it obligatory for dental hygienists to be familiar with the latest instruments and available techniques, in accordance with the increasing aesthetic demands of patients. The role of dental hygienists begins with the recognition of deformations and sending patients to an orthodontic consultation. After the compilation of the treatment plan, the next steps are completing preparatory tasks before interventions and teaching daily dental hygienic tasks. During orthodontic treatments that usually last for several years, professional cleaning and maintenance should gain significant attention, regarding the increased plaque accumulating features of orthodontic appliances. Finally, at the end of the interventions, dental hygienists still play a significant role in maintaining and enhancing achieved orthodontic results.


**12.1. Orthognathic surgery**

Irregular teeth and jaw deformities lead to aesthetic and functional problems. In most cases, tooth alignment and facial aesthetics can be improved by orthodontic treatment. More severe cases (dentsoskeletal deformities) require a combination of orthodontics and surgery.

These deformities can result in impairment of the orofacial function in different ways. Malocclusion can affect mastication, and especially in severe cases, this impairment can lead to maldigestion and as a consequence, to general nutritional problems. Lip incompetence results in mouth breathing, which eliminates the physiologic functions of the nose on breathing. Speech can also be affected. Irregular dentition may have a negative effect
on maintaining proper oral hygiene, and this can result in dental caries and periodontal disease. Dentofacial deformities may lead to temporomandibular dysfunction.

The physiological effects of these deformities cannot be neglected, but the psychosocial impact on the individual is often paramount. The quality of life can be severely impaired life-long.

Combined orthodontic treatment and surgery makes it possible to treat dentofacial deformities that could not have been corrected orthodontically alone.

Orthognathic surgery refers to “alignment of the jaws”. The aim of orthognathic surgery is to normalize the relationship of the jaws between themselves and to the rest of the craniofacial complex.

**COMMON DENTOFACIAL DEFORMITIES**

**Maxillary anteroposterior excess**

Protrusive maxilla is where there is overgrowth in the anterior horizontal direction. The clinical picture is characterised by a convex facial profile. There is a short, curled upper lip and the lower lip is curled under the maxillary incisors. The latter results in the development of a deep labiomentonal sulcus. There is a large overjet. The nose often appears to be prominent.

**Maxillary anteroposterior deficiency**

Retrusive maxilla is where there is undergrowth in the anterior horizontal direction. The clinical picture is characterised by the hypoplasia of the midface that results in a concave facial profile. The paranasal areas are flat. The lower lip is ahead of the upper lip. The occlusion is characterised by anterior (bulldog bite) and posterior crossbites. The upper incisors are usually labially inclined that results in an acute nasolabial angle. There is a lingual inclination of the lower front teeth (dentoalveolar compensation).

**Mandibular anteroposterior excess**

Protrusive mandible is where there is overgrowth in the anterior horizontal direction. The mandible appears strong. The lower lip is ahead of the upper lip. The labiomentonal fold is obtuse. There is malocclusion with an anterior and posterior crossbite. The mandibular incisors are often lingually inclined (dentoalveolar compensation). A midline discrepancy and a tendency toward open are often present. The tongue is often enlarged.

**Mandibular anteroposterior deficiency**

Retrusive mandible is where there is undergrowth in the anterior horizontal direction. The clinical picture is characterised by a convex facial profile. The chin is retruded and weak. The nose seems to be large due to the deficient mandible. There is a short chin-to-throat length. The lower lip is everted and wedges in behind upper incisors. The labiomentonal fold is deep. There is a large overjet. There is an increased overbite; the lower incisors often bite into the palate.

**Apertognathia (open bite)**

In contrast with the previous ones, apertognathia is a vertical and not sagittal discrepancy. There is a vertical maxillary excess, more posterior than anterior. The anterior facial height is increased (long face), particularly in the lower third. Open bite is often characterised by an increased interlabial gap, mouth breathing and an enlarged tongue. In this condition, the teeth in opposing jaws fail to contact. In mild cases, only the front teeth are not in contact. In more severe cases, premolar or even the first molar teeth are not in occlusion. Usually there is an excessive anterior maxillary and mandibular dentoalveolar height as partial compensation.

**ORTHOGNATHIC PLANNING**

The objective of orthognathic treatment is to achieve an optimal balance between aesthetics, function and stability.

The aim of the treatment planning is to

- obtain a functional occlusion with teeth in most ideal position to aid stability and aesthetics.
• correct underlying skeletal deformity.

• achieve maximum aesthetic result that does not compromise occlusal or skeletal deformity.

Patient examination and diagnosis

History taking: to determine the patient’s feelings about the problems and their expectations for treatment results

Clinical evaluation

• Facial form: long, short, concave, convex, flat
• Facial proportions: relationship of facial thirds
• Relationship of soft tissues to dentition: smile line, occlusal cant, dental and soft tissue midlines
• Clinical measurements: vertical, transversal, anteroposterior, intra-arch dimensions

Radiographic examinations

• Orthopantomogram
• Lateral and antero-posterior cephalometric X-rays
• Occasionally computer tomography

Dental study models

• Accurate bite registration - facebow transfer indicated in two jaw surgery
• Two jaw surgery requires duplicate models

Photo documentation: Standardised facial and intraoral photographs are essential to analyse facial form and harmony.

Cephalometric analysis: Cephalometric analysis depends on cephalometric radiography to study relationships between bony and soft tissue landmarks and can be used to diagnose facial growth abnormalities prior to treatment.

TREATMENT

Dental extractions

In order to achieve the ideal result, dental extractions might be necessary. Most frequently the first bicuspids are extracted to facilitate orthodontics. Lower third molars have to be removed at least six months prior to mandibular surgery (sagittal split osteotomy) in most cases.

Treatment sequence

1. Any dental restoration or periodontal treatment should be performed prior to orthodontic and surgical intervention.

2. Presurgical orthodontics
   • Align and level the teeth
   • Co-ordination of upper and lower arch
   • Dentoalveolar decompensation

3. Surgery

4. Postsurgical orthodontics
• Final tooth alignment

Once ready for the surgical procedures, the orthodontist and maxillofacial surgeon will review photographs, x-rays, and dental models to finalise the surgical plan. The operation may involve a single jaw (maxilla or mandible) or both jaws (bimaxillary procedure). The surgery may also be combined with other procedures, such as rhinoplasty (nose correction) or genioplasty (chin correction) to improve the general appearance of the face.

**Model surgery and occlusal wafer**

Following presurgical orthodontics **model surgery** is performed. The primary goal of model surgery is to simulate the patient’s jaws and dental structures as accurately as possible to allow accurate simulation of the planned surgery. The surgical movement of the jaws or dentoalveolar segments is simulated on the casts. Reference lines are drawn on the mounted casts to record their positions in three planes of space. The casts are cut into pieces according to the surgical plan. Following that the desired occlusion is determined and the segments are repositioned and glued together. The reference lines show the direction and distance of the movements of the segments.

Following model surgery, **occlusal wafers** are made that will determine the new position of the jaws during real surgery.

**Surgery**

There are several types of mandibular and maxillary orthognathic surgical procedures. These are classified according to the design of the osteotomy (bone cut) and the region where the surgery is undertaken.

**Sagittal split osteotomy** is the most versatile and most frequently performed **mandibular** osteotomy. The ramus is split in the sagittal plane, and the whole body of the mandible along with the dental structures is mobilised. The segment is repositioned into its new position determined by the occlusal wafer.

The most common orthognathic procedure on the **maxilla** is **Le Fort I osteotomy** that separates the palate together with the maxillary dentoalveolar process and the dental structures from the rest of the skull. Following mobilisation of the segment, its new position will be determined by the previously fabricated occlusal wafer.

When two-jaw surgery is performed, two **occlusal wafers** are fabricated. The role of the first (intermediate) wafer is to set the new position of the firstly operated jaw (usually the maxilla) relative to the intact jaw (mandible). The second (final) wafer determines the position of the secondly osteotomised jaw (usually the mandible) relative to the previously repositioned segment (maxilla).

Depending on the type of the surgery, the jaws may be fixed together (**inter-maxillary fixation**) at the end of the procedure. Intermaxillary fixation can be established with the use of elastic rings or stainless steel wires. Some surgeons prefer keeping the occlusal wafer intraorally for some time after surgery. Intermaxillary fixation facilitates bone healing, the adaptation of the masticatory muscles and maximal intercuspidation.

**Complications**

In general, complications of orthognathic surgery occur infrequently. Like in any other surgery, there can be **bleeding** or **infections** may develop. **Damage to teeth** is rare. **Vascular compromise** due to stretching or tearing of the vascular pedicle may lead to **inadequate bone healing**, **necrosis** or **bone resorption**. **Malocclusion** is either due to relapse or poor intraoperative dental positioning. **Temporomandibular joint dysfunction** may develop after surgery. The only frequent and severe complication of orthognathic surgery is **neurological dysfunction** after sagittal split osteotomy or after other mandibular procedures. The inferior alveolar nerve may be stretched, avulsed, torn or compressed during split, manipulation or fixation of the segments. This results in some numbness in the lower lip and chin. The lack of feeling is usually temporary, but in certain occasions it can be permanent.

**Postoperative period**

Intermaxillary fixation can be maintained for a period of time; therefore, patients are often required to adhere to an all-liquid diet. Diet is very important after surgery, to accelerate the healing process. Some weight loss due to lack of appetite and the liquid diet is common.
Effective oral hygiene is especially important for those undergoing orthognathic surgeries. Fixed orthodontic appliances, intermaxillary fixation, elastics; soft tissue swelling and postoperative pain can prevent both effective tooth brushing and the mechanical cleaning action of mastication. Scrupulous oral hygiene is essential to prevent plaque accumulation and infection of the healing wounds. Oral hygienists have an important role in this field. Emphasis must be placed on routine hygiene, including professional tooth cleaning and home care instructions.

Most of the orthognathic patients are released from hospital 2–5 days after surgery.

After the jaws are healed, 4–6 weeks postsurgically, orthodontic treatment is continued to bring the teeth to their final position.

12.2. Treatment of patients with cleft lip and palate

Clefts of the lip and palate appearing approximately 1:700 live births are one of the most common craniofacial birth defects. Due to modern ultrasound diagnostic procedures, these patients can already be diagnosed in early foetal age; however, their treatment puts extraordinary burden to both parents and other family members of the patient, as well as to medical staff members participating in the complex treatment until the patient’s adulthood. In-utero diagnosis offers parents the opportunity to be prepared for the corrective surgery to be executed in accordance with a precise schedule after birth, as well as for living in altered conditions. Corrective surgery and rehabilitation need synchronised actions from several specialists, where the main providers are health visitors, otolaryngologists, speech therapists, paediatric dentists, orthodontists, cranio-maxillofacial surgeons, and plastic surgeons.

The main control and management of dental treatment are performed by a team consisting of an orthodontist and a cranio-maxillofacial surgeon. Orthodontic care can be divided into four separate developmental periods. Apart from the patient’s age, milestones of these periods are defined by different developmental stages:

- Neonate and infant stage (birth to 2 years of age)
- Primary dentition stage (2 to 6 years of age)
- Mixed dentition stage (7 to 12 years of age)
- Permanent dentition stage (over 12 years of age).

**NEONATE AND INFANT STAGE**

Neonatal maxillary orthopaedic interventions having been widely applied earlier aimed at aligning cleft lips to each other, creating more favourable conditions for lip closure surgery. Follow-up studies could not support strong evidence on advantageous growth results in patients receiving this treatment; therefore, it has not been much preferred recently. Primary surgical lip repair restores the continuity of the mucous membrane, skin, and perioral musculature in the infant’s cleft lip with a single procedure. Definitive lip repair is usually achieved by the time the infant is 3 to 6 months old, while repair of the palate is typically delayed until 12 months to 2 years of age. Palatal repair is anticipated by hardness in speech development of the patients, which typically evolve around this age. At the same time, scarring of the surgical area will prohibit further improvement, impeding transversal and sagittal development of the maxilla. The narrow and short maxilla developed in this way is going to initiate crossbite of the anterior and posterior teeth at a later age.

**PRIMARY DENTITION STAGE**

Primary dentition developed by 2 to 3 years of age is already characterized by the main features of dentooskeletal disorders expected later. The facial soft tissues may mask the underlying skeletal deficiency of the midface in young children. Parallel to growth, redistribution of facial soft tissue proportions is getting to unmask the skeletal discrepancy, which is compensated by dental position temporarily. Therefore, retro-inclination of mandibular incisors and proclination of the maxillary incisors are both typical accompanying the phenomena of the anomaly in this period. In cases of a bilateral cleft, anterior medial prominence of the maxilla (premaxilla) frequently extrudes in front of the lateral plate, and their closure leads to the formation of an extremely narrow dental arch.
Bilateral cleft lip and palate (a., extraoral; b., intraoral; c., occlusal) in mixed dentition period

Early correction of both transversal (lateral crossbites) and sagittal (frontal crossbite) deformities is highly recommended; however, undergoing orthodontic treatments in primary dentition stage is affected by several age-related aspects as follows:

• the ability of the patient to cooperate
• the severity of the malocclusion
• timing of secondary bone grafts
• the need for future orthodontic treatment.

Contemporary opinion recognises an essential need for orthodontic treatment in the early mixed and permanent dentitions in case of patients with a cleft. At the same time, no strong evidence supports long-term results of treatments stimulating growth started at the primary dentition stage. Taking the age-related characteristics into account, long-lasting orthodontic treatment does not promise unambiguous improvement and should be considered rather disadvantageous by delaying speech development and adequate nutrition. Indeed, in line with the above-mentioned cooperation problems, it could even jeopardize subsequent, already inevitable, treatment. Nowadays, modern treatment strategies put emphasis mainly on the adequately structured and performed maxillofacial corrective surgery in order to provide an appropriate basis for any subsequent orthodontic treatment.

MIXED DENTITION STAGE

At this age, the further growth of the craniofacial complex often accentuates a previously mild skeletal discrepancy. This period is characterised by the eruption of the permanent incisors adjacent to the cleft site, which can be performed in different ways. Lateral and central incisors are frequently characterised by deviations in position, moreover by difference in form, size, or even number (aplasia or supernumerary teeth).

Bilateral cleft lip and palate (a., extraoral; b., intraoral) in mixed dentition period

The lateral crossbite having developed earlier consolidates through the eruption of lateral teeth. In this period, the treatment of lateral crossbite is the main challenge in order to ensure further transversal growth of the maxilla.
Unilateral cleft lip and palate (a., extraoral; b., intraoral; c., occlusal) in mixed dentition period

Elimination of discontinuity in the cleft-side alveolar ridge is crucial in terms of preventing further teeth eruption problems. Timing is mainly determined by root development of the cleft-side canine. According to the recent knowledge, permanent canine root should be developed at two-thirds at the time the graft is placed. This oral surgery intervention offers the following benefits for the patient:

• provision of bone support for unerupted teeth and those ones adjacent to the cleft
• closure of oronasal fistula (duct between oral and nasal cavities)
• support and elevation of the alar base on the cleft side
• construction of a continuous alveolar ridge, which is essential for moving adjacent teeth and/or for implants to be placed during adulthood
• achieving stabilisation and some repositioning of the premaxilla in patients with bilateral cleft.

Movement of adjacent teeth should be delayed until 2 to 6 weeks following the surgical intervention. Early movement of the roots into the grafted bone appears to promote the integration and survival of the implanted bone tissue.

PERMANENT DENTITION STAGE

With the eruption of the canines and premolars, the permanent dentition is established. During this time, the adolescent growth spurt and onset of puberty occurs. In this period skeletal discrepancy becomes accentuated, and facial appearance and occlusal relationships deteriorate. Impairment of speech is also characteristic, which may intensify further hypernasality being typical of patients with a cleft.

Both facial appearance and speech characteristics impose serious psychological burden on the adolescent patients with a cleft; therefore, installation of necessary braces and completion of treatments are particularly difficult to perform. The interdisciplinary treatment of the acquired deformities cannot be provided unless a very close step-by-step collaboration of orthodontists, cranio-maxillofacial surgeons and speech therapists is provided.

Precise forecast of direction and pace of continuous growth tendency is essential in terms of the planned treatment. Conservative mandibular-orthopaedic interventions may be successful in this period; however, it requires close cooperation from the patients. It is vital to establish the expected growth of the patient as precisely as possible as it determines whether the movement of teeth itself would be enough in the correction, and the result would be stable or not.

If further growth may occur after the treatment, orthodontic treatment should also serve the preparation of maxillofacial corrective surgical interventions. By planning any surgical intervention, particular attention should be paid to functions of soft tissues located in the operation field with special care on their role in speech. One of the main objectives of orthodontic surgical treatments is to keep and increase, if necessary, the results of earlier soft tissue correction interventions.

Orthodontic treatment being part of surgery preparation is always performed by fixed appliances, and lasts for 12–16 months at least. Operation planning is preceded by a joint consultation with the patient together with an orthodontist and a cranio-maxillofacial surgeon.
Unilateral cleft lip (a., extraoral; b., intraoral) in permanent dentition period at the time of dysgnathic surgical planning

Determination of the direction and extent of the planned mandibular movements is followed by a test operation performed on plaster models based on facial arch registration. The main purpose of this operation is to obtain surgical splints containing all movement information, since they are going to be used by the cranio-maxillofacial surgeon during the surgical intervention for fixing the new position of moved maxillary bones. The patient should go on wearing the installed surgical splints using intermaxillary elastics after the mobilisation as well, which is usually performed a couple of weeks after the operation.

Movement of maxillary bones is not conducted in a single step during distraction osteogenesis, but it is carried out step-by-step through screw-activation of the distractor after instalment surgery. It is also a surgical splint that determines the final position of the maxillary bones during such interventions. Both types of surgical interventions are followed by an additional orthodontic treatment lasting for at least 6 months; when new position of the moved maxillary bones is finally get stabilised, and a balanced occlusal position is going to be established.

When the growth of the patient will have finished, dentoskeletal treatments are usually completed by plastic surgery correction of facial soft tissues.

The final aim of the interdisciplinary treatment based on close cooperation is the full rehabilitation of the patient in terms of both dentofacial aesthetics and function of speech and occlusion.


**13.1. Treatment of pregnant patients**

Preventive measures for the oral care of pregnant patients are crucial to maintain the health of the mother. They are not only responsible for their own health, but that of the baby as well.

**FOETAL DEVELOPMENT**

During the first trimester the foetus is highly susceptible to injuries and malformations because all organ systems develop during this period.

Factors that can harm the embryo

- Infections (rubella, HIV, HBV, rubeola, varicella, syphilis, gonorrhoea). Severe periodontitis and chronic oral infections increase the risk for pre-term birth with low birth weight.
- Medications (LMWH, anticonvulsive agents, tetracycline, streptomycin, antiretroviral drugs, antineoplastics, psychotropic drugs and hormones)
- Drug and alcohol abuse
- Smoking
- Herbal dietary supplements

Developmental anomalies of the jaws, lips and teeth can be found in Chapter 1.2, 1.4, 2.1. In the second and third trimester the organs are fully developed and growth and maturation continue. The foetus is less susceptible to harm compared to the previous period.

**SPECIFIC FINDINGS IN PREGNANCY**

- Gingivitis and gingival enlargement (see Chapter 2.15.)
- Enamel erosion due to morning sickness with vomiting
• High risk of carious lesions because of frequent snacks and frequent sugar intake

**MEASURES TO PREVENT ENAMEL EROSION**

• Eat small amounts to prevent vomiting

• Take acid neutralising agents

• Avoid tooth brushing immediately after eating

• Use sugarless chewing gum

• Use topical gels with higher concentrations of calcium and phosphorous ions

**ASPECTS OF PATIENT CARE**

**Appointments**

• Professional oral hygiene appointments should be scheduled early in pregnancy

• Must pay attention to the emotional changes of the mother

• Shorter appointments because of frequent urination

**Treatment position**

• Discomfort can be felt if one position is maintained too long

• Backache

• Without supine or Trendelenburg positions: the major vessels are pressed by the foetus and the blood supply of the placenta will be affected. Patients must lie on their left side; elevate the right hip to displace the uterus to the left to prevent damage to the baby and for more comfort.

**Nausea and gagging**

• Avoid the use of strong smells, instruments that can elicit the gag reflex and radiographic films as much as possible

**ORAL CARE PERFORMED BY DENTAL HYGIENISTS**

• Dental biofilm control

• Prevention of periodontal diseases

• Smoking cessation

• Dietary instructions

• Dental caries control

**13.2. Role of sexual hormones in oral health**

Female sexual hormones are responsible for certain changes in the gums under physiological and non-physiological (hormone therapy, contraceptives) circumstances. During the menstruation cycle first oestrogen (follicular epithelial cells), then progesterone (in the yellow body, i.e. corpus luteum) is produced, which influences mucosal conditions in the uterus. The process is regulated by the hormone secretion of the hypophysis. (FSH, LH). These hormones cause changes not only in the uterus but in the oral mucosa as well.

Clinical signs can be noticed during major hormonal changes (puberty, menstruation, pregnancy, menopause) Hormonal changes result in the following: oestrogen influences the antimicrobial effect of salivary peroxidase enzyme, stimulates collagen metabolism and angiogenesis, induces the production of some growth factors, influences vascular responses and connective tissue degradation in inflammatory reactions together with
progesterone in reaction with other mediators. The latter factor explains why inflammatory reactions are more pronounced during hormonal changes. Progesterone decreases the level of IL-6, which is responsible for inhibiting MMP (matrix-metalloproteinase), which initiates connective tissue decomposing. It also stimulates the production of TIMP (Tissue Inhibitor of Metalloproteinases) in fibroblasts, decreases the level of TNF cytokines, which are responsible for defence mechanisms, as well as acute-phase proteins, which play a similar role. The result of these processes is more pronounced inflammatory reactions.

**Puberty and menstruation**

Some authors experienced increased inflammation of the gingiva with a higher bleeding index but unchanged oral hygiene (no changes in plaque scores). Some periodontopathogenic bacteria appear in women during puberty: increase in the number of Prevotella strains can be observed parallel to hormonal changes, since oestrogen and progesterone are secreted into the sulcular fluid, where they function as nutrients for the bacteria (replacing Vitamin K, necessary for reproduction).

During menstruation inflammatory signs are more pronounced, the amount of sulcular fluid increases. Although most women do not experience any changes in their gums during menstruation, some studies suggest increased bleeding accompanied by increased amount of sulcular fluid and hypermobility of the teeth.

**Pregnancy**

During pregnancy, the level of oestrogen and progesterone is increased from the embedding of the embryo till delivery. Plaque formation is increased in the second and third trimesters due to hormonal changes. Increase in probing depth and bleeding index can be observed, with a parallel increase in the amount of sulcular fluid. Clinical symptoms can be avoided through appropriate oral hygiene. In the second and third trimesters inflammatory parameters rise; however, the plaque level remains the same. The inflammatory parameters normalise three months after giving birth.

Pregnancy gingivitis is not rare according to the literature; it can affect one to two thirds of pregnant women. This is of great concern considering that some studies showed high correlation between premature delivery and oral inflammations.

In some cases fibrogranulomatous lesions may develop (pregnancy epulis, granuloma). In most cases they resolve after the delivery, or after breast-feeding at the latest. If they do not, they can be removed surgically, but only after delivery, otherwise they could reoccur.

**Menopause**

Hormone levels in ovaries drop, levels of IL-6 increase, which facilitates RANKL production, this way facilitating bone resorption processes (see chapter 2.15.1). This is one explanation for postmenopausal osteoporosis. It is more severe in smokers, due to increased oestrogen catabolism in the liver. The positive effects of hormone replacement therapy on osteoporosis confirm these facts.

**PERIODONTAL THERAPY**

Hormonal changes in puberty and pregnancy require a rigorous oral hygiene program. Instruction and motivation are of great importance, since the main reason for this inflammation is inadequate oral hygiene. Special aspects of treating pregnant woman were mentioned in the previous chapter.

**13.3. Elderly Patients**

Aging may be defined as a progressive decline of functional capacity that follows the period of reproductive maturity. Aging is an inescapable, genetically determined process, which begins well before old age, and gradually increases the likelihood of death. Aging causes self-regulatory mechanisms to deteriorate, which means that the body gradually loses its ability to adapt to its changing environment.

**AGE**

**Chronological and biological age**

Based on chronological age, we differentiate between the aging (≫55 years), the elderly (≫65 years), the aged (≫75 years) and the old (≫85 years).
Biological age is determined on the basis of the actual physiological state of the body. This is not a synonym of chronological age; a complete overlap between the two is not a rule. For instance, there are people who may be classified as biologically aging at the chronological age of 45, but others’ bodies fail to show the characteristic alterations even at the chronological age of 75.

**Functional age**

With a view to the general state of health and physical activity in the elderly, they can be classified as self-sufficient, partially self-sufficient and inactive persons.

**Primary and secondary age**

Primary age is determined without taking external influencing factors (like trauma, stress, chronic illnesses) into consideration. Secondary age is determined with these factors in mind.

**PHYSIOLOGICAL CHANGES OCCURRING WITH AGE**

**Musculoskeletal system**: over 40 years of age, the volume and functionality of the muscles decreases, and the same is true for the bones.

**Skin**: thinner, drier, the turgor is decreased, the sweat glands undergo atrophy, lentigo (age-related hyperpigmented spots) appears.

**Cardiovascular system**: hypertrophy of the left ventricle, decreased elasticity of the vessel walls, the blood supply of the individual organs drops, arteriosclerosis may occur.

**Respiratory system**: decreased vital capacity, less efficient gas exchange, poorer cough reflex, increased vulnerability to infections.

**Gastrointestinal system**: decreased amount of digestive fluids, slower peristalsis, less efficient absorption (this affects the absorption of both nutrients and medications).

**Nervous system**: poorer short-term memory, less efficient cognitive functions.

**Vision**: presbyopia, colour discrimination anomalies, disorders of light sensation.

**Hearing**: presbyacusis (age-related hearing loss), thicker and drier earwax, which further deteriorates hearing, a selective loss of higher frequencies, tinnitus.

**Endocrine organs**: decreased thyroid activity, increased sensitivity to cold due to the suboptimal functioning of thermoregulation.

**Immunity**: increased vulnerability to infections as a result of decreased B- and T-cell activity.

**THE ORAL CAVITY OF THE ELDERLY**

The oral and dental status of the elderly is significantly different from that of younger patients, which is a result of several factors acting at the same time.

**I. Soft tissues:**

Lips: dried, scaly lips, chronic chelitis (often due to systemic diseases), also chelitis angularis due to limited mouth opening and candidiasis (the latter as a result of dry mouth).

Oral mucosa: atrophic, less elastic, less vascular, sometimes hyperkeratotic (due to sharp tooth edges or missing teeth, which leads to an overload of the mucosa during chewing), petechiae, oropyrosis.

Tongue: glossopyrosis, atrophic tongue due to anaemia, varicositas linguae

Xerostomia: there are several known aetiological factors (e.g. diabetes mellitus, Sjögren syndrome, side effect of medications, irradiation).
Candidiasis oris: this is often seen in patients wearing dentures. This type of atrophic candidiasis often presents with cheilitis angularis, especially as a side effect of medications and after chemotherapy or irradiation.

II. Teeth

The teeth are characteristically discoloured (usually yellowish). The pulp chamber and root canals are narrowed. Calcifications are often present in the root canal. Attrition, abrasion and root caries are often encountered.

III. Periodontium

Bone: osteoporosis, decreased vascularity, poorer healing. Periodontitis is frequently seen in the elderly.

Cementum: it is thickening.

Gingiva: recession, decreased blood supply.

DENTAL AND DENTAL HYGIENIC INTERVENTIONS

First of all, a thorough medical history is of utmost importance. As life expectancy is on the rise, approach to the treatment of the elderly has generally shifted from a sort of palliative care to comprehensive dental care, involving regular oncological screening. Edentulousness occurs less frequently than earlier, therefore aesthetic and operative interventions have gained importance in this patient population.

Hindrances to dental treatment in old age:

Old-age patients tend to be less worried about their dental status, they have limited financial resources and sometimes they have difficulties getting to the dental office. These are all obstacles to the professional dental care of this population.

History:

As said before, the medical history should be comprehensive; that is it should mention all diseases/conditions and medications that may possibly influence dental outcomes. Examples for such conditions are: hypertension, diabetes mellitus, tumours, heart conditions, post-infarction states, disorders of blood coagulation, osteoporosis, heart valve prosthesis, hip prosthesis.

Medications potentially influencing the dental treatment/outcomes include: anticoagulants, antihypertensive drugs, chemotherapeutic agents, bisphosphonates, sedatives and antidepressants.

Antibiotic prophylaxis is indicated in a wider range of conditions: after irradiation, when heart valve or hip prostheses are present, for half a year following acute myocardial infarction (AMI) or if the patient takes bisphosphonates regularly. Although these are not absolute indications, antibiotic prophylaxis is recommended after chemotherapy and in steroid therapy.

Prevention:

Plaque control and debridement are first priority, as the decline of cognitive, mental and physical abilities usually brings about poorer oral hygiene. It should also be regularly checked if the denture-wearing patient is able to clean the dentures properly.

Examination of the biofilm, plaque removal:

Increasing recession and secondary cementum exposition, along with decreased saliva production favour biofilm attachment.

Removal of the biofilm is relatively easier from the surface of the roots, while tooth replacements (bridges, crowns) may be more challenging. Some systemic diseases (like Parkinsonism) can make plaque removal more difficult than normal.

Instruction:
Sufficient amount of time must be allocated for this; that is, it should never be left for the end of the treatment. Instruction must always be individualised, and, whenever possible, the patient’s carer/relative should be involved. The instructions should be repeated at each visit.

**Special recommendations:**

Different brushing techniques should be presented, and it must be emphasised that physical weakness can be compensated by the use of hard toothbrushes. Granular toothpastes should be avoided to prevent root abrasion. Desensitisation may become necessary wherever dentine tubuli are open. To manage xerostomia, ample liquid intake and citric acid-containing tablets are recommended, but special toothpastes, mouthwashes and gels are also commercially available. Electric toothbrush is strongly suggested.

**Periodontological treatment:**

Regular depuration and root smoothing/ scaling, root planning and polishing (talán jobb lenne, mint a depuration and smoothening). It is recommended to be done frequently, by quadrants, repeating instructions at each session. It is practical to do this together with caries control and prevention (e.g. application of local fluoride).

**Diet:**

The elderly often have to keep some kind of special diet, which can easily lead to deficient states (e.g. atrophic, burning tongue and/or cheilitis angularis in vitamin B deficiency). Irregular meals and poor quality food can also cause such problems.

It can be recommended that the elderly patient decreases calorie/cholesterol intake (as the energy needs are lower), but intake of a sufficient amount of proteins, minerals, vitamins (especially vitamin D), folic acid, calcium and water is crucial.

<table>
<thead>
<tr>
<th>Problem</th>
<th>Recommended solution</th>
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<tbody>
<tr>
<td>Problems of vision</td>
<td>pay attention to optimal lighting</td>
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<tr>
<td></td>
<td>instructions should be written in capitals</td>
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<tr>
<td></td>
<td>if the patient wears glasses, they should be worn when written instructions are explained</td>
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<tr>
<td>Problems of hearing</td>
<td>slow, loud, articulated speech</td>
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<td></td>
<td>be face to face with the patient while giving instructions- they often read the speaker’s lips</td>
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<tr>
<td></td>
<td>background music and rotary instruments should be turned off</td>
</tr>
<tr>
<td>Withdrawal, disturbances of complex thinking</td>
<td>frequent recall, communicate small units of information at a time</td>
</tr>
<tr>
<td>Difficulties of understanding</td>
<td>slow learning and complete inability to learn the instructions must be differentiated</td>
</tr>
<tr>
<td>Memory problems</td>
<td>in these cases the instructions must always be put in writing</td>
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<tr>
<td></td>
<td>repeat the instructions frequently</td>
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<td></td>
<td>the accompaniment should be involved</td>
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<tr>
<td>Limited physical abilities and secondary frustration</td>
<td>once the frustration has been noticed, offer positive reinforcement frequently</td>
</tr>
<tr>
<td>Depression</td>
<td>the depressed state should be noted and acknowledged, but still a positive approach is to be taken</td>
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Some frequent problems of the elderly and recommendations on how to address them

**13.4. Treatment of patients with cleft lip and palate**

Hygiene care of patients with cleft lip and palate is crucial for preservations of teeth and their supporting tissues and for their habilitation. Incidence of dental caries more frequent (3,5 times more decayed tooth surface)
Patient with cleft lip and palate compared to the healthy patients. Caries risk is more evident in the primary teeth among individuals with orofacial clefts, particularly the teeth adjacent to the malformations and molars.

Reasons which increase a risk of dental caries:

- longer oral clearance time of foods
- soft food consumption
- inadequate individual oral hygiene due to poor accessibility of tooth surface because of consequences of alterations
- fearing from pain and injury

Children treated with obturator have a 7.6 times higher prevalence of caries compare to the children without intraoral appliances. Increased risk of caries can be found at older patient with cleft lip and palate because of insufficient toothbrushing due to developmental anomalies and wearing orthodontic appliances.

**PREVENTION OF CARIOUS LESIONS AND PERIODONTAL DISEASES**

Good conditions of oral health may maintained by adequate oral hygiene incorporating Fones and the Bass technique (from age of adolescent stage) and interdental cleaning methods. More frequent appointments of professional oral hygiene are indicated. KEEP IN MIND: surgically repaired lip and the anatomy of cleft result in restriction of toothbrushing and self-cleaning ability of the mouth! Systemic and topical fluoridation, utilizing of xylitol-containing products and dental sealants may decrease a risk of carious lesions. Daily care of removable appliances is necessary for adequate oral hygiene, prevention of oral infections and preservation of teeth.

**ROLE OF DENTAL HYGIENIST IN THE ORAL CARE**

Dental hygienist has an important role in the treatment patient with left lip and palate. Dental hygienist should play role parental counselling, motivation, patient education, both presurgical and postsurgical oral care and take an important part in a continuous supportive treatment (professional oral hygiene). Dental team members must pay attention to prevention of cross-contamination by inhalation because the patient with cleft palate is more susceptible to infection of the upper respiratory tract and middle air.

**13.5. Maxillofacial prosthetics**

**THE PATIENT POPULATION**

Maxillofacial prosthetics serves the purpose of the correction of facial and intraoral defects of various aetiologies. Although the primary closure method of choice is surgery, it has its own physical limits (e.g. because of the localisation or size of the defect), beyond which a surgical solution would not guarantee an optimal outcome- especially in terms of quality of life. Such defects are managed with prosthetic methods.

From an aetiological point of view, the most frequently seen patients are:

1. patients after surgery and oncotherapy for cancers of the head and neck region
2. traumatised patients
3. patients with cleft lip or palate
4. patients with congenital developmental defects (like the lack of the outer ear in Goldenhar syndrome)

Regardless of the aetiology, prosthetic rehabilitation is recommended when the intraoral or facial defect is so large that it causes serious functional or aesthetic problems (Figures 5.131.).

Rehabilitation is practically the restoration of lost physical abilities. The affected abilities are usually swallowing, speech and eating.
Prosthetic rehabilitation is recommended when the intraoral or facial defect is so large that it causes serious functional or aesthetic problems.

1. Status after hemimaxillectomy (tumor resection) 2. Reconstructive results of an operation following a car accident 3. Distorted maxillo-mandibular relations in CPGS 4. Congenital lack of the external ear

**THE METHODS OF MAXILLOFACIAL PROSTHETIC REHABILITATION**

**Intraoral rehabilitation:**

**Maxillary defects:**

Hemimaxillectomy, which is the removal of the maxilla on one side is usually indicated when a tumor infiltrates the maxillary sinus. An iatrogenic cause may be avascular bone necrosis of patients receiving bisphosphonate therapy. In these cases the goal of the intervention is to remove the necrotic debris. As a result, the oral cavity can become continuous with the maxillary sinus, the nasal cavity, and in extreme cases (when enucleation of the eye is also necessary) even with the orbit. Such defects are covered by obturators. An obturator is a special prosthesis, which serves the dual purpose of covering the defect and replacing the missing teeth. In terms of tooth replacement, these can be conventional full dentures, combined prostheses and implant-based replacements. If implantation is the method of choice, the recommended sites are the premaxilla and the area of the tuber maxillae (Figure 5.132.).

**Palatal defect restored by obturator**

1. Palatal defect following tumor resection 2. Obturator with removable partial tooth replacement 3. Defect covered with an obturator

**Defects of the mandible:**

Operation of the tumours of the mouth floor, the mandibular gingiva or the mandible itself can result in a situation where the individual structures are hardly discernible (Figure 5.133.).
Postoperative status after tumour resection and reconstruction in the mouth floor

Sometimes the picture does not even resemble the anatomical situation. Naturally, the prosthesis has to be adapted to the actual circumstances, especially regarding its shape. Stability is of utmost importance, therefore it is recommended that the prosthesis should be fastened to an implant basis (Figure 5.134.).

Implant-supported prosthesis

1.-2. Postoperative status after tumour resection and reconstruction in the mouth floor Implant-based tooth replacement 3. The same replacement in the mouth

Extraoral rehabilitation:

Loss of parts of the face is caused dominantly by accidents or tumour therapy, and it rarely occurs as a congenital disorder (Figures 5.135.).
Such defects are managed with epitheses. Epitheses are made of silicon, and they are removable by the patient. Fastening can be done with special glue, but if the condition of the patient and the prognosis of the disease allow it, extraoral implants can be inserted to provide a firm basis for the epithesis (Figures 5.136). The latter solution significantly enhances stability and patient comfort, but it has to be kept in mind that extraoral implantation means additional surgical intervention, by which the duration of the rehabilitation process is prolonged until the integration of the implant, which means that the defect is covered months later than without implantation.

THE SPECIFIC PROBLEMS OF THE PATIENT POPULATION

1. As previously written, the oral cavity of these patients is often continuous with the maxillary sinus and the nasal cavity. This makes personal oral hygiene particularly difficult. Disinfectant mouth rinses and professional cleansing have a key role.

2. Eating, swallowing and speech may be compromised, and the defect can also be aesthetically disadvantageous. This can seriously affect the patient’s social life.

3. Trauma patients may receive an intermaxillary fixation device postoperatively, for a period of approximately 6 weeks. The device limits mouth opening, which hinders eating and personal oral hygiene. For more details on the care of such patients, please see the chapter on bed-bound patients.

4. More often than not, head and neck cancer patients come from poor homes, and they also smoke and drink excessively. These factors add up to poor oral hygiene. The importance of patient education cannot be overemphasised in this group.

5. Some of these patients also receive chemotherapy, which results in immunosuppression. In turn, the chance of bacterial, viral or fungal infections is increased, abscesses and bleeding of the gums should be expected. Chemotherapy is a contraindication of any kind of dental intervention which may cause bleeding. Also, no such intervention should be done until the blood test values of the patient return to normal after the chemotherapy.
6. Pre- or postoperative irradiation causes problems in two ways:

- a. Irradiation causes multiple abscesses in the oral cavity. Local anaesthesia and disinfection should be applied (CHX or Listerine). Patients should be instructed how to apply hydrogen peroxide at home for everyday use.

- b. Although the abscesses disappear when radiation therapy is over, xerostomia (dry mouth) can persist, as irradiation also affects the salivary glands. This is usually a longer-term effect, which sometimes requires the application of antimycotics, so as to fight candidiasis. Dry mouth leads to an increased risk of caries, especially in the cervical region. This makes patient education and professional hygienic measures even more important.

**THE ROLE OF THE DENTAL HYGIENIST**

The circumstances laid out above indicate that in this population professional oral hygiene (i.e. the oral hygienist) has an especially important role. The dental hygienist can support the work of the oral surgeon/prosthetist in the following ways:

1. **Education regarding the hygiene of the replacement**

   At the regular check-ups not only the oral hygiene, but also the hygiene of the replacement must be checked.

   The replacement should be cleaned at least two times a day, but preferably after each meal with a toothbrush or a specially designed replacement cleaning brush. Soap and granule-free toothpaste can be used. Two times a week, the replacement should be put in a disinfectant solution for a few hours. If plaque is formed on the replacement, it should be removed with a depurator or ultrasonic instrument.

   In special cases, when no teeth of the patient’s own are left, the use of adhesives is unavoidable. The task of the dental hygienist is to teach the patient how to apply the adhesive on the replacement and how to remove it. Patients tend to be quite prejudiced against adhesives, because they think that dentures fastened by adhesives are not safe or they do not function properly. Care must be taken that the patient understands that the anatomical situation allows only this type of fastening, and to ease the anxiety caused by various misconceptions.

2. **Instruction, motivation**

   The necessity and importance of regular control and check-ups must be emphasised at all visits. Check-ups allow the dental hygienist to assess the efficiency of personal oral care. Plaque control is a regular task. If something is not done properly, corrections (and re-education) should be performed.

3. **Professional oral hygiene, advanced caries prevention**

   Plaque control and depuration must be regular (2-3 month recall), but it must not be forgotten that irradiated patients require special attention: as mentioned before, interventions that can cause bleeding are contraindicated in irradiated patients- if for some reason such an intervention becomes necessary, it is to be done only after consultation with the patient’s dentist, and preferably under antibiotic prophylaxis.

4. **Tobacco cessation support**

   Sadly enough, head and neck cancer patients find it difficult to quit smoking, even after the diagnosis has been set up. Thorough patient education on the connection between smoking and cancer, and on the local and systemic effects of smoking in general can also be a task for the dental hygienist, just as supporting the idea of cessation and providing information on where the patient can get help with giving up smoking.

5. **Supporting therapy**

   These tasks should be done regularly, preferably at each check-up.

### 13.6. Care of Oral Surgical Patients

**ORAL SURGICAL PATIENTS**

All patients undergoing oral surgical interventions either on an outpatient or hospital basis are considered oral surgical patients.
The interventions performed most frequently on an outpatient basis (i.e. dentoalveolar surgery) are:

- simple or complex extraction, maxillary sinus closure, cystectomy, resection, incision and drainage, removal of salivary stones, removal of wisdom teeth and impacted teeth in general, implantation, preprosthetic and periodontal surgery.

The conditions treated most frequently on a hospital basis (i.e. maxillofacial surgery) are:

- head and neck trauma, tumours of the head and neck region, orthognathic surgery, cleft lip and/or palate, and diseases of the salivary glands.

**POSTOPERATIVE STATES REQUIRING SPECIAL ORAL HYGIENE AND CARE**

They occur most frequently after major operative interventions. For instance, palatal defects are frequently seen in orthognathic surgery, and in cases of trauma, and they require special attention both in terms of the patient’s diet and oral hygiene.

**THE ROLE OF THE DENTAL HYGIENIST IN THE CARE OF ORAL SURGICAL PATIENTS**

**Preparation: planning, counselling and care before the intervention**

Before any oral surgical intervention, attention of the patient should be called to the following:

1. The proper timing of the last meal before general anaesthesia.
2. Alcohol must not be consumed, and any regular medications should be taken before the intervention (except for anticoagulants), or brought along to the hospital.
3. If accompaniment by a family member seems necessary.

Enhancement of oral hygiene and reduction of the oral microflora is of utmost importance before oral surgical interventions. For that reason, depuration may be necessary a few days before the planned intervention (or even the same day), and immediately before the intervention (but after the initiation of anaesthesia), rinsing with 2% chlorhexidine for 2 minutes.

**Instructions**

They should be given a few days prior to the intervention. If plaque or biofilm is present on the teeth, the patient’s attention should be called to it, and more efficient tooth cleaning techniques should be taught (including the right technique of brushing, and the right type of toothbrush and flossing).

**Postoperative care**

It is advisable to have a printed list of postoperative instructions at hand, a copy of which can be given to the patient.

1. Bleeding control: The gauze tampon must be kept in the mouth for 20–30 minutes. If there is still some leakage after taking it out, another one may be put on the wound for further 30 minutes.
2. Drinking is allowed while under local anaesthesia, but eating is not advisable, as this can lead to bitten wounds on the tongue and the buccal mucous membrane.
3. No disinfectant rinsing (like chlorhexidine) is allowed for approximately 24 hours post op. This may lead to the decomposition of the thrombus.
4. Careful personal oral hygiene cannot be overemphasised, but attention should also be called to the importance of a gentle approach to the operative area.
5. Pain control: The patient should be instructed on the proper use of over-the-counter analgesics (especially their dosage), and if it seems necessary, prescription only medication should be prescribed.
6. Cooling: it should prevent the eventual oedema occurring as a result of extreme soft tissue damage or bone drilling. Special cryogels are available, but deep-frozen products wrapped in a piece of cloth, placed over the affected area for about 20 minutes for two or three hours can serve this purpose just as well.

7. Dietary considerations: Smoking, alcohol consumption and consumption of small seeds (like sesame seeds) must be avoided for two days after the intervention. At the same time, the diet should contain ample amounts of vitamins and protein.

8. Complications: extreme pain, massive bleeding, or fever.

The patient should be aware that these might occur, and the availabilities of the nearest caregiver on duty must also be provided, should the patient experience any of these.

13.7. Immunosuppressed patients

**IMMUNOSUPPRESSION, IMMUNODEFICIENT STATES**

Immunodeficiency is a term to describe states in which the immunity of the body is (partially) compromised, and therefore, the risk of infection is increased. It is not only that the risk of infection is increased in these states, but already existing infections might progress more rapidly and to more serious stages, eventually to become life-threatening. It is also very characteristic of these states that otherwise harmless microbes (like Streptococcus epidermidis, Corynebacterium jeikeium, and various Bacilli) become pathogens. In the following, we give a brief summary of the diseases that can cause immunodeficiency.

**Insufficiency of the humoral immunity**

This is often a congenital state, but it can occur in myeloma multiplex, chronic lymphocytic leukaemia and after splenectomy (resection of the spleen). The leading laboratory finding is low antibody titre.

**Granulocytopenia (Neutropenia)**

This occurs after bone marrow transplantation, accompanying solid tumours, in acute leukaemia and as a result of myelosuppressive chemotherapy. When the neutrophil count drops below 1000/µL, the risk of infections is increased, but under 100/µL, the situation becomes life-threatening and requires hospitalisation.

**Insufficiency of the cellular immunity**

Patients suffering from AIDS, Hodgkin’s lymphoma and those taking immunosuppressive medications belong into this group. The latter group includes patients having undergone transplantation, receiving antitumour therapy and high-dose corticosteroid therapy (e.g. in asthma and chronic inflammatory and autoimmune diseases).

**Bone marrow transplantation (haemopoietic stem cell transplantation)**

Serious neutropenia is characteristic in the first three weeks following the transplantation. The risk of both Gram-negative and Gram-positive infections is increased, along with viral (especially herpes simplex) and fungal (especially Candida albicans) infections. Complications might occur, however, even three months after the intervention. In this period Varicella zoster, Cytomegalovirus and Aspergillus infections are often seen.

**Patients after organ transplantation**

Immediate postoperative infections mostly affect the transplanted organ itself. Infections occurring 2 to 4 weeks after the transplantation are usually nosocomial or iatrogenic (i.e. related to the hospital treatment and stay). Examples are wound infections and infections related to foreign bodies (like drains). In this period, Herpes simplex, Varicella zoster, Cytomegalovirus, Candida albicans and Toxoplasma gondii frequently cause infections. After six months, when immunosuppression is decreased to a maintenance dose, infections occurring in the normal population are to be expected.

**Miscellaneous immunodeficient states**

Some patients do not suffer from immunodeficiency in a classical sense, that is, their immunocompromised state is secondary to some other condition. They might have suffered a severe burn or trauma or undergone invasive
interventions. Notably, diabetics also belong to the immunodeficient patient population requiring extra care in this respect.

**ACQUIRED (IATROGENIC) IMMUNOSUPPRESSION**

Acquired immunosuppression is the state when the activity of the immune system is suppressed by medications for some specific therapeutic purpose. In a range of inflammatory diseases, in autoimmune diseases, after organ transplantation and in tumour therapy such measures are necessary. What follows is a brief summary on the most important groups of immunosuppressive medications.

**Corticosteroids**

In the everyday language this is often referred to as 'steroid therapy'. The primary effect of corticosteroids is the suppression of the inflammatory reaction, which is achieved by the regulation of several cellular processes. Immunosuppression is achieved by the suppression of the proliferation of the lymphocytes and the cellular immune response.

**Cytotoxic agents**

The most frequently used cytotoxic agents are the antimetabolites and cyclophosphamide. Cyclophosphamide damages cells by forming DNA crosslinks, but it also suppresses both B- and T-cell immunity and inflammation.

**Antimetabolites**

The most important antimetabolites include methorexate, azathioprine and leflunomide. Azathioprine suppresses the proliferation of T- and B-lymphocytes and macrophages. Methorexate specifically suppresses quickly proliferating cells (like tumour cells), but it also antagonises inflammation and both the cellular and humoral types of immunity. Such suppression is always necessary after organ transplantation. Without this, graft-versus-host rejection would occur in almost 100% of the cases, but by the application of immunosuppression, this can be reduced to as low as 20–30%.

**Cyclosporine**

An agent of fungal origin, cyclosporine is the first choice after organ transplantation. It suppresses both the proliferation of T-lymphocytes and the T-cell mediated immune response. However, it is toxic to the liver and the kidneys.

**Tacrolimus (or Fujimycine)**

This agent is about a hundred times more effective than cyclosporine, for which it seems to gradually supersede cyclosporine. Tacrolimus targets T-cell immunity, too. Side effects include a rise in blood glucose, as well as renal and nerve toxicity.

**ORAL SYMPTOMS IN IMMUNOSUPPRESSION**

As a result of the seriously weakened immunity, canker sores and abscesses occur all along the gastrointestinal tract. These are usually deep and tend to heal only very slowly. Frequent recurrences are also to be expected.

**THE PROTOCOL OF DENTAL CARE IN IMMUNOSUPPRESSION**

If sores or abscesses are present, the treatment of these must be the first priority. By the application of local agents, epithelisation, disinfection and analgesia are done. No dental intervention should be performed until the sores have healed. An ongoing immunosuppressive therapy is also an absolute contraindication of dental interventions. Should emergency care become necessary, oral antibiotic prophylaxis must be administered.

**THE ROLE OF THE DENTAL HYGIENIST**

**History taking**

A detailed patient history should be recorded in the dental file, including the specific immunodeficient state and all systemic diseases, regularly taken medications and, of course, the immunosuppressants.
**Intraoral examination**

The first priority also here is to check for sores on the mucosa. These may be so painful that the patient cannot tolerate the dental hygienic treatment either. In such cases, the treatment should be postponed, similarly to other dental treatments.

**Plaque control**

Given the burden that the main disease puts on these patients, they often fail to pay enough attention to their personal oral hygiene. For this reason, the oral hygienist must put emphasis on plaque control/home care at each visit.

**Depuration, root smoothing/planning**

Before any intervention that may involve bleeding, it is advised to consult the GP or the internist. Although immunosuppression is not an absolute indication of antibiotic prophylaxis in these cases, it is recommended that it should be administered anyway.

**Instruction**

Beyond the general oral hygiene education, care must be taken that the patient knows how sore disinfection is performed with 3% hydrogen peroxide at home.

**13.8. Bed-bound patients**

**DEFINITION**

From an oral hygienic point of view, it is practical to divide bed-bound patients into groups based on their different needs. We recommend the following classification:

**Patients on the maxillofacial ward**

These patients require the most attention, as they have surgical wounds, mostly intraorally, the healing of which necessitates excellent oral hygiene. Preoperative oral hygiene is not less important. This is further discussed in the last subchapter.

**Patients on other chronic wards (e.g. internal wards, homes for the elderly)**

These patients also need help with otherwise simple issues like tooth brushing and mouth washing. If the patient wears dentures, care must be taken that these are disinfected regularly.

**Bed-bound patients at home**

Generally the same applies as under B, but here the various tasks are not performed by professional patient care personnel, but the family members or by home care personnel.

**SPECIAL PROBLEMS**

Bed-bound patients are usually in a very poor condition, and they can pay very little attention to their oral health- if they can at all. Most of the time they are limited in their movement, which means that they cannot brush their teeth or clean their dentures alone.

**THE ROLE OF THE DENTAL HYGIENIST**

The specific aspects mentioned here apply to maxillofacial wards. We chose this because it covers everything that has to be observed on any other ward too.

**Difficulties**

1. **Gingivitis, periodontitis**

   If normal salivary flow or the self-cleaning effect stemming from the movements of the tongue, lips and facial muscles is reduced, gingivitis develops in 9-19 days.
b. Loss of appetite

Patients on a mashed food diet lose weight, which leads to weakened immunity.

c. Mouth opening difficulties

Postoperative wound healing, intermaxillary fixation, or sometimes the presence of a tumour can limit mouth opening. These states can be really challenging in terms of optimal oral hygiene.

Special intraoral devices, states


b. The period after the removal of special devices: A few weeks after the removal of the intermaxillary fixation, and if the grade of mouth opening allows it, professional calculus/plaque removal and root smoothing is necessary. Regular follow-up is recommended.

Dietary measures

Weight loss due to an unsatisfactory diet and insufficient calorie intake is often seen in bed-bound patients.

a. Nutrients: In the postoperative period, the body covers the nutrient needs of tissue remodelling and wound healing from the food intake. Beyond the essential elements, protein intake is crucial, just like the intake of Vitamins A and C, Calcium and Phosphorus.

b. Means of nutrition:

1. Straw: This is useful when mouth opening is limited, or the patient cannot maintain a sitting position.

2. Spoon feeding: This is the method of choice when the patient cannot move their arms (e.g. after stroke).

3. Feeding tube (nasogastric tube): after extensive head/neck operations, in head/neck trauma and for burn patients.

c. Smashed food diet: Limited movement of the lips and the tongue and limited mouth opening may indicate this, but it is also a good intermediary step between liquid and solid diets, especially because it means less mechanical irritation for the healing tissues than the immediate reintroduction of solid food.

Individual oral hygiene

a. Tooth brushing and mouth washing: Individual tooth brushing without help should be encouraged as soon as possible after the operation (with a soft toothbrush). In the first few days, mouth washing with chlorhexidine or some fluoride-containing solution is indispensable.

b. Professional oral hygiene and motivation

13.9. Patients with physical disabilities

DEFINITION OF DISABILITY, PREVALENCE, ITS RELATION TO DENTAL CARE

Under ADA (The Americans with Disabilities Act of the United States), an individual with a disability is a person who: (1) has a physical or mental impairment that substantially limits one or more major life activities; OR (2) has a record of such impairment; OR (3) is regarded as having such impairment.

The rate of disabled persons in the population is one in five, and their number is increasing, given the higher life expectancy due to advanced medical care, regardless of whether the disability is congenital or acquired.

For the purposes of dental planning, it is useful to classify the disabled in three categories, that is, those who can be treated on an outpatient basis (e.g. lack of one arm), patients requiring a wheelchair and bed-bound patients.
1. **Congenital malformations**: cleft lip and palate, disorders of mandibular development, malocclusion, positional irregularities of the teeth, amelogenesis imperfecta and dentinogenesis imperfecta.

2. **Injuries of the lips and oral cavity**: attrition, damage to the teeth proper, dental avulsion, injuries of the lips and tongue (e.g. during an epileptic attack).

3. **Facial paralysis**: in unilateral paralysis, symmetrical chewing is impossible, plaque is formed more rapidly on the affected side.

4. **Malocclusion**: often seen as part of developmental disorders. Skeletal and muscular deformity, macroGLOSSIA, lack of dental buds, tongue thrust swallowing, mouth breathing.

5. **Iatrogenic alterations**: gingival hyperplasia caused by hydantoin therapy (makes plaque removal difficult), xerostomia, mucositis due to chemotherapy or irradiation, multiple abscesses in the oral cavity, bleeding, petechiae.

**FREQUENTLY-SEEN DIFFICULTIES IN SPECIAL NEEDS DENTISTRY**

Getting the patients to the dental office is often a problem in itself. Many physically disabled people are bound to a wheelchair. Hence, public caregivers are legally obliged to provide disabled access to their facilities, including parking lots, pavements, entrances, toilets and corridors. This applies to the interior of the dental office too, where a wheelchair-bound patient should be able to move around without difficulty. If possible, a mobile headrest and a wheelchair with tiltable back is used, so that the patient does not have to get out of the chair for the treatment. If such conditions cannot be provided, the wheelchair-bound patient should be treated in a hospital setting.

These patients rely completely on their families, and therefore they usually arrive at the office with members of their family.

**SPECIAL TASKS, TREATMENTS AND INSTRUCTIONS IN PHYSICAL DISABILITIES**

**Pre-treatment consultation**

Information should be acquired on what type and what degree the patient’s disability is, on how the patient will arrive and whether a nurse is required. If the patient is bed-bound and cannot be mobilised, treatment in a hospital ward is recommended, possibly under general anaesthesia (however, the degree of sedation must be determined on an individual basis). It should also be known if the patient has any other (systemic) diseases and whether (what) medications are taken regularly.

**Timing**

The treatment of such patients is preferably carried out in the morning, without the patient having to wait for a longer time. The treatment should be allocated more time than an average treatment, partially due to the inherent difficulties and also because complications occur more often in these cases.

**Preparation**

Pre-medication if necessary (antibiotics, sedatives, etc.). Bite blocks or finger protection so as to avoid the injury of the treating person.

**Prevention, patient education**

Teaching of different brushing techniques, plaque-control and local fluoridation. Assessment of cooperation factors (e.g. lack of motivation, unsatisfactory physical condition for personal oral hygiene) and the extent to which the patient requires help with personal oral care (full, partial, none) via guided questions or questionnaires. The accompanying person should be involved in all steps.

Special attention must be paid to the toothbrushing technique of the disabled person. For instance, there are patients whose arm is dysfunctional, so they hold the toothbrush with difficulty. Specially designed toothbrush accessories are commercially available for a range of conditions. There are toothbrushes that can be fastened on the back of the hand, there are ones with extra long shafts, or even rubber balls in which regular toothbrushes can be inserted, so that even patients who cannot close their fingers can hold the toothbrush alone. Dental floss
can be used with the help of a dental floss frame. The aim is always to find the solution by which the patient can carry out the widest range of oral care techniques alone.

If the patient wears removable dentures, a regular check on the efficiency of cleaning is of high importance.

**Plaque-control, depuration, local fluoridation**

These should be done in one session if possible, so that the patient is not exposed to the unpleasantries of several visits.

**Instructions to the accompanying person**

It must be emphasised that the single most important aim is that the patient carries out his or her personal oral care alone - as much as possible. The environment of the patient should instruct the patient regularly to do so.

At the same time, if the patient is dependent on help, instruction should be given on the proper way of brushing for another, disabled person, including the proper positions for holding the patient’s head, etc.

### 13.10. Patients with mental retardation

**MENTAL RETARDATION: DEFINITION, FORMS, AETIOLOGY**

The mentally retarded (also: mentally disabled) are mostly children and young adults, whose mental capacity is significantly lower than that of an average person of his/her biological age and significant behavioural, sensory or expressive difficulties are also present.

Mental retardation may be seen as part of approximately 200 different conditions. In most cases, mental disability has prenatal origins, but it can be perinatal or postnatal as well. In many cases it is not possible to identify the aetiological factor(s).

**Classification:**

Levels: mild, moderate, severe, total. The classification is based on standardised IQ tests.

- mild: IQ 50-69,
- moderate: IQ 35-49,
- severe: IQ 20-34, total: under IQ 20.

**THE SYSTEMIC AND ORAL SYMPTOMATOLOGY OF MENTAL RETARDATION**

**Bodily symptoms**

These are determined by the type and severity of the condition. Characteristic alterations include differences in the shape and size of the face and skull, facial asymmetry, deformities of the external ear, the face and the nose, and the non-physiological curvature of the vertebral column.

**Oral symptoms**

- Lips: full, enlarged lips with bite marks.
- Dentition: delayed eruption, lack of buds, amelogenesis imperfecta, increased risk of caries.
- Periodontium: increased vulnerability to infections.
- Bad habits: bruxism, mouth breathing, tongue thrust.
- Bad oral hygiene

**DIFFICULTIES OF MAINTAINING ORAL HYGIENE IN THE MENTALLY RETARDED**
Mentally compromised patients usually have serious sensorimotor problems, therefore their personal oral hygiene is particularly bad as they cannot even brush their teeth alone. More often than not, their environment also fails to pay enough attention to this question, as it seems to be a minor problem as compared to the host of other difficulties accompanying the patient’s general state. For this reason, the dental status of most of the mentally compromised patients is extremely poor.

Their nourishment also tends to be pretty one-sided, as they accept a limited range of foods, and they usually fancy sweets and caries-promoting food. This way, caries-prevention, which would be especially important in this age group, becomes almost impossible.

Compliance is almost impossible to expect, which leads to most interventions having to be carried out under general anaesthesia. This also necessitates a team of anaesthetists who have experience with this patient population, which, in turn means that such interventions can only be carried out in a hospital, where parents’ accommodation has to be arranged, too.

Informed consent of the parent/custodian is crucial.

In milder forms of retardation, sedation with nitrous oxide might be enough, but this should not be done without the presence of an anaesthetist either.

Due to the poor compliance and dental status, one must find an optimal intervention, which takes the lowest number of sessions and provides a long-term solution. Having that in mind, extraction often remains the only method of choice.

**THE ROLE OF THE DENTAL HYGIENIST IN THE TREATMENT OF THIS PATIENT POPULATION**

In mild retardation or autism it sometimes happens that the patient cooperates relatively well. Such instances should be used for professional oral care, which can be the task of the dental hygienist. Parents should always be involved (in contrast to an average paediatric intervention). If patient compliance is good, the following are recommended:

- „Introduction” of the patient to the instruments and the dental office.
- Plaque-control: the task here is mostly the education of the parent or custodian regarding proper oral care by which plaque formation can be prevented (electric toothbrush+ antiseptics use, CHX, Listerine).
- Scaling and root planning.
- Local fluoridation (5% oldattal öblítés, DH -ecsetelés).

The importance of empathy cannot be overemphasised in these cases. The assistant or dental hygienist working with this population of patients is optimally an experienced one with advanced skills and willingness to work in a team. This latter is of key importance, as the anaesthetist, the anaesthetic assistance, the dentist, the dental hygienist and also the parents must form a team to achieve optimal results.

**13.11. Alcohol and drug dependence**

**THE SYSTEMIC EFFECTS OF ALCOHOL**

Symptoms of alcohol dependence:

1. An irresistible urge to consume alcohol.
2. Excessive alcohol consumption.

Regular alcohol consumption damages the physiological functioning of almost all organs and organ systems.

1. Liver: fatty liver, cirrhosis, hepatitis, early fibrosis.
2. Immunity: increased vulnerability to infection (pneumonia, TB).
3. Gastrointestinal system: increased gastric acid secretion, gastritis, oesophagitis, rupture of the oesophagus, diarrhoea, weight loss, vitamin deficiency.

4. Disorders of food intake: loss of appetite, malabsorption, various deficiencies.


6. Cancer: regular alcohol intake (esp. when accompanied by smoking) increases the risk of cancers of the gastrointestinal system, the respiratory system and the head and neck region.

7. Nervous system: both the peripheral and the central nervous system are damaged. Wernicke-Korsakoff syndrome.

8. Reproductive organs: these are damaged because of the dysregulation of the endocrine system.
   - Female: disturbances of the menstrual cycle, lack of ovulation, early menopause.
   - Male: testicular atrophy, low levels of testosterone, infertility.

**TREATMENT OF ALCOHOL DEPENDENCE**

The treatment is aimed at total abstinence, and the key to success is the patient’s inner motivation. Usually psychotherapy is combined with pharmacotherapy. Methods fall into one of the following categories:

1. Early intervention (before the dependence has developed);
2. Detoxification: treatment of acute intoxication and withdrawal symptoms;
3. Pharmacotherapy;
4. Rehabilitation (behavioural and group therapies);
5. Follow-up

**THE EFFECTS OF THE MOST COMMON DRUGS ON THE BODY**

The most frequently used drugs may be categorised as:

1. Cannabis derivatives (e.g. hashish, marijuana).
2. Hypnotics and sedatives.
3. Dissociative anaesthetics (e.g. phencyclidine or PCP, originally designed for general aesthetic use).
4. Hallucinogenic drugs (e.g. LSD).
5. Opiates (morphine, heroine).
6. Stimulants (e.g. cocaine, methamphetamine).
7. Miscellaneous (e.g. aromatic compounds for sniffing, anabolic steroids).

Regular drug abusers may suffer damage to the following organs and organ systems:

1. Cardiovascular system (e.g. hypertension, vasoconstriction, arrhythmias, angina, sudden cardiac death).
2. Nervous system (e.g. stroke, intracerebral haemorrhage, amnesia, delirium, dementia, mood disorders, sleep disturbances).
3. Gastrointestinal system (nausea, vomiting).
5. Liver damage.


7. Respiratory system (asthma, bronchitis, lung cancer).

8. Foetal damage

9. Infection (abscess and cellulitis at injection sites, osteomyelitis, HIV)

THE DEPENDENT PATIENT IN THE DENTAL OFFICE

History

Personal history taking is crucial when one suspects dependence. Even more importantly, extra attention should be paid to the patient’s pharmaco-history. It must also be noted that these patients often have several diseases at the same time. In elderly alcoholics, regularly taken medications must be recorded, and it should never be forgotten that they might have memory problems and that they belong to a high risk group because of their various diseases.

A variety of questionnaires is available to obtain information on dependence in a discreet way. However, good personal rapport is of utmost importance.

Clinical examination

A. Extraoral part:

Tell-tale signs of alcohol abuse:

- breath smelling of alcohol and tobacco,
- a tremor of the hands, tongue and eyelids (withdrawal symptom),
- ruddy complexion,
- mild icterus due to liver dysfunction,
- red and baggy eyes,
- various smaller injuries.

Tell-tale signs of drug abuse:

- a readily visible neglect of personal hygiene,
- frugal clothing,
- long sleeves to cover up needle marks,
- dramatic weight loss,
- abnormal pupil size (either too wide or narrow),
- subconjunctival injection of the eye (and sunglasses to cover it),
- needle marks, continuous sneezing,
- yawning,
- drowsiness,
- an empty look,
- behavioural disturbances and hallucinations
B. Intraoral part:

Parotid glands: enlarged.

Lips, tongue, mucous membrane: cheilitis angularis (due to malnutrition), xerostomia, glossitis, vulnerable mucous membrane.

Gingiva: bad oral hygiene, plenty of calculus, acute gingivitis, increased bleeding tendency, direct lesions of the mucous membrane (in cocaine abuse), periodontal infections.

Teeth: attrition (due to bruxism), chipped or broken coronal part, erosion (due to regular vomiting and alcohol abuse), increased vulnerability to caries.

Oral hygiene is usually very poor, as these patients spend all their energy and financial resources on their dependence. Most often they see a dentist only when they actually suffer from an acute (and painful) problem.

Treatment

Preparation: antibiotic prophylaxis, antiseptic mouthwash, sürgősségi beavatkozáshoz való előkészület (droghatás, vasokontrikció stb.).

Repetiting scaling and root planning, polishing: due to bad oral hygiene, these must be done in most of the cases. An increased tendency to bleeding, poor healing, poor immunity and vulnerability to infections must all be taken into consideration. The use of sand blasters is contraindicated.

Motivation and education are of crucial importance, and the restoration of normal oral hygiene is also part of the rehabilitation in general.

Nutrition: Sufficient calorie intake is of primary importance, and care must be taken that the protein- and vitamin-intake (especially Vitamin A) is also sufficient.

13.12. Treating patients with circulatory diseases

Cardiovascular diseases in Hungary are endemic diseases, which are among the leading causes of death together with cancer. The reason for special attention is that medications for their treatment can cause disorders on the gums and may lead to complications during treatment. Treatment of these patients can only be performed after appropriate preparation in most cases.

Ischaemic heart diseases

The disease is caused by coronary artery obstruction or complete coronary occlusion. If treatment is not performed soon enough, cardiac muscles may necrotise, which can result in their death, i.e. cardiac infarction, if exceeding a certain extent because of lack of oxygen.

Ischaemic heart disease is the result of atherosclerosis. Injuries of the vessel walls in inflammatory processes develop an atheroma plaque, which reduces the diameter of the vessel, this way compromising circulation. Periodonto-pathogenic bacteria can be related to vascular inflammations. Periodonto-pathogenic bacteria strains can be isolated form atheroma plaques. Many of the risk factors are mutual in periodontitis and atherosclerosis.

For therapy, insertion of a stent is possible, which can release medications in certain cases. If this is not the case, antibiotic prophylaxis is recommended for surgeries with the risk of bacteraemia. In many cases after heart infarction and static heart deficiency, blood thinners are prescribed but at least thrombocyte aggregation inhibitors. As a result of cardiac muscle necrosis, changes in cardiac blood flow can lead to thrombosis. In these cases, coumarin derivatives are given to the patient. Precautions need to be made before invasive surgeries (setting INR level with LMW heparin).

Within one year following myocardial infarction, the recurrence has a high lethal rate. Therefore, sedative drugs are recommended to prevent patient stress during dental treatment. It is best to postpone treatment if possible, or perform treatment in several shorter sessions.

Hypertension
Patients suffering from high blood pressure have a high incidence. It increases peripheral resistance, the heart pumps blood with higher pressure, which can lead to cardiac growth, thickening of the left heart chamber wall. Medications for treatment often include Ca-channel blockers, which can cause pronounced hyperplasia of the gums in plaque induced gingivitis. The method of treatment is improving oral hygiene, removing excess gingival growth, and switching to different medications.

High blood pressure can be primary or essential. Risk factors for development are smoking, genetics, overweight, high salt consumption, gender, age, race, and stress. Secondary hypertension can originate from kidney diseases, endocrine disorders, or taking of certain medications (e.g. steroids).

**INFECTIVE ENDOCARDITIS**

It is caused by bacterial infection of the heart valves and the endocardium. The first disease directly linked to bacteraemia related to dental treatment. The risk factors are previous endocarditis, artificial heart valve, and intravenous drug abuse, bacterial deposits on the endocardium or the valves.

As a preventive measure, these patients need to go through antibiotic prophylaxis. The patient may receive blood thinners in case of artificial heart valves, therefore, the patient needs to be prepared for treatment as described before.

**PACEMAKER**

Pacemakers are used to treat heart rhythm disorders. Prophylaxis is applied if the underlying disease indicates it. You should always ask the patient whether he/she takes blood thinners or thrombocyte aggregation inhibitors. Debridement is forbidden with magnetostrictive ultrasonic devices.

**INHERITED DISORDERS**

Congenital disorders require antibiotic prophylaxis prior to dental treatment, when the risk of infective endocarditis is high.

**13.13. Treatment of haematological diseases**

Haematological diseases, depending on the cell strain affected, can be classified as anaemia, neutropenia, haemostasis disorders, and myeloproliferative, lymphoproliferative diseases. It must be emphasised that in many cases the disease is not isolated, all blood cell strains are affected. This condition is called pancytopenia.

**RED BLOOD CELL DISORDERS: ANAEMIA**

The number and parameters of red blood cells drop below normal values. This can be due to either lack of production or increased decomposition of the cells. It can present oral symptoms. One type, called aplastic anaemia, comes with pancytopenia, which can lead to haemorrhage and infections. It can be inherited or acquired (related to chemotherapy, radiotherapy, radiation, medications such as antibiotics, phenylbutazone, phenytoin, carbamazepine, quinacrine, tolbutamide, or autoimmune diseases such as systemic lupus erythematosus, hepatitis, or it can even be related to pregnancy). Gingival growth can be a typical sign.

A rare form of anaemia is autosomal inherited Fanconi anaemia, which accompanies other inherited disorders. These patients are prone to leukaemia and mucosal tumours.

Another inherited disease is sickle-cell anaemia, which was named after the sickle shaped red blood cells typical for this disease. The cells are more rigid, haemoglobin is less soluble in venous blood, they are prone to aggregation leading to thrombosis in capillaries, and decompose in an early stage.

Regarding periodontal treatment, it is a general rule to avoid surgical treatment in severe anaemia because of the prolonged wound healing. In sickle cell anaemia, it is prohibited to provide dental treatment during the acute phase. Surgeries may only be performed if the haemoglobin level is above 70%. General anaesthesia is contraindicated. Local anaesthesia may cause necrosis at the point of insertion. Perfect soft tissue coverage needs to be performed, due to the increased risk of osteomyelitis. Oral inflammations can have severe effects, therefore, antibiotic prophylaxis is advised. The disease comes with liver and kidney dysfunction, this must be considered regarding medication therapy!

**DISORDERS OF WHITE BLOOD CELLS**
Summary of white blood cells is presented in the following:

1. Decrease in production – Neutropenia
2. Haemopoietic stem cells, clonal disease – Myeloproliferative diseases
3. Leukocytes qualitative disorders
   - Migration within blood stream
   - Attachment onto vessel surface
   - Exiting blood stream
   - Chemotaxis
   - Phagocytosis
   - Degranulation

Neutropenia, the quantitative dysfunction of white blood cells can be traced back to the following reasons:

1. Bone marrow diseases
   - Aplastic anaemia
   - Isolated white blood cell aplasia
   - Congenital – rare
   - Cyclic neutropenia
   - Medications (antibiotics, procainamide, phenytoin, chlorine-promazin, methimazo, etc.)
   - Chronic benign
2. Peripheral diseases
   - Hypersplenia
   - Sepsis oImmunity
   - Felty’s syndrome

Periodontal treatment principles in neutropenia are conservative treatment if possible, and radical therapy might be necessary in this case (extraction of questionable prognosis teeth). Antibiotic prophylaxis is necessary before treatment, continued also after treatment.

Inherited diseases of leukocyte functional disorders lead to fast progression, destructive periodontitis, and ulcerative inflammations may develop on the mucosa. The underlying diseases are the following:

- LAD (Leukocyte Adhesion Deficiency)
- Chediak–Higashi syndrome
- Lazy leukocyte syndrome
- Papillon–LeFevre syndrome
- Down syndrome

**HAEMOSTASIS DEFICIENCIES**

Diseases can be categorised as following:
• Pathological thrombocyte count
• Pathological thrombocyte function
• Dysfunction of fibrin (clot) formation

Diseases with pathological thrombocyte count:

Aetiology:

1. Bone marrow diseases
2. Non-bone marrow diseases Immune diseases
   • Idiopathic thrombocytopenic purpura
   • Medication induced
   • Secondary
   • Post-traumatic
3. Hypersplenia
4. DIC (Disseminated intravascular coagulation)
5. TTP (Thrombotic thrombocytopenic purpura)
6. HUS (haemolytic uraemia syndrome)
7. Sepsis
8. Haemangioma
9. Viral infections, AIDS

*Oral symptoms:* petechiae, ecchymosis, haemorrhagic vesicles, haematoma, bleeding gums, and prolonged bleeding on trauma.

*Treatment:* establishing good oral hygiene, and avoiding invasive interventions if thrombocyte count is lower than 50000/mm3. Administration of salicylates and NSAIDs is contraindicated.

**Haemophilia**

It is an inherited disease caused by gene mutation on the X chromosome. The two forms are Type-A (factor IIIIV) and Type-B (factor IX). It comes with either lack of production or dysfunction of the mentioned factors.

Inheritance is sex dependent, and recessive. Carrier women are typically symptom free, only 20% of them suffer from clinically relevant factor dysfunction.

If factor Xa suffers lack of production, thrombin production becomes prolonged, the fibrin structure becomes instable, the blood clot will be small and fragile. The fibrinolytic system inactivates the clot rapidly.

*Diagnosis:* APTT (partial thromboplastin time) becomes prolonged, however, haemorrhage time, prothrombin time, and thrombin time show normal values.

*Therapeutic considerations:*

Substitution therapy is needed:

• Occurrence of bleeding
• Danger of bleeding
• In case of invasive intervention

• For prophylactic purposes

Special considerations: antibiotic prophylaxis is recommended in case of gastrointestinal or oral haemorrhages (but: contraindicated in haematuria!). Administration of salicylates and NSAIDs is forbidden. Vaccination can only be done under factor protection. (APPLIES TO LOCAL ANAESTHESIA!!!). Individual oral hygiene is performed gently with a soft toothbrush avoiding injuries is important when using any other oral hygiene instruments.

Surgical perioperative treatment can only be performed if the required haemostatic factor is at our disposal, and necessary haemostatic tests have been performed pre-surgically. Only selective COX-2 inhibitors can be given as painkillers.

Treatment can be performed:

• One dose factor replacement

• Continuous (infusion) factor replacement (major surgeries)

• Desmopressin administration

• Fibrinolytic treatment (Tranexam-acid)

• Use of fibrin-glue

Factors determining operative and postoperative factor replacement:

• Patient’s factor activity

• Patient’s body weight

• Extent of surgery

• Anaesthesia exceeding 30 minutes

• Risk of internal bleeding

• Local haemostatic cannot be used

• Minor bleeding can cause major trouble

• Localisation of surgery

• Alternative haemostatic options

• Isolation of specific factor form blood serum

• Half-life time of specific factors: VIII: 8–12h, IX: 16–18h

Haemophilic patients need to be treated the following way in case of invasive treatment:

• Continuous factor replacement is only necessary under major surgeries.

• Desmopressin (DDAVP) is recommended in mild and moderate cases of Haemophilia "A" for smaller surgeries.

• Anti-fibrinolytic therapy (tranexaminic acid) may be performed for oral surgeries, combined with factor replacement or Desmopressin.

• Fibrin-glue is a good combination to factor replacement for oral interventions.
Dental and surgical treatment of patient with haemophilia

**Haemophilia caused by anti-factor antibodies**

This can be inherited. In this case, treatment consists of F VIII, rFVIIa factor replacement, administration of prothrombin complex, which can be combined with additional antifibrinolytic treatment.

In the acquired form (caused by early pregnancy, medications, malignancies, infections, iatrogenic interventions, etc.) FVIII (human), rFVIIa or porcine FVIII factor replacement is recommended. In both cases, establishment of immune tolerance is the main goal.

**Von Willebrand disease**

Lack or dysfunction of von Willebrand factor, which helps thrombocytes attach to the subendothelial cells. It binds to the coagulant protein of factor VIII, and protects it from degradation. If it is not present, secondary coagulopathy can develop. It is inherited autosomal dominantly (severe forms: recessive). Establishing the diagnosis is difficult, sometimes further examinations are needed. APTT may be prolonged. In many cases, severe, uncontrollable bleeding after non-surgical periodontal therapy of adults can be an obvious sign.

Treatment: Desmopressin (DDAVP), in case of life-threatening bleeding or major surgeries: administering virus inactivated factor concentrate (FVIII/vWF). Adjuvant treatment: administration of Tranexam.

**LEUKAEMIA**

Myeloproliferative disease accompanied by pancytopenia. Consequences can be skin and mucosal lesions, haemorrhage, high risk of infection. In 25–33% of the cases, oral symptoms develop at first.

Principles of periodontal therapy:

- Aggressive treatment (elimination of all irritating factors, questionable teeth) is effective in avoiding oral symptoms
- Only emergency treatment during acute phase
- Perform all dental treatment in the remission phase to re-establish and preserve oral health
- Importance of individual oral hygiene, it should be performed with caution due to risk of haemorrhage
- Bleeding: local treatment, if it is not effective: thrombocyte suspension
- Application of gelatine or collagen sponge
- Use of Antibiotic rinse or spray
Prevention of spreading of ulcerations and development of bacteraemia must be our aim. Local antibiotic and antifungal therapy, as well as use of chlorine-hexidine (facilitates healing, and prevents secondary infection) is recommended. Due to the risk of viral infections, antiviral therapy may often be beneficial.


There are two types of Diabetes Mellitus: Type I (insulin dependent) and Type II (non-insulin dependent). Consequential dysfunctions in related organs are due to hyperglycaemia. On the one hand, enzymatic transformation of glucose produces sorbitol, which is directly toxic to tissues. On the other hand, proteoglycans are produced, different protein molecules attach to hexoze molecules. The structure of essential proteins change due to this attachment, and their function also becomes impaired.

Proteo-glycates facilitate the production of pro-inflammatory cytokines, and collagenase (MMP), collagen production of fibroblasts reduces. The immune system reacts with increased pro-inflammatory cytokine production to bacterial irritation. Regenerative potential of mesenchymal cells in the periodontal ligaments is lower, and reaction to growth factors is less pronounced.

Diabetic patients are more susceptible to periodontal diseases: both destructive periodontitis and gingival recession have a higher occurrence among these patients. Several studies demonstrated a connection between uncontrolled or poorly controlled diabetes mellitus and periodontitis. There are also data supporting easier regulation of blood sugar level after successful periodontal treatment.

**INFLUENCE OF DIABETES MELLITUS ON THE ORAL FLORA AND HOST RESPONSES**

Patients with uncontrolled diabetes have a higher tendency to develop periodontitis and carious lesions. Changes in microbiological composition are most significant in Capnocytophaga species, Prevotella intermedia, Campylobacter rectus, Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans species increase in composition rate.

Diabetes mellitus also affects host responses. PMN dysfunctions develop, especially chemotactic disorders. The sulcus fluid contains higher level of collagenase, originating form PMN cells. Other enzymes (elastase, beta-glukoronidaze) may also be released in greater quantities from PMN cells. Increased amount of elastase may be responsible for vascular damage.

Some pro-inflammatory cytokine levels (IL-1ß and PGE2) reach a higher level in diabetic periodontal patients. The same enzymes are also released in a higher amount from monocytes, and T NFα levels are also higher in diabetic patients compared to systemically healthy ones. Increased production of inflammatory cytokines induced by proteo-glycane has already been mentioned.

Among the effects on collagen, the effect of glycated proteins on blood vessel collagens must be mentioned. These vascular changes participate in compromised immune functions and wound healing by decreased perfusion. The basal membrane of capillaries becomes thinner, and oxygen diffusion through the vessels, elimination of harmful metabolic products, PMN migration, and antibody diffusion become compromised. Proteo-glycans can induce formation of microthrombus on the inner surface of the vessels, which further impair tissue perfusion.

**EFFECTS ON WOUND HEALING**

The combined effects of the factors mentioned above are that the collagen synthesis of fibroblasts decreases, decomposition increases, glycolised collagen on the wound margin impairs healing, and remodelling functions suffer damage.

**TREATMENT OF DIABETIC PATIENTS**

Results of periodontal treatment in well-controlled diabetic patients are equal to systematically healthy patients. Uncontrolled blood sugar level has a negative effect on treatment outcome: less clinical attachment gain, and higher tendency to recurrence of the disease.

**COMPLICATIONS IN THE DENTAL PRACTICE**

Particularly Type-I diabetes can cause acute symptoms, which can be the result of both hypo- and hyper-glycaemia. Dropping of blood sugar level is more common. Symptoms are the following: blood sugar level in
venous blood is lower than 3.5 mmol/L, autonomous nervous system is activated resulting in symptoms which provoke elevated blood sugar level: hunger, nervousness, anxiousness, sweating, and discomfort. Symptoms differ person by person. If glucose level continues dropping, symptoms of cerebral hypoglycaemia take over: vision becomes blurry, speech becomes slurry, concentration problems and frustration can occur. Lower blood sugar level can result in tiredness of the patient or even aggression. Severe persisting hypoglycaemia may lead to unconsciousness. Treatment should be fast absorbing sugar intake.

Symptoms leading to hyperglycaemic coma: dry skin, nausea, stomach ache, shortness of breath, acetone breath, disturbed vision, thirst and tiredness. Treatment is accomplished by the administration of insulin. It is essential to have the patient’s medication at our disposal.

13.15. Patient infectious diseases

See in Chapter 6.3.!
Chapter 6. MULTIDISCIPLINARY ISSUES

1. Acute sickness in the dental chair (Judit Méray MD)

Sickness and fainting in the dental office are often caused by anxiety and fear of the treatment, therefore, its incidence can be reduced by appropriate reassurance or if necessary, by sedative premedication.

In case of a sudden fainting, the most important question is always how severe the situation is, and whether it is life threatening or not.

The observation of the symptoms, the course of events can help in the decision, but in an ideal case, one can rely on the previously documented history and further information in the patient’s file (e.g. accompanying diseases, like cardiac disease, asthma or previous problems, like recurrent loss of consciousness, etc.).

The file of every new patient should contain a precise history, which has to be refreshed on every occasion with new data. It is recommended to ask the questions in a regular sequence, and in order to speed up the process, a lot of the necessary data can be obtained through questionnaires filled in by the patients while waiting for the treatment. The important questions are listed in Fig. 6.1.

![Medical questionnaire before dental treatment](image)

If there is a history of previous sickness during dental treatment, we ask the patient about its course, and with suitable questions we try to find out what it was (e.g. asking whether it occurred before or after the local anaesthesia, whether it was prevented by a feeling of weakness, or itching, skin reactions, etc. Were there difficulties of breathing or swallowing? Did it need medical intervention or the sickness subsided spontaneously? Did any medical check-up happen afterwards?).

It is also important that the present general medical problems and untoward events that occurred during the actual treatment should be documented. The adequate record may protect the dentist in case of later litigation as
it is a legally important document. It also serves the interest of the patient since it serves as a reminder at further treatments and helps us avoid complications and to plan the interventions accordingly. The patient’s file should contain the following items:

- Description of the symptoms (and of the probable cause if possible)
- Type of the sickness (e.g. vasovagal fainting or allergic reaction, or “transitory loss of consciousness” if the cause is unknown)
- Time of the occurrence (e.g. after giving i.v. antibiotics, or before starting the treatment, or during treatment, etc.)
- Symptoms (e.g. red skin, rashes or blood pressure of 210/130 mmHg, or a short episode of loss of consciousness with convulsions, etc.).
- Therapy applied and the dose of the given drugs (e.g. raising the legs, or terbutaline aerosol 2 puffs or epinephrine 0.1 mg s.c., etc.)

Figure 6.2. helps overview and document the symptoms.

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>skin color</td>
<td>pale, red, cyanosis (lips, nails)</td>
</tr>
<tr>
<td></td>
<td>fainting, anemia, hypoglycemia, stress, fever, hyperglycemia, hypertonia</td>
</tr>
<tr>
<td></td>
<td>hypoxia, central/peripheral circulatory insufficiency</td>
</tr>
<tr>
<td>edema</td>
<td>on the legs, on the face, head</td>
</tr>
<tr>
<td></td>
<td>cardiac insufficiency, kidney disease, orthopedic causes, allergy, anaphylaxis</td>
</tr>
<tr>
<td>pulse</td>
<td>bradycardia, tachycardia</td>
</tr>
<tr>
<td></td>
<td>vagal action, disturbances of conduction, excitement, fever, hypovolemia, cardiac causes</td>
</tr>
<tr>
<td>blood pressure</td>
<td>hypotension, hypertension</td>
</tr>
<tr>
<td></td>
<td>vasovagal collaps, hypovolemia, anaphylaxis, shock, hypertonic disease, anxiety, fear</td>
</tr>
<tr>
<td>thoracic tightness, oppression</td>
<td>angina</td>
</tr>
<tr>
<td></td>
<td>coronary spasm, myocardial infarction</td>
</tr>
<tr>
<td>shortness of breath</td>
<td>difficult exhalation</td>
</tr>
<tr>
<td></td>
<td>asthma, allergic bronchospasm, COPD, cardiac diseases</td>
</tr>
</tbody>
</table>

Evaluation of symptoms

THE MOST COMMON TYPES OF SICKNESS DURING DENTAL TREATMENT

Because of their relative frequency and simplicity, let us start with the less dangerous situations.

The hysterical fit is usually characterised by certain theatrality. Sometimes the previous behaviour of the patient, his manner helps us with the diagnosis. No pallor or cyanosis can be observed, the blood pressure is normal or slightly elevated, and the skin colour is normal or reddish. These patients are often young with no organic diseases. Hyperventilation can often be seen. The complaint related to certain organs (“stabbing” heart pain, shortness of breath without its clinical signs, difficulties of swallowing, etc.) may be of psychic origin; however the patients really experience and suffer from these feelings. One should never forget the possibility of a “true cause” either!
The most important factor is the empathic, sympathetic, but at the same time resolute, assuring attitude of the medical personnel. We ask for cooperation, try to achieve positive compliance. No drugs are necessary. (Sometimes sending away the relatives may help.)

Sometimes the alkalosis caused by excessive hyperventilation provokes tetany with characteristic “deer-head”-like spasm of the hand. The symptom, however frightening for the non-professional observer, can be terminated by making the patient hold his breath or breathe into a plastic bag.

Common fainting, vasovagal syncope is the most frequent cause of sudden loss of consciousness in the dental office. It usually occurs in young men with no known disease, and more than once in their history there are recurrent episodes of fainting during medical interventions, accidents or at the sight of blood.

It is characterised by short peripheral circulatory insufficiency (collapse), with temporary loss of consciousness (syncope). It can be preceded by getting pale, sweating, feeling weak, nauseated, experiencing blurred vision, and shortness of breath. Sometimes vomiting, short muscle twitching (sign of cerebral hypoxia!) may create an alarming picture. Another time the loss of consciousness comes suddenly, without any warning sign. The pulse is easily suppressible, filiform, in the typical case it is slow (vagal action!), seldom tachycardiac.

The pathophysiological basis is vasodilatation in certain parts of the vasculature (mainly in the striated muscles) and the consecutive fall of blood pressure. (In physiologic stress situations, the active functioning of the muscles – running, struggle – promotes the flow back of blood to the heart, whereas in passively “suffered”, “not refusale” stress, this balancing effect does not occur, while instead of a tachycardia, a vagal effect (bradycardia) prevails. The vessels are relaxed, but the heart rate does not compensate to maintain blood flow to the brain. (Of course, there may be other components of psychic fainting, but the full pathomechanism is not yet completely cleared.) The redistribution of blood and the fall of blood pressure can cause transitory cerebral ischaemia, which leads to loss of consciousness, and may even cause convulsions, muscle twitching.

The patient should be laid (if the fainting happened in the dental chair, it has to be brought into horizontal position). Both legs have to be elevated in order to promote the flow of venous blood to the heart. Tight clothing should be loosened, the window opened, and we may try to stimulate the fainted person by briskly tapping, or putting a wet compress on the forehead. In case of vomiting, the head or the whole body should be turned to the side.

If the consciousness recurs, the patient should be encouraged to move the limbs actively. Blood pressure and pulse have to be checked repeatedly. In case of excessive vagal effect, if the syncope does not improve quickly, atropine (0.3–0.5 mg in adults) can be given. After regaining consciousness, the patient should remain in the lying position for at least 10–15 minutes.

If the patient regained consciousness, his/her blood pressure returned to normal, and he/she feels all right, the intervention that has already been started can be finished. Fainting after the injection of the local anaesthetic may raise the question whether the intervention can be done using the already developed anaesthetic state, when the patient totally regained alertness; nevertheless, we have to be absolutely sure that the reaction was not an allergic one. (One should, however, not forget that fainting may reappear.) If the intervention has not yet been started, or if the sickness prolongs, we have to postpone the treatment, and do it only following due preparation, under maximal attention.

Possible other causes of syncope with hypotension are allergic, anaphylactic reactions to the local anaesthetic, other drugs or disinfectants, epinephrine effect (tachycardia, arrhythmia), conduction disturbances (atrioventricular blocks), carotid sinus hypersensitivity, “primary” cardiac insufficiency, cardiac ischaemia, hypoglycaemia, hypoadrenia, epileptic fit, etc. Previous history, the course of the fainting and accompanying signs (e.g. oedema, rash) can help in the decision. The lack of a quick recovery may be the sign of a more serious condition: if raising the legs does not improve the situation, but it shows a tendency to get worse, it is better to ask for immediate help and call the ambulance.

**Allergic reactions, anaphylaxis**

Allergy and particularly anaphylaxis, which is its more severe, life threatening form, has to be taken seriously. Drugs and substances which are often responsible for an antigen–antibody reaction are antibiotics, analgetics, contrast media, and latex. In some cases, the anaphylaxis seems to be “idiopathic”. (While allergic reactions are IgE mediated, anaphylactoid reactions are not; they are based on complement activation, but cause a similar clinical syndrome.) If the patient claims to be allergic to anything (drugs, disinfectants, or other substances) used...
in the dental praxis, we better accept it until proven otherwise. An allergology examination is strongly recommended in these situations.

In case of an allergic (or possibly allergic) reaction, the therapeutic plan depends on the severity of the symptoms.

- **Mild** allergic reactions are usually characterised by urticaria (rash) or erythema (reddish spots of the skin), itching, or running nose, but the breathing is undisturbed, no dizziness, sickness occurs and the blood pressure does not fall.

- **Medium severe** allergic reactions may cause oedema (mainly on the face), sickness, shortness of breath, light fall in blood pressure, usually with tachycardia (as opposed to bradycardia in simple vasovagal episodes). In this case, immediate intervention is necessary!

- **Severe** anaphylaxis usually presents with oedema, difficulty swallowing and breathing, wheezing, blurred speech, confusion, nausea and vomiting, severe fall in blood pressure, circulatory shock, and loss of consciousness. It is a dangerous, life threatening state!

Mild cases can be solved without intervention, or antihistamines may be given in tablet form. In the latter case, the patient should be warned of the danger of getting drowsy afterwards and of its incompatibility with alcohol. (The use of antihistamines is not recommended in more severe cases because of the risk of drowsiness and hypotension.)

In medium severe or severe cases, prompt action is necessary. While we call the ambulance, epinephrine should be given immediately. The first dose (0.1 mg) can be given subcutaneously, but then it is recommended to fix an intravenous line and use 0.5–1 mL fractions of diluted epinephrine (1 mg in 10 mL) intravenously, followed by infusion of a crystalloid solution. Methylprednisolone or hydrocortisone (100–200 mg) can be given. In case of asthma-like wheezing, a bronchodilator, beta 2 agonist spray should be applied. Until the arrival of the ambulance, the patient should be kept under tight control. Give oxygen and keep the airways free.

A low blood pressure with a petechial or purpuric rash can be a sign of a septic shock. Seek help early if there are doubts about the diagnosis or treatment!

**Sickness of cardiovascular and hypertensive origin**

The importance of strong palpitation, rhythm disturbances depends mainly on their haemodynamic action: decrease in the pump function of the heart, fall of the blood pressure and of the oxygen supply to the heart muscle, or the tendency of the symptoms toward a dangerous ventricular rhythm (ventricular tachycardia, ventricular fibrillation) has to be taken seriously.

Palpation of the pulse shows the fact of arrhythmia. By counting the heartbeats over the heart and on the periphery, we get the pulse deficit. The type of the arrhythmia can only be established by ECG, which can show the presence of myocardial ischaemia as well. In case of sudden arrhythmia with a sickly feeling, the patient should urgently be seen by an internist, especially if there is no such problem in the history, or if the heart rate is very high or very low, the tendency is worsening as well as in case of the development of angina. In these cases, it is better to call the ambulance immediately. In case of a sudden paroxysmal tachycardia, we can try to stimulate the vagus nerve by pressure on the carotids or on the eye bulb.

Pectoral angina or symptoms of myocardial infarction (epigastric discomfort, pressure, fullness, squeezing, or pain in the centre of the chest that may spread to the shoulders, neck, or arms or may be located in the upper abdomen, back, or jaw, clammy skin, rapid or irregular heart beat, pallor, and feeling of impending doom) means urgency: the patient should be taken by the ambulance and receive expert help.

In patients with ischaemic heart disease (IHD), prevention is very important: the patient should take his/her regular drugs before the dental treatment, and it is sometimes recommended to order a sedative premedication. If the patient is using coronary dilating medication (e.g. nitroglycerine), this also has to be applied before the treatment.

On the basis of IHD, every stress situation because of the consecutive tachycardia and hypertension may cause acute left ventricular failure, and this can lead to pulmonary oedema. Beside the history the appearance may also be helpful in the diagnosis: sudden sickness with shortness of breath, which is worsening in horizontal position. The neck veins are full, the blood pressure may fall, “wet lung”, the presence of frothy pulmonary secrets are
rather characteristic. Because of the worsening of the pump function of the heart, the peripheral circulation is usually poor with cyanosis and with a characteristic ashen coloured skin. Call the ambulance immediately and give oxygen! If necessary, apply BLS (basic life support).

A sudden rise of the blood pressure, hypertensive crisis situation, usually develops on the basis of a hypertensive disease. The precipitating factor can be anxiety and fear, especially if the patient skipped the usual antihypertensive medication. It is recommended to postpone the treatment if the blood pressure is over 160/110 mmHg, or if there are untoward clinical signs (e.g. headache, nausea, visual disturbances, or angina). The so-called “white coat hypertension” is caused by the stress of preparing for the dental appointment, and it may disappear after a short time. In case of a mild elevation of the pressure, it is recommended to let the patient take his habitual drugs and a sedative tablet and wait until the blood pressure returns to normal. Hypertensive patients with normal or nearly normal blood pressure can be treated in the usual way, which means that also a vasoconstrictor (epinephrine) can be used with the local anaesthetic. Nevertheless, it is important to warn these patients to come to the appointment after taking their antihypertensive drugs, and it is highly recommended to order a sedative for them to be taken one hour before the treatment. (One should avoid a sudden fall in blood pressure as well because this can lead to cerebral ischaemia in hypertensive patients.)

In crisis situations with extremely high blood pressure values (over 190/120 mmHg) that do not improve soon, the patient should be immediately seen by an internist. Chest pain or severe headache, nausea or vomiting can occur, and in the worst cases, acute left ventricular insufficiency may occur with dyspnoea and pulmonary oedema. It is an emergency situation! A hypertensive crisis damages the blood vessels and can lead to the development of stroke or myocardial ischaemia. The patient should be brought into a sitting position, and while waiting for the ambulance (“life squad”), an angiotensin converting enzyme inhibitor (e.g. captopril 12.5–25 mg tablet) or nitroglycerin (sublingual) may be given.

Respiratory problems

A part of respiratory problems (shortness of breath, pulmonary oedema) may be caused by heart (left ventricular) insufficiency. These patients are usually already known and treated by the cardiologist. A sudden deterioration of the patient can be precipitated by fear, tachycardia and elevation of blood pressure or coronary spasm and myocardial ischaemia. In these cases, urgent expert help is necessary. Call the ambulance, give oxygen and keep the patient under tight control. The problem is better prevented by letting the patient take his usual medication and ordering a sedative before the dental treatment.

Upper airway obstruction may be caused simply by the relaxation of the pharyngeal structures and falling back of the tongue in the unconscious patient lying on the back. Elevation of the mandible, eventually with the use of an oropharyngeal (e.g. Guedel or Mayo) airway can usually solve the problem. It is important to clear the mouth of secretion and foreign material! In patients suffering from obstructive sleep apnoea (OSA), even superficial sedation may lead to complete obstruction of the airways. Sedatives should better be avoided in these patients.

Aspiration of a foreign body or foreign substance may create a life threatening situation. As long as the patient is able to speak and cough effectively, the obstruction is mild. Encourage him/her to continue coughing. In severe cases, if we are not able to remove the obstructing body easily, try to give 3–5 sharp back blows, and check if the obstruction has cleared. If these fail to relieve the obstruction, the application of the so-called “Heimlich manoeuvre” (quick, strong abdominal thrusts using our both arms from behind the sitting patient) is recommended. These methods can be repeated once more. In case of a complete obstruction, the only solution may be the incision of the cricothyroid membrane on the neck.

Partial obstruction of the upper airways is characterized by an inspiratory stridor. It can also be caused by laryngospasm as a reaction to irritation of the glottis or by laryngeal oedema in severe allergic reaction (see treatment for anaphylaxis!).

A typical example of the obstruction in the lower airways (small bronchi, bronchioles) is bronchial asthma, and the same reaction (bronchospasm) caused by allergy. In asthmatic patients, beside the bronchospasm, there is a viscous secretion and an inflammatory component as well causing the small airways close before the end of expiration. Wheezing sounds can be heard all over the lungs, and the patient seems to “struggle for air”. The history of the patient usually helps us with the diagnosis. Asthmatic patients should keep their usual inhalator (beta2 agonist spray) at hand when coming for the dental treatment, and use it as soon as they feel signs of a beginning attack. In crisis situations, epinephrine (0.1 mg s.c., i.m. or i.v.) can be administered, and the
ambulance has to be called. The stock of emergency drugs in the dental office must contain a beta2 sympathomimetic aerosol (e.g. terbutaline) as well.

A patient suffering from **chronic restrictive or obstructive pulmonary disease (COPD)** needs extra attention at dental treatments. Sometimes the reduced respiratory capacity may cause shortness of breath if the mouth is partly obstructed by dental instruments, and anxiety may worsen the situation. Acute infections can cause exacerbation of the respiratory disease, and if possible, one should avoid dental treatment in these periods.

**Hormonal/metabolic disorders**

In case of active **hyperthyroidism**, which is rare nowadays, only very urgent interventions can be done, every elective, planned treatment has to be postponed until the patient is in stable condition. Refer the patient to his/her endocrinologist before starting the treatment and make sure that his/her disease is under control. The patient with hyperthyroidism is very sensitive to the action of epinephrine and of beta sympathetic agonists, and even the small amount of epinephrine applied with the local anaesthesia may cause rhythm disturbances and can lead to critical situations. Uncontrollable tachycardia, tremor, sweating may be the signs of a serious problem, and it is better to call the ambulance immediately. Before unavoidable interventions, it is highly recommended to give sedative premedication.

On the other hand, in cases of **hypofunction of the thyroid gland**, commonly due to autoimmune thyroid destruction, sedatives are contraindicated and could lead to worsening of the situation. These patients are prone to hypotension, hypoglycaemia and ischaemic heart disease. They should only be treated in a euthyroid state; nevertheless, one should never forget about the possibility of the above problems.

**Adrenocortical insufficiency, hypoadrenia** is not necessarily a consequence of a primary endocrine disease, more often it is caused by prolonged steroid treatment. Exogenous steroids suppress excretion of adrenocortrophic hormone, and consequently, the function of the adrenal cortex. In stress situations (anxiety, fear, surgery, inflammation, etc.), when the need for corticosteroids is elevated, the adrenals cannot meet the requirements, and acute hypoadrenia occurs. The first sign can be nausea, hypotension, pale skin and hypoglycaemia. The initial treatment does not differ from the common treatment of fainting, but in these patients the causal therapy is the administration of a single dose of a corticosteroid (metylprednisolone, hydrocortisone). Always ask the patients about recent steroid treatment! In all patients with primary or secondary adrenocortical insufficiency, it is recommended to consult the endocrinologist before the start of the dental treatment.

**Diabetes mellitus** is a frequent disease in civilised countries, and the dental treatment of diabetic patients requires particular attention.

Type I diabetes – previously called "insulin dependent diabetes mellitus" (IDDM) means absolute insulin deficiency, that is these patients can not tolerate prolonged periods without exogenous insulin. (Insulin is necessary for everybody, even when fasting to maintain glucose homeostasis and balance stress hormones.) The serum glucose level of these patients is usually unstable, and they are prone to hyperglycaemic ketoacidosis as well as to severe hypoglycaemia on the other side.

Type II diabetics can be characterised mainly by insulin resistance, that is they produce some endogenous insulin, and their metabolic state is not so much endangered during fasting. The disease is called "non insulin dependent diabetes mellitus" (NIDDM); however, some of these patients are on regular insulin treatment beside the usual tablets (e.g. metformin, glitazone, etc.). Some of these my cause hypoglycaemia if the patient does not eat regularly.

Diabetics should get their usual medication also on the day of dental treatment, and eat according to their usual daily schedule. It is recommended to plan the appointment in accordance with the patients needs. The best is to start the intervention in the morning, after the patient has given his/her usual dose of insulin or taken his/her tablets and has eaten some breakfast. Infection, surgery, every stress situation may elevate the insulin requirement. In order to keep the blood sugar level at acceptable (near physiological) levels, frequent check up and possibly tight glucose control is recommended before, during, and after dental treatment. In order to avoid hypoglycaemia, one should aim at a still tolerable blood sugar value of about 8.5 mmol/L.

**Hypoglycaemia**, the fall of blood sugar value under 4 mmol/L is the most frequent and most dangerous cause of sudden sickness in diabetics. It can happen suddenly and can be treated quickly and easily if we think of it in time. The typical clinical signs are: pallor, wet skin, sweating, hypotension, weakness, shakiness, confusion,
dizziness, tremor, in most severe cases loss of consciousness. Severe hypoglycaemia is more likely to occur in people with type I diabetes. The danger of central nervous system damage is high! (A possible cause is that the patient has administered the usual insulin injection, but he/she could not eat afterwards.) We always have to consider the possibility of hypoglycaemia if a diabetic patient gets suddenly sick!

_Therapy:_ as long as the patient is conscious, sugar (preferably glucose tablets, but any other type of sugar would do), or sweet drinks (tea with sugar, juice) should be given. Unconscious patients have to get an intravenous glucose infusion as soon as possible! Repeated blood sugar checks are necessary afterwards!

Mild hypoglycaemia may occur also in patients who do not have diabetes: postprandial hypoglycaemia occurs 2–4 hours after meals, whereas fasting hypoglycaemia is often related to an underlying disease.

Hyperglycaemia may occur in septic states of diabetic patients, especially if they neglect the tight control of their disease. Every infection elevates the need for insulin (absolute or relative insufficiency), and precise care is necessary to keep the blood sugar between acceptable limits (4–9 mmol/L). Older type II diabetics are more prone to hyperosmotic states, while type I patients may get into severe metabolic disturbances with ketoacidosis.

The characteristic picture of the hyperglycaemic state is dry, hot, usually red skin, poor turgor, confusion, and in extremely severe cases, loss of consciousness. The treatment of the patient by a diabetologist is urgently necessary. (High blood sugar is usually accompanied by complex metabolic imbalance, and the right treatment means not merely the correction of the glucose level!)

_Emergency states of neurologic origin_

A _transient ischemic attack (TIA)_ and the _ischaemic or haemorrhagic stroke_ may present with similar symptoms, but while the previous is a short lived phenomenon, stroke is a more serious state with usually permanent damage. The degree of the damage may depend on the urgent expert help: immediate intervention is necessary to save the brain tissue in the peripheral zone of the lesion (penumbra).

The clinical signs (always unilateral) may vary from mild neurologic deficit (weakness or numbness on one side) to coma. Sickness with unilateral difference requires immediate transport to a medical centre.

Seizures may be the consequence of many pathologic states (e.g. cerebral trauma, subarachnoid bleeding, brain tumour, toxaemia gravidarum, cerebral hypoxia, intoxication, metabolic disorders, and high fever – the latter mainly in children). The most important help in establishing the diagnosis is the knowledge of the patient’s history and the course of the events. While in an epileptic patient, who may have “regular” short fits, it is not necessary to call the ambulance immediately, some other cases have to be considered urgent emergency situations.

Every _convulsion and spastic state_ may endanger the normal reflex activity, and pulmonary aspiration, or injuries can happen. It is important to keep the airways free by positioning the patient, suctioning the vomitus or secretions from the mouth, and prevent the patient from falling down. Cushion the patient’s head against banging it against hard surfaces, and loosen tight neckwear to ease breathing. Do not restrain the patient during a seizure unless there is danger, because some persons may get aggressive if you do so. Remove sharp or solid objects around.

Epileptic seizures usually present with a sudden short attack. Patients and their family already know about the disease, but sometimes they do not like to admit it without directly asking them. The fit is usually over in a couple of minutes, and the only thing one has to do is to keep the patient from injuries. However, it is necessary to call the ambulance if the seizure does not cease (lasts for more than 5 minutes), or if it reappears after a time (two or more seizures in a row), or if the patient remains unconscious after the fit, or if she is pregnant.

It is important to know that although intravenous benzodiazepines may terminate the seizures, they usually depress or “terminate” the spontaneous breathing as well, and their injection should only be applied by experts in preserving the airways and in artificial ventilation.

_Toxic action of local anaesthetics_

While local anaesthetics are present in a high concentration at the site of the injection, normally their blood concentration does not achieve toxic limits. This can only happen if one exceeds the maximal dose, uses extended local infiltration without vasoconstrictors, or if the local anaesthetic is accidentally given directly into a vessel. Toxic effects affect the heart or the central nervous system. The best thing to avoid this is prevention:
respect the dose limits, use epinephrine always if not contraindicated (contraindication is rare!), carefully avoid intravasal injection.

In the central nervous system, the first sign of toxic effect is a kind of excitement: perioral tingling, numbness, restlessness, undiscerning behaviour, blurred speech, confusion, tremor, or seizures. This is then followed by loss of consciousness, apnoea, and coma.

The first thing to do is oxygen inhalation, assisted ventilation through a bag-mask unit, if necessary. In case of confusion, seizures or coma, urgent help is necessary, call the ambulance! Start cardiopulmonary resuscitation (BLS) if necessary.

Cardiac symptoms (rhythm disturbances, myocardial depression) are often caused by high doses of bupivacaine. This problem is extremely rare in the dental praxis.

**BASIC LIFE SUPPORT (BLS). CARDIOPULMONARY RESUSCITATION**

The most severe problem in the dental office is the sudden death of the patient, which is of course a rare accident and may cause paralysing perplexity, whereas in these situations only a proper, immediate intervention can give the patient the chance of survival. It is very important that the medical personnel should be prepared and educated to act immediately in these situations.

BLS is the first step of cardiopulmonary resuscitation that can be provided by trained persons also in the prehospital setting. BLS generally does not include the use of drugs or invasive skills, while advanced life support (ALS) does. The first minutes after the circulatory arrest may be decisive, and an action started immediately increases the time available for higher medical responders to arrive and provide ALS care.

In the first seconds, the observer realises the “blackout” of the patient. A sudden loss of consciousness, lifelessness, change of the skin colour from normal to bluish–ashen may be the first warning sign to act immediately. If there is no palpable pulse, or if it is uncertain, the case should be taken as a circulatory arrest. The brain cells must get oxygen within 3–4 minutes and this can only happen if there is circulation. The probability of success is less and less with every missed minute.

The most important steps (The first 4 steps should not take more than 1–2 seconds each!):

1. Check the patient’s reaction to stimuli (brisk tapping, shaking at the shoulders, shouting: “Are you okay?”)
2. Send for help immediately, somebody should call the ambulance.
3. Check for breathing! While doing so, elevate the lower jaw and apply mild retroflexion of the head (head tilt/chin lift or jaw thrust maneuvre), eventually introduce oral airway.
4. In a patient who is unresponsive and not breathing, CPR should be started immediately.
   • bring the patient into horizontal position and start cardiac compressions (in a rhythm of about 100 compressions per minute) immediately, allowing chest to recoil in between. The depth of the compressions should be 5 cm applied at the middle of the chest.
   • at every 30 compressions, inflate the lungs twice through the nose (lips closed) or through the mouth (nostrils closed) trying to keep the airways free. (In the USA, the mouth to mouth technique is not recommended unless a face shield is present). Verify that the chest rises and falls if it does not, try to open the airway again
   • Continue chest compressions and the rescue breaths in a 30:2 rhythm until help arrives.
   • After every five cycles or 2 minutes, reassess the pulse at the carotid artery on your side for an adult – the palpation should not last longer than 5 seconds.

In a well equipped dental office adequately trained medical personnel can use self refilling bags to ventilate the patient. The presence of an automatic defibrillator (AED) can be of great help in cardiopulmonary resuscitation. Attach it as soon as it is available and follow prompts.

If well palpable pulse is detected, continue artificial ventilation and transport the patient to a medical centre.
If the patient is already breathing spontaneously, and pulse is present, he should be placed in the recovery position and monitored. After resuscitation everybody needs continuous medical care.

The above steps should be practiced on mannequins to achieve satisfactory skills and to be able to apply the technique without delay in a critical situation.

A simplified guideline of the necessary steps in case of acute sickness in the dental chair

### 2. Smoking and Oral health (Judit Kádár-Nagy DMD)

**THE COMPOSITION OF TOBACCO SMOKE, FORMS OF TOBACCO USE**

As opposed to the commonly held belief, nicotine is not the most harmful component of cigarette smoke. Tobacco itself contains organic substances, nicotine and additional substances. Tobacco smoke contains more than 1000 noxious materials such as carbon dioxide, carbon monoxide, tar, hydrogen-cyanide, sulphur dioxide, nitrosamine, benzopirene, formaldehyde, polonium 210 and cadmium. Passive smokers are also exposed to most of these substances.

Tobacco can be smoked in the form of cigarettes, cigars, pipe, and waterpipe, but it can also be inhaled (snuff) and it is also chewed.

**THE EFFECTS OF SMOKING ON THE BODY**

Smoking affects all organs and organ systems. It is important to mention that about half of the smokers will certainly die of some kind of cancer. Regular smokers are often regular drinkers at the same time. This makes the damage even more serious, especially in the oral cavity.

Systemic and local effects

1. Cardiovascular system: arteriosclerosis, disease of the coronary vessels, hypertension, aortic aneurysm, stroke, and arterial thrombosis
2. Respiratory system: COPD (chronic obstructive pulmonary disease), emphysema, bronchitis, constant cough due to increased secretion, asthma, tuberculosis, pneumonia, and decreased vital capacity.

3. Cancer: Tumours of the following anatomical locations have been associated with smoking: the oral cavity, the pharynx, the larynx, the lungs, the oesophagus, the stomach, the urinary bladder and the cervix of the uterus. According to US statistics, 87% of lung cancers are associated with smoking, while cancers of the oral cavity are associated with smoking in 75% of the cases.

4. Constant irritation of the eyes and the nose (especially second hand smokers)

5. Miscellaneous: osteoporosis, Alzheimer’s disease, quickened aging of the skin, early menopause, increased affinity to other substances.

PASSIVE SMOKING (SECOND-HAND SMOKING)

Passive smoking means being exposed to tobacco smoke, even if one is not a smoker. A typical example is the smoking of parents with small children, or it used to be workplace smoking. Fortunately, workplace smoking is prohibited by law in several countries nowadays, including Hungary. The goal of such a prohibition is exactly the protection of non-smokers’ health.

Unfortunately, passive smokers are exposed to more or less the same harmful effects as active smokers. It also has to be noted that while active smokers gradually adapt to smoke, in passive smoking it is not seen, and some carcinogenic components appear in the unfiltered smoke in higher concentrations.

SMOKING DURING PREGNANCY

The foetuses of second-hand smoking mothers are just as much at risk as those of active smokers. Such foetuses exhibit slower postnatal development and poorer performance in several respects than their peers from non-smoking families.

1. Intrauterine effects: Nicotine and carbon monoxide readily pass the placenta barrier and enter the body of the foetus. Spontaneous abortion, premature birth, detachment of the placenta, cleft lip and palate, low birth weight and increased perinatal mortality are all common consequences.

2. Neonatal effects: Various chemical compounds enter the body of the newborn with breast milk. Lower respiratory tract infections occur more frequently, and the risk of sudden infant death is increased.

3. Minors exposed to smoke: Constant cough, intense mucus secretion, and asthmatic breathing. Asthma bronchiale. Frequent occurrence of otitis media. These children often find it difficult to concentrate, they have behavioural problems, and they may keep falling back at school due to frequent illnesses.

NICOTINE ADDICTION AND CESSATION SUPPORT

Nicotine is the psychoactive component of tobacco smoke. It acts by increasing the levels of dopamine in the brain, thus causing a "general feeling of well-being". Regular consumption leads first to tolerance, and then to dependence. Tolerance means that an increasing amount of the substance is necessary to achieve the same feeling. When dependence develops, the patient loses control over nicotine intake, and if nicotine is withdrawn for 24 hours, withdrawal symptoms are present.

Smoking can be quit individually and with external support. Individual methods include the gradual decrease of the amount of tobacco, special chewing gums, electronic cigarettes, and a complete change in one’s lifestyle. Professional support involves counselling, developing individualised strategies, and the involvement of the patient’s environment in the process. Cessation can also be supported with pharmacological agents (e.g. Bupropion, nicotine-containing chewing gums, nicotine-containing nasal spray, nicotine patches or varenicline). The application of these agents, however, requires close attention, as they are contraindicated in a number of conditions (like nicotine in cardiovascular disorders or asthma), and they may also have adverse effects.

MANIFESTATIONS IN THE ORAL CAVITY

They depend on the form of tobacco use (smoking or some smokeless form), and also its duration and frequency, in addition to the dose.
Smoking is readily recognisable by extra- and intraoral examination. Breath smelling of cigarette smoke, yellow discolouration of the fingers and fingernails, deep, hoarse voice and cough are all tell-tale signs. Intraoral symptoms can be categorised as follows:

1. Precancerous states, cancer: leukoplakia, squamous cell carcinoma
2. Periodontological problems: ANUG, ANUP, frequent peri-implantitis after implantation, recession, and loss of attachment, and increased progression of periodontal disease.
3. Alterations of the mucous membranes: stomatitis nicotina palati, melanoplakia, glossitis rhombica mediana, glossitis pilosa nigra, chronic hyperplastic candidosis, leukoedema, hyperkeratosis, ostitis alveolaris, and prolonged wound healing
4. Alterations of the hard tissues: abrasion, cervical wear, dehiscence, and tooth loss
5. Aesthetic and social problems: halitosis, discoloration, disturbance of smell and taste
6. The oral symptoms in AIDS and diabetes are always aggravated if the patient smokes.

**THE ROLE OF THE DENTAL HYGIENIST**

The treatment of a smoking patient is always a challenge for the entire dental team. A common ground is that the patient should be encouraged to quit smoking. Smoking patients are characterised by increased calculus formation and poor periodontal status, which means that their professional oral hygiene treatment always takes a longer time, and they also require more frequent visits and it means more costs.

**History**

Smoking history must be taken at each visit. For this purpose, various questionnaires are available which cover a wide range of smoking-related questions. It is also important to gather information on substance use.

**Extraoral examination**

As mentioned before, smoking is readily recognised by a number of external signs, even without asking a single question (see 6).

**Intraoral examination**

Watch out for intraoral lesions that are possibly related to smoking. If such a lesion is found, the patient’s attention must be called to it, and it must be explained that the lesion is in connection with smoking. If a lesion does not heal in two weeks, the patient must be referred for biopsy. This must be marked in the patient file.

An important part of patient education is the teaching of oral self-examination.

**Plaque control**

Optimally, the patient should be able to perform plaque control individually (e.g. with an indicator). Smear the lips with vaseline and chew the dissolving tablet, spread the dye on the tooth surfaces and rinse it with water. Start to remove the dye from the tooth surfaces with a toothbrush. It should always be emphasised with these patients how important plaque control is as they do not tend to pay enough attention to their personal oral hygiene, which leads to an increased vulnerability to develop calculus, periodontal infections and abnormalities of the mucous membranes.

**Scaling and root planning**

The patient must know that smoking significantly deteriorates the expectable outcomes of these interventions.

Care must be taken that the patient should not inhale water or particles of the removed plaque during the treatment, as most of these patients suffer from chronic respiratory and cardiovascular problems, too.

**Instruction, cessation support**
Diet: Smokers tend to have less appetite as compared to non-smokers; therefore, the importance of regular and rich meals must always be pointed out. Regular check of body weight is also advisable.

Alcohol-free mouthwashes are recommended. These patients often use alcohol-containing mouthwashes to cover up halitosis, but a long-term use of these products may cause xerostomia (dry mouth), and a regular exposure to alcohol (together with smoking) may increase the possibility of oral cancer.

3. Infection control in dentistry (Péter Vályi DMD)

AIM OF THE INFECTION CONTROL

Infection control refers to policies and procedures used to minimise the risk of spreading infections, especially in hospitals and human or animal health care facilities. Legal and ethical responsibility of the dental staff is applying the methods of protocols to prevent nosocomial infections. The principles of infection control:

• All of the dental staff has to know and apply the preventive measures of nosocomial infection

• The standard precautions of the infection control protocol have to be applied regularly and strictly in the dental office. The infection control protocol is an important part of the quality management system of the dental office.

• Both the dental staff and the patient have to be protected from infectious diseases.

• Reduction of pathogenic microorganisms to a level which may be eliminated by the intact immune system.

• The transmission of infectious diseases has to be prevented from the patient to the other patient or the dental staff or other person relation to the dental team.

• The methods must be simplified and easy to apply according to the rules.

INFECTIOUS DISEASES

Infectious diseases are disorders caused by organisms such as bacteria, viruses, fungi or parasites. Numerous steps are necessary for the development of an infectious disease. The six steps of the “chain of infection” is required for the development of an infection. The chain of infection consists of an infectious agent, a reservoir (source), a portal of exit, mode of transmission, a portal of entry to the host and susceptible host.

The infections caused microorganisms are classified into five groups: viruses, bacteria, protozoa, fungi and prions. We classified the microorganisms into two groups: the opportunistic pathogens, which do not cause infections in a healthy host but only in a subject with compromised immune system. The other pathogens cause the disease in most subjects with intact immune system.

The reservoir of the microorganisms is the source of infectious agents: individual, equipment, supplies, food, water, animals or insects. The pathogens must leave the source of infectious agents to infect another individual: portal of exit. The portal of exit may be continuous (respiratory droplet) or dependent: the body fluid exiting the body under several circumstances (bleeding, sexual contact, etc.).

The spreading of microorganisms is the mode of transmission: direct contact, airborne transmission, blood borne transmission, ingestion, or indirect contact. The portal of entry allows the infectious agent to enter the susceptible host settling and proliferating of pathogens to cause an infections. The portal of entry may be an injured skin or the intact or injured mucous membranes (eyes, nose, mouth, airways, or gastrointestinal tract) exposed to the external environment. The last chain link is the susceptible host who is unable to resist the pathogens.

The pathogenicity is ability of the microorganisms to cause an infection. It depends upon the virulence of the microorganism, susceptibility of the host and host responses. Infectious diseases may be classified based on the source of the microorganisms: endogenous infection caused by microorganisms inside the individual’s body: habitats of the commensal flora will be infectious agents. Exogenous infections originate from a source that may be found outside the infected person.
The relationship between the pathogens and host: the pathogens can cause the disease, may persist in the host without symptoms (source of infection!) and may be eliminated by host response without damage to the body. The development of infectious diseases can be found in the next figure (Figure 6.4):

The pathogens may have attached to the susceptible host and entry the body (invasion) to destruct the tissues after proliferation of the microorganisms by direct (virulence factors) or indirect way (harmful effect of the host response).

The pathogens have to migrate to the location of infection: spreading may be in a direct way (cell to cell, anatomic space) or indirect way (blood or lymph vessels, cerebrospinal fluid, or axons). The location of colonisation depends on the portal of entry, the special affinity of pathogenic agents for particular tissues or organs of the body (organotropism), amount of invading microorganisms and the susceptibility of the host.

The stages of the infectious disease

1. Incubation period (asymptomatic period) between the initial contact with the microbe and the appearance of the first symptoms. Duration of incubation period depends on the disease (pathogens).
2. Prodromal symptoms (initial symptoms): non-specific general symptoms may occur with variable duration.
3. Invasive period (increasing severity of symptoms): fever, inflammation and swelling with tissue damage may occur in this period. Infection may spread to other sites. Alteration can be observed at the portal of entry, in the blood and the location of the infection. Pathogens may appear in these locations and in the body fluids. Acme (Fastigium) will be happen. Pathogens provoked systemic host responses
4. Declining period: declining signs and symptoms
5. Convalescence: no signs and symptoms

The infectious diseases may occur with short-term or long-term incubation period. The damage of the host depends on the longevity of this period: when the symptoms developed immediately after the invasion of the pathogens, the destruction caused by virulence factors of microbes, while the development of diseases has occurred after long-term incubation period, the tissue damage has been formed by harmful effect of immune reactions, especially by systemic responses.
The severity of infections influenced by virulence of microorganisms and susceptibility of the host, which are determined by numerous factors, can be found in the next figure (Figure 6.5):

**Factors influencing exposition and development, severity of infection**

*DISEASES OF CONCERN IN THE DENTAL OFFICE*

Causative agents of infectious diseases, which may be a risk for the dental team and patients, are classified based upon spreading: there are blood borne pathogens, respiratory pathogens, microbes spread by oral fluids and waterborne disease agents. The pathogens of the most common nosocomial infections in the dental office can be seen in the next figure (Figure 6.6):

**Nosocomial infection and their spreading, causative agents and danger in the dental office**

*THE IMMUNE SYSTEM*
Immunity is the ability of the host to resist an infectious disease. The functions of the immune system are: protection against microorganisms, transplanted organs and cancer cell growth. The immune system may be classified into two subsystems: non-specific (innate) immune system and adaptive (acquired, specific) immune system. Within these systems the humoral and cellular components can be distinguished. The innate immune system consists of cells and proteins that are always present and ready to mobilise and fight microbes at the site of the infection: the respond is linearly increasing, fast, without immunological memory. The adaptive immune system requires some time to react to an invading organism, requires the recognition of specific antigens (antigen presentation): the adaptive immune system is antigen specific. The acquired immune system demonstrates immunological memory: these specific memory cells react more rapidly on subsequent exposure to the same organism. There are four forms of specific immunity:

- acquired immunity (mentioned above),
- artificial active immunity: which is achieved by administration of vaccines
- congenital passive immunity: when antibodies are passed to a foetus from the mother (short-term immunity of newborn babies)
- passive immunity: which is achieved by administration of ready-made antibodies (preventing or treating infectious diseases by e.g. gamma globulins.

However, the two arms of the immune system have distinct functions, there is interplay between these systems.

Immunisation provides individuals artificial active immunity to numerous infectious diseases based on the immunological memory effects. The aim of the immunisation is to introduce an antigen from a pathogen in order to stimulate the immune system and develop specific immunity against that particular pathogen without causing disease associated with that organism. The immunisation may be achieved by live attenuated viruses, killed whole organisms and acellular components of micro-organisms including harmless toxin components. The effectiveness of vaccination may persist lifelong (rubeola) or in shorter periods (influenza). Vaccines may be produced against one or more serotypes of microbes, but there are vaccines based on alterable antigenicity of microorganisms.

**THE PRINCIPLES OF INFECTION CONTROL IN DENTISTRY**

The following principles are designed to reduce the risk of cross-infection in the dental office:

- patient screening
- office design
- work organisation
- disinfection and sterilization procedures
- barriers
- personal protective equipment
- immunisation
- waste management

**PATIENT SCREENING**

We classified the patients into two groups according to the nosocomial infection: individuals as the potential source of infection and patients as more susceptible hosts. However, every patient is treated as the potential source of infection and being particularly susceptible to the infection; thus, the screening of both risk groups is very important. Since the discovery of the HIV virus, risk patients treated in “high-risk units” have been separated from healthy patients. Unfortunately, several patients with infectious diseases have hidden their disease due to the stigmatisation of the infected patients, and presented to the dental office with the healthy subjects, thus threatening both the dental team and otherwise healthy patients treated in the dental surgery, as a result of lower standard of infection control.
The medical/dental history provides an excellent opportunity to recognise the patients at risk. The medical/dental history form may inform us by simple questions about the health conditions and medications of the patient. The patient’s signature validates the recorded specific information and motivates patients to complete this form and should not leave blank spaces because of later (legal) consequences. Having the written documentation is a key in collecting information on the patient’s previous medical conditions compared to the dental interview alone.

Prior to treatment, the current information may allow the application of safety measures at treatment of high risk patients (HIV, TBC, Hepatitis B, C patients) such as: treatment at the end of opening hours, special barriers, minimising the use of cooling spray, etc.. Information required by the medical/dental history interview may arise the suspicion of the patient’s risk factors including dwelling in an endemic area; behavioural habits; sexual orientation; mental disability; people who frequently require blood or blood products, dialysis patients, recipients of solid organ transplantations; people interned in prisons; IV drug users; people with multiple sexual partners, etc. During the medical/dental history interview, treatment planning and procedures have to serve all patients without discrimination considering their sensitivity.

Attention should be paid to the treatment of immunocompromised patients. The immunocompromised patient will be susceptible to infections caused by common microbes such as Pseudomonas aeruginosa, which are normally ubiquitous in dental unit waterlines. Patients can be immunocompromised by drugs, congenital or acquired diseases and conditions. We should pay particular attention to the dental treatment of individuals suffering from haematological and liver diseases.

**DENTAL OFFICE DESIGN**

A particularly requirement of the dental office’s design is to separate rooms and corridors according to the functions. The route of patients must not be crossed by the route of transportation of contaminated instruments and waste, and within the treatment room the hygienic zones must be clearly demarcated:

- treatment (contaminated) zone (all surfaces and items within the contaminated zone must be deemed contaminated by the treatment in progress),
- clean zone (those surfaces and drawers where clean or sterilized instruments are stored and that never come in contact with contaminated instruments or equipment.)

The treatment room designed for easy cleaning and disinfection with smooth, uncluttered surfaces, junction between floor and wall in the dental room shall be smooth curved lines without angles. Carpets, curtains, not cleanable decoration must be avoided. There must be good ventilation inside the dental procedure room and inside the dental facility in general. Ventilation should be enough to removes odours, fumes, condensation and keep the facility cool and comfortable to work in. Use of an air cleaner is recommended, however, the sterile air filtration is not required. Attention should be pay for lighting in the treatment room. Maximise provision of natural light to areas where staff spend a large proportion of their working day. The rate of lighting intensity between operating area and environments is 10:1.

**WORK ORGANISATION**

The preliminary treatment planning and work organisation are important parts of the infection control. Schedule of treatment should be based on a detailed treatment plan. Patient treatment should also be planned in advance in a logical sequence, which allows time for preparation of the treatment room: only the necessary instruments and materials should be found in the contaminated area. Utilising of single-dose materials, single-use instruments, preset cassettes may decrease the cross-contamination in the dental office. These are the tenets of the four-handed dentistry.

**DISINFECTION, STERILIZATION**

The modes of cross-contamination in the dental office are transmission of microbes from contaminated hands, instruments, surfaces, dental unit (surfaces, waterlines) and spreading by droplet (aerosol, slatter). The successful prevention of disease prevention depends on the elimination of pathogens from the contaminated area. Disinfection and sterilization are both decontamination procedures. While disinfection is the process of eliminating or reducing pathogenic microorganisms, sterilization is the process of killing all microorganisms and also destroying the spores.

The following items have to be decontaminated in the dental office:
• instruments
• impressions
• dental unit
• x-ray system
• walls, floors, cabinets (housekeeping area)

**Instrument processing**

Dental instrument may be classified into three groups: critical instruments are those used to penetrate soft tissue or bone, or enter into or contact the bloodstream or other normally sterile tissues. Semi-critical instruments are those that do not penetrate soft tissues or bone but contact mucous membranes or non-intact skin such as mirrors, reusable impression trays and amalgam condensers. Non-critical instruments are those that come into contact only with intact skin such as external components of x-ray heads, blood pressure cuffs and pulse oximeters. The critical and semicritical instruments should be sterilized after each use. In some cases, however, sterilization of semicritical items is not feasible and, therefore, high-level disinfection is appropriate.

The flow-chart of instrument processing is presented in Fig. 6.7:

![Instrument processing flow-chart](image)

After the treatment, the contaminated instruments have to be completely submerged into the pre-soaking solution to prevent the drying of protein contained debris. After the transportation of the instruments to the recycling area, instruments should be rinsed by lukewarm tap water (to prevent denaturation of proteins), and then cleaned mechanically by manual brushing or ultrasonic cleaner or washer disinfecter machine.
Ultrasonic cleaner
Washer disinfector machine

The duration of immersion disinfection required by the manufacturer for the intended use depends on the concentration of the solution. Instruments to be sterilized should be rinsed and dried after disinfection.
Instruments that are sterile at the time of use must be packaged (bagged or wrapped) prior to sterilization. The adequate storing and undamaged wrapping maintain the sterility of the instruments. Storing of sterilized packaged instruments depends on the type of the sterilizers and the mode of packaging.

Requirements of disinfectant agents

The perfect disinfectant agent must be able to kill the pathogenic microorganisms in a short time period, remaining effective long-term, must be effective on items contaminated by protein-containing body fluids and debris without harming humans and items (e.g. non-corrosive), be odourless and inexpensive. Disinfectants fulfilling all the requirements are missing in the market.

Quaternary ammonium compounds, aldehyds, alcohols, oxidizing agents (iodine, halogens, etc.), and silver are most commonly used as disinfectants.

Sterilization

Sterilization is the elimination of all forms of microbial life. Sterilization can be achieved by physical (heat, high pressure, or filtration), chemical processing, the heat and moist sensitive instrument can be sterilized by irradiation.

In a dental practice, the most efficient and simplest approved methods are the types of heat sterilization. Heat sterilization can be caused by moist (steam under pressure) or dry heat.

The consistent and constant temperature is carried out by ventilation generated by forced air system or gravitation in the dry heat sterilizers. Specific conditions of temperature and time for certain preparations are: 160 °C for 45 minutes, 180 °C for 25 minutes, 200 °C for 10 minutes. The whole cycle of sterilization takes more time because of preheating and cooling down before and after effective sterilizing period.

Autoclaves uses steam under pressure to destroy all forms of microbial life. Steam should penetrate all surfaces and areas of instruments placed inside the chamber of machine achieved by air evacuation. In the N-class type sterilizers, steam pushes the air downwards using gravity and forces it out via a port in the bottom of the chamber. This machine can be used for sterilizing unwrapped, solid items. The S-class sterilizers used with multi-pulse vacuum may allow us the sterilization of wrapped hollow objects, where the ratio of the length of the hollow portion to its diameter is no more than 1:5. B-class machines are necessary for the sterilization of hollow, porous and wrapped goods. These pre-vacuum or Class-B sterilizers remove air from the load prior to the chamber being pressurised with steam by fractioned vacuum.
Loading the sterilizer

Heat-sensitive items can be sterilized with **plasma sterilizers**. Plasma sterilizers use hydrogen peroxide vapour and low-temperature gas plasma to sterilize most devices quickly with no toxic residues. The temperature should be kept lower than 60°C and the cycle time is under 1 hour, which prevents heat and moisture damages to sophisticated medical instruments, and prolongs the life expectancy of them. Chemical vapour sterilization and ethylene oxide treatments are no longer accepted because of their toxicity.

Each sterilization cycle must be **documented** and **monitored**. This information is typically recorded in a sterilization log. Documentation is more simple with electronic devices: the sterilization log can be printed by connected or built-in printer or saved on PC or server by flash card, network devices.

*Disinfection methods of impressions*

Dental impressions are contaminated by saliva, debris and large amount of microbes during procedures. Rinse the impression, after removal from the patient's mouth, gently with tap water. Sprinkle dental stone into impression before rinsing to aid in cleaning. Impressions should be disinfected following cleaning and rinsing. Methods of decontamination are spraying, dipping, and immersing. Exposure time depends on the composition, and the concentration of disinfectants should be as recommended by the manufacturer of disinfectant for tuberculocidal disinfection. Rinse and dry impressions prior to their delivery to labs. Place them in a labelled plastic bag annotated with the description of the disinfecting procedure (composition and exposure time of disinfectant). Shorter exposure time may cause inadequate disinfection, however, longer exposure time may damage the impression.

*Surface and equipment decontamination*

Equipment (Dental unit, x-ray, and various machines), cabinets, walls, floors should be decontaminated. Disinfectant can be applied via spray or wipes according to the recommendation of the manufacturer. The floor should be cleaned daily, visible parts and front of cabinets should be disinfected weekly. The inner part of cabinets must be cleaned monthly. Purity painting should be performed in every year.

**INFECTION CONTROL OF THE DENTAL UNIT**

Treatment of waterlines and suction unit of the dental equipment is an important part of an infection control protocol. Water in dental lines is stagnant 99 percent of the time, thus allowing the accumulation of microbes on the inner tubing surfaces. Flushing waterlines may reduce biofilm accumulation. Air and waterlines from any device connected to the dental water system that enters the patient’s mouth (e.g. handpieces, ultrasonic scalers, and air/water syringes) should be flushed for a minimum of two minutes at the start of the day and for 30 seconds between patients. All handpieces should be fitted with anti-retraction valves to prevent retrograde contamination of the lines by fluids from the oral cavity. An independent water supply contains less amount of microbes compared to tap water and can reduce the accumulation of biofilm. Decontamination of dental unit waterlines may be performed by sodium hypochlorite solution (cc. 5%) by filling it into the water bottle or perfusing it by special accessories of the dental unit.
Disinfector of dental unit waterlines

Regular flushing out of suction units with water is not enough to fight biofilm build-up. Regular application (between every patient) of disinfectants always protects against biofilm formation according to the recommendation of the manufacturer.

BARRIERS

A common method of infection control is to utilise barriers. Barriers may be classified into two groups: protective materials of team members, personal and protective materials covering of equipment and accessories. Dental instrument sleeves, chairs, x-y sensors and tubes, keyboards of dental units and PC, lamps, etc. may be contaminated by saliva, splatter, blood and debris protected by several types of barrier film, and disposable plastic covers.
Protective films and plastic covers

Single-use bibs or drapes should be used to protect the patient’s clothing, and reduce their exposure to spatter and debris created during dental procedures. Sterile, single-use drapes should be used to the isolation of surgical sites to protect the contamination of suture materials, the operator’s hand by hair, skin, and clothes of the patient.

PERSONAL SAFETY AND BARRIER PROTECTION

Protective wear and personal protective equipment (PPE: gloves, mask, eyewear) should be worn by all members of the dental team during procedures.

Medical wear must cover the whole body and arms. Wrists should be covered by gloves.

Protective eyewear is designed to safeguard the eyes from splatter and mechanical damage coming from the lateral side. Eye protective devices may be glasses, goggles, glasses with shields and a face shield.

A surgical face mask should cover the nose and mouth during dental procedures. Masks lose efficiency over time, as they become moist from the team member’s breathing. Accordingly, masks should be changed between each patient or sooner if they become visibly soiled. Masks are classified according to their filtration efficiency. Filtration efficiency means the percentage of filtered particles of dental aerosols (size: 0.1-0.3 micron). Requirements of the face mask: good bacterial filter efficiency, good sub-micron particulate filtration efficiency and resistance to penetration by synthetic blood over 160 mmHg and good breathability and formfitting to the nose and face.
Personal barrier protection

The dental hygienist must remove all jewellery, and should perform hand hygiene procedures before putting on and after removing the gloves. The hands should be dried before gloving. Gloves must be changed between patients or during lengthy procedures (more than 1 hour duration). In case of latex sensitivity (dental team members or patient!), latex-free gloves have to be worn by the dental staff.

Hand hygiene should be performed:

- following personal body functions (e.g. blowing nose or using the bathroom);
- before and after direct contact with individual patients;
- before putting on and after removing gloves;
- after contact with environmental surfaces, instruments or other equipment in the dental operatory;
- after contact with dental laboratory materials or equipment;
- before and after eating or drinking.

Hand hygiene should be performed by washing with plain or antimicrobial soap and running water, or by using a 70–90% alcohol-based hand scrub. Both methods are equally effective, unless hands are visibly soiled (including powder from gloves) or contaminated with body fluids, in which case hands should be washed with soap and water.

Hand hygiene should be performed by using a 70–90% alcohol-based hand scrub if hands are not visibly soiled or not contaminated with body fluids. Hand alcohol should be thoroughly rubbed into the hands and on the lower forearm for a duration of at least 30 seconds, and then allowed to air dry. Hand washing with antimicrobial soap and running water should be performed if hands are visibly soiled (including powder from gloves) or contaminated with body fluids. The use of alcohol-based hand gels dries the skin less, leaving more moisture in the epidermis, than hand washing with antiseptic/antimicrobial soap and water preventing the
microinjury and fissures of the skin and protecting from microbial penetration. Before surgical procedures, surgical hand-washing should be performed by all sterile team members.

Aim of the surgical hand preparation to eliminate the transient (on the epithelial cell) and reduce the resident (microbes residing under the superficial cells of the stratum corneum and can also be found on the surface of the skin) flora and it should also inhibit the growth of bacteria under the gloved hand.

Nail cleaning should be performed prior to surgical hand washing. The nail cleaning consists of removing debris from underneath fingernails using a nail cleaner under running water and cleaning of outer surfaces of nails and adjacent skin. Requirements of surgical hand hygiene:

- fingernail cleaning performed by single-use nail cleaner.

- careful nail cleaning without injuries.

- fingernails should be short and smooth.

- fingernail cleaning should be performed out of surgical theatre or a day prior to procedures.

- surgical procedures should be performed by intact skin and nails covered by gloves.

- in case of wounded skin, dermatitis or infection, surgical procedures must be cancelled

- remove rings, watches, and bracelets before beginning the surgical hand scrub. Fingernails must be free of any artificial nail.

- after surgical procedures, treat hands with hand cream or lotion to protect the skin following hand washing

**Scrub** each side of each finger, between the fingers, and the back and front of the hand. Proceed to scrub the arms, keeping the hand higher than the arm at all times. This prevents bacteria-laden soap and water from contaminating the hand. Wash each side of the arm to three inches above the elbow. Duration of hand washing should be at least 1 minute, but no more than 2 minutes.

**Rinse** hands and arms by passing them through the water in one direction only, from fingertips to elbow. Do not move the arm back and forth through the water. Inadequate rinsing and remaining soap on the skin result in decreasing antimicrobial efficiency of hand scrubs.

**Drying** of hands: Single-use, non-sterile paper towel is used in operating theatres to dry wet hands after surgical hand antisepsis (e.g. Hagleitner Luna, Papertowel/Spender, Tork Hand Towel H3 Classic).

**Surgical hand scrub** methods require surgical hand scrubs approved by the authorities. Hand scrubs should be applied without dilution according to the recommendations of the manufacturer. An effective surgical hand scrub should take no longer than five minutes: 3-minute scrub could be as effective as the 5-minute scrub, depending on the formula of the scrub agent. After application of the alcohol-based product as recommended, allow hands and forearms to dry thoroughly before putting on the sterile gloves.

**IMMUNISATION, OCCUPATIONAL SAFETY AND HEALTH**

It is the responsibility of all dental team members to maintain a high level of personal health. The health care personnel must be vaccinated according to occupational safety and health regulations. Vaccination of the staff must be documented.

Significant exposures include percutaneous injuries with contaminated needles, burs or other sharp instruments, as well as accidents in which blood, saliva or other body fluids are splashed onto non-intact skin or the mucosa of the eyes, nose or mouth. However, percutaneous injuries pose the greatest risk of transmission of blood-borne pathogens to dental hygienists. In the event of a significant exposure, immediate first-aid measures should be instituted:

- For percutaneous injuries, allow the wound to bleed briefly and freely. Then, gently wash the wound with soap and water, and bandage as needed.

- For exposures involving the eyes, nose or mouth, flush the area with copious amounts of water.
• For exposures involving non-intact skin, wash the site with soap and water.

Any kind of occupational injury should be reported according to occupational safety and health regulations.

**WASTE MANAGEMENT**

Released waste in the dental office may be classified into two groups: municipal solid waste and hazardous waste. Hazardous waste may be medical waste or chemical waste. Hazardous waste must be removed from the office according to the regulations. Any type of disposable sharps must be disposed in puncture-resistant, colour-coded and labelled, closable, leakproof containers approved by governmental agencies. Other types of medical waste disposed of in closable, leakproof bags. The processes of waste management must be documented.

![Container of hazardous medical waste](image)

**4. Practice management (Angyalka Segatto DMD)**

**HEALTH POLICY**
Dramatic changes began in the health care industry in the late 1980s. As a result, health care institutions continue to face a turbulent, confusing and often threatening environment. Significant changes have come from many sources including legislative and policy initiatives; international as well as domestic economic and market forces; demographic shifts and lifestyle changes; technological advances; and fundamental health care delivery changes.

Health policy determines the rules of the game that apply to all consumers and providers in the field. The role of health policy is to determine the preferences of the society and to develop and fine tune institutions that can efficiently meet those preferences. Meeting preferences may mean defining the ground rules under which insurers and providers compete.

**ENVIRONMENTAL ANALYSIS**

*External environment:*

- Composed of a number of organisations and individuals in the general health care environment.

Some of the organisations and individuals in the external environment have little direct involvement in the health care industry while others are directly involved. The distinction is not always clear. These organisations and individuals, through their normal operations and activities, are generating changes that may be important for the future of other organisations. Changes in the general environment are always “breaking through” to the health care environment.

*Strategic issues:*

- Are trends, developments, dilemmas and possible events that affect an organisation and its position within its environment.

The scanning function serves as the organisation’s “window” or “lens” on the external world. The scanning process categorises, organises, accumulates and evaluates issues.

*Information sources:*

- There are a variety of sources for environmental information.

Although organisations create changes as well. Essentially, people and publications both inside and outside the organisation serve as the lens to the external world. Many organisations collect patient and consumer information. Outside the health care organisation, patients, physicians, nurses, suppliers, third-party payers, pharmaceutical representatives may be considered important direct sources. Indirect sources involve mostly newspapers and journals, internet, TV, libraries, and public and private databases.

Environmental scanning is perhaps the most important part of environmental analysis. In the scanning activity issues and changes are specified and sources identified. An important aspect of environmental scanning is that it focuses leaders attention on what lies outside the organisation and enables them to create an organisation that can adapt to and learn from that environment.

**STAKEHOLDERS**

*Stakeholders:*

- Forces affecting the organisation.

Stakeholders may be categorised as internal, interface and external.

*Internal:* those who operate primarily within the bounds of the organisation such as managers and employees.

Interface: those who function both internally and externally (medical staff and corporate officers).

*External:* operate outside the organisation (suppliers, third-party payers, competitors, regulatory agencies, media, and the local community).

Some of the stakeholders are almost always powerful or influential; others are influential regarding only certain issues; still others have little influence or power.
COMPETITOR ANALYSIS

Service area competitor analysis is a process of understanding the market identifying and evaluating competitors. Together with general and health care trends and issues, service area and competitor analysis must be synthetised into strategic issues facing the organisation. Competitors providing services in the same category in the service analysis must be analysed. Competitors can be positioned against the important dimension of the market and assessed as to their likely strategic moves.

Dentistry represents a highly competitive and low market share industry. Typically there is a number of general dentists competing not only among themselves but also against other providers. The first step in service area competitor analysis is to specify the service category to be analysed. The service area is considered to be a geographic area surrounding the health care provider from which it gains the majority of its customers/patients. A health care organisation should not only define its service area but must also analyse the details of all relevant and important aspects of the service area.

The service area is defined by customer’s preferences and by the health care providers that are available.

THREAT OF NEW ENTRANTS

New entrants into a market are typically a threat to existing organisations because they increase the intensity of competition. New entrance may have substantial resources and often attempt to rapidly gain market share. Such actions may force prices and profits down. The barriers may be assessed to determine the current or expected level of competition within industry or service area. In health care markets, the barriers to entry for new “players” may be substantial. Certificate of need, laws and regulations can present significant barriers!

COMPETITIVE ADVANTAGES

Organisations are successful when they create value for their customers. Similarly, health care organisations are successful to the extent that they create value for the patients and other stakeholders. Value is defined as the amount of satisfaction received relative to the price paid for a health care service. Despite the high price, the perception of social acceptance increased feeling of self-esteem and improve self-conscience may provide so much satisfaction that the patient perceives very high value. Value is the perceived relationship between satisfaction and price, and it is not based solely on price.

Being aware of the strengths and weaknesses of an organisation can help us create a competitive advantage. A competitive advantage is created within the organisation in the form of strengths that are important in the external analysis. A competitive advantage for many health care organisations was primarily a matter of “position” where the physician’s office occupied well-defined competitive niche. In today’s environment, strengths can quickly become weaknesses as successful strategies are challenged by competitors.

Strengths are subjective in that they represent the opinion of people who are doing the evaluation. Competitive advantages of an organisation may be based on having rare or abundant resources, special competencies or skills or management or logistical capabilities. Similarly, competitive disadvantages may result from the lack of resources, competences or capabilities.

One of the most important factors is identifying the target market. One of the difficulties with health care marketing is that there are many very diverse customers to satisfy. Segmentation is the process of identifying a recognisable group that makes up the market and then selecting a group as the target market. A proper segmentation can be the future’s key success factor.
Chapter 7. References

1. Books


25. HARDT N, KUTTENBERGER J: Craniofacial Trauma. Springer, 2010


29. KOVÁCS ÁDÁM: Maxillofacial trauma traumatológia, Semmelweis Kiadó, 1999


35. MCNEILL C: Science and Practice of Occlusion, Quintessence 1997.


41. PERTES RA, GROSS SG: Clinical management of temporomandibular disorders and orofacial pain. Quintessence, 1995


References

49. SONKODI ISTVÁN: Orális és maxillofaciális medicina. Semmelweis Kiadó, Budapest, 2006
60. WILKINS EM: Clinical Practice of the Dental Hygenist. 10th Lippincott Williams and Wilkins, Baltimore, 2009.

2. Articles

7. BERNIE KM: Clinical Considerations for the Dental Hygienist in Orthodontic Therapy. CDHA Journal Vol.23 No.2.


References


72. RAAP U, STIESCH M, REH H, KAPP A, WERFEL T. Investigation of contact allergy to dental metals in 206 patients. Contact Dermatitis 2009; 60: 339-343


74. ROHRER N, WIDMER AF, IMO T, KULIK EM, WEIGER R., FILIPUZZI-JENNY E, WALTER C: Antimicrobial efficacy of oral antiseptics containing octenidine, polyhexamethylene biguanide, or Citroxx: can chlorhexidine be replaced? Infect Control Hosp Epidemiol. 2010; 31: 733-739


