A TEXTBOOK

OF ORAL AND MAXILLOFACIAL SURGERY

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Plovdiv, 2018
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PUBLISHER:

MEDICAL UNIVERSITY – PLOVDIV, BULGARIA


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SALIVARY GLAND DISORDERS

SOFT TISSUE CYSTS

JAW CYSTS

DEFICIENCY SYNDROME (AIDS)

HUMAN IMMUNODEFICIENCY VIRUS (HIV)

SPECIFIC INFLAMMATION

MAXILLARY SINUSITIS

LYMPHADENITIS

ODONTOGENIC OSTEOMYELITIS

NONSTEROIDAL ANTIINFLAMMATORY DRUGS (NSAIDs)

ANTIBIOTICS

SURGICAL TREATMENT OF ACUTE INFLAMMATIONS

ODONTOGENIC SEPSIS

LIFE-THREATENING CONDITIONS IN ACUTE ODONTOGENIC INFECTION

IN ACUTE ODONTOGENIC INFECTION

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(Petia Pechalova)

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INFLAMMATORY PROCESSES IN ORAL CAVITY
AND MAXILLOFACIAL AREA
(Petia Pechalova)

Inflammation of oral cavity and maxillofacial area could be divided in two main groups, depending on their origin:

1) Odontogenic inflammations:
   - Periapical periodontitis
   - Marginal periodontitis
   - Pericoronitis and paracoronitis
   - Infected odontogenic cysts
   - Odontogenic sinusitis
   - Odontogenic lymphadenitis
   - Odontogenic osteomyelitis

2) Non-odontogenic inflammations:
   - Rhinogenic sinusitis
   - Infected soft tissue wounds
   - Adenophlegmons
   - Peritonsillar abscesses
   - Infected fractures of the jaws
   - Non-odontogenic osteomyelitis
   - Sialadenitis

The main clinical forms of inflammations are:

- **An abscess** (from Latin, abscessus) is a localized collection of pus, a result of necrotic suppuration inflammation, caused by bacteria, surrounding by a fibrous wall – “pyogenic membrane” - an expression of the body's attempt to isolated inflammation from the healthy tissues.

- **A phlegmon** (cellulitis) is a diffuse, rapidly progressive, suppurative inflammation with poorly defined margins (without pyogenic membrane) and more severe clinical presentation compared to an abscess.

- **An adenophlegmon** is a subtype of phlegmones which originates from inflamed lymph nodes. It characterized with prolonged clinical course compared to regular phlegmon, because a period of time is necessary for the infection to dissolve the membrane of the lymph nodes and spread of the inflammation in the surrounding tissues.

Based on mode of involvement, facial spaces could classified as:

I. Primary (in these spaces could develop primary odontogenic inflammation):
   1) Primary maxillary (canine fossa and infraorbital, buccal, infratemporal)
   2) Primary mandibular (siblingual)

II. Secondary (in these spaces development of primary odontogenic inflammation is impossible due to anatomical reasons and the inflammation spreads from the surrounding primary spaces):
   1) Masseteric,
   2) Temporal
   3) Pterygomandibular,
   4) Parapharyngeal,
   5) Retropharyngeal,
   6) Parotid
Canine fossa abscess and infraorbital abscess

Anatomy: The canine fossa is a small space between the levator labii superioris and the levator anguli oris muscles. The boundaries of the infraorbital space are:

- the nasal cartilages (anteriorly)
- the buccal space (posteriorly)
- the levator labii superioris (superiorly and superficially)
- the oral mucosa of the maxillary vestibular fold (inferiorly)
- the levator anguli oris muscle (the deep border)

Infraorbital region contains angular artery and angular vein, infra-orbital nerve (a branch of the maxillary division of the trigeminal nerve).

Etiology: Infected root canals of maxillary premolars and canines are considered to be responsible for the development of abscesses of the canine fossa.

Clinical features: Canine fossa abscess is characterized by edema, localized in canine fossa, which spreads towards the eye, and side of the nose as far as the corner of the mouth. The lower eyelid is swollen, but painless in palpation (collateral edema due to lymphostasis without inflammation), the upper eyelid is without changes. There is also obliteration of the nasolabial fold, and somewhat of the mucolabial fold. The edema at the canine fossa region is painful during palpation, and later the skin becomes taut, reddish and shiny.

Infraorbital abscess is characterized by the same features as the canine fossa abscess. The differences are: (1) the inflammatory edema of lower eyelid, which is painful on palpation, (2) the upper eyelid is swollen but painless (collateral edema) and (3) the eye is closed. Infraorbital abscess is more severe compared to canine fossa abscess.

Surgical treatment: The incision for is performed intraorally and horizontally at the mucobuccal fold, parallel to the alveolar bone), in the canine region A hemostat is then inserted, which is placed at the depth of the abscess until it comes into contact with bone. Finally, a rubber drain is placed. Extraoral incision could be made in the nasolabial fold.

Buccal space abscess

Anatomy: The boundaries of the buccal space are:

- the angle of the mouth (anteriorly)
- the masseter muscle (posteriorly)
- the zygomatic process of the maxilla and the zygomaticus muscles (superiorly)
- the depressor anguli oris muscle and the attachment of the deep fascia to the mandible (inferiorly)
- the buccinator muscle (medially)
- the platysma muscle, subcutaneous tissue and skin (laterally)

The buccal space contains the buccal fat pad (also called Bichat’s fat pad), the parotid duct (Stenson’s duct), the anterior facial artery and vein, the transverse facial artery and vein.

The buccal space communicates posteriorly with the pterygomandibular space, infratemporal space, submasseteric space or even to the lateral pharyngeal space. Superiorly the buccal space communicates with the infraorbital space and canine space.

**Etiology:** The buccal space abscess may originate from infected root canals of posterior teeth of the maxilla and mandible.

**Clinical sings:** Buccal space abscess is characterized by swelling of the cheek, which extends from the zygomatic arch as far as the inferior border of the mandible, and from the anterior border of the ramus to the corner of the mouth. The skin appears taut and red, with or without fluctuation of the abscess. The most severe pain is provoked during simultaneously intra- and extraoral palpation (for the right cheek the index finger is placed on the mucosa of the cheek, the thumb of the same hand – on the skin of the cheek or opposite for the left cheek - the thumb is placed on the mucosa of the cheek, the index finger of the same hand – on the skin of the cheek).

**Surgical treatment:**

Horizontal intraoral incision, parallel to the Stenson’s duct (above or below the projection of the duct in order to prevent injury of the salivary duct, which could lead to strictures, or narrowing of the duct, and salivary gland diseases) is preferable method.

An extraoral incision is made when intraoral access could not ensure equate drainage, or when the pus is near to the skin. The extraoral incisions (black lines) could be solely or multiple, parallel to the projections of the facial nerve - parallel to the imaginary lines between the tragus of the ear and lateral angle of the eye, ala nasi, and angle of the mouth (red lines). In severe cases both intraoral and extraoral incision are made. As a rule intraoral drainage is by rubber drain, while in extraoral incision tube drains are inserted.

*Pandey et al, 2011*
Infratemporal abscess

**Anatomy:** Infratemporal (below the temporalsis muscle) space is located posterior to the maxilla, between the lateral pterygoid plate of the sphenoid bone, medially and by the base of the skull superiorly. The boundaries of the infratemporal fossa are formed by bone and soft tissue:

- ramus of the mandible (lateral)
- lateral pterygoid plate of the sphenoid (medial)
- posterior surface of the maxilla (anterior)
- carotid sheath (posterior)
- The floor of the infratemporal fossa is comprised of the medial pterygoid muscle
- The roof is formed by the greater wing of the sphenoid bone (two foramina open out on the roof – the foramen ovale and foramen spinosum; they provide a connection with the cranial cavity)

The infratemporal fossa acts as a pathway for neurovascular structures passing between the cranial cavity, pterygopalatine fossa and temporal fossa. It also contains some of the muscles of mastication - the medial and lateral pterygoids are located within the fossa itself, whilst the masseter and temporalsis muscles insert and originate into the borders of the fossa. Infratemporal space contains branches of the maxillary artery and the pterygoid venous plexus, as well as the mandibular nerve, auriculotemporal, buccal, lingual and inferior alveolar nerves, chorda tympani and otic ganglion. The communications of the infratemporal space are the pterygomandibular space (inferiorly), the buccal space (anteriorly and inferiorly), the cavernous sinus via the pterygoid plexus of veins.

**Etiology:** Infections of the infratemporal space may be caused by: (1) infected posterior maxillary teeth, especially the wisdom teeth, (2) the inflammation of the pterygomandibular space, and (3) may also be the result of the maxillary nerve block (tuberosity approach).

**Clinical presentation:** Severe trismus (III grade) is the main clinical symptom. The pain is provoked during palpation behind the tuber maxillae or in an attempt to opening the mouth. The swelling is discrete and could be missing or could be observed as a small edema below and above the zygomatic arch, which is presented as a slight depression.

**Surgical treatment:** Intraoral horizontal incision, in the mucobuccal fold, behind the last maxillary molar is suggested. With curve hemostat, inserted into the incision, moving upward, inward and backward, the abscess is opened and rubber drain is inserted.
Temporal abscess

Anatomy: Temporal space is divided into superficial and deep temporal spaces. The superficial temporal space is bounded laterally by the temporal fascia and medially by the temporalis muscle, while the deep temporal space is found between the medial surface of the temporalis muscle and the temporal bone. Temporal space contains temporal arteries and veins, auriculotemporal nerve. The superficial temporal space communicates with buccal space via Bichat’s fat pad, the deep temporal space communicate with infratemporal space.

Etiology: Infection could be spread from surrounding anatomical spaces (by neighborhood), by hematogenous or by lymphogenous way. The reason for development of the temporal abscess never ever could be odontogenic.

Clinical presentation: large and very painful edema of the temporal area, severe trismus due to involvement of the temporalis and medial pterygoid muscles. The general health status is impaired.

Surgical treatment: The puss evacuation could be made by extraoral incisions only. The hair in temporal area is shaved. Superficial temporal abscesses are incised by multiple, radial incisions (puncture lines), parallel to the muscle fibres of temporalis muscle.

In cases of deep temporal abscess, multiple incisions are suggested, parallel to the linea nuchae superior and solely incision below the zygomatic arch (solid line); with hemostat, inserted into the zygomatic incision (under the soft tissues in contact with temporal bone), is made a temporary canal to each of linea nuchae incisions; plastic drains are inserted and are sutured to the skin. In cases of phlegmomes, that involved both superficial and deep part of the space, two types of incisions are made.

Submental abscess

Anatomy: The submental triangle, is an unpaired suprahypoid area lying inferior to the chin. It is limited by the body of the hyoid bone inferiorly, laterally by the right and left anterior bellies of the digastric muscles. These bellies of the digastrics muscle taper superiorly and forward towards the apex of the triangle. The apex of the submental triangle is at the mandibular symphysis. The hyoid bone forms the base of the triangle, while the roof is formed by the two mylohyoid muscles, which meet in a median fibrous raphe. The submental triangle contains one to four lymph nodes (the submental lymph nodes), a submental
artery and some small submental veins, which unite to form the anterior jugular vein. The submental space communicated with the submandibular spaces posterolaterally and the sublingual space superiorly (via erosion through the mylohyoid).

**Etiology:** Infection of the submental space usually originates from the mandibular anterior teeth, if their apices are located below the mylohyoid muscle or is the result of spread of infection from other anatomical spaces.

**Clinical presentation:** The infection presents as an indurated, painful and fluctuated submental edema. The skin is reddish. The swallowing is painful and difficult.

**Surgical treatment:** An extraoral incision of the skin and subcutaneous tissues is suggested. The incision could be vertical – from the chin to the hyoid bone, or horizontal – parallel to the mandible.

### Submandibular abscess

**Anatomy:** The submandibular triangle is bordered superiorly by the inferior part of the mandible and the mastoid process, posteriorly by the posterior belly of digastric and stylohyoid, and anteriorly by the anterior belly of digastric. The roof of the triangle is the skin, superficial fascia, the platysma and the deep fascia. The floor is formed by the mylohyoideus muscle (anteriorly) and by the hyoglossus muscle (posteriorly).

The submandibular triangle contains:
- The external and internal carotid artery
- The internal jugular vein
- The deep cervical lymph nodes
- Vagus nerve
- The submandibular gland
- The submandibular lymph nodes
- The Facial artery and vein
- Hypoglossal nerve

The submandibular space communicated with: (1) the submental space; (2) the sublingual space (located above the mylohyoid muscle); (3) the lateral pharyngeal space.

**Etiology:** Infection of submandibular space may originate from the mandibular second and third molars, if their apices are found beneath the attachment of the mylohyoid muscle (if their apices are above the mylohyoid muscle, the infection could involve the sulcus mandibulo-lingualis). The submandibular abscess may also be the result of spread of infection from the sublingual or submental spaces.
Clinical presentation: The infection presents as moderate swelling at the submandibular area, which spreads, creating greater edema that is indurated and redness of the overlying skin. Also, the angle of the mandible is obliterated, while pain during palpation and moderate trismus due to involvement of the medial pterygoid muscle are observed as well.

Surgical treatment: The incision for drainage is performed extraorally, approximately 2 cm beneath and parallel to the inferior border of the mandible (the marginal mandibular nerve, a branch of the facial nerve, is located over the inferior border of the mandible). During the incision, the course of the facial artery and vein should be taken into consideration (the incision should be made posterior to these). A hemostat is inserted into the cavity of the abscess to explore the space. A rubber drain is placed.

Sublingual abscess and abscess of mandibulo-lingual sulcus

Anatomy: There are two types of sublingual spaces above the mylohyoid muscle: (1) sublingual area that is located in the anterior part of the space, between the inner surface of the mandible (from the left to the right canines) and the tongue; and (2) sulcus mandibulo-lingualis – left and right, that is located in the distal regions between the corpus of the mandible (from canine to third molar) and the tongue. These three spaces are located above the mylohyoid muscle. The roof of the spaces is oral mucosa. Sublingual area contains the sublingual salivary gland. Saliva from the sublingual gland drains through several small excretory ducts in the floor of the mouth. Sometimes a more distinctive duct can be recognized, known as Bartholin's duct. The mandibulo-lingual space contains a number of blood vessels and nerves, e.g. the lingual artery and nerve, the hypoglossal nerve and the glossopharyngeal nerve, the deep part of the submandibular gland and the submandibular duct (Wharton's duct) and some extrinsic tongue muscle fibers. The sublingual space communicates posteriorly around the posterior free border of the mylohyoid muscle with the submandibular space. Infections of the sublingual space may also erode through the mylohyoid, or spread via the lymphatics to the submandibular and submental spaces.

Etiology: Infection of sublingual area may originate from the incisors and canine, if their apices are found above the attachment of the mylohyoid muscle (if their apices are found beneath the mylohyoid muscle, the infection could involve the submental area). The abscess may also be the result of spread of infection from the mandibulo-lingual sulcus, submental space or tongue. Infection of the mandibulo-lingual sulcus may originate from the mandibular premolars and molars, if their apices are found above the attachment of the mylohyoid muscle (if their apices are found beneath the mylohyoid muscle, the infection could involve the
The abscess of mandibulo-lingual sulcus may also be the result of spread of infection from the submandibular area, sublingual area or tongue.

**Clinical presentation:** The abscess of the sublingual space presents with characteristic swelling of the mucosa of the floor of the mouth, resulting in elevation of the tongue towards the palate. The patient speaks with difficulty, because of the edema, and movements of the tongue is painful.

The abscess of the mandibulo-lingual sulcus presents as a swelling of the area, elevation of the tongue laterally, slight trismus, difficulty in swallow and pain.

**Surgical treatment:** The incision for drainage of sublingual area is performed intraorally, parallel to the inner surface of the mandible (the blade of the scalpel is oriented perpendicular to the bone). The incision of the mandibulo-lingual inflammation is intraoral, parallel to the mandible in the distal area of the region, laterally, and along Wharton’s duct and the lingual nerve. In order to locate the pus, a hemostat is used to explore the space inferiorly, in an anteroposterior direction and beneath the submandibular gland. After incision, a rubber drain is placed.

**Lingual abscess**

**Anatomy:** The tongue is built by four intrinsic (superior longitudinal, inferior longitudinal, transverse, vertical muscle) and four extrinsic muscles (genioglossus, hyoglossus, styloglossus, palatoglossus muscle) covered by a mucous membrane. Although the muscles do not act in isolation, intrinsic muscles generally alter the shape of the tongue, whereas extrinsic muscles alter its position. From anterior to posterior, the tongue has three surfaces: tip (apex), body, and base. The mobile part of the tongue consists of the tip and the body. The tip is the highly mobile, pointed anterior portion of the tongue. Posterior to the tip lies the body of the tongue, which has dorsal (superior) and ventral (inferior) surfaces. The median sulcus of the tongue separates the body into left and right halves. The terminal sulcus is a V-shaped furrow that separates the body from the base of the tongue. At the tip of this sulcus is the foramen cecum, a remnant of the proximal thyroglossal duct. The base of tongue
contains the lingual tonsils, the portion of Waldeyer’s ring. The tongue derives arterial blood supply from the external carotid artery by the lingual artery. Motor innervation for all of the muscles of the tongue comes from the hypoglossal nerve with the exception of the palatoglossus, which is supplied by the pharyngeal plexus (fibers from the cranial root of the spinal accessory nerve carried by the vagus nerve). General sensation of the anterior two thirds of the tongue is supplied by the lingual nerve, a terminal branch of the third division of the trigeminal nerve. Taste sensation for this portion of the tongue is carried by the chorda tympani branch of the facial nerve. The posterior third of the tongue relays general and sensation via the lingual-tonsillar branch of the glossopharyngeal nerve. Some general and taste sensation from the base of tongue anterior to the epiglottis is carried by the internal laryngeal branch of the superior laryngeal nerve. Lymphatics from the tip of the tongue travel to the submental lymph nodes. This can be ipsilateral or bilateral depending on the site of the lesion. Lymph from the medial anterior two thirds of the tongue travels to the deep cervical lymph nodes, and lymph from the lateral anterior tongue goes to the submandibular nodes. The tongue-base lymphatics drain bilaterally into the deep cervical lymph nodes.

Etiology: Infection of the tongue is from nonodontogenic origin.

Clinical presentation: The inflammation of the mobile part of the tongue is presented as unilateral edema, painful in palpation. This painful swelling makes difficult tongue movement. The swallowing is not affected. The inflammation of the base of the tongue is presented as an extraoral swelling around the hyoid bone, which worsen swallowing because the severe pain. The protrusion of the tongue outside of the oral cavity is impossible. If the edema of the base of the tongue became larger, there is a risk of stenosis of the trachea and suffocation could observed.

Surgical treatment: The incision for drainage of the mobile part of the tongue is performed intraorally, parallel to the lengthy of the tongue, preferably near to the lateral border.

The incision for drainage of the tongue base is performed extraorally under general enesthesia. The incision is made in the submental region, near to the hyoid bone.

Floor of the mouth and tongue phlegmon (cellulitis)

Anatomy: Floor of the mouth is composed from six anatomical spaces around the mandible – three of them are located above the mylohyoid muscle and three are below it, and from the base of the tongue.
I. Above the mylohyoid muscle are located:
   1) Sublingual space;
   2) Left sulcus mandibulolingualis;
   3) Right sulcus mandibulolingualis.

II. Below the mylohyoid muscle are located:
   1) Submental space; 2) Left submandibular space;
   3) Right submandibular space.

    **Definition:** Phlegmon of the floor of the mouth developed when the inflammation involves **at least two** primary mandibular spaces.

    **Clinical signs:** Clinical presentation depend on the anatomical spaces are involved. The patient complains from pain on swallowing and general malaise. Breathing is often difficult. Patients will be characterized by a forced posture: sitting, head bowed forward, hands resting on the edge of the bed or chair. Pronounced symptoms of intoxication could be observed: body temperature can rise to 40° C, the number of white blood cells is increased to 12-15x10^9/L or more (leukocytes normal range 4.00-11.0x10^9/L), erythrocyte sedimentation rate (ESR) increases dramatically.

    **Surgical treatment:** During the diagnosis, the dentist must to found which spaces are inflamed. All affected spaces have to be incised as described above. Usually, the treatment of phlegmon of the floor of the mouth is done in a hospital base because of a great risk of life threatening complications.

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**Submasseteric abscess**

    **Anatomy:** Submasseteric space is located between the external surface of the mandible ramus and the inner surface of the masseter muscle. The boundaries of submasseteric space are: the anterior margin of the masseter muscle anteriorly, the parotid gland posteriorly, the zygomatic arch superiorly, the inferior border of the mandible inferiorly, the outer surface of the mandibular ramus medially (the submasseteric space is superficial to the mandible), the masseter muscle laterally. The submasseteric space contains the masseteric artery and vein. The submasseteric space communicates with the buccal space, the pterygomandibular space, the parotid space, the infratemporal space.

    **Etiology:** The reason could be paracoronitis of the third mandibular molars.

    **Clinical presentation:** It is characterized by a severe trismus and painful edema of the masseter muscle, which involved the angle of the mandible, that’s lead to impossible palpation of the mandibular angle. Intraoral examination is difficult, but edema at the retromolar area and at the anterior border of the ramus could be observed. The swallow is not affected.
Surgical treatment: In initial stages vertical intraoral incision, parallel to the anterior border of the mandibular ramus is suggested. In later stages extraoral incision, made by general anesthesia is proposed method. Extraoral incision is made around the mandibular angle, at a distance of 2 cm below the mandible in attempt to preserve the integrity of the branch of the facial nerve (marginal mandibular nerve).

Ptérygomandibular abscess

Anatomy: Ptérygomandibular space is located between the medial ptérygoid muscle and the inner surface of the ramus of the mandible. The boundaries of ptérygomandibular space are: the posterior border of the buccal space anteriorly, the parotid gland posteriorly, the lateral ptérygoid muscle superiorly, the lingual surface of the ramus of the mandible inferiorly, the medial ptérygoid muscle medially (the space is superficial to medial ptérygoid), the ascending ramus of the mandible laterally. The ptérygomandibular space contains the mandibular division of the trigeminal nerve, the inferior alveolar artery and vein. The ptérygomandibular space communicates with the buccal space, the lateral pharyngeal space and peritonsillar space, the submasseteric space, the parotid space, the infratemporal space.

Etiology: An abscess of ptérygomandibular space is caused mainly by infection of mandibular third molars or as the result of an inferior alveolar nerve block if the penetration site of the needle is infected.

Clinical presentation: Severe trismus and slight extraoral edema beneath the angle of the mandible are observed. The angular edema is typical in cases of dental etiology. When inflammation is from iatrogenic origin (nerve block with infected needle), edema is located intraorally. Displacement of the uvula to the opposite side could be observed. Patient’s complaints include painful swallowing.

Surgical treatment: The incision for drainage is performed intraorally or extraorally. Intraoral incision is a vertical incision, along the mesial temporal crest. The curve hemostat is inserted and moved inward keeping contact with the inner surface of the ramus. The angular extraoral incision would be performed. The hemostat is inserted via the incision and is moving upward keeping a contact with the inner surface of the mandibular ramus.
**Parapharyngeal abscess (lateral pharyngeal abscess)**

*Anatomy:* The parapharyngeal space is shaped like an inverted pyramid. The parapharyngeal space has complex fascial margins, and occupies the space between the muscles of mastication and the muscles of deglutition. The boundaries are:

- **superior margin:** base of skull
- **inferior margin:** greater horn of the hyoid bone
- **medial margin:** m. tensor et levator veli palatini, m. constrictor pharynges superior, pretracheal layer of the deep cervical fascia
- **lateral margin:** medial pterygoid muscle and aponeurosis interpterygoidea
- **anterior margin:** raphe pterygomandibularis
- **posterior margin:** processus styloideus, mm. stylohyoideus, styloglossus, stylopharyngeus, lig. stylomandibulare, lig. stylohyoideum, prevertebral layer of the deep cervical fascia

Behind the parapharyngeal space and carotid space lies the retropharyngeal space. The parapharyngeal space is continuous with the retropharyngeal space. It also communicates with other cervical and cranial fascial spaces, as well as the mediastinum. The parapharyngeal space contains glossopharyngeal nerve, Vagus nerve, Internal carotid artery, internal jugular vein, Accessory nerve, Hypoglossal nerve, sympathetic trunk and superior cervical ganglion of the trunk, ascending pharyngeal artery, deep cervical lymph nodes.

*Etiology:* Infections of this space originate in the region of the third molar and are the result of spread of infection from the submandibular and pterygomandibular spaces.

*Clinical Presentation:* Extraoral edema at the lateral region of the neck that may spread as far as the tragus of the ear, displacement of the pharyngeal wall, tonsil and uvula towards the midline, pain that radiates to the ear, trismus, difficulty in swallowing, significantly elevated temperature, and generally malaise are noted.

*Surgical treatment:* The incision for drainage is performed intraorally or extraorally. Intraoral incision is a vertical incision, along the mesial temporal crest, similar to incision made for drainage the pterygomandibular abscess. The curve hemostat is inserted medially from the medial pterygoid muscle and moved inward. The angular extraoral incision would be performed. The hemostat is inserted via the incision and is moving upward medially from the medial pterygoid muscle.
LIFE-THREATENING CONDITIONS IN ACUTE ODONTOGENIC INFECTION
(Dimitar Atanasov)

Facial vein thrombosis

**Etiology:** Facial veins are affected by inflammatory processes in case of acute odontogenic lymphadenitis, osteomyelitis, sialadenitis, sinusitis, abscesses and cellulitis, carbuncles and furuncles in the facial area.

**Clinical presentation:** The disease is characterized by the appearance of painful “strings” along the angular vein and the facial vein. The soft tissues of the face are infiltrated, hyperemia of the skin is present, with a bluish hue. Edema extending beyond the infiltrate is present. The subcutaneous veins are dilated. General symptoms develop rapidly, including: pronounced intoxication, fever, chills, general weakness, leukocytosis with left shift and elevated erythrocyte sedimentation rate (ESR). According to a number of authors, changes in homeostasis occur – shortened venous blood clotting time, increased fibrinogen content in the blood, increased factor XIII activity and suppression of fibrinolysis.

**Treatment:** Treatment is focused on limiting the spread of inflammation and normalizing the homeostasis. If urgent measures are not taken, sepsis or metastatic abscesses in the internal organs may develop. The responsibility of the dental practitioner is to make a proper diagnosis and to immediately refer the patient to a maxillofacial surgery clinic for hospital treatment, where the following should be done:

- **intensive antibiotic** (broad-spectrum) therapy - detoxifying and desensitisation therapy; immunotherapy.
- **anticoagulation therapy** - heparin is prescribed (2500-3000 U at every 4-6 hours) to prevent intravascular clotting of the blood.
- **Acidum nicotinicum** (50 mg 1%, 2 daily) is prescribed to reduce factor XIII activity and increase fibrinolysis
- **incision and drainage should be performed in case of abscess in the thrombotic veins**

Cavernous sinus thrombosis

**Topographic anatomy:** Cavernous sinus is a cavity with multiple septa, which gives it a cavernous appearance. It is located in the middle cranial fossa on the lateral surface of the sella. In cross section, it looks like a triangle with an upper side (cranial nerves III and IV), an
outer side (branch I of cranial nerve V, with cranial nerve VI passing between it and cranial nerve IV) and an inner side. The abducens nerve (cranial nerve VI), the internal carotid artery and the carotid plexus pass through this sinus. The left and right cavernous sinuses are connected via venous anastomoses which form sinuses in the anterior and posterior part of the diaphragm of the sella - anterior intercavernous sinus and posterior intercavernous sinus. Thus, a large cavity is formed around the pituitary gland located in the sella.

**Etiology:** It may occur as a complication of inflammatory processes in the facial area:
- otitis and mastoiditis
- disorders of the nose and maxillary sinuses
- odontogenic inflammatory processes
- after tooth extraction
- facial vein thrombophlebitis
- inflammation of tonsils and pharynx
- inflammation of scalp

**Routes of entry of infection**
- orbital veins – in purulent processes in the upper lip, eyelids and forehead.
- pterygoid venous plexus - in abscess on the face and neck, sinusitis or tonsilitis
- inferior petrosal sinus - in otitis
- mixed type - if there are several inflammatory foci

**Clinical presentation:** The first clinical description was made by Morgagni (1761).
In the early stages of the disease development, Oskolkova, G. and Sukachev, I. (1974) observed pronounced cyanosis of the mucous membranes of the lips, nose, ears and the skin of the forehead, which in their view, was a sign of intoxication of the body and impaired activity of the cardiovascular system. With the progression of the disease, other symptoms develop that can be subdivided into 3 main groups:
• **General (infectious) syndrome**

It is characterized by a severe headache, severe pain in the eyes, general weakness and fever up to 38-40°C with chills, accelerated erythrocyte sedimentation rate (ESR) (40-60 mm/h), neutrophilic leukocytosis, sometimes anemia, commonly - enlarged spleen.

• **Circulatory syndrome**

Swollen and hyperemic skin of the forehead and eyelids, infiltrated orbital soft tissue, chemosis and exophthalmos. Circulatory changes are characteristic of anterior route of entry of infection and are poorly expressed in posterior route.

• **Neurological syndrome**

Affects the functions of some or all of the cranial nerves located on the walls of the sinus or passing through it (III, IV, VI and branch I of cranial nerve V). It is presented clinically with:

- external ophthalmoplegia (complete absence of eye movement)
- ptosis of the upper eyelid (levator palpebrae superioris muscle is innervated by oculomotor nerve).
- miosis (narrowed) or mydriasis (dilated) pupil - iris sphincter muscle is innervated by oculomotor nerve
- pain in the eye or the forehead
- hyperalgesia or hypalgesia in the area of the supraorbital nerve
- neuritis and secondary atrophy of the optic nerve (reduced vision, white pupillary reflex)

**Diagnosis:** Diagnosis is based on the presence of an inflammatory process in the face or in the neck, and manifested general infectious, circulatory and/or neurological symptoms. To confirm or rule out the diagnosis, a consultation with a neurologist is necessary (finding elevated cerebrospinal fluid pressure, protein content and cylinders content in the cerebrospinal fluid).

**Differential diagnosis:** The dentist needs to distinguish thrombophlebitis of the cavernous sinus from orbital phlegmon. Common symptoms are presence of general
infectious syndrome and circulatory syndrome. Difference: phlegmon does not cause changes in the cerebrospinal fluid.

**Treatment:** Treatment is an extremely difficult and important task, and it should be conducted in hospital setting. The responsibility of the dental practitioner is to be aware of this serious complication and to diagnose and promptly refer patients to adequate comprehensive treatment. The following should be performed:

- sanitation of the primary suppurative focus
- combined antibiotic therapy (2-3 antibiotics should be administered)
- administration of antipyretics - antipyretics have anti-inflammatory, antipyretic and mild anticoagulant effect
- administration of anticoagulants - heparin. With treatment not including anticoagulants, mortality is significant, whereas, according to Gruzdev, A. (1978), with combined antibiotic and anticoagulant treatment, the mortality is reduced to 15-28%.

**Complications:** Possible complications of delayed or improper treatment of sinus cavernous thrombosis are:

- meningitis
- meningoencephalitis
- cerebral abscess
- sepsis
- thrombosis of the internal carotid artery
- pituitary gland necrosis (Cushing's syndrome)
- Horner's syndrome (enophthalmos, narrowed palpebral fissure, ptosis and myosis)
- optic nerve atrophy (blindness)

**Mediastinitis**

**Topographic anatomy:** The mediastinum is a space of the central part of the thoracic cavity which is restricted by: laterally - by the two mediastinal pleuras (left and right); ventrally – by the sternum (from the jugular notch of the sternum to the xiphoid process); dorsally – by the thoracic vertebrae and the initial part of the ribs; caudally – by the diaphragm, and cranially it passes into the upper tissue areas of the neck without distinct anatomical boarders (the upper border is generally assumed to the upper margin of the clavicles). It consists of the following parts in which vital organs are located:

- anterior mediastinum (mediastinum supracardiale) – here the thymus, trachea, esophagus and the aortic arc are located.
- middle mediastinum (mediastinum cardiale) – here the heart and the lungs, enclosed in the pericardial sac, are located.
- **posterior mediastinum** (mediastinum retrocardiale) - between the posterior wall of
the pericardium and the spine, the lower esophagus and the thoracic aorta are situated.

Etiology: An inflammatory process in the mediastinum may develop as a complication of purulent inflammatory processes of the floor of the oral cavity, the parapharyngeal space or the neck (abscesses and phlegmons). The pus spreads along the neurovascular bundle of the neck and along the facial and pretracheal cellular spaces. The most common microbial agents that can cause mediastinitis are staphylococcus and streptococcus, less commonly pneumococcus, coli, proteus or anaerobic organisms. Cases of mediastinitis caused by Enterobacter, Piocianeus, Klebsiella are extremely severe.

Clinical presentation: Odontogenic mediastinitis may have a fulminant course and occur simultaneously with a phlegmon of the floor of the oral cavity, which makes its diagnosis difficult. The general infectious syndrome is characteristic of both phlegmon and mediastinitis - fever with chills, low blood pressure, rapid heartbeat, mental disorders (in some cases), severe intoxication, laboratory findings typical for acute inflammation.

In mediastinitis, it is important to know that despite the incision and adequate medication, the general condition of the patient suddenly worsens with rapid onset of intoxication - body temperature rises to 40°C, the heartbeat becomes arrhythmic and accelerates to 140/min, breathing is difficult, shallow and rapid (45-50 per minute). Clinical symptoms are severe and depend on the localization of the process and the nature of its development (diffused as in phlegmon, or abscess). Mediastinitis is characterized by the presence of retrosternal pain (behind the sternum), which intensifies with deep inhaling and swallowing. Breathing becomes shallow and respiratory failure may develop. The pain also intensifies when the head is turned back (Gerke’s sign).

A patient with mediastinitis is forced to stay in a position, where his head leans forward or sideways, with chin pressed against the chest and legs bent towards the abdomen. Upon examination, a swollen neck and swollen jugular veins can be seen. Later on, swelling and redness of the skin occurs in the area of the jugular fossa and the supraclavicular fossa, as well as parasternal edema with dough-like texture.

Diagnosis: The diagnosis should be based on the clinical and radiographic findings. Making a diagnosis is difficult, especially in the presence of a phlegmon of the neck, when its symptoms mask those of the mediastinitis. Radiographic study is important for early and accurate diagnosis. It is made in 3 projections - anteroposterior, lateral and oblique. The presence of an enlarged mediastinal shadow indicates mediastinitis.

Differential diagnosis: It is important to differentiate between severe phlegmon of the floor of the oral cavity or of the neck and mediastinitis. What is common among them is the well-pronounced general acute inflammatory syndrome, swelling of the neck and the upper compartments of the mediastinum. Difference - retrosternal pain occurs with mediastinitis and the pain becomes more severe when the head is flexed backwards. Radiographic study contributes to the differential diagnosis of the disease. In phlegmons, radiological changes in the mediastinum are not found.
Treatment: The treatment should be carried out in a hospital. The dentist must know the symptoms and, after making proper diagnosis, should refer the patient to a surgical clinic for consultation and treatment. The outcome of the disease depends on early diagnosis and on timely administered comprehensive therapy, which includes mediastinotomy (performed by a thoracic or general surgeon), and intensive antibiotic and detoxification therapy.

Asphyxia

Asphyxia is disruption or cessation of gas exchange in tissues with subsequent choking. All conditions characterized by hypoxia (acute tissue oxygen deprivation) result in asphyxia. The end-stages of cessation of life, regardless of the cause, are characterized by oxygen starvation and asphyxia.

**Etiology:** Inflammatory processes of the floor of the oral cavity or the root of the tongue may result in asphyxia due to narrowing of the airways as a result of compression. The so-called mechanical stenotic asphyxia may develop.

**Clinical presentation:** Asphyxia clinically develops in 5 stages:

- **Preasphyxial stage**
  Due to oxygen deficiency, breathing becomes more rapid and deeper. This way the body is trying to compensate for the deficiency of oxygen. Consciousness is preserved, the patient is pale, scared. It last for 2 to 2 ½ minutes.

- **Blue asphyxia stage**
  The decrease of oxygen or the accumulation of carbonic acid in the body above the permissible limits result in breathing disorder. Deep inspiration and short expiration, with increasing dyspnea and cyanosis (lips and nails turn blue) are observed. Elevated arterial pressure, decreased heart rate (vagus pulse), dilated pupils (mydriasis) are observed. The patient is conscious, agitated and frightened, and symptoms of vertigo and altered
consciousness gradually appear. This last for 30-40 seconds and is followed by a loss of consciousness and seizures. Some authors define it as a stage of dyspnea and seizures.

- **Transient apnea (white asphyxia) stage**
  With the accumulation of carbonic acid in the body, the tone of the respiratory center and muscles weakens, which causes periods of absence of breathing (apnea) lasting from several seconds to one minute. During these periods, consciousness is lost, reflexes disappear and there is a paralytic dilation of pupils (mydriasis). The skin of the face becomes livid. The patient is still alive, clinical death has not yet occurred, and if quick action is taken, the patient’s breathing may be restored. Depending on the individual, this stage lasts from 1 to 1½ minutes. If timely and proper treatment is not given, the next stage begins.

- **Terminal breath**
  It is characterized by separate, deep breathing movements at one- or two-minute intervals, after which breathing stops. At this stage, respiratory movements have the opposite characteristics of the ones observed in stage 2 - inspiration is short and expiration is deep. After the breathing stops, clinical death occurs.

- **Terminal automatic heartbeat stage**
  At this stage, the patient is unconscious and not breathing, but the heart continues to beat. Biological death has occurred, but the heart continues to beat and pump blood. It continues for 15-20-30 minutes, but it is of no practical significance, because the patient can only be saved within 5 to 8 minutes from the onset of asphyxia. After this, hypoxia in the central nervous system occurs, resulting in the destruction of pyramidal cells and inability to return to a normal function.

In inflammatory processes and gradually occurring asphyxia (hours, day), compensatory mechanisms, with longer development of clinical symptoms, may be triggered in the body. The position of patients in this case is the following: they stand with their head and body inclined forwards and downwards, with a constantly stretched neck, open mouth, tongue out of the mouth and eyes showing strong fear. Due to fear of suffocation, patients do not sleep and look tired and exhausted by thirst and starvation. The face is usually pale, lips are cyanotic and nails are blue (P. Z. Arzhanzev, G. M. Ivashchenko, T. M. Lurie, 1975).

*Treatment:* In case of a severe phlegmon, the dentist should focus on making a rapid and correct diagnosis and refer the patient to a specialized hospital treatment. The life of the patient can be maintained by providing oxygen (8-12 L/min), intubation or coniotomy, which should only be done in case of emergency. Emergency tracheostomy and extensive opening of the suppurative focus, followed by an adequate medication treatment is of the utmost importance when treating such patients. Tracheotomy should be performed under local anesthesia with the patient in seated position or after intubation. It should be performed only by a well-trained surgeon, and it is advisable to be performed by an ENT specialist.
Purulent meningitis (Meningitis purulentа)

Etiology: Inflammation of the meninges may occur with phlegmon of the infratemporal fossa, due to purulent destruction of the walls of the cavernous sinus.

Clinical presentation: The disease has an acute onset, with fever (39-40°C), a severe headache, nausea and vomiting. Consciousness is depressed. Clinical presentation develops with the appearance of two major syndromes:

1. Meningeal-radicular irritation syndrome - includes the following symptoms:
   • **Headache** - the pain is severe and increases with bright light, sharp head movement or loud noise. It is caused by irritation of the peripheral nerve terminals of the trigeminal nerve and the vagus nerve in the meninges, as well as by increased cerebrospinal fluid hypersecretion and increased intracranial pressure. Headache is one of the most persistent symptoms.
   • **Vomiting** - usually accompanies the headache and is due to irritation of the vagus nerve or its nuclei in the oblongated marrow.
   • **Rigidity** of neck muscles - the head is curved backwards (opisthotonos) and its passive contraction is impossible.
   • **Contracture of the flexor muscles of the legs.**
   • **Kernig’s sign** - The patient is placed in supine position and his legs are flexed at the hip and knee at 90°. When attempting to straighten them, a resistance in the lower legs can be felt, which means a positive Kernig’s sign.
   • **Brudzinski’s sign:** (1) lower - passive flexion of one leg in the knee and hip causes a similar movement of the other; (2) upper - when the head is abruptly and passively bent forward, flexion of the legs to the abdomen occurs.

2. Cerebrospinal fluid syndrome: It is characterized by:
   • cloudy and purulent cerebrospinal fluid
   • elevated cerebrospinal fluid pressure (when performing a lumbar puncture, the cerebrospinal fluid streams or drains with rapid drops), positive Rivalta test - presence of protein in the cerebrospinal fluid (elevated albumins and globulins over 80-300-600 mg%)
   • low glucose level in the cerebrospinal fluid - reduced to 20-30 mg% (normal limits - 50-60 mg%)
   • pleocytosis - over several thousand in mm³, this is an important sign for the diagnosis.

Diagnosis: It is based on the medical history and the objective clinical examination, confirmed by the laboratory minimum (blood test - leukocytosis with left shift and elevated erythrocyte sedimentation rate (ESR), and cerebrospinal fluid test).

Treatment: In case meningeal-radicular irritation is suspected, urgent consultation and subsequent treatment by a neurologist (infectious diseases specialist) are required.
Cerebral abscess

*Etiology:* Limited inflammation in the brain may occur due to a facial abscess or phlegmon. The most common causes of pyogenic infection in the brain are staphylococcus, streptococcus and anaerobes (peptococcus, peptostreptococcus, bacteroides).

*Clinical presentation:* The development of the brain abscess goes through 4 stages:

Stage I - appearance of a purulent collection in the brain, characterized by manifestations of increased intracranial pressure - headache, vomiting and somnolence. In some patients focal neurologic symptoms are also found.

Stage II - An abscess cavity is formed in the brain. The acute symptoms resolve, and this condition may last for weeks or months.

Stage III - period of enlargement of abscess cavity. Different clinical symptoms appear: Symptoms of increased intracranial pressure, convulsions, focal symptoms (focal symptom of loss). Mental changes, subfebrile body temperature, leukocytosis with elevated erythrocyte sedimentation rate (ESR), elevated cerebrospinal fluid pressure are also observed. When examining the eye fundi, congested papillae are found.

Stage IV - with untimely diagnosis or improper treatment, complications may develop, as pus may enter the subarachnoid space or the cerebral ventricles, resulting in cerebral edema and death.

*Diagnosis:* The diagnosis is based on evidence of an inflammatory process in the maxillofacial area, signs of space-occupying lesion in the brain and elevated cerebrospinal fluid pressure. The diagnosis should be confirmed after CT scan.

*Treatment:* An urgent consultation and treatment by a neurosurgeon must be conducted. The mortality rate is approximately 20%.
ODONTOGENIC SEPSIS
(Dimitar Atanasov)

Sepsis is a general purulent non-specific infection characterized by the entry of a large amount of virulent microorganisms into the blood stream from a suppurative focus in the maxillofacial area and proliferation of microorganisms in the capillary network resulting in the release of toxins into the bloodstream.

**Etiology and pathogenesis:** Sepsis occurs when the humoral and cellular immunity of the macroorganism is impaired and is conditioned by:
- existence of a primary suppurative focus (pathogen)
- entry of a sufficient number of microorganisms ($10^5$ per 1 gram of tissue), so that after the bactericidal action of the blood, the amount necessary to induce a severe reaction remains
- response of the macroorganism

The following microbial agents may cause sepsis: Staphilococcus aureus, Streptococcus haemolyticus, E.coli, Proteus, Pseudomonas aeruginosa, Enterobacter, Klebsiella, Salmonella, Diplococcus, Meningococcus, Candida albicans, non-spore-forming anaerobic bacteria.

Factors for the development of sepsis include:
- delayed surgery
- inadequate detection of the suppurative focus
- ineffective drainage
- **inadequate antibiotic treatment**

People susceptible to sepsis include:
- elderly people and children
- diabetic and cancer patients in cachexia stages
- people with anemia
- people with hematopoietic system diseases
- people who have suffered a traumatic shock, acute blood loss, hypovolemia or hypoxia
- people receiving corticosteroid, cytostatic or X-ray therapy

Infected wounds, postoperative infections, primary purulent diseases (abscess, phlegmon and osteomyelitis) may become an entry door allowing the development of sepsis. The primary suppurative focus may be an entrance door only, but it can also be an active purulent process supporting the sepsis. According to Struckov, G. (1987) sepsis may develop with a normergic (inflammatory processes predominate), hyperergic (destructive-degenerative changes) and anergic (poorly manifested changes due to weak protective response or acquired immunity) response.

The main pathogenetic factor for sepsis is the presence of toxemia and bacteremia, i.e. the infection can be called sepsis if bacteremia along with clinical symptoms is present. In a
number of odontogenic inflammatory processes (abscesses, phlegmons) there may be transient bacteremia, but this is not sepsis.

Clinical presentation: In sepsis, the general symptoms as in the general acute inflammatory syndrome are present (abscesses and phlegmons), but they are more pronounced and worsening of the general condition occurs despite antibacterial therapy administered. Often, the onset of sepsis is preceded by a worsening of the local inflammatory process, increase in pain, reddening of the skin (mucous membranes), increased edema or exudation. The following stages are defined in the clinical progression of sepsis:

Stage I (absorption fever). The general symptoms are related to the local inflammatory process and if there is normoergic response, they are consistent with the local changes.

Stage II (initial phase of sepsis). After treatment of the local inflammatory process (incision and medication therapy), the general symptoms do not resolve and the pathogenic microorganism can be found in the blood. This is diagnosed as an initial phase. If intensive treatment is administered, the disease can be completely cured.

Stage III (septicemia). Despite the treatment administered, the general condition of the patient remains poor (severe headache, insomnia, fever, chills), pathogenic microorganisms are found in the blood. There are no suppurative foci in other tissues or organs of the body. This condition is diagnosed as septicemia (toxic stage).

Stage IV (septicopyemia). Metastatic abscesses are found in various organs of the body, as a result of bacteria spreading from the primary focus via haematogenic route. A manifested general acute inflammatory syndrome is present with changes in various organs and systems - fever with chills, severe headache, dizziness or somnolence, malaise, dry and coated tongue, excessive thirst, vomiting, decreased urine production, presence of albumin and cylinders in the urine, accelerated (over 100/min) and sometimes irregular heart rate, drop in arterial blood pressure, anemia, leukocytosis with left shift, elevated erythrocyte sedimentation rate (ESR) (over 60 mm/h), hypoproteinemia and pathogenic microorganisms in the blood. There are great differences in the values of body temperature taken in the morning and in the evening.

Pyemic foci are most commonly formed in the lungs (the first filter of the lesser circulation), liver (the first filter of the portal vein system), brain, kidneys. According to Struchkov, the most common causes of pyemic foci are staphylococci (55%), streptococci (35%) and coli bacteria (10%).

Stage V (exitus letalis). In patients with pyemic foci, the following conditions may develop: metastatic pneumonia, empyema, pericarditis, peritonitis, nephrosis, meningitis with a subsequent dangerous onset of acute respiratory failure, kidney failure, cerebral coma and endotoxic shock, which in the most cases end with exitus letalis.

Diagnosis: Sepsis is diagnosed based on the presence of clinical symptoms in combination with a "set of laboratory data " (Pophristov, D., 1998). The presence of an infection entry point, a temperature curve of septic nature accompanied by chills before raising, anemia, hypoproteinemia, leukocytosis with left shift, lymphopenia, elevated ESR
and tachycardia is typical of sepsis. According to Pophristov, a positive blood culture is not mandatory for the diagnosis.

Differential diagnosis: It is necessary to distinguish between sepsis and:

a) abscesses and phlegmons – a common symptom is a manifested general acute inflammatory syndrome; difference - in the treatment of abscesses and phlegmons (incision, medication therapy), rapid improvement occurs, but not in sepsis.

b) absorption fever - a quick improvement occurs after sanitation of the local focus, unlike in sepsis.

Treatment: Sepsis is a particularly dangerous general infection which the dentist needs to know, be able to diagnose and should refer the patient to a hospital for treatment.

The basic principles of the treatment of sepsis include:

- sanitation of the primary focus (incision, removal of necrotic tissues)
- fighting the infection (antibacterial treatment)
- general intensive treatment (infusion treatment)
- ongoing correction of the functions of vital organs and systems
- stimulating the body's immune response (non-specific and/or specific immunotherapy)
SURGICAL TREATMENT OF ACUTE INFLAMMATIONS
(Dimitar Atanasov)

Surgical treatment plays a leading role in the comprehensive multi-modality treatment of acute odontogenic infections (AOI). The old principle of purulent surgery "ubi pus ibu evacua" - "if there's pus about, let it out", has not lost its significance even today, in the era of powerful antibiotics. Surgical treatment is aimed at:

- removing the purulent necrotic focus
- limiting the purulent inflammatory process
- preventing the development of complications
- interrupting the intoxication of the body
- removing the source of infection

Surgical treatment - incision, is based on the topographic location of the inflammatory process and the anatomical and topographic features of the area. In abscesses, the length of the incision is usually approximately 2-3 cm, and in phlegmons - 5-6 cm. In incision, today, we take into account Lexer's aphorism, “the incision should be as large as necessary, and as small as possible”. Incision is the most common surgical procedure, which in clinical practice is often underestimated as a minor procedure. We need to remember that there is no minor and major surgery and that each surgical procedure should comply with certain rules - asepsis and antisepsis, reliable analgesia and creating surgical comfort.

After the incision and exposure of the abscess, a drain should be placed, aimed at achieving:

- evacuation of pus, exudate and blood from the wound
- prevention of hematoma formation or accumulation of serum
- reducing the number of dressings
- maintaining a pathway for the elimination of necrotic tissue
- creating conditions for antibacterial rinse
- reducing the use of antibiotics
- creating conditions for post-operative bacteriological diagnosis

Rubber, silicone, polychlorvinyl tubes, corrugated rubber bands or gauze strips can be used as a drain (Steinberg, C. M., 1986; Popkirov, St., 1998). The action of drains is based on gravity - they are placed at the lowest point of the abscess after its exposure, or on suction (vacuum) when placed in closed body cavities. Thus, drain systems are subdivided into open and closed. Rubber bands or tube drains are used in open drain systems. It is not recommended to use gauze strips because their action is based on hygroscopicity and after 6-8 hours they become completely soaked with pus and blood, and already act as pads. This necessitates their frequent change - 3-4 times a day (Struchkov, I., 1975; Popkirov, St., 1998). Silicone drains are non-flexible, which facilitates both their insertion and the irrigation of the wound (Krishnan, V. et al., 1993). Closed drain system is used in hermetically sealed cavities filled with pus and is connected to a suction system, e.g. Redon drain (a vacuum drain).
To remove germs and their toxins, and the products from bacterial and tissue breakdown, wound flushing is performed. For this purpose, antiseptic solutions (0.5:1000 Hybitan), proteolytic ferments (trypsin), antibiotic solutions (0.5:1000 Chloramphenicol, 1:1000 Kanamycin) may be used. Flushing can be performed with a syringe under pressure or with a blood transfusion set.

To improve draining of exudate from the wound, sterile gauze dressings soaked in hypertonic solutions (10% sodium chloride, 25% magnesium sulphate) are placed on the wound. Dressings should be applied daily, and drains should be removed after the exudation stops, i.e. after the last drop of pus.
ANTIBIOTICS
(Petia Pechalova)

Antibiotics are a type of antimicrobial used in the treatment and prevention of bacterial infection. They may either kill the bacteria (bactericidal antibiotics) or inhibit the growth of bacteria (bacteriostatic antibiotics). However, there is not always a precise distinction between bacteriostatic and bactericidal antibiotics. High concentrations of some bacteriostatic agents are also bactericidal, whereas low concentrations of some bacteriocidal agents are bacteriostatic.

**Mechanism of action:** Antibacterial action generally falls within one of four mechanisms:

1) the inhibition or regulation of enzymes involved in cell wall biosynthesis
2) the inhibition or regulation nucleic acid metabolism and repair
3) the inhibition or regulation protein synthesis
4) the disruption of membrane structure

**History of antibiotics:** The serendipitous discovery of the first antibiotic - penicillin is usually attributed to Scottish scientist Alexander Fleming (1881–1955), though others had earlier noted the antibacterial effects of *Penicillium*. In 1928, Fleming, at his laboratory in St. Mary's Hospital in London, noticed a halo of inhibition of bacterial growth around a contaminant blue-green mold on a *Staphylococcus* plate culture. Fleming concluded that the mold was releasing a substance that was inhibiting bacterial growth and lysing the bacteria. He was inspired to further experiment and he found that a mold culture prevented growth of staphylococci, even when diluted 800 times. He named the active substance penicillin.

On 1942, John Bunstead and Orvan Hess became the first in the world to successfully treat a patient using penicillin. During World War II, penicillin made a major difference in the number of deaths and amputations caused by infected wounds. Availability was severely limited, however, by the difficulty of manufacturing large quantities of penicillin and by the rapid renal clearance of the drug necessitating frequent dosing. Penicillins are actively secreted and about 80% of a penicillin dose is cleared within three to four hours of administration. During those times, it became common procedure to collect the urine from patients being treated so that the penicillin could be isolated and reused.
The chemical structure of penicillin was determined by Dorothy Crowfoot Hodgkin in the early 1940s, enabling synthetic production. A team of Oxford research scientists led by Australian Howard Walter Florey and including Ernst Boris Chain and Norman Heatley discovered a method of mass producing the drug.

The Nobel Prize in Physiology or Medicine in 1945 was awarded jointly to Sir Alexander Fleming, Ernst Boris Chain and Sir Howard Walter Florey "for the discovery of penicillin and its curative effect in various infectious diseases".

**Beta (β)-lactam antibiotics** are a broad class of antibiotics, consisting of all antibiotic agents that contain a β-lactam ring in their molecular structures. They are the most widely used group of antibiotics and includes Penicillin derivatives, Cephalosporins, Monobactams, Carbapenems. β-lactam antibiotics work by inhibiting cell wall biosynthesis in the bacterial organism. Immunologically mediated adverse reactions to any β-lactam antibiotic may occur in up to 10% of patients receiving that agent (a small fraction of which are truly IgE-mediated allergic reactions). Anaphylaxis will occur in approximately 0.01% of patients. There is a 5%-10% cross-sensitivity between penicillin-derivatives, cephalosporins, and carbapenems.

**Classification of (β)-lactam antibiotics:**

1. **Penicillin derivatives**
   1. Narrow-spectrum:
      a) β-Lactamase-sensitive (natural penicillins):
         - *Penicillin G* (benzylpenicillin) for parenteral use
         - *Penicillin V* (phenoxyethylpenicillin; Ospen) for oral use
      b) β-Lactamase-resistant:
         - *Methicillin*
         - *Oxacillin*
   2. Broad spectrum penicillin derivatives:
      - *Amoxicillin* (*Ospamox*)
      - *Ampicillin*
3. Extended-spectrum penicillin derivatives:
   - Azlocillin
   - Carbenicillin
   - Mezlocillin
   - Piperacillin
   - Ticarcillin

4. Penicillins with beta-lactamase inhibitors: Penicillins may be combined with β-lactamase inhibitors to increase efficacy against β-lactamase-producing organisms. The addition of the beta-lactamase inhibitor does not generally, in itself, increase the spectrum of the partner penicillin:
   - Amoxicillin + clavulanic acid = Augmentin
   - Ampicillin + sulbactam = Unasyn
   - Ticarcillin + clavulanic acid = Timentin
   - Piperacillin + tazobactam = Zosyn

II. Cephalosporins:
   1. First generation (moderate spectrum)
   2. Second generation (moderate spectrum):
      a. with anti-Haemophilus activity
      b. with anti-anaerobic activity
   3. Third generation (broad spectrum)
   4. Fourth generation (broad spectrum)
   5. Fifth generation (broad spectrum)

III. Carbapenems: broad-spectrum antibiotics that are active against virtually all groups of organisms, with only a few exceptions (e.g., resistance has been reported for all oxacillin-resistant staphylococci, selected Enterobacteriaceae and Pseudomonas, and other Gram negative bacilli). Carbapenems are a part of strategic antibiotic reserve and they must use only in life-threatening diseases when all other options are exhausted.
   - Imipenem (fl. 500 mg, 20 ml) - 2-3x1 flacon/ 24 h, IV
   - Meropenem (fl. 500 mg, 1 g powder) - 2-3x0,500-1,0/ 24 h, IV

IV. Monobactams: narrow-spectrum antibiotics that are active only against aerobic, Gram negative bacteria. Anaerobic bacteria and Gram positive bacteria are resistant. The advantage of narrow spectrum antibiotics is that they can be used to treat specific infections without disrupting the patient's normal, protective bacterial population.
   - Aztreonam (Azactam) - 1g powder for injection, 2-3x 1g/ 24 h IV

Aminoglycosides are antibiotics that inhibit protein synthesis. These antibiotics exert their effort by passing through the bacterial outer membrane (in Gram negative bacteria), cell wall, and cytoplasmic membrane to the cytoplasm, where they inhibit bacterial protein synthesis by irreversibly binding to the 30S ribosomal proteins. Aminoglycoside antibiotics display bactericidal activity against Gram-negative aerobes and some anaerobic bacilli where
resistance has not yet arisen, but generally not against Gram-positive and anaerobic Gram-negative bacteria.

- Gentamycin 80 mg, 2 ml (2x1 amp/ 24 h, IM)
- Kanamycin 500 mg, 2 ml (15 mg/kg/24 h, IM, IV)
- Amikin 500 mg, 2 ml (15 mg/kg/24 h, IM, IV)

**Tetracyclines** are broad-spectrum, bacteriostatic antibiotics that inhibit protein synthesis in bacteria by binding reversibly to the 30S ribosomal subunits. Tetracyclines are effective in the treatment of infections caused by *Chlamydia, Mycoplasma, Rickettsia*, and other selected Gram-positive and Gram-negative bacteria.

- Tetracycline 0.250 caps (4x1 caps/ 24 h)
- Doxycycline 0.100 caps (I day: 2x1 caps/ 24 h; II day and etc: 1 caps/ 24 h)

Tetracyclines may cause permanent staining of the teeth (discoloration) from yellow to dark brown, if used in children less than the age of 8 or during the 2nd and 3rd trimester of pregnancy.

**Macrolides** are bacteriostatic antibiotics with a broad spectrum of activity. They have been used to treat pulmonary infections caused by *Mycoplasma, Legionella*, and *Chlamydia* species, as well as to treat infections caused by *Campylobacter* species and Gram positive bacteria in patients allergic to penicillin.

- *Erythromycin* (tab. 250 mg and 500 mg) - 3-4x0,250-0,500/ 24 h orally
- *Clarithromycin* (tab. 250 mg and 500 mg) - 2x0,250-0,500/ 24 h orally
- *Azithromycin* (tab 250 mg and 500 mg) - I day: 2 tab/ 24 h; II, III, IV and V days: 1 tab/ 24 h

**Lincosamides** are derivates of amino-acid and sulfur-containing octose. First lincosamide to be discovered is *Lincomycin*, isolated from *Streptomyces lincolnensis*. Lincosamides, macrolides, and chloramphenicol, although not structurally related, seem to act at the same site. The lincosamides are bacteriostatic or bactericidal depending on the concentration. Activity is enhanced at an alkaline pH. Lincomycin (tab. 500 mg, every 6 – 8 hours) has narrow spectrum and is effective against gram-possitive microorganisms. Lincomycin has been superseded by *Clindamycin*, which exhibits improved antibacterial activity. Clindamycin (caps. 0.300 and 0.600, every 6 – 8 hours) is active against staphylococci and anaerobic Gram negative bacilli but is generally inactive against aerobic Gram negative bacteria. Clindamycin may prolong effects of neuromuscular blocking agents during general anesthesia.
**Metronidazole** was originally introduced as an oral agent for the treatment of *Trichomonas* vaginitis. However, it was also found to be effective in the treatment of serious anaerobic bacterial infections. Metronidazole has no significant activity against aerobic or facultatively anaerobic bacteria.

- *Trichomonacid* (tab 0.250, 3x1-2 tab/ 24 h orally)
- *Flagyl* (tab 0.250 and 0.500, 3x1-2 tab/ 24 h orally)

**Principles of combination antibiotic therapy**

**Additive (indifferent) effect:** the activity of two drugs in combination is equal to the sum of their independent activity when studied separately:

\[
\text{combination of two beta-lactam antibiotics}; 2+2=4
\]

(Augmentin + Imipenem)

**Synergistic effect:** the activity of two drugs in combination is greater to the sum of their independent activity when studied separately:

\[
\text{beta-lactam antibiotics + aminoglycosides}; 2+2>4
\]

(Ampicillin + Gentamycin)

**Antagonistic effect:** the activity of two drugs in combination is less to the sum (or a partial sum) of their independent activity when studied separately:

\[
\text{beta-lactam antibiotics + bacteriostatic antibiotics}; 2+2<4
\]

(Penicillin + Tetracyclin)

As a general principle, an infection should be treated with a single antibiotic. Combinations of antibiotics are only occasionally justified. The main bacteriological reasons for the use of combinations are to extend the spectrum of activity, to delay the development of resistance, to achieve synergy. Common antibiotic combinations are:

\[
\beta\text{-lactam antibiotics} + \text{Aminoglycoside};
\]

\[
\beta\text{-lactam antibiotics} + \text{Aminoglycoside} + \text{Metronidazol}.
\]

**Indications for the clinical use of antimicrobial combinations:**

1) Prevention of the emergence of resistant organisms
2) Polymicrobial infection (mixed aerobic and anaerobic organism)
3) Initial therapy
4) Decreased toxicity
5) Synergism
Analgesics fall into three categories: opioid (narcotic) analgesics, nonopioid analgesics, and adjuvant analgesics (drugs that are usually given for reasons other than pain but that sometimes relieve pain).

Most nonopioid analgesics are classified as nonsteroidal antiinflammatory drugs (NSAIDs). NSAIDs are used to treat mild to moderate pain and may be combined with opioids to treat moderate to severe pain. NSAIDs not only relieve pain, but they also reduce the inflammation that often accompanies and worsens pain. All NSAIDs are taken by mouth. One NSAID, ketorolac, can also be given by injection into a vein (intravenously) or muscle (intramuscularly). Indomethacin can be given by suppository. Although widely used, NSAIDs can have side effects, sometimes serious ones. Problems in the digestive tract: All NSAIDs tend to irritate the stomach’s lining and cause digestive upset (such as heartburn, indigestion, nausea, bloating, diarrhea, and stomach pain), peptic ulcers, and bleeding in the digestive tract (gastrointestinal bleeding). Coxibs (COX-2 inhibitors) are less likely to irritate the stomach and cause bleeding than other NSAIDs. However, if people take a coxib and aspirin, these problems are just as likely. Taking NSAIDs with food and using antacids may help prevent stomach irritation. The drug misoprostol can help prevent stomach irritation and ulcers, but it can cause other problems, including diarrhea. Proton pump inhibitors (such as omeprazole) or histamine-2 (H₂) blockers (such as famotidine), which are used to treat peptic ulcers, can also help prevent stomach problems due to NSAIDs. Taking NSAIDs for a short time is unlikely to cause serious problems. If NSAIDs are taken for a long time, certain tests are done regularly. They include blood pressure measurement, blood tests (such as a complete blood count and tests to check kidney and liver function), and tests for blood in stools. For older people, the risk of side effects due to NSAIDs, particularly indomethacin and ketorolac, is increased. For people who drink alcoholic beverages regularly and take NSAIDs, the risk of digestive upset, ulcers, and liver damage may be increased. The risk of heart attacks and stroke may be higher for people with coronary artery disease, other heart and blood vessel (cardiovascular) disorders, or risk factors for these disorders. Older people and people who have heart failure, high blood pressure, or a kidney or liver disorder require a doctor’s supervision when they take NSAIDs. Some prescription heart and blood pressure drugs may not work as well when taken with these analgesics. NSAIDs vary in how quickly they work and how long they relieve pain. Although NSAIDs are about equally effective, people respond to them differently. One person may find a particular drug to be more effective or to have fewer side effects than another.

Aspirin (acetylsalicylic acid) has been used for about 100 years. Aspirin is taken by mouth and provides 4 to 6 hours of moderate pain relief. Because aspirin can irritate the stomach, it may be combined with an antacid (in a buffered product) to reduce this effect. The antacid creates an alkaline environment that helps aspirin dissolve and may reduce the time aspirin is in contact with the stomach lining. However, buffered aspirin can still irritate the stomach because
aspirin also reduces the production of substances that help protect the stomach’s lining. These substances are a type of prostaglandin, which are similar to hormones. Enteric-coated aspirin is designed to pass through the stomach intact and dissolve in the small intestine, thus minimizing direct irritation of the stomach. (Enteric refers to the small intestine.) However, enteric-coated aspirin may be absorbed erratically. If food and enteric-coated aspirin are ingested at about the same time, the aspirin is not absorbed as quickly because food delays the emptying of the stomach. Consequently, pain relief is delayed. Aspirin increases the risk of bleeding throughout the body because it makes the particles that help blood clot (platelets) less likely to do so. People who bruise easily may be especially vulnerable to this effect. People who take aspirin and anticoagulants (such as warfarin) are closely monitored to avoid life-threatening bleeding. Usually, aspirin should not be taken in the week before scheduled surgery. Aspirin can aggravate asthma. People with nasal polyps are likely to develop wheezing if they take aspirin. A few people, who are sensitive (allergic) to aspirin, may have a severe allergic reaction (anaphylaxis), leading to a rash, itching, severe breathing problems, or shock. Such a reaction requires immediate medical attention. In very high doses, aspirin can have serious side effects such as abnormal breathing, fever, or confusion. One of the first signs of an overdose may be noise in the ears (tinnitus).

NSAIDs such as ibuprofen, ketoprofen, and naproxen are generally believed to be gentler on the stomach than aspirin, although few studies have compared the drugs. Like aspirin, these drugs can cause digestive upset, ulcers, and gastrointestinal bleeding. They can make asthma worse and increase blood pressure. Taking one of these drugs probably slightly increases the risk of stroke, heart attack, and blood clots in the arteries of the legs. Although ibuprofen, ketoprofen, and naproxen generally interfere with blood clotting less than aspirin does, people should not take these drugs with anticoagulants (such as warfarin) except under a doctor’s close supervision. People who are allergic to aspirin may also be allergic to ibuprofen, ketoprofen, and naproxen. If a rash, itching, breathing problems, or shock develops, medical attention is required immediately.

Coxibs (COX-2 Inhibitors), such as celecoxib, differ from other NSAIDs. Other NSAIDs block two enzymes: COX-1 (cyclooxygenase-1), which is involved in the production of prostaglandins that protect the stomach and play a crucial role in blood clotting; COX-2, which is involved in the production of prostaglandins that promote inflammation. Coxibs tend to block mainly COX-2 enzymes. Thus, coxibs are as effective as other NSAIDs in the treatment of pain and inflammation. But coxibs are less likely to damage the stomach and to cause nausea, bloating, heartburn, bleeding, and peptic ulcers. They are also less likely to interfere with clotting than are other NSAIDs. Because of these differences, coxibs may be useful for people who cannot tolerate other NSAIDs and for people who are at high risk of certain complications (such as gastrointestinal bleeding) from use of other NSAIDs. Such people include older people, people who are taking anticoagulants, those who have a history of ulcers, and those who must take an analgesic for a long time. However, blockage of COX-2 enzymes appears to make blood clots more likely to form. Thus, taking coxibs, like taking other NSAIDs, is likely to
increase the risk of heart attack, stroke, and blood clots in the legs. How much the risk is increased depends on the drug used. The risk is higher when higher doses are taken and when the drug is taken for a longer time. Thus, before people who have a cardiovascular disorder (such as coronary artery disease), who have had a stroke, or who have risk factors for these disorders are given a coxib, they are told about the risk and the need to be closely monitored. Coxibs are not appropriate for people who have heart failure or who are at increased risk of heart failure (such as those who have had a heart attack).

Acetaminophen is roughly comparable to aspirin in its potential to relieve pain and lower a fever. But unlike NSAIDs, acetaminophen has virtually no useful anti-inflammatory activity, does not affect the blood’s ability to clot, and has almost no adverse effects on the stomach. How acetaminophen works is not clearly understood. Acetaminophen is taken by mouth or suppository, and its effects generally last 4 to 6 hours. Acetaminophen appears to be a very safe drug. However, high doses can lead to liver damage, which may be irreversible. People with a liver disorder should use lower doses than those usually prescribed. Whether lower doses taken for a long time can harm the liver is less certain. People who regularly consume large amounts of alcohol are probably at highest risk of liver damage from overuse of acetaminophen. People who are taking acetaminophen and stop eating because of a bad cold, influenza, or another reason may be more vulnerable to liver damage. Taking high doses for a long time may lead to kidney damage.

Nonsteroidal anti-inflammatory drugs (NSAIDs) work in two ways. They reduce the sensation of pain. At higher doses, they reduce the inflammation that often accompanies and worsens pain. NSAIDs have these effects because they reduce the production of hormone-like substances called prostaglandins. Different prostaglandins have different functions, such as sensitizing pain receptors to mechanical and chemical stimulation and causing blood vessels to dilate. Most NSAIDs reduce prostaglandin production by blocking both cyclooxygenase (COX) enzymes (COX-1 and COX-2), which are crucial to the formation of prostaglandins. One type of NSAID, the coxibs (COX-2 inhibitors), tend to block mainly COX-2 enzymes. Only COX-2 enzymes are involved in the production of prostaglandins that promote inflammation and the resulting pain. These prostaglandins are released in response to an injury – burn, break, sprain, strain, or invasion by a microorganism. The result is inflammation, which is a protective response: The blood supply to the injured area increases, bringing in fluids and white blood cells to wall off the damaged tissue and remove any invading microorganisms. Prostaglandins that are formed through the action of COX-1 enzymes help protect the digestive tract from stomach acid and play a crucial role in blood clotting. Most NSAIDs block COX-1 enzymes and thus reduce the production of these prostaglandins. Consequently, these NSAIDs may irritate the stomach’s lining and cause digestive upset, peptic ulcers, and bleeding in the digestive tract. Because coxibs block mainly COX-2 enzymes, they are less likely to cause these problems. However, coxibs block some COX-1 enzymes, so even coxibs may slightly increase the risk of these problems.
Bone is the basic unit of the skeletal system and provides shape and support for the body, as well as protection for some organs. There are 206 bones in the human skeleton: 80 axial skeletal bones (e.g. skull, vertebral column and sacrum) and 126 appendicular skeletal bones (e.g. bones of extremities, scapula, pelvis). Each bone is constructed of periosteum, cortex (composed of cortical bone or compact bone) and medullary space (composed of cancellous or spongy bone):

Bone is composed of 35% organic (cells, proteins) and 65% calcium hydroxyapatite (99% of body's calcium; 85% of phosphorus; 65% of sodium; magnesium).

Classification of bones:
- Based on location:
  - Axial skeleton – bones of the skull, scapula, vertebral column
  - Appendicular skeleton – bones of the pectoral girdle, pelvis and limbs
- Based on shape:
  - Flat bone – bones of the skull, sternum, pelvis and ribs
  - Tubular bone – long tubular bones are bones of the extremities (e.g. femur, humerus); short tubular bones are bones of hands and feet
  - Irregular bone – bones of the face and vertebrae
  - Sesamoid bones – patella
- Based on size:
  - Long bone – tubular bones of extremities (e.g. femur, humerus)
  - Short bone – cuboidal in shape, in the foot (tarsal bones) and wrist (carpal bones)
- Based on texture:
  - Compact bone
  - Sponge bone
- Based on matrix arrangement:
  - Lamellar bone
- Lamellae of sponge bone are arranged parallel to each other
- In contrast, lamellae of compact bone are organized concentrically to around vascular canal (haversian canal)
- Woven bone (eventually converted to lamellar bone)

The facial skeleton serves to protect the brain, the sense organs of smell, sight, and taste and provide a frame on which the soft tissues of the face can act to facilitate eating, facial expression, breathing, and speech. The primary bones of the face are mandible, maxilla, frontal bone, nasal bones, zygoma.

Under the term osteomyelitis it is understood not only the inflammatory process in bone marrow, but also in all structural parts of the bone and in surrounding its soft tissue. Osteomyelitis of the jaws represents infectious process, which develop in the bone and in its surrounding tissues under the influence of aggressive factors of physical, chemical or biological nature against the preliminary sensibilization and neurohumoral changes. Osteomyelitis of the jaws can be: odontogenic, traumatic, toxic, hematogenic and specific.

There are distinguished three phases (periods) of disease course: acute, subacute and chronic. Depending on the spread of the process, the osteomyelitis can be circumscribed and diffuse. During the circumscribed osteomyelitis the pathological process is localized. Diffuse osteomyelitis is characterized with affection of large area of the jaw.

**Classification of the odontogenic osteomyelitis:**

![Diagram of osteomyelitis classification]

**Clinical signs:**

**Acute phase** of the odontogenic osteomyelitis it is characterized by the diffuse suppurate inflammation of all bone elements without process of bone demarcation and sequestration. In the chronic phase of odontogenic osteomyelitis the regions of osteonecrosis becomes apparent. They are surrounded by healthy bone. Without treatment, in a period of 1-2 moths, the formation of sequesters (full separation of regions with osteonecrosis from the unaffected bone)
usually stop. In some cases these are singular or multiple small sequesters (military sequesters), in other cases - a big regions of the jaw is involved (full-thickness sequesters).

The clinical presentation of the odontogenic osteomyelitis is defined for a variety of courses: microbes’ virulence caused the disease, the state of immunologic reactivity and nonspecific defense factors, patient age, the appearance of the affected maxilla.

In the acute phase of disease, patients complain of the pain in the region of one tooth, which is the source of infection. The signs of periodontitis could be noticed. The pain increases, becomes tearing, irradiating on temporal region and ear. One of typical complaints connected with osteomyelitis of the mandible is disturbance of the sensitivity of the region of inferior alveolar nerve (Vincent’s symptom in honor of the author who described it) - numbness of the lower lip, gingiva and the skin of the chin of the relevant part. When inflammatory process involved the soft tissues, the pain moves to outside of the jaw. A patient’s local complaints are swelling of the soft tissues, trismus, and pain during mastication. General complaints include headaches, weakness, fever, loss of appetite and sleep disturbances. It is assumed that the acute phase of osteomyelitis is attended by leukocytosis with appearance of new form of neutrophil leukocytes. Blood Sedimentation Rate as a rule is raised till 40-60mm/h.

Patients are pale, the pulse is rapid, in some cases arrhythmic. In the region of affected jaw infiltration and edema of soft tissues was appeared. The tooth - a cause for the development of osteomyelitis, at the beginning is mobile, but soon stabilizes, while the neighbor teeth become mobile and painful on percussion (symptom of multiple periodontitis). The gingivae and vestibular fold in the region of osteomyelitis are edematous and hyperemic. Their palpation is painful. During the development of osteomyelitis, abscesses and phlegmons can occur. As the result of the inflammation, lymphadenitis often appear. The most permanent and early osteomyelitis symptoms of the mandible are thickness of its border, Vincent’s symptom, and changes of tooth electroexcitability.

Acute phase of the maxillary osteomyelitis is characterized with short duration and absence of large destructive bone areas compared to mandibular osteomyelitis (the osteomyelitis of the mandible is more common and more severe). The maxillary osteomyelitis rare is complicated by the phlegmons. This specific clinical characteristics of the maxillary osteomyelitis are due to the anatomic pattern of maxilla – good vascularization, presence of big quantity of apertures in cortical substance, which assists in quick evacuating of the purulent exudate under the periosteum or under the mucosa, and the lack of massive muscles.

In acute phase of odontogenic osteomyelitis, the only findings on a roentgenography, is the pathological process of the tooth (periodontitis more common) that cause the disease. No changes into the bone can be observed yet.

**Subacute phase** is short-term, but often it is lasted 1.5-2 weeks. Subacute phase of the jaw’s osteomyelitis is characterized by the stabilization of the inflammatory process. Sockets of the extracted teeth become clean, without the necrotic tissues, granulation occur, and the flow of pus and edema decreased. Teeth located in the region of the inflammatory nidus,
becomes more mobile. Typical symptom of the osteomyelitis acute phase is improvement of the general status of the patient: disappear weakness, the sleep and appetite change to normal, body temperature is decreased, leukocytes count and erythrocyte sedimentation rate significantly diminished.

**Chronic phase** of the jaw’s osteomyelitis is more prolonged. Pain and infiltration of soft tissues decreases. The main characteristic of this fase is formation of the sequesters – necrotic parts of the bone. Mucous and cutaneous sinus tracts develop. The spontaneous evacuation of the sequesters is accompanied by the growth of the granulation tissues that prominent from the sinus tracts. In the region of the osteomyelitis core the jaw is thickened, teeth are usually mobile. In the cases of the extensive destruction of the mandible body can due to pathological fracture of the jaw.

Important place in the diagnosis of the chronic odontogenic osteomyelitis has X-ray examination. In a subacute phase, focal or diffusive osteoporosis can be noticed. Later one or more focuses of destruction of irregular shape become apparent. The major diagnostic value during the chronic phase of osteomyelitis has sequestration. The sequester border is clearly distinguished on the background of more transparent surrounding bones’ elements. During the osteomyelitis of the mandible the sequestration is visible in the end on the 3-rd to 4-th week. Sequesters can be differentiated by form - rounded, oval, multangular, with irregular edges, and by location – central, peripheral, complete.

**Differential diagnosis (DD):** The acute phase of the odontogenic osteomyelitis should differentiate from following diseases: 1) acute purulent parodontitis, 2) acute purulent periostitis, 3) isolating inflammatory process of the soft tissues (abscesses, phlegmons), 4) suppurate cysts in maxilla facial region (odontogenic and nonodontogenic - dermatoid, epidormoid).

The chronic odontogenic osteomyelitis should be differentiate from specific jaw’s inflammation (actinomycosis, tuberculosis, syphilis), benign and malignant tumors.

**Treatment:** The removal of the “causative” tooth, localised on roentgenography, in the early acute odontogenic osteomyelitis is mandatory. Immediately extraction of the correct tooth - a cause of the osteomyelitis, is a crucial for the fast healing process, as well as proper medical therapy – antibiotics, especially those that cumulated into a bone tissue (osteotropic activity) – Clindamycin 0,600 orally every 8 hours or Lincomycin 0,500 orally every 6 hours. Appropriate drugs in acute stage of osteomyelitis also are analgesics, antipyretics, and vitamins.

In subacute phase of the odontogenic osteomyelitis antibacterial therapy is suggested. In the chronic phase parallel with antibacterial and anti-inflammatory medical treatment, surgical procedures are provide – removal of necrotic bone (sequestrotomy and sequestrectomy) after elevation of mucoperiosteal flap and extraction of affected teeth. Additional medical treatment includes: NaCl 0.09% in doses 30-60 ml/ kg (for example 80 kg patient needs of 2,4 l NaCl 0,09% intravenously), Glucose 50%, 500-1000 ml intravenously (after level of blood glucose was assessed); antiallergic solutions - Suprastini, Dimedrol 1%-1ml; immunotherapy.
LYMPHADENITIS
(Petia Pechalova)

The lymphatic system represents an accessory route through which fluids flow from the interstitial spaces into blood. It is essential part of body’s immune system. The lymphatic system consists of primary organs (red bone marrow, thymus) and secondary/ peripheral organs (lymph nodes and vessels, spleen).

Of the 800 lymph nodes in the human body, 300 are in the neck. Only two areas of head and neck have no direct lymphatics – orbit and muscles, their lymph drains in fascial planes between muscles and around the blood vessels that supply them. Lymph vessels are not present in central neuron system, bones and alveoli of lungs.

Classification of face and neck lymph nodes:

Submental lymph nodes are located into the triangle space bounded by the anterior bellies of digastric muscles (left and right), anterior part of the mandible and hyoid bone, the mylohyoid muscle, and skin. Their number usually fluctuates from 1 to 4. Submental lymph nodes receive the lymph from tissues around mandibular incisors, from the anterior part of the alveolar bone and mandibular body, from the apex of the tongue and anterior area of the floor of the mouth, from lower lip and adjacent structures.

Submandibular lymph nodes are located in the submandibular triangles (left and right) and lie outside submandibular salivary gland capsule, as a chain, along the mandibular
edge. In submandibular lymph nodes fall lymph vessels from mandibular canine, premolars and molars, from the alveolar bone and mandibular body in this area.

**Facial lymph nodes** are located parallel to the facial vein – from the mandibular angle to the internal corner of the eye.

**Parotid lymph nodes** lie both, superficially, under the parotid fascia, and deeply - into the parotid gland. These nodes collected lymph from the parotid salivary gland and from the lateral face’s parts, from the buccal mucosal membrane, nose, eyelids, brows, ears.

*Definition:* Lymphadenitis is an inflammation of the lymph nodes. The progress of the inflammation leads to an abscess of the lymph node. In this pathological process all structures of the lymph node are destructed except the outer membrane, which isolated the pus from the adjacent structures. When the inflammation break through the lymph node’s capsule and destroy it, due to the pus spreading into the surrounding soft tissues, adenophlegmone develops.

**Lymphangitis** is inflammation of the lymph vessels. Lymphangitis develops as the result of penetration of microorganisms and their toxins in lymph tracts.

**Ethiology:** Lymphadenitis could be a result of acute or exacerbated periodontitis, suppuration of the radicular cyst, osteomyelitis, sialadenitis, suppurated wounds, etc. When lymphadenitis of oral and maxillofacial area are result of an odontogenic infection, they are named “odontogenic”. Lymphadenitis also can develop as a result of general infectious diseases and damage of oral mucosal membrane (stomatogenic), from skin inflammation (dermatogenic), from tonsilitis (tonsillogenic), from inflammation of the ear (otogenic).

The most common reason of the acute pyogenic lymphadenitis is nonspecific bacterial infection (nonspecific lymphadenitis). In rare cases, lymphadenitis can be caused by the tuberculosis mycobacteria, Treponema pallidum, actinomyces species (specific lymphadenitis), by virus (infectious mononucleosis) or fungi (histoplasposis, blastomycosis).

*Clinical signs:* Two main types of lymphadenitis exist: acute and chronic lymphadenitis. Acute lymphadenitis can proceed in serous and purulent forms. Chronic lymphadenitis proceeds in the hyperplastic and proliferative forms, and it can exacerbate.

**Acute serous lymphadenitis** (lymphadenitis acuta) is the initial stage of the inflammatory process in lymph nodes. In this form there is an inflammation of the lymph nodes, but no pus. The disease starts with discomfort, stretching of the tissues, dull pain, difficulty swallowing and head movements. The lymph nodes are tenderness, enlarged, mobile with tight-elastic consistency, round and oval shape. An asymmetrical face usually looks and skin over swelling is without changes. The general condition is not affected, but in
many cases can observe high temperature (38 °C). By effective treatment of basic disease caused lymphadenitis, inflammation process of lymph nodes undergo a back development: lymph nodes decrease in size, become soft, reduce pain. The major complaints of acute serous odontogenic lymphadenitis are: appearance movable, painful “ball” during the palpation in the certain area. The child would note that it was toothache before of the lymph node enlargement. During examination the dentist can note a spherical lump which is painful during palpation. Mobility of the lump could be limited and it would be the sign of spreading of the process to the surrounding tissues. The skin above the lymph node is without changes. The process is localised homolateraly - no enlargement of the lymph nodes from the other site can be observed.

Acute serous lymphadenitis can lead to acute purulent lymphadenitis. Affected lymph node is presented as a painfull, increased in size, with limited mobility. In palpation soft consistency and fluctuation can be noticed. The overlying skin could be reddish and heat. The general condition is affected.

**Chronic lymphadenitis** is the outcome of the acute process in lymph node or is a primary chronic. The chronic lymphadenitis is characterized by enlargement of a lymph node, by roundish or oval form, solid-elastic consistency, mobile, no solder with surrounding tissues can observed, the overlying skin is with no changes. The lymph node is painless. A palpation can produce a slight pain. The general state of the patients is not disturbed.

In separate cases, the moderate granulation tissue enlargement replace by itself lymphoid tissue, and spreads outward the node, grows to skin thereby make it thinner. During the progression, the thinner skin is perforated, and the skin sinus tract develops. The chronic hyperplastic lymphadenitis should be differentiated with odontogenic subdermal and dermal granuloma, subdermal form of the actinomycosis, scrofuloderma and tumor metastasis.

**Differential diagnosis:**

- Malignancies: metastasis, tumors of lymphoreticular system (lymphomas, leukemias, Kaposi’s sarcoma)
- Autoimmune diseases: lupus erythematosus, rheumatoid arthritis
- Miscellaneous: sarcoidosis
- Others

*Treatment:* During the acute stage, the main surgical procedure is the removal of the reason - tooth extraction, to prevent the future entry of microorganisms in the lymph nodes. In serous lymphadenitis the treatment could be conservative – antibiotics (Zinnat 0,500 every 8 hours), nonsteroidal antiinflammatory drugs, and physiotherapy (heating procedures aimed increasing of the blood supply in the area and eradication of the inflammation due to bactericidal effect of the blood). In acute purulent lymphadenitis antibiotics are mandatory, nonsteroidal antiinflammatory drugs are necessary, the physiotherapy includes ice pack only (for limitation of the inflammation). In same cases of chronic lymphadenitis extirpation of lymph node is a method of choice.
The paranasal sinuses are maxillary, ethmoid, frontal, and sphenoid. The maxillary sinus is the largest of the paranasal sinuses. The maxillary sinus is also known as the antrum or the antrum of Highmore. The term antrum is derived from the Greek word meaning “cave.” Nathaniel Highmore, an English physician in the 1600s, described a sinus infection associated with a maxillary tooth, and his name has long been associated with sinus nomenclature.

The maxillary sinuses are air-containing spaces that occupy maxillary bone bilaterally. Their development begin embryonically - in the third month of fetal development as mucosal invaginations. The initial maxillary sinus development is termed primary pneumatization. Secondary pneumatization begins in the fifth month of intrauterine life as the initial invaginations expand into the developing maxillary bone. After birth, the maxillary sinus expands by pneumatization into the developing alveolar process of the maxilla and extends anteriorly and inferiorly from the base of the skull. As the dentition develops, portions of the alveolar process of the maxilla, vacated by the eruption of teeth, become pneumatized. Around age 13 years, the maxillary sinus will have expanded to the point at which its floor will be on the same horizontal level as the floor of the nasal cavity. The complete development of maxillary sinus is achieved at age 20 years. In adults, the apices of teeth may extend into the sinus cavity. The mean volume of each maxillary sinus is 15 ml. The maxillary sinus is significantly larger in edentulous adults compared with patients with full dentition in the posterior maxilla.
The maxillary sinus is described as a four-sided pyramid, with the base lying vertically on the medial surface and forming the lateral nasal wall. The apex extends laterally into the zygomatic process of the maxilla. The upper wall (roof) of the sinus is also the floor of the orbit. The posterior wall extends the length of the maxilla and dips into the maxillary tuberosity. Anteriorly and laterally, the sinus extends to the region of the first maxillary premolar or molar. The floor of the sinus forms the base of the alveolar process of the maxilla.

The sinuses are primarily lined by respiratory epithelium. The cilia and mucus into the respiratory epithelium, are necessary for the drainage of the sinus because the sinus opening, or ostium, is not in a dependent (inferior) position but lies two thirds the distance up the medial wall and drains into the nasal cavity. The maxillary sinus opens into the inferior end of the semi-lunar hiatus, which lies in the middle meatus of the nasal cavity, between the inferior and middle nasal conchae.

The inflammation of maxillary sinus (maxillary sinusitis) could be from odontogenic or non-odontogenic (rhinogenic) origin. Odontogenic maxillary sinusitis usually manifested unilateral, while rhinogenic maxillary sinusitis more commonly involved both—left and right sinuses.

According to clinical presentation, odontogenic maxillary sinusitis are acute, chronic and exacerbated. The main **clinical signs** are pain in the lateral areas of the face and nasal obstruction with nasal discharge from the nostril of involved side. In acute sinusitis symptoms include purulent collection in the nasal cavity, severe pain and affected general condition—fever, headache and fatigue. The chronic sinusitis is characterized with mild pain, blockage of the homolateral (from the same side as sinusitis) nostril without discharge, lack of general symptoms. When a chronic sinusitis exacerbated, clinical signs are similar to the acute sinusitis.

**Sources of odontogenic infections** that involve the maxillary sinus include acute and chronic periapical diseases and periodontal diseases. Infection and sinusitis may also result from trauma to the dentition or from surgery in the posterior maxilla, including removal of teeth, alveolectomy, tuberosity reduction, sinus lift grafting and implant placement, or other
procedures that create an area of communication between the oral cavity and the maxillary sinus.

**Clinical evaluation** of a patient with suspected maxillary sinusitis should begin with a careful visual examination of the patient’s face and intraoral vestibule for swelling or redness. Nasal discharge may be evident during the initial evaluation. The examination of the patient with suspected maxillary sinusitis should include palpation of the sinus externally and palpation intraorally on the lateral surface of the maxilla between the canine fossa and the zygoma. Patients with maxillary sinusitis frequently complain of dental pain, and pain to percussion of several maxillary posterior teeth is often indicative of an acute sinus infection.

The next step in clinical evaluation may include transillumination of the maxillary sinuses. Transillumination of the maxillary sinus is done by placing a bright fiberoptic light against the mucosa on the palatal or facial surfaces of the sinus and observing, in a darkened room, the transmission of light through the sinus.

![Transillumination](image)

The involved sinus shows decreased transmission of light because of the accumulation of fluid, debris, or pus and the thickening of the sinus mucosa. Transillumination may help distinguish maxillary sinusitis from other pain of dental origin associated with molars and premolars.

![Transillumination of the normal maxillary sinus](image) ![Transillumination in patient with left maxillary sinusitis](image)

*Nogueira Júnior et al, 2010*

**Radiographic examination** of the maxillary sinus may be accomplished with a wide variety of exposures. Standard dental radiographs that may be useful in evaluating the maxillary sinus include periapical, occlusal, and panoramic views. A periapical radiograph is limited in that only a small portion of the inferior aspect of the sinus can be visualized. In some cases, the apices of the roots of posterior maxillary teeth may be seen to project into the sinus floor. Panoramic radiographs may provide a “screening” view of the maxillary sinuses. Panoramic radiograph can be obtained to provide a view of both maxillary sinuses for
comparison. Because a panoramic radiograph provides a focused image within a limited focal trough, structures outside of this area may not be clearly delineated. If additional radiographic information is required, Waters’ and lateral views are two plain film radiographs that are frequently useful. The Waters’ view is taken with the head tipped 37 degrees to the central beam. CT is a useful technique for imaging of the maxillary sinuses and other facial bony structures.

Interpretation of the radiographs of the maxillary sinus is not difficult. The sinus should appear radiolucent and should be outlined in all peripheral areas by a well-demarcated layer of cortical bone. Comparison of left with the right is helpful when examining radiographs. Thickened mucosa on the bony walls, air-fluid levels (caused by accumulation of fluids), or foreign bodies lying free should not be present. Partial or complete opacification of the maxillary sinus may be caused by the mucosal hypertrophy and fluid accumulation of sinusitis, by blood filling the sinus following trauma, or by neoplasia. Radiographic changes are to be expected with acute maxillary sinusitis. Mucosal thickening caused by infections may obstruct the exit of the sinus and allow accumulation of mucus, which will become infected and produce pus. The characteristic radiographic changes may include an air-fluid level in the sinus, thickened mucosa on any or all of the sinus walls, or complete opacification of the sinus cavity.
Radiographic changes indicative of chronic maxillary sinusitis include mucosal thickening, sinus opacification, and nasal polyps. Air-fluid levels in the sinuses are more characteristic of acute sinusitis but may be seen in chronic sinusitis during periods of acute exacerbation.

Differential diagnosis of odontogenic sinusitis include non-odontogenic sinusitis, tumors and cysts of maxillary sinus.

Acute maxillary sinusitis is a painful, potentially serious condition that requires immediate attention and aggressive medical and surgical care. The first step in treatment of acute odontogenic sinusitis include removal of the source of infection. The radical method is extraction of the tooth if it is untreatable with other methods, and creation of an oro-antral communication for evacuation of the pus from the maxillary sinus. Via the communication, the sinus is washed several times daily with saline or antiseptic solutions under pressure by syringe. Antibiotic treatment is prescribed (Ospamox 1.0 every 8 hours for example). Nasal decongestants (Xylometazoline 0.1%, 10ml) have to apply in both nostrils. The second step of treatment is assessment of the results of the medication and washing procedures by imaging studies. If the acute sinusitis is completely heal and there is no evidence of inflammation in the maxillary sinus, the treatment ending with operation for closure of the oro-antral communication. If the residual inflammation of the sinus mucosa is suspected, the method of the choice is radical antrotomy by Caldwell-Luc. In this technique, the anterior wall of the sinus is accessed in the area of the canine fossa through a vestibular approach. The sinus is opened, and abnormal tissue or foreign bodies are removed. The ostiomeatal area is evaluated and opened, or a new opening for more dependent drainage into the nose (termed antrostomy) may be created near the floor of the sinus. Because this operation lead to many long lasting complications, connected with the lack of mucous membrane of the sinus, now it was replaced by endoscopic techniques that are less aggressive. The goal of sinus surgery is to remove abnormal tissue from within the sinus cavity and restore normal drainage through the ostium.

Diagnosis and treatment of chronic maxillary sinusitis is difficult and may include allergy testing, nasal or septal surgery, and surgical débridement of the sinuses.
SPECIFIC INFLAMMATION
(Petia Pechalova)

Specific inflammation has a known or single cause. Specific inflammation is a chronic granulomatous disease in which certain immune system cells do not function properly. This leads to ongoing and severe infection. In chronic granulomatous disease immune system cells (phagocytes) are unable to kill some types of bacteria and fungi. This disorder leads to long-term (chronic) and repeated (recurrent) infections. The infection is progressive without spontaneous remission. The granuloma in specific inflammation has a specific structure:

<table>
<thead>
<tr>
<th>Disease</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tuberculosis</td>
<td>Mycobacterium tuberculosis</td>
</tr>
<tr>
<td>Syphilis</td>
<td>Treponema pallidum</td>
</tr>
<tr>
<td>Actinomycosis</td>
<td>Actinomyces Israelii</td>
</tr>
<tr>
<td>Leprosy</td>
<td>Mycobacterium leprae</td>
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Tuberculosis (TB)

TB is a multisystemic disease with myriad presentations and manifestations. Classic clinical features associated with active pulmonary TB are as follows (elderly individuals with TB may not display typical signs and symptoms): cough, weight loss due to loss of appetite, fever, night sweats, hemoptysis, chest pain (can also result from tuberculous acute pericarditis), fatigue. TB was affected humans for millennia and historically is known by variety of names – consumption, wasting disease, yellow guest, etc.

Mycobacterium tuberculosis (Koch's bacillus) was discovered by Dr. Robert Koch in 1882. This microorganism is a rod-shaped, aerobic bacterium, small in size, slow-growing. There are different subtypes of Mycobacterium tuberculosis - humanis, bovis, gallinaceus, others.
Frequency: Almost 20 persons develop tuberculosis and 4 persons die from the disease every minute, somewhere in the world (data from 2011); in Bulgaria frequency is 120: 100 000 (data from 2013). Almost one-third of the human population is infected with the bacillus, but less than 10% of those infected go on to develop the disease. In the other infected individuals, of whom there are thought to be two billion worldwide, the disease remains in a latent state. These individuals serve as a reservoir of the bacterium and, if they become immunodepressed, they may present a reactivation of the disease, leading to the spreading of the infection.

The prognosis of the disease depends on the ability of the host to eliminate the bacillus.

The respiratory route is the principal route of infection. The disease starts when droplets from actively or latently infected individuals reach the respiratory tract of healthy individuals. These droplets contain a small number of bacilli that enter the lung, where they infect primarily alveolar macrophages, type 2 pneumocytes, and polymorphonuclear neutrophils. Transmission could be also by ingestion (tonsilar and intestinal form), by inoculation, by transplacental route (congenital tuberculosis).

In the oral cavity TB could penetrate trough hematogenous way, lymphogenous way, intracanalicular way (via digestive and pulmonary system) or by contact way.

Risk factors for TB are age (babies or young children and elderly people more common are affected), HIV infection (the virus that causes AIDS), substance abuse, silicosis, diabetes mellitus, severe kidney disease, low body weight, organ transplants, head and neck cancer, medical treatments such as corticosteroids or organ transplant, specialized treatment for rheumatoid arthritis or Crohn’s disease.

Some people develop TB disease soon after becoming infected (within weeks) before their immune system can fight the TB bacteria. Other people may get sick years later, when their immune system becomes weak for another reason. Overall, about 5 to 10% of infected persons who do not receive treatment for latent (sleeping) TB infection will develop TB disease at some time in their lives. For persons with weak immune systems, the risk of developing TB disease is much higher than for persons with normal immune systems.

Types: TB could be primary (seen as an initial infection) or secondary (seen as a reactivation of previous infection - reinfection, particularly when health status declines); symptomatic or asymptomatic, pulmonary or extrapulmonary.

Classification of TB in oral cavity and maxillofacial area is:

I. Primary isolated tuberculosis:
   1. TB of the mucous membrane of oral cavity:
      a) tuberous
      b) ulcerous
   2. TB of the jaws
   3. TB of the skin
   4. TB of the salivary glands
   5. TB of the lymph nodes

II. Secondary tuberculosis
Primary TB affects usually the skin of the face and mucous membrane of the oral cavity, as well as lymph nodes. Several weeks after penetration of the infection, papulous, bullate or pustulous formation is occur. This formation turns into a painful ulcer with abrupt edges and floor, fulfilled by the grainy granulations, yellowish or pinkish in color. Primary TB of lymph nodes of maxillofacial region is due to the entry of the Mycobacterium tuberculosis through damage teeth, via tonsils, mucous membrane, or skin.

Secondary skin TB – scrofuloderma, occurs predominantly at children and is localized in the skin of submandibular, submental, cervical, or parotid regions. Skin over the focus is sharp drawn and on its surface are seen separate fistulous tracts or ulcers of incorrect form, filled by abundant granulations. On the skin you can be seen the “phenomena of apple jelly” – change of the lesion color during the diascopy. In the oral cavity, during the gingiva affection, the process can spread on the alveolar bone. The affection of the mucous membrane of the oral cavity by miliary-ulcerous tuberculosis, happens during the severe tuberculosis form of lungs and throat. Mucosa is affected in the result of entry in the oral cavity of the significant number of Mycobacterium tuberculosis with expectoration. The disease more often proceeds in chronic form and is accompanied by subfebrile temperature, fatigue and loss of appetite. Maxillary tuberculosis appears secondary in the result of spread of Mycobacterium tuberculosis by hematogenous or lymphogenous route, from the affected internal organs.

The clinical presentation of orofacial TB

Tuberculous gingivitis may appear as nodular or papillary proliferation of gingival tissues which is diffuse and hyperaemic. There may be absence of any clinical attachment loss, alveolar bone loss or significant cervical lymphadenopathy. Such diffuse gingival enlargements fail to respond to initial usual therapy consisting of supragingival debridement. Sometimes tuberculous gingivitis can be seen simultaneously with marginal periodontitis and enlarged cervical lymph nodes or may present as periodontal loss of tooth support leading to loose teeth and gingival enlargement. A biopsy of the lesion is mandatory for arriving at the diagnosis of TB.

Tuberculous dental periapical granuloma: Tuberculous involvement of the periapical tissue has often been reported. Three routes can be perceived to be entry portals for the tubercle bacillus to become implanted in the periapical tissues. The first is the invasion of the dental pulp through a deep carious lesion by the acid-fast bacilli in the saliva. Should the pulp degenerate and breakdown, a tuberculous periapical infection might result. A second route is the hematogenous and third is the deep periodontal pocket. Patients not responding to the usual periodontal treatments may be harboring tuberculous infection of the paradental tissues even though its presence is not evident. The lesions are usually painless and sometimes involve a considerable amount of bone by relatively rapid extension.

Tuberculous involvement of extraction sockets of teeth: Healing of the tooth extraction sockets is delayed and the socket gets filled with “tuberculous granulation tissue” consisting of many pink to red elevations. Outbreak of TB following dental extractions at two
community dental clinics has been reported where 15 patients developed primary TB lesions, out of them 8 patients had primary tooth socket involvement. The dentist who performed the extractions at both the clinics was found to have active bilateral pulmonary TB.

**Tuberculous osteomyelitis of maxilla and mandible:** Tuberculous osteomyelitis is rare and constitutes less than 2% of skeletal TB. Jaw involvement is even rarer. The mandibular involvement is more frequent than maxilla and alveolar and angle regions have greater affinity. Tuberculous osteomyelitis commonly affects the adults; however, in some cases children are also affected. The spread of infection may be by direct transfer from infected sputum, through an extraction socket or mucosal opening associated with an erupting tooth or by regional extensions of soft tissue lesions to underlying bone or by hematogenous spread. TB of the jaw causes slow necrosis of the bone and formation of a sub-periosteal abscess (lumpy jaw) appearing as a painless, soft swelling. This sub-periosteal abscess may burst resulting in single or multiple sinuses intraorally or extraorally. Pathological fracture of mandible or sequestratation may also occur. In the early stages, when plain radiographs appear normal, MRI or CT may help to localize lesions. The radiographic picture of tuberculous osteomyelitis usually presents as a blurring of bone details leading to diffuse radiolucent picture and cortical plate erosion. It can also present as mixed radiopaque–radiolucent appearance or “worm-eaten” appearance of bony lesions with fistula formation through which small sequestra are exuded. The findings are similar to that of the destructive disease if the periodontal tissues get involved. More advanced lesions may appear as osteoporosis, bone lysis bone lysis, sclerosis and periostitis that mimic chronic pyogenic osteomyelitis and it is often difficult to differentiate the two conditions. Joint involvement may be present but unlike pyogenic osteomyelitis, articular margins and cartilage space are spared. A solitary lytic lesion can also appear sometimes which can mimic neoplasia. A biopsy is mandatory for the diagnosis, and anti-TB drugs along with surgical debridement if required are the main mode of treatment.

**TB of maxillary sinus** is usually a disease of adults and remains an under-diagnosed entity. It is usually secondary to pulmonary or extrapulmonary TB resulting from the bloodstream or by direct extension. Primary sinonasal TB is rare probably due to bactericidal secretion, ciliary movement and mechanical filtering by vibrissae of nose. Most commonly, it presents as nasal discharge, stuffiness of nose, crust formation and sometimes with epistaxis. It can also present as fluctuant swelling (Pott's puffy tumor) and may resemble a malignant lesion. Three types of sinonasal TB have been described:

- **Mucosal involvement** leading to formation of polyps with minimal pus discharge, this type is more common
- **Bony involvement** and fistula formation with abundant discharge of acid-fast bacilli (AFB); this type can lead to midfacial defect
- **Hyperplastic type** has granuloma formation and mimics a malignancy.

If not treated early, it can lead to complications like brain abscess and deterioration of vision. Antral lavage examination for AFB (test Acid fast bacilli) and culture for
Mycobacterium tuberculosis can facilitate early diagnosis. The diagnosis of TB sinusitis is usually based on the absence of response to usual antibiotics, the presence of a caseous granulomatous inflammatory lesion and by bacteriological culture or polymerase chain reaction assay.

The most common salivary glands involved in primary TB are parotid glands, whereas in systemic TB submandibular glands are most commonly involved. There are two clinical forms of tuberculous parotitis: the localized form which is common and involves intraglandular/periglandular lymph nodes and the diffuse parenchymatous form is very rare and is considered to be an acute pathology involving whole of the gland. It may be secondary to the nodal infection. Initially, mycobacterium manifests in the nodes of the preauricular area. It presents as slow growing, non-tender localized swelling in front and below the ear. The pain, abscess, fistula, and facial nerve involvement are the late features. The constitutional symptoms of TB like chronic cough, fever, weight loss may be present but are rare. The diagnosis of tubercular parotitis is very difficult because of the absence of symptoms and may often be misdiagnosed as a benign parotid tumor.

Tuberculous lymphadenitis in the cervical region, also known as scrofula, is the most common site of extra pulmonary TB and accounts up to 5% of the cervical lymphadenopathy. It often affects children and young adults in age range of 30–40 years and shows female predilection. It can present as a single or bilateral neck mass, affecting deep lymph nodes and may be associated with supraclavicular and axillary node involvement. Patients present with slowly enlarging asymptomatic lymph nodes in the neck which is persistent. The mass is referred to as a cold abscess, due to lack of local color or warmth and the overlying skin presents a violaceous color. Other symptoms of disease, such as fever, chills, malaise and weight loss, are present in about 43% of the patients. As the lesion progresses, skin becomes adhered to the mass and may rupture, forming a sinus and an open wound. Fine-needle aspiration cytology and direct microscopic screening for acid-fast bacilli are recommended for the routine diagnosis of tuberculous lymphadenopathy along with culture that remains the gold standard for diagnosis. Diagnosis often requires biopsy. Therapy includes various types of anti-TB chemotherapy, surgical excision, or a combination of surgery and chemotherapy.

Lupus vulgaris is the most common form of cutaneous TB found in individuals with moderate immunity and high degree of tuberculin sensitivity. It is caused by M. tuberculosis and can involve the skin by hematogenous or lymphatic route. Eighty percent of the lesions are on the head and neck and most often on the face around the nose, eyelids, lips, cheeks, ears. Females are affected two to three times more often than males. Skin lesions are of five types: plaque, ulcerative, vegetating, tumor-like, papulonodular.
The plaque form is the most common form (32%), whereas ulcerative form is the most destructive and deforming of all lesions. A single or several, unilateral, reddish-brown papules first appearing on face, neck or arms and then coalescing into erythematous plaques. The surface of the papules exfoliates and the centers scar. Papules recur on the scarred areas, gradually and repeatedly enlarging and coalescing. This leads to the formation of large, firm, elevated plaques. At the periphery are small reddish-yellow or brown nodules. The characteristic lesion is a reddish-brown plaque, composed of nodules which show an “apple-jelly” color when pressed with a glass spatula (diascopy). The lesions may ultimately develop into disfiguring skin ulcers if left untreated. In long-standing scarred lesion, squamous cell carcinoma can develop. Lupus vulgaris is diagnosed by the clinical features, pathology, and strong positive in tuberculin skin test. The disease treatment consists of systemic anti-TB drugs.

**Diagnosis:**

1) **The skin test** *(The Mantoux test)* involves injecting a small amount of fluid (called tuberculin) into the skin in the lower part of the arm. Then the person must return after 48 to 72 hours. Result depends on the size of the raised hard area or swelling. The larger the size of the affected area the greater the likelihood that the person has been infected with TB bacteria at some time in the past. The TB skin test also cannot tell if the person has latent TB or active TB disease. A prior BCG vaccination may cause a false positive result.

2) **The Interferon Gamma release assays:** blood tests that measure a person’s immune response to the bacteria that cause TB. The immune system produces some special molecules called cytokines. These TB tests work by detecting a cytokine called the interferon gamma cytokine. Results can be available within 24 hours, and prior BCG vaccination does not cause a false positive result.

3) **Serological tests** for TB (serodiagnostic tests) are carried out on samples of blood, and they claim to be able to diagnose TB by detecting antibodies in the blood. They are inaccurate and unreliable.

4) **Biopsy** and histopathological evaluation.

5) **Sputum smear microscopy:** is inexpensive and simple, the results are available within hours. The sensitivity though is only about 50-60%.

6) **Fluorescent microscopy**

7) **Culturing bacteria**

**Treatment of orofacial tuberculosis** is the same as standard antimycobacterial treatment regimens used for treating pulmonary TB. The five basic or “first line” antibiotics that form the core of TB treatment are:

- Isoniazid (10 mg/kg)
- Rifampicin (10-20 mg/kg)
- Pyrazinamide (20-35 mg/kg) i.v.
- Ethambutol (25 mg/kg)
- Streptomycin (15 mg/kg IM/24 h; no more than 1 g/24 h)
*Second line* or reserve drugs are used when first-line drugs are not effective. This group consists of:

- **Group I**: first-line oral agents like pyrazinamide, ethambutol, rifabutin;
- **Group II**: injectable agents like kanamycin, amikacin, capreomycin, streptomycin;
- **Group III**: fluoroquinolones like levofloxacin, moxifloxacin, ofloxacin;
- **Group IV**: oral bacteriostatic second-line agents like para-aminosalicylic acid, cycloserine, terizidone, ethionamide, prothionamide;
- **Group V**: drugs with an unclear role in the treatment of drug-resistant TB like clofazimine, linezolid, thiacetazone, amoxicillin/clavulanate, high-dose isoniazid, imipenem/cilastatin, clarithromycin.

After decades of stagnation in the development of anti-TB medications, two new drugs have been approved. The first novel drug **bedaquiline** for treatment of multi-drug-resistant tuberculosis was approved in 2014 in USA. **Delamanid** is the second drug that has been was granted conditional approval by the European Medicine Agency in 2014 for the treatment of drug resistant TB. Several other novel compounds are being evaluated and are in various phases of preclinical or clinical trials.

**Syphilis (Lues)**

Syphilis is a sexually transmitted infection caused by the spirochete bacterium *Treponema pallidum*. Humans are the only source of treponemal infection; there are no known nonhuman reservoirs. Treponema pallidum was discovered by Schaudinn and Hoffmann (1905) in the chancre and inguinal lymph nodes of syphilitic patients.

Syphilis can present in one of four different stages: primary, secondary, latent, tertiary. Syphilis may also occur congenitally - from mother to fetus during pregnancy or at birth. It was referred to as "the great imitator" by William Osler due to its varied presentations.

Untreated syphilis is a slowly evolving chronic disease that transpires in stages separated by asymptomatic intervals. The spirochete bacterium penetrate mucous membranes or enter minuscule breaks in the skin. In women the initial lesion is usually on the labia, the walls of the vagina, or the cervix; in men it is on the shaft or glans of the penis. An initial lesion also may occur on lips, tongue, tonsils, anus, or other skin areas. Blood from a patient with incubating syphilis may be infectious long before the appearance of an initial lesion.
Treponemal infections are unique in that they are characterized by distinct clinical stages. Multiplication of the organisms at the initial site of entry produces the primary stage. The dissemination of treponemes to other tissues results in the secondary stage. After a relatively prolonged period, in some cases 20 to 30 years, the tertiary or late stage evolves.

The primary syphilis: After an incubation period of 10 to 90 days, extensive multiplication of treponemes at the site of entry produces erythema and induration. The resultant papule eventually progresses to a superficial ulcer with a firm base called a hard chancre. Numerous treponemes are present in this highly contagious, open lesion. Regional lymph nodes enlarge, causing regional lymphadenopathy. After 2 to 6 weeks of symptoms, this primary lesion heals, leaving only remnants of scar tissue.

Qiao and Fang, 2011

The secondary syphilis: After an asymptomatic period of 2 to 24 weeks, the secondary or disseminated stage begins. Organisms multiply in many different tissues. Clinical manifestations include slight fever, generalized lymphadenopathy, malaise, and a mucocutaneous rash.

The rash initially appears on the palms and soles and eventually spreads to other areas or could be generalized. The rash may be macular, papular, follicular, papulosquamous, or pustular.
Superficial sores (mucous patches) may occur on mucous membranes of the mouth, vagina, or anus, while wart-like lesions called condylomata lata may form in moist intertriginous areas. All of these lesions teem with treponemes and are highly contagious.

Deposition of immune complexes consisting of treponemal antigens and host antibodies in glomerular basement membranes may produce **nephrotic syndrome**. Two to six weeks after the onset of secondary syphilis, host defenses bring about healing. About 25% of untreated patients experience recurrences of this secondary stage in the first several years following infection.

**The latent syphilis:** The period between secondary and tertiary syphilis, termed latency, can last for many years. Early latency refers to the first 4 years when secondary relapses may occur; late latency is the asymptomatic period beyond 4 years. During this latter period, the patient harbors infectious organisms, especially in the spleen and lymph nodes and blood serology remains positive.

**Tertiary syphilis** can affect almost any tissue. Approximately 80% of fatalities are caused by cardiovascular involvement, while most of the remaining 20% are from neurologic involvement. Gummas are highly destructive tertiary syphilitic lesions that usually occur in skin and bones but may also occur in other tissues.
They are necrotizing granulomas with numerous lymphocytes, giant cells, and epithelioid cells, but few treponemes.

A delayed hypersensitivity response to the small numbers of treponemes in the lesions may be responsible for the development of gummatous disease. Gummas have become rare in the post-antibiotic era. Tertiary syphilis could lead to complication as cardiovascular and neurological problems. Cardiovascular complications are usually attributed to local inflammation induced by the multiplication of treponemes within the wall of the thoracic aorta. The subsequent aortitis produces complications such as aneurysms and coronary artery stenosis.

Neurologic syphilis could be meningeal, meningovascular, parenchymatous and various combinations there of. If the parenchymatous form involves the brain, it is called generalized paresis; if it involves the spinal column, it is called tabes dorsalis. Complications of neurosyphilis include dementia, loss of proprioception, strokes, and blindness.

The congenital syphilis: Besides the three stages of disease in adults, Treponema pallidum also damages fetuses. If a woman is pregnant and has symptomatic or asymptomatic early syphilis, hematogenously disseminating organisms may pass through the placenta to infect the fetus. Approximately 50% of fetuses are aborted or stillborn; the rest exhibit diverse syphilitic stigmata. The congenital syphilis could be early (within first 2 years) or late (later than 2 years). Early congenital syphilis is a result of direct bacterial infection. The late congenital syphilis is a result of phenomenon of hypersensitivity. In early congenital syphilis, signs are apparent before the age of two years. These include rhinitis, mucocutaneous lesions, osteochondritis (especially within the long bones), anemia, and hepatosplenomegaly. In late congenital syphilis, an infected child appears normal past two years of age and then exhibits syphilitic manifestations, such as interstitial keratitis and blindness, tooth deformation (notched incisors and moon molars), eighth-nerve deafness, neurosyphilis, rhagades (fissures at mucocutaneous junctions), cardiovascular lesions, Clutton's joints (fluid accumulation on knee), and bone deformation of the legs, nasal septum, and hard palate.
Diagnosis: Definitive diagnosis of syphilis is complicated by the inability to cultivate Treponema pallidum in vitro. Clinical manifestations, demonstration of treponemes in lesion material, and serologic reactions are used for diagnosis. Serologic tests are mainstay of syphilis diagnosis. They are the only means of identifying asymptotically infected individuals. More than 200 serologic tests have been developed over the years. The perfect test, not yet developed, would detect 100% of the treponemal infections and would be nonreactive in all other diseases. Serologic tests fall into two general categories: “nontreponemal” tests, which measure antibodies directed against lipid antigens, principally cardiolipin, thought to be derived from host tissues; “treponemal” which detect antibodies directed against protein constituents of Treponema pallidum. Congenital syphilis is difficult to diagnose in asymptomatically infected neonates because maternal antibodies (IgG) which pass through the placenta and enter the fetal circulation cause reactivity in both nontreponemal and treponemal tests. In uninfected infants, such maternal antibodies disappear by 3 months. Because of the presence of maternal antibodies in the newborn, quantitative tests should be performed monthly over the first 6 months. If the titer increases or stabilizes and does not decrease, congenital syphilis is indicated and the baby should be treated accordingly.

Treatment: Penicillin remains the drug of choice for treating syphilis:
- Primary, secondary and early latent syphilis - Benzathine penicillin G 2.4 million units intramuscularly (IM) in a single dose
- Late or latent syphilis of unknown duration - Benzathine penicillin G 7.2 million units total, administered as 3 doses of 2.4 million units IM each at 1-week intervals

Penicillin-allergic, nonpregnant patients with early syphilis can be treated with tetracycline.
Actinomycosis

Actinomycosis is an infectious bacterial disease caused by filamentous Gram positive anaerobic bacteria from the Actinomycetaceae family that normally colonize the mouth, colon, and urogenital tract. At least 30 Actinomyces species have been isolated, of which the most common microorganisms causing infections in humans are: \textit{Actinomyces Israelii}, \textit{Actinomyces Viscosus}, \textit{Actinomyces Odontolyticus}, \textit{Actinomyces Naeslundii}, \textit{Actinomyces Meyeri}, \textit{Actinomyces Gerensceriae}. Actinomyces are closely related to Nocardia species. Actinomycosis has unique features that distinguish it from other microorganisms but, surprisingly, it has some amazing similarities to fungal infections.

Human actinomycosis was first described in 1878 by Israel, who along with Wolfe first isolated the causative agent in culture and defined the organism's anaerobic nature. In early reports of infections caused by filamentous gram-positive organisms, no distinction was made between disease caused by actinomyces and nocardias. It was not until 1943 that this genera were clearly differentiated by Waksman and Henrici, enabling separation of the diseases they caused. The pathogenic Actinomyces species do not exist freely in nature but are commensals and normal inhabitants of the oropharynx, gastrointestinal tract, and female genital tract in humans. Hence, humans are themselves the natural reservoir of the Actinomyces species that cause actinomycosis. No external environmental reservoir such as soil or straw has been documented. There is no person-to-person transmission of the pathogenic Actinomyces species. Actinomyces are commensals. When tissue integrity is breached through a mucosal lesion they can invade local structures and organs and become pathogenic. Actinomycosis is therefore mainly an endogenous infection. The portal of entry of Actinomyces species is typically a break in the mucosa of the gastrointestinal tract, anywhere from the mouth to the rectum; such a break may occur as a result of a dental procedure, overt or covert dental sepsis, bacterial suppuration, diverticulitis, appendicitis, surgery, or trauma.

Actinomycosis may occur both in immunocompetent persons and persons with diminished host defenses. Anyone can be infected with actinomyces, but the disease is essentially rare. In the 1970s the incidence in Cleveland, USA, was reported to be one per 300,000, compared with Germany and the Netherlands in the 1960s where it was estimated to be one per million. Risk factors associated with the acquisition of actinomyces are age between 20-60 years, male (except for pelvic actinomycosis, which mainly affects women), Diabetes mellitus, immunosuppression due to steroids or bisphosphonates use, leukaemia with chemotherapy, HIV, lung and renal transplant, alcoholism, local tissue damage caused by trauma, recent surgery, irradiation.

Four clinical forms of actinomycosis account for the majority of infections in humans: cervicofacial, thoracic, abdominopelvic, cerebral. \textit{Oro-cervico-facial actinomycosis} is the most common form of the disease and comprises about 50% of all reported cases. It usually follows dental manipulation or trauma to the mouth, although it can arise spontaneously in patients with poor dental hygiene. Actinomyces species are normally present in high concentrations in
the tonsillar crypts and gingivodental crevices, and many actinomyces infections are odontogenic in origin. In addition to poor dentition and recent dental manipulation, chronic tonsillitis, otitis, and mastoiditis are important risk factors. External trauma may result in the introduction of Actinomyces species into head and neck tissues. Cervicofacial actinomycosis occurs almost exclusively by direct invasion and rarely by hematogenous spread. A hallmark of disease is the tendency to spread without regard for anatomical barriers, including fascial planes and lymphatic channels.

Typically, the disease presents as a slowly progressive painless indurated mass, evolving into multiple abscesses with draining sinus tracts on the skin surface or oral mucosa, sometimes expressing a typical thick yellow exudate with characteristic sulfur granules. At advanced stages, pain and trismus can occur, linked with mastication muscles infiltration. Palpation reveals swelling of a woody consistency. The overlying skin may adhere to the mass and display a venous congestion that imparts a purplish coloration.

Acute suppurative forms with rapid abscess formations are less common and are usually febrile and painful. Regional adenopathy is rare except in late stages of disease. Cervicofacial actinomycosis may extend to the underlying mandible or facial bones, leading to periostitis or osteomyelitis. Bone involvement is observed in approximately 10% of cases. When clinical symptoms are not typical, actinomycosis can be erroneously diagnosed as a soft tissue tumor. Many cases of actinomycosis are odontogenic in origin, and cervicofacial lesions are frequently located at the angle of the jaw or in the submandibular regions. Infections of the periapical regions, nose, paranasal sinuses, oropharynx, hypopharynx, tongue, and trachea may also be seen. Occasionally, patients can develop involvement of the masseter muscle or thyroid, and rarely some individuals may have intracranial or thoracic extension of infection.


Diagnosis: Only 10% of actinomycotic infections are correctly diagnosed at the time of initial presentation. The diagnosis of actinomycosis is made most accurately by isolation of Actinomyces species in cultures of clinical specimens. The visualization of actinomycotic granules in exudates or in histopathologic tissue sections is strongly supportive of the
diagnosis. Multiple biopsy sections from different tissue levels are recommended to improve histopathologic diagnosis.

**Treatment:**

I. Antibiotics:

1) **Penicillin G** (50–75 mg/kg/day intravenously in four daily divided doses) for 4 to 6 weeks followed by per oral **penicillin V** (30–60 mg/kg/day administered in four divided doses) for 2 to 12 months.

2) For patients with a penicillin allergy, the **tetracyclines** (doxycycline 2 to 5 mg/kg per day administered in two equal portions at 12-hour intervals) probably offer the best treatment alternative for cervicofacial infections.

For example, mild cervicofacial actinomycosis may be managed with just 2 months of oral penicillin V or doxycycline without surgical intervention.

II. Surgical treatment include curettage of bone, resection of necrotic tissue, excision of sinus tracts, drainage of soft tissue abscesses. Although surgery facilitates recovery, it is usually not curative by itself.

III. Immunotherapy is a type of treatment that enhance the body's natural defenses to fight the disease. It uses substances made by the body or in a laboratory to improve or restore immune system function. Immunotherapy is applied in complete lack of signs of acute inflammation, because a risk of worsening of the disease. Immunotherapy is a part of treatment of actinomycosis in a chronic stage of the disease. The most appropriate type of immunotherapy in cases of specific inflammation is non-specific immunotherapy. Two common non-specific immunotherapies are:

- **Interferons:** an interferon made in a laboratory is called an interferon alpha (Roferon-A, Intron A, Alferon). Side effects of interferon treatment may include flu-like symptoms, an increased risk of infection, rashes, and thinning hair.

- **Interleukins:** an interleukin made in a laboratory is called interleukin-2, IL-2, or aldesleukin (Proleukin). Common side effects of IL-2 treatment include weight gain and low blood pressure. Some people may also experience flu-like symptoms.
HUMAN IMMUNODEFICIENCY VIRUS (HIV)
AND ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS)
(Petia Pechalova)

HIV is an RNA based virus that causes AIDS by attacking the immune system and destroying the body’s defenses against diseases.

Body becomes vulnerable to infections and cancers that don’t normally develop in healthy people.

History: HIV was first reported as a new and distinct clinical disease by the Centers for Disease Control and Prevention (USA) in June 1981. At the time, the condition did not have a name. Doctors in San Francisco, Los Angeles and New York had documented an unusual cluster of diseases in young homosexual men. These diseases, Kaposi’s sarcoma and Pneumocystis carinii pneumonia, were previously unknown to this group. All of the subjects were suffering from general immune deficiency. Their bodies were vulnerable to rare “opportunistic” infections. The subjects were otherwise healthy. The earliest known cases are those of five young homosexual men in Los Angeles were diagnosed with Pneumocystis carinii pneumonia. Twenty six homosexual men, from both New York and San Francisco, were diagnosed with Kaposi sarcoma. Since all of the first cases of this newly identified disease involved homosexual men, researchers initially considered sex among gay men the route of transmission. The condition was named Gay-related immune deficiency syndrome (GRIDS). However, HIV cases were soon reported in other populations as well. The spread to other populations was observed in intravenous drug users, hemophiliacs, blood transfusion recipients, adults from Central Africa, Haitians living in the United States and infants born to intravenous drug using mothers. Researchers then hypothesized that because the virus was primarily affecting homosexual men and intravenous drug users, the agent causing the disease was probably both blood-borne and sexually transmitted. By 1982, research was going on in both the United States and France to identify the virus. In the United States, Robert C. Gallo of the National Cancer Institute called the virus HTLV-III (human T-cell lymphotropic virus type III). In France, Luc Montagnier and his colleagues at the Pasteur Institute in Paris called the virus LAV (Lymphadenopathy-Associated Virus). Both groups of researchers were working on the same virus. In subsequent years, there was a dispute as to who discovered the virus. In 1983, the disease was said to be caused by the Human Immunodeficiency Virus (HIV). Credit for the discovery was given jointly to the U.S. team and the French group. The
term AIDS is the name given to the condition associated with the HIV. It replaced the name
GRIDS when it became apparent that the disease was not just limited to gay men.

A review of the medical literature dating back to the 1950s found 19 cases of what
appear to be AIDS cases. The illnesses fit the Centers for Disease Control and Prevention
criteria for HIV/AIDS with regards to risk factors, symptoms and progression. The mean age
of patients was 37 years. Males were more than females. Sixteen patients had opportunistic
infections without Kaposi's sarcoma, the remainder had Kaposi's sarcoma. Two patients were
reported to be homosexual. Three others had been living in Africa. One patient was born in
Haiti. In two instances concurrent or subsequent opportunistic infection occurred in family
members. All patients died 1 month to 6 years after the initial manifestation of disease.
Frozen tissue and serum samples stored at the University of Arizona were available for one of
these possible early AIDS cases. The patient was a 15-year-old black male, named Robert R.
from St. Louis, Missouri, who was hospitalized in 1968. The patient had extensive swelling of
the genitalia and lower extremities and swelling of the lymph nodes in his neck. Chlamydia
was also found in his body, indicating that he was sexually active. During the following year,
Robert R. deteriorated. He died on May 16, 1969. Tests done in the mid 1980s found that he
had HIV-antibodies. The patient had never received a blood transfusion, nor had he ever
traveled outside of the United States. The researchers concluded that the virus may have been
introduced into the human population long before the first cases were officially reported.

Soon after AIDS was recognized in humans, researchers began to report cases of a
similar virus in colonies of monkeys. The virus, called Simian Immunodeficiency Virus
(SIVsm) was found in African green monkeys, white-collared monkeys and the sooty
mangabey monkey, among others. Another strain of the virus, named SIVcpz, was found in
chimpanzees. The SIV was found in more than 30 African primate species. However, the
virus was not causing illness or death to these animals. The questions was did this Simian
Immunodeficiency Virus infect humans and how is it possible? It has been known for some
time that certain viruses can pass between species, including from animals to humans. The
transfer of disease from animals to humans is known as zoonosis. Other examples of zoonosis
are anthrax, bubonic plague, avian Influenza (Bird flu), “mad-cow” disease, severe acute
respiratory syndrome (SARS). It is theorized that the virus at some point crossed species -
from primates to humans. Several theories tried to explain the mechanism:

Natural process theory: This theory proposes that hunters of chimpanzees contracted
the virus as early as the 1940s. This time frame was arrived at based on a study done in 2000
using a computer model to track the evolution of the HIV. The hunters cut themselves while
preparing infected chimpanzee meat. The virus then mutated into HIV and was passed along
through millions of humans. The virus could have also been transferred to humans through the
selling and consumption of primate bushmeat sold in African markets, an ongoing practice in
parts of Africa. Both preparers of the meat and those who ate it could have easily become
infected.
Oral polio vaccine theory: Polio vaccine was developed in Africa (Belgian Congo, Rwanda and Urundi) during the 1950s. The theory is that the vaccine was produced using the kidney cells of infected chimps infected with SIV. This then led to the subsequent infection of humans with HIV. This theory was disproved when samples of the original polio vaccine were analyzed and no traces of either HIV or SIV were found. Other tests showed that the kidney cells were taken from the Asian macaque monkey only, which has been shown to be incapable of being infected with either SIV or HIV.

Contaminated needle theory: Healthcare professionals in Africa during the 1950s used needles on multiple patients as a way to save money on syringes. The virus could have been spread from one person (ex. a chimp hunter) to another with relative ease.

The Colonialism theory: People across Africa, under colonial rule, were subjected to harsh conditions in labor camps, leading to food scarcity, poor sanitation and poor health. As a result, SIV could have infiltrated those camps and taken advantage of the weakened immune system of the workers. Additionally, workers may have been inoculated with contaminated needles and may have become contaminated via prostitution because many of the camps employed prostitutes to keep the workers happy. Labor camps were set up around the time that HIV was first believed to have passed into humans - the early 20th century.

The Conspiracy theory: HIV is a man-made virus and was designed to be part of a government biological warfare program against blacks and gays.

Prevalence of HIV/AIDS: HIV/AIDS has spread to virtually every continent on the planet. Approximately 33 million people worldwide have the disease. Presently, the country with the largest population of people living with HIV/AIDS is South Africa (5.5 million). There are about 1.1 million Americans living with HIV/AIDS. Fifty-five thousand new infections occur every year.

HIV types: There are two types of HIV: HIV-1 and HIV-2. Both types are transmitted by sexual contact, through blood, and from mother to child, and they appear to cause clinically indistinguishable AIDS. However, it seems that HIV-2 is less easily transmitted, and the period between initial infection and illness is longer in the case of HIV-2. Worldwide, the predominant virus is HIV-1, and generally when people refer to HIV without specifying the type of virus they will be referring to HIV-1. The relatively uncommon HIV-2 type is concentrated in West Africa and is rarely found elsewhere. The strains of HIV-1 can be classified into three groups: the "major" group M, the "outlier" group O and the "new" group N. These three groups may represent three separate introductions of simian immunodeficiency virus into humans. Within group M there are known to be at least nine genetically distinct subtypes (or clades) of HIV-1. These are subtypes A, B, C, D, F, G, H, J and K.

Until about 1994, it was generally thought that individuals do not become infected with multiple distinct HIV-1 strains. Since then, many cases of people coinfected with two or more strains have been documented. All cases of coinfection were once assumed to be the result of people being exposed to the different strains more or less simultaneously, before their immune systems had had a chance to react. However, it is now thought that "superinfection"
is also occurring. In these cases, the second infection occurred several months after the first. It would appear that the body's immune response to the first virus is sometimes not enough to prevent infection with a second strain, especially with a virus belonging to a different subtype. It is not yet known how commonly superinfection occurs, or whether it can take place only in special circumstances.

Transmission could be by vaginal intercourse, anal intercourse (10x higher infection rate than vaginal way, because of higher rate of tissue tears), oral intercourse, blood transfusion, needles, infected mother to child.

**Clinical signs of HIV:** No physical findings are specific to HIV infection. The physical findings are those of the presenting infection or illness.

*Generalized lymphadenopathy* is common. Weight loss may be apparent.

*Oral candidiasis* presents as a thick, white, curdlike coating of the tongue, hard and soft palates, and buccal mucosa, which can be easily removed with a tongue blade to reveal a hemorrhagic area beneath. Oral candidiasis is recognized as an early clinical marker of immunosuppression, an initial manifestation of symptomatic HIV infection, and a poor prognostic marker for progression.

*Hairy leukoplakia* is a verrucous, white, corrugated lesion, which, unlike candidiasis, cannot be scraped off with a tongue blade, is usually asymptomatic, and is usually located on the lateral surface or under surface of the tongue. It represents oral infection with Epstein-Barr virus, which has been demonstrated on electron microscopy.

Gingival and periodontal disease has recently been recognized as a possible early presentation of HIV infection. Herpetic infections of the skin and mucous membranes are common conditions in the general population, but in HIV-infected individuals can be a presenting sign or symptom. *Herpes simplex* lesions can involve the mouth, or any skin surface, producing single or multiple painful ulcerations.

*Herpes zoster* represents reactivation of varicella zoster infection in ganglia, leading to acute neuritis with a dermatomal rash, sometimes followed by post-herpetic neuralgia in 10% of patients, which can occur in immunosuppressed individuals and could affect trigeminal nerve. Multidermatomal or disseminated zoster is clearly associated with HIV infection, although whether it is predictive of the development of AIDS is controversial.

Cutaneous *molluscum contagiosum* is a common viral skin infection which is caused by a DNA virus from the poxviruses (Poxviridae) family. It is most frequently found in children. However, it is also found in adolescents by sexually transmission, which usually appears on the genital area. Extragénital molluscum contagiosum occurs almost exclusively in HIV-infected patients or immunocompromised patients. They are asymptomatic pearly-colored slightly irritated papules with characteristic central umbilication.

*Kaposi's sarcoma* is a neoplastic vascular disorder. It can be solitary or multiple, nodular or plaquelike, and can involve the cutaneous surface, mucous membranes, or parenchyma of virtually any organ.
HIV infection should be considered in any patient with unusual or recurrent serious infections without another cause, especially in those with risk factors for HIV infection. Any of the opportunistic infections or cancers associated with acquired immune deficiency syndrome (AIDS) can also occur in the absence of HIV infection, although they usually develop in patients with some other form of immune suppression or defect.

**Clinical classification of HIV infection:**

*Group 1.* Acute infection

*Group 2.* Asymptomatic infection

*Group 3.* Persistent generalized lymphadenopathy

*Group 4.* Other disease: constitutional disease, neurologic disease, secondary infectious disease, secondary cancers, other conditions.

Early clinical signs and symptoms of HIV infection are protean and range from mild non-specific fatigue and malaise to fever, night sweats, and weight loss. They can reflect the direct effects of the HIV or represent an opportunistic infection or malignancy. Acute HIV infection is defined as the period between exposure to the virus and completion of the initial immune responses. This period varies but generally lasts 2-3 months. During this time, antibody tests may be negative for HIV, but the serum viral load (the amount of HIV virus in the blood) is detectable and can be quite high (millions of copies per milliliter). Patients have signs and symptoms of acute viral infection 1 to 8 weeks after exposure: fever, myalgia, nausea, pharyngitis. Some of these patients can present with symptoms of aseptic meningitis: headache, meningismus, photophobia. On examination, a macular rash, aphthous mouth ulceration, lymphadenopathy, and hepatosplenomegaly can be noted. Asymptomatic HIV infection begins after antibodies to the virus have fully developed and the initial immune response is complete. HIV disease with active virus replication usually progresses during this asymptomatic period, and the rate of disease progression correlates directly with HIV RNA levels. Individuals with high levels of HIV RNA progress to symptomatic HIV disease faster than patients with low levels of HIV RNA. In third stage some individuals develop symptoms or organ dysfunction during chronic infection due to direct effects of the virus rather than a defect in cell-mediated immunity. Some infected persons who are otherwise asymptomatic develop persistent generalized lymphadenopathy during this time. With few exceptions, CD4 cell counts decline progressively during this asymptomatic period, at an average rate of approximately 50 cells/µL/y. The fourth stage, acquired immunodeficiency syndrome (AIDS), is the condition that results from long-term (chronic) HIV infection and is defined by an absolute CD4 cell count of less than 200 cells/µL and specific opportunistic infections or malignancies. The interval between acute HIV infection and AIDS is highly variable, with a median time of approximately 10 years. In many infected individuals, an opportunistic disease is the first manifestation of HIV infection. When the CD4 cell count falls to below approximately 200 cells/µL, the resulting state of immunodeficiency places the individual at high risk for opportunistic infections and neoplasms (clinically apparent HIV disease).
**Diagnosis:** Screening for human immunodeficiency virus (HIV) infection is paramount, since infected individuals may remain asymptomatic for years while the infection progresses. Serologic tests are the most important studies in the evaluation for HIV infection. Secondary testing that may be performed to assist with diagnosis or staging includes the viral culture, lymph node biopsy, proviral DNA polymerase chain reaction (PCR), genotyping of viral DNA/RNA. In June 2014, the Centers for Disease Control and Prevention (USA) issued new recommendations for HIV testing in laboratories that are aimed at reducing the time needed to diagnose HIV infection by as much as 3-4 weeks over previous testing approaches. Diagnosis starts with a fourth-generation test that detects HIV in the blood earlier than antibody tests can; it identifies the viral protein HIV-1 p24 antigen, which appears in the blood before antibodies do. If this test is positive, an immunoassay that differentiates HIV-1 from HIV-2 antibodies should be performed; results from such assays can be obtained faster than they can from the Western blot test. In patients with positive results on the initial antigen test but with negative or indeterminate results on the antibody differentiation assay, HIV-1 nucleic acid testing should be performed to determine whether infection is present.

**Staging of HIV disease** is based partially on clinical presentation, but other laboratory tests can help in deciding whether to initiate or modify treatment. Baseline laboratory studies for other infections (eg, tuberculosis) are important in the initial workup of a patient with newly diagnosed HIV infection. In addition, baseline levels of factors that may be affected by antiretroviral therapy (eg, lipids) should be measured. The CDC classifies HIV infection into 3 categories, according to the presence of certain infections or diseases. These conditions may be exacerbated by the HIV infection or represent true opportunistic infections. *Category A* is asymptomatic HIV infection without a history of symptoms or AIDS-defining conditions. *Category B* is HIV infection with symptoms that are directly attributable to HIV infection including bacillary angiomatosis, oropharyngeal candidiasis, vulvovaginal candidiasis, pelvic inflammatory disease, cervical dysplasia/cervical carcinoma in situ, oral hairy leukoplakia, idiopathic thrombocytopenic purpura, constitutional symptoms - fever (>38.5°C) or diarrhea lasting more than 1 month, peripheral neuropathy, herpes zoster. *Category C* is HIV infection with AIDS-defining opportunistic infections. People with healthy immune systems can be exposed to certain viruses, bacteria, or parasites and have no reaction to them—but people living with HIV/AIDS can face serious health threats from what are known as “opportunistic” infections. Opportunistic infections are signs of a declining immune system. Most life-threatening opportunistic infections occur when your CD4 count is below 200 cells/mm3. Opportunistic infections are the most common cause of death for people with HIV/AIDS. The United States Centers for Disease Control and Prevention developed a list of more than 20 opportunistic infections that are considered AIDS-defining conditions — if patients have HIV and one or more of these opportunistic infections, they will be diagnosed with AIDS, no matter what their CD4 count happens to be. List of opportunistic infections:

- Candidiasis of bronchi, trachea, esophagus, or lungs
- Invasive cervical cancer
- Coccidioidomycosis
- Cryptococcosis
- Cryptosporidiosis, chronic intestinal (greater than 1 month's duration)
- Cytomegalovirus disease (particularly CMV retinitis)
- Encephalopathy, HIV-related
- Herpes simplex: chronic ulcer(s) (greater than 1 month's duration); or bronchitis, pneumonitis, or esophagitis
- Histoplasmosis
- Isosporiasis, chronic intestinal (greater than 1 month's duration)
- Kaposi's sarcoma
- Lymphoma, multiple forms
- Mycobacterium avium complex
- Tuberculosis
- Pneumocystis carinii pneumonia
- Pneumonia, recurrent
- Progressive multifocal leukoencephalopathy
- Salmonella septicemia, recurrent
- Toxoplasmosis of brain
- Wasting syndrome due to HIV

Baseline studies for other infections that are important in the initial workup of a patient with newly diagnosed HIV infection include the following: skin testing for tuberculosis, cytomegalovirus testing, syphilis testing, tests for gonococcal and chlamydial infection, hepatitis A, B, and C serology, anti-Toxoplasma antibody, ophthalmologic examination.

The CD4 T-cell count is a reliable indicator of the current risk of acquiring opportunistic infections. CD4 counts vary, and serial counts are generally a better measure of any significant changes. The reference range for CD4 counts is 500-2000 cells/µL. After seroconversion, CD4 counts tend to decrease (around 700/µL on average) and continue to decline over time. The CD4 count under 200/µL is considered AIDS-defining owing to the increased risk of opportunistic infections at this level. The three HIV categories are further subdivided based on the CD4+ T-cell count. Categories A1, B1, and C1 are characterized by CD4+ T-cell counts greater than 500/µL. Categories A2, B2, and C2 are characterized by CD4+ T-cell counts between 200/µL and 400/µL. HIV infections in patient with CD4+ T-cell counts under 200/µL are designated as A3, B3, or C3. Importantly, once an HIV infection has been staged into a higher clinical category, it remains in that category permanently. In addition, the infection is classified based on the lowest CD4+ T-cell count in that patient.

The treatment of HIV disease depends on the stage of the disease and any concomitant opportunistic infections. In general, the goal of treatment is to prevent the immune system from deteriorating to the point that opportunistic infections become more likely. Immune reconstitution syndrome is also less likely in patients whose immune systems are weakened to this point. Highly active antiretroviral therapy (HAART) is the principal method for
preventing immune deterioration. In addition, prophylaxis for specific opportunistic infections is indicated in particular cases. Successful long-term highly active antiretroviral therapy results in a gradual recovery of CD4 T-cell numbers and an improvement of immune responses and T-cell repertoire (previously lost antigen responses may be restored). The peripheral T-cell counts initially surge after therapy is initiated, but this represents redistribution of activated T cells from the viral replication centers in the lymph nodes rather than a true increase in total-body CD4 T-cell counts.

Nearly 30 antiretroviral drugs have been approved for use in HIV-infected adults and adolescents; 18 of these have an approved pediatric treatment indication and 15 are available as a pediatric formulation or capsule size. Of the 27 antiretroviral drugs that have been approved, 3 are no longer being manufactured either because of the development of improved formulations (ie, amprenavir replaced by fosamprenavir) or because of limited use (ie, delavirdine and zalcitabine).

Classification of antiretroviral agents:
I. Nucleoside reverse transcriptase inhibitors (NRTIs)
II. Protease inhibitors (PIs)
III. Non-nucleoside reverse transcriptase inhibitors (NNRTIs)
IV. Fusion inhibitors
V. Cellular chemokine receptor (CCR5) antagonists
VI. Integrase inhibitors

The introduction of HAART has significantly improved mortality rates. One study of nearly 7000 men with HIV infection found that annual mortality rates decreased from 7% in 1996 to 1.3% in 2004, although the findings highlighted the fact that non-AIDS-related illnesses were accounting for a greater proportion of deaths. Treatment failures are most closely related to the timing of therapy initiation (and, therefore, of timeliness of diagnosis). CD4 counts under 200/μL and evidence for AIDS are strong predictors of mortality (risk ratios of 2.7 and 1.6, respectively). Antiretrovirals should be prescribed by an infectious disease specialist. Antiretroviral regimen selection is individualized, on the basis of the following:

- Virologic efficacy
- Toxicity
- Pill burden
- Dosing frequency
- Drug-drug interaction potential
- Drug resistance testing results
- Comorbid conditions

Treatment guidelines for HIV infection are age-specific. Because of advances in management, HIV-infected patients are now having fewer complications and surviving longer; as a result, they are increasingly experiencing common health problems seen in the general population, and these problems must be addressed.
JAW CYSTS
(Petia Pechalova)

Jaw cysts are known from high antiquity. Lesions of the jaws interpreted as cysts were found in mummies, 4500 years b.c. In the beginning of the I-st c.a.c. Aulus Cornelius Celsus described cystic lesions of the jaws. Later Pierre Fauchard (1690 – 1762) and John Hunter (1729 – 1793) published description of jaw cysts. Written reports for the nature and treatment of jaw cysts became more frequent after 1850. In 1866 Paul Broca created classification of odontomas, including odontogenic tumors, cysts and malformations. In 1974 Kramer defined cyst as a “pathological cavity fully or separately epithelized, having fluid, semifluid or gaseous contents and which is not created by the accumulation of pus”. Recent century continues enriching knowledge about jaw cysts – the latest classification of World Health Organization (WHO) is done in 2005. WHO defines radicular cysts as pathologic bone cavities with inflammatory genesis, epithelized, developed around teeth’s apexes an rarely laterally to the teeth’s roots in connection with extra lateral root channels. Nair writes about presence of two types radicular cysts – “real”, that represent an independent from the microorganism closed system and “pocket” or “cove”, connected with the apical foramen and the root channel.

Classification of jaw cysts:
A. Epithelial-lined cysts:
   I. From developmental origin:
      1. Odontogenic:
         a) Gingival cyst of infants
         b) Odontogenic keratocyst
         c) Dentigerous cyst
         d) Eruption cyst
         e) Gingival cyst of adults
         f) Developmental lateral periodontal cyst
         g) Botryoid odontogenic cyst
         h) Glandular odontogenic cyst
         i) Calcifying odontogenic cyst
      2. Non-odontogenic:
         a) Midpalatal raphé cyst of infants
         b) Nasopalatine duct cyst
         c) Nasolabial cyst
   II. From inflammatory origin:
      1) Radicular cyst, apical and lateral
      2) Residual cyst
      3) Paradental cyst and juvenile paradental cyst
      4) Inflammatory collateral cyst
B. Non-epithelial-lined cysts

1) Solitary bone cyst
2) Aneurysmal bone cyst

In the pathogenesis of radicular cysts conventionally can be divided three stages: initiation, formation of epithelial capsule and growth of the cyst.

I. Initiation: Radicular cysts arise out of epithelial cell of Malassez, which proliferation is stimulated by inflammation – a result from pulp necrosis and released necrotic tissue and bacterial antigens the key part among which take the bacterial endotoxines from Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis and Escherichia coli, thought to be the most important initiation factor in pathogenesis of radicular cysts. A substantial meaning for proliferation of epithelial cells of Malassez have the inflammatory cytokines predominant place among which take immunoglobulin (Ig) G, followed by IgA, IgE, IgM. In the periapical cysts lymphocytes B and T have been established which suggests that the origin of these legions is a result of immune reactions with participation of cellular and humoral immunity. Immunohistochemical studies determine presence of interleukin (IL)-1 α, IL-1 β, IL-6 in the epithelial cover, in the endothelial cells of the vessels, tumor necrosis factor (TNF) and IL-8 – in macrophages.

II. Formation of epithelial capsule: There are two famous theories. The first one suggests that the proliferating epithelium covers the uncovered connective tissue surface of the abscess’s cavity or the surface of the cavity formed as a result of connective tissue destruction under the action of proteolysis enzymes. The other theory which has more upholders says that the cystic cavity is formed after epithelial tissue proliferation in the apical region through degeneration and cells’ death in the center. Participation of matrix metalloproteinase (MMP)-13, secreted from epithelial cells, fibroblasts and plasmatic cells is supposed in the development of this process. Important is also the role of MMP-1, MMP-8, MMP-2, MMP-9.

III. Growth of the cyst: It is well known that the pressure of the fluids in the cystic cavity is statistically significant higher than the local blood pressure as the inter cystic pressure is in reverse proportion with the cyst size, which means that it has a leading role in the earliest stages of cyst’s growth. In the process of cyst’s growth participation of glucosaminoglycans is suspected, presented in the cellular wall and cystic contain. It is supposed that the process is attended by destruction of the enclosed connective tissue and bone resorption with participation of prostaglandins. The role of RANKL, found in the cellular wall, has also been established.

The clinical signs of radicular cysts include bone expansion; development of soft, fluctuant, and bluish in color, moderate painful swelling when covered bone was destructed. If aspirate the content of the cyst, you will see a straw-coloured fluid with a cholesterol crystals.
Many radicular cysts are symptomless and are discovered when periapical radiographs are taken of teeth with non-vital pulps. Over the years, the cyst may remain static or grow in size.

**Dentigerous cysts** embrace fully or partially the crown of unerupted tooth and accomplish a contact with it in the region of cemento-enamel junction. In the literature predominates the statement that dentigerous cyst can be of extra- or intra follicular origin, as the second type develops as a result of fluid accumulation between the reduced enamel epithelium and the enamel as well as in the enamel organ. Another theory for the origin discusses the possibility for falling of the permanent tooth’s crown during eruption into dentigerous cyst formed around the apex of its transient predecessor. It has been also discussed the opinion that inflammatory changes in the apex of the transient tooth predecessor can be responsible for developing of dentigerous cysts around the corresponding permanent teeth.

Dentigerous cysts are always associated with an unerupted tooth or a developing tooth bud and are found most frequently around the crown of the mandibular premolars in children or mandibular third molars in adults followed, by the maxillary canines, maxillary third molars and, rarely, the maxillary central incisors. The cyst may cause swelling, teeth displacement, tooth mobility and sensitivity if it reaches a size larger than 2 cm in diameter. In the radiograph, the dentigerous cysts usually show a well-defined unilocular radiolucency, often with a sclerotic border, surrounding the crown of an unerupted tooth.

The term “**odontogenic keratocyst**” is proposed by Philipsen in 1956. With the publication of the last classification of jaw cysts WHO recommends the name “odontogenic keratocyst” to be replaced by “keratocystic odontogenic tumour”. But the opinion of the International Association of Oral Pathologists from June 2006 is that the name “odontogenic keratocyst” should be preserved until receiving of undisputed evidences in molecular level proving the neoplastic character of the lesion. It arising from epithelial odontogenic rests. The typical microscopic characteristic of this lesion is the presence of variable amounts of aberrant epithelial cells, without nuclei, which are named “ghost cells”. In the wall of the odontogenic keratocyst a collagenolytic activity is established and later α: polymorphonuclear collagenase is isolated which helps the connective tissue destruction at absence of inflammatory cells by degranulation of sub cellular particles under the action of specific antigen – induced immunocomplex or by direct contact. Basal cell nevus syndrome or Gorlin-Goltz syndrome, an autosomal dominant inherited disorder, with a high penetration, clinically characterised by
a series of associated manifestations, the most common being maxillary keratocysts and cutaneous basal cell carcinomas, and other less frequent ones which can be present, such as cardiac disturbances (persistent ductus arteriosus), characteristic facies (mandibular prognathia, marked superciliary arches, wide bridged nose and hypertelorism), skeletal (brachymetacarpalism of the 4th and 5th fingers, vertebrae problems, costal synostosis or bifid ribs), skin (dermoid cysts, lipomas), neurological (congenital hydrocephalus, calcification of the cerebral falx, learning difficulties), sight (blindness, congenital cataracts, strabismus), hormonal (hypogonadism) or associated with other malignant neoplasias. Pathogenesis of the syndrome is attributed to abnormalities in the long arm of chromosome 9 (q22.3-q31) and loss or mutations of human patched gene (PTCH1 gene). Diagnosis is based upon established major and minor clinical and radiological criteria and ideally confirmed by deoxyribonucleic acid analysis.

**Nasopalatine duct cysts** are epithelial cysts that are considered to be the most common (32.8-73.2%) of the nonodontogenic cysts. The nasopalatine duct leads from the incisive fossa in the oral cavity to the nasal floor, in which it ends in the nasopalatine infundibulum. Nasopalatine duct cyst is one of many pathologic processes that may occur within the jaws, but it is unique in that it develops in only a single location, which is the midline anterior maxilla. Nasopalatine duct cysts usually present as unilateral pathology, but they may also occur bilaterally (approximately 0.25% of all cases). The nasopalatine ducts ordinarily undergo progressive degeneration. However, the persistence of epithelial remnants may later become the source of epithelia that gives rise to a nasopalatine duct cyst, from either spontaneous proliferation or proliferation following trauma (eg, removable dentures, dental implant treatment), bacterial infection, or mucus retention. Genetic factors have also been suggested. The mucous glands present among the proliferating epithelium can contribute to secondary cyst formation by secreting mucin within the enclosed structure. Nasopalatine duct cysts can form within the incisive canal, which is located in the palatine bone and behind the alveolar process of the maxillary central incisors, or in the soft tissue of the palate that overlies the foramen, called the cyst of the incisive papilla. Patients may be asymptomatic, with the lesion being detected on routine radiographs. Complaints are often found to be associated with an infection of a previously asymptomatic nasopalatine duct cysts and consist primarily of swelling, drainage, and pain. The vitality of surrounding teeth should not be affected, but it is not uncommon to see evidence of endodontic therapy because the nasopalatine duct cyst was previously clinically misdiagnosed as a periapical cyst or granuloma.

Panoramic and periapical radiographs showed a well-circumscribed radiolucency in the midline of the anterior maxilla. The lesion was apical to the central incisors and appears to abut the mesial surfaces of both associated lateral incisors. The radiolucency has a heart shape, due to the superimposition of the nasal spine. No root resorption is noted.
Treatment is determined by cyst’s aetiology and localisation, which means that the causal tooth must be treated or removed, and the cystic lining, which produces the cystic content, must be removed. Several methods of treatment exist depending on the size and localisation. Cysts are most frequently treated surgically.

Treatment options differ with regard to the classification of cysts into large and small. The name Carl Partsch (1855-1932) is connected with a method for surgical treatment of large mandibular cysts which he introduced in 1910, and which is still today called Partsch I method (marsupialization). By the marsupialization the large bone cavity in the mandible is drained into the oral cavity by the technique of marsupialization. The principle of the method is to make an opening, i.e. fenestration, on the outer wall of the cyst through which the cystic content drains into the oral cavity. In this way pressure on the cyst is reduced, resulting in a reduction of the cystic cavity. After fenestration jodoform gauze is inserted into the cystic cavity in order to prevent infection and closure of the edges of the opening. The edges of the cystic capsule are previously sutured with the oral mucous membrane. The gauze is left for 1-2 days, after which an impression is taken of the bone cavity and an obturator fabricated from acrylic. The obturator must not cover the whole of the cavity as this interferes with cicatrization, i.e. reduction of the bone cavity. Its function is to maintain communication between the cyst and the oral cavity. The obturator is filed every 8 days and in this way becomes smaller. It remains in the mouth until the bone cavity has become so small that the obturator drops out of the cavity and the mucous membrane of the cyst is changed by metaplasia into oral mucous membrane. Drainage of the cyst ensures that fluid no longer accumulates, and its stimulus on the cystic capsule disappears, which is considered one of the potential causal agents of its progressive growth. Consequently its autonomic growth is arrested and accordingly further development. Although marsupialization satisfies certain therapeutic requirements, it has significant drawbacks, such as slow healing and cicatrization.

The Partsch II method (enucleation) of treatment of small jaw cysts (smaller than 2.5 cm in diameter) was introduced later. The method presents a completely removal of epithelial cyst capsule. Although the enucleation was a method for treatment of small cysts, it should be used whenever possible as it has certain advantages over marsupialization. After enucleation the bone cavity will fills with a blood clot and become the basis and initiator of new bone formation. In cases of large cysts organisation of a blood clot is not possible and hence the formation of new bone. After enucleation of large cysts a blood clot is at great risk, as it can easily become infected and lead to development of inflammation. It can also lead to the formation of a large haematoma, which occurs as a result of uncontrolled accumulation of blood in the bone cavity and its penetration into the soft tissue. That’s why surgeons fill the bone cavities with autogenous, allogenous or xenogenous transplants (grafts). Most frequently used autologenous grafts are bone or cartilage from the crista illiaca, rib, tibia, chin and mandible. The rejection of the implants due to instability, poor blood supply, increased
possibility of infection and the need to open a further operation field and resorption of the autotransplants are possible complication. Allogeneous bone used for bone cavity filling is a human demineralised bone, the antigenicity of which is reduced to a minimum by a process of dry freezing. The most frequent complications connected with allogeneous bone implants are the immunological reaction by rejection of a foreign body, resorption, deformation, HIV or hepatitis infection. Due to their biocompatibility and great porosity, which enables rapid vascularisation and adherence of osteoblasts, xenogenous grafts have osteoconductive ability, i.e. cicatrization of the recipient bone occurs in xenogenous implants. A disadvantage of xenogenous grafts is the possibility of their rejection due to infection.

Enucleation completely removes the cystic capsule from the bone, therefore reducing the possibility of recurrence, and also the possibility of spreading carcinoma from the epithelium of the cystic capsule. Several months are necessary for complete regeneration of the bone, and in the case of large cysts - several years.

In order to improve the marsupialization method and to remove the drawbacks, a modified method was proposed, the so-called dual-phase method according to Hermann. This author developed a technique, the object of which was to speed up postoperative rehabilitation. The first phase consists of the standard Partsch I method and in the second phase, which begins after 12-16 months, the remains of the cystic capsule are removed and the wound primarily closed. Another drawback of the method is the long period of treatment and the need to subject the patient to two operations.

The methods of cyst’s treatment by chemicals were not adequate, mainly because it is impossible to accurately determine and follow up the effect of the chemical on the cystic capsule and surrounding bone tissue.

The clinical characteristic of odontogenic keratocysts is the frequency of recurrence, which occurs within the five-year postoperative period. In order to avoid this various authors have attempted to solve the problem by different methods of treatment. Various methods for the treatment of keratocysts by cryotherapy and application of chemicals (Carnoy's liquid) were suggested. After enucleation of the cystic capsule cryotherapy is performed by freezing the walls of the bone cavity by spraying with liquid nitric oxide (create a temperature of -70°) for a period of one minute each. Frequent postoperative complication was nerve damage – a result of difficulty in determination of the depth of the effect of low temperature in tissue by cryotherapy. A treatment of odontogenic keratocysts by a technique of decompression in the first phase and cystectomy in the second was described. The first phase consists of decompression by means of small polyethylene tubes, which are used for drainage of the cystic fluid. The method of decompression achieves a reduction in the cystic volume, thinning of the cyst walls and regeneration of bone. The second phase consists of cystectomy, which begins when reduction in the cyst volume of 50%-60% has been achieved approximately 9 to 12 months after initial decompression and cyst reduction is followed up every 4 months by an orthopantomogram.
SOFT TISSUE CYSTS
(Petia Pechalova)

Soft tissue cysts represent abnormal epithelium-lined cavities, which are filled with liquid (usually glandular secretions), tissue remnants or cell products. They can arise before birth or postnatally. The most affected areas of the face and neck are lips, floor of the mouth, and salivary glands. In soft tissue cysts the emphasis is on clinical symptoms. Soft tissue cysts are slow-growing, well demarcated, painless, fluctuant swellings of a soft to firmly elastic consistency. It is not uncommon for soft tissue cysts to be associated with sinus tracts (fistulae). Fistulae may also arise in isolation at typical sites. Pain or other inflammation reactions only occur in the event of secondary infection of the cyst.

Aspiration, contrast visualisation procedures, ultrasound, CT, MRI are helpful in the diagnosis of soft tissue cysts. Sialography can be diagnostically useful for cysts of the large salivary glands.

**Epidermoid cysts** are lined with keratinised squamous epithelium and contain a thick keratinic liquid. **Dermoid cysts** contain keratinised squamous epithelium and skin appendages (hair follicles and sebaceous glands) and contain a greasy keratinic liquid with hairs. Both, epidermoid and dermoid cysts arise from epithelial enclosures in the area of the embryonic facial furrows and clefts. They may be present at birth or develop subsequently (rarely also as a result of traumatic displacement of the epithelium downwards). The main localisations of epidermoid and dermoid cysts are the medial line of the floor of the mouth (sublingual dermoid cyst) in the oral cavity and the medial line of the submental area extraoraly (submental dermoid cyst).

Sublingual dermoid cysts are recognisable as rounded, painless swellings of the floor of the mouth. Where the mucosa is thin, a translucent yellowish appearance is seen.

A submental dermoid cyst can give the appearance of a double chin. The rounded cyst is movable along the skin of the throat and surrounding tissue.
Treatment consists of complete extirpation (removal) of the cyst. The surgical approach could be by intraoral incision (for sublingual cysts) or by extraoral way (for submental cysts).

Cysts of the salivary glands arise as a result of obstruction of drainage ("retention cysts") or as extravasation cysts. If sacculation of a salivary gland duct occurs as a result of salivary congestion, this is referred to as a salivary retention cyst or a retention mucocele. These are true cysts with an epithelial lining. Extravasation mucoceles are mucus-containing cavities arising as a result of duct obstruction due to inflammation or trauma (e.g. bite injuries). This obstruction leads to penetration of secretions into the gland interstitium and thus to the formation of a cyst. These cysts are a translucent bluish colour as a result of the covering mucosa and give rise to hemispherical swellings. They may burst, releasing a colourless, mucoid liquid. Circumcision and excision is indicated as a treatment. The edges of the wound are stitched in the area of the mobile soft tissues. On the hard palate, the wound is allowed to heal by open granulation. Unexcised cyst remnants will lead to recurrence.

Ranulae (derived from latin word “rana” means "frog", ranula = "little frog") present an extravasation cysts of the sublingual gland and are located in the mucosa of the anterior part of the floor of the mouth lateral to the lingual frenum. Congenital atresia or obliteration of the duct as a result of recurrent inflammation play a role in pathogenesis. Ranula almost always has a paramedian location or above the mylohyoid muscle in the area of the sublingual plica.

If ranula is extensive, this can give rise to swallowing problems, speech problems and reduced tongue mobility. Ranula could drain spontaneously and then refill (recurrent swelling).

Differential diagnoses include sublingual abscess, obstruction of the submandibular duct by salivary calculi (Wharton's duct), dermoid cysts, lymphangioma, tumours of the sublingual
gland, congenital cystic hygroma of the neck in neonates, haemangioma of the floor of the mouth.

Treatment of ranula is a total extirpation. It is important to avoid damage to the duct of the submandibular gland and the lingual nerve during extirpation, for which reason this intervention should only be performed by experienced surgeons. Larger cysts can also be treated using marsupialisation. In this case the oral cyst is excised together with the covering mucosa. The wound edge of the oral mucosa is stitched to what is left of the cyst.

Median cervical cysts are congenital anomalies arising from remnants of the thyreoglossal duct epithelium. The thyroid gland forms from a diverticulum (median thyroid anlage) located between the anterior and posterior muscle complexes of the tongue at week 3 of gestation. As the embryo grows, the diverticulum is displaced caudally into the neck and fuses with components from the fourth and fifth branchial pouches. The descent continues anterior to or through the hyoid bone with the median anlage elongating into the thyroglossal duct. By weeks 5 to 8 of gestation, the thyroglossal duct obliterates, leaving a proximal remnant, the foramen cecum, at the base of the tongue and a distal remnant, the pyramidal lobe of the thyroid. If the duct fails to obliterate before the formation of the mesodermal anlage of the hyoid bone, it persists as a cyst. Thyroglossal duct remnants occur in approximately 7% of the population, although only a minority of these is ever symptomatic.

Median cervical cyst is a firm, rounded swelling located on the median line of the throat, immediately below and attached to the hyoid bone, from the foramen caecum linguæ to the caudal cervical area. The cyst slides upwards when swallowing or when the tongue is stuck out. One fourth of patients present with a draining sinus that results from spontaneous drainage or surgical drainage of an abscess. This drainage can result in a foul taste in the mouth if the spontaneous drainage occurred by way of the foramen cecum.

One fourth of patients present with a draining sinus that results from spontaneous drainage or surgical drainage of an abscess. This drainage can result in a foul taste in the mouth if the spontaneous drainage occurred by way of the foramen cecum. Median cervical

cysts also fluctuate in size. Other rare presentations can be severe respiratory distress or sudden infant death syndrome from lesions at the base of the tongue, a lateral cystic neck mass, an anterior tongue fistula, or coexistence with branchial anomalies. Two thirds of thyroglossal duct anomalies are diagnosed within the first 3 decades of life, with more than half being identified before age 10 years.

The preoperative evaluation for a patient who has a suspected median cervical cyst includes a complete history and physical examination, preoperative ultrasound, and a screening thyroid stimulating hormone (TSH) level. Patients who have history, examination findings, or elevated TSH levels suggesting hypothyroidism or a solid mass should undergo scintiscanning to rule out an ectopic thyroid. When ectopic thyroid is present, all of the patient’s functional thyroid tissue can be located within the cyst, and its removal would render the patient permanently dependent on thyroid replacement. The management of ectopic thyroid is controversial. Some investigators believe these patients can be treated with exogenous thyroid hormone to suppress the gland, whereas others advocate for resection for reasons that are discussed later. Although ectopic thyroid only occurs in rare of median cervical cyst, some authors advocate for scintiscans in all patients.

Differential diagnoses include enlarged submental lymph nodes, submental abscessus, suprahyoid dermoid and epidermoid cysts, lateral cervical cysts, tumours of the thyreoglossal duct.

A major problem in the surgical treatment of median cervical cysts is the high frequency of recurrence. The required treatment is complete extirpation of the cyst via transverse skin incision, because malignant degeneration has been described. Embryonic considerations indicate an important causal role for the hyoid bone in incidences of recurrences. To avoid recurrence, a partial resection of the central portion of the body of the hyoid bone was provide and the tract is further dissected with a core of tissue from the muscle at the base of the tongue to the foramen cecum.

**Lateral cervical cysts** are developmental abnormalities of the visceral clefts or cystic transformation of epithelium (salivary gland epithelium) enclosed in lymph nodes during embryogenesis have been discussed as possible mechanisms of pathogenesis. The branchial arches are the embryological precursors of the face, neck and pharynx. Branchial anomalies compose approximately 30% of congenital neck masses and can present clinically as cysts, sinus tracts, fistulae or cartilaginous remnants with typical clinical and radiological findings. Cysts are remnants of the cervical sinus without an external opening. Sinuses are the persistence of the cervical sinus with its external opening, whereas a fistula also involves persistence of the branchial groove with breakdown of the branchial membrane resulting in a pharyngocutaneous fistula.

Branchial anomalies usually present in childhood or early adulthood. By the end of the fourth week of gestation, there are four well-defined pairs of arches and two rudimentary arches. These are lined externally by ectoderm, internally by endoderm, with mesoderm in between. The mesoderm contains the dominant artery, nerve, cartilage rod, and muscle for
each arch. Each arch is separated by clefts externally and pouches internally. The branchial arches develop between the fourth and seventh week of gestation and form the embryological precursors of the ear and muscles, blood vessels, bones, cartilage and mucosal lining of the face, neck and pharynx. In total, six pairs of branchial arches form on either side of the pharyngeal foregut in cranio-caudal succession. Each arch transforms throughout gestation into a defined anatomic pattern. Knowledge of this pattern of transformation and its relationship to normal structures in the neck is essential in the diagnosis and treatment of these anomalies.

Branchial anomalies can be lined with either respiratory or squamous epithelium. Cysts are often lined by squamous epithelium, whereas sinuses and fistulae are more likely to be lined with ciliated, columnar epithelium. Lymphoid tissue, sebaceous glands, salivary tissue, or cholesterol crystals in mucoid fluid can also be seen. Squamous cell cancer can be found within branchial lesions in adults, although it is rare. It is difficult to distinguish between a primary lesion arising from within an anomaly and a metastatic lesion from an occult primary.

The evaluation of lateral cervical cysts begins with a complete history and physical, which may include upper airway endoscopy to locate the pharyngeal opening. The pyriform sinus and the tonsillar fossa should be carefully examined. In adults, fine needle aspiration should be performed to rule out metastatic carcinoma or clarify the diagnosis. This clarification is not necessary in children and incisional biopsy should not be performed because this makes the resection more difficult. Ultrasound, CT, and MRI can be used to help define the lesion and its course, but CT is the current study of choice.

Clinically lateral cervical cysts present as circumscribed, flexible, painless, slow growing swelling on the lateral side of the neck, below the mandibular angle in the upper third of the sternocleidomastoid muscle. Differential diagnoses include lymph node disorders, tumours and diseases of the submandibular gland, cervical lymph node metastases.

The definitive treatment of all branchial anomalies is complete surgical excision. Unresected cysts and sinuses have a high risk for infection and incomplete resection results in high rates of recurrence.
1. TMJ Anatomy

The temporomandibular joint (TMJ) or jaw joint is a bi-arthroidal hinge joint that allows the complex movements necessary for eating, swallowing, talking and yawning. Dysfunction of the TMJ can cause severe pain and lifestyle limitation. Temporomandibular disorders are common and sufferers will often seek physiotherapeutic advice and treatment.

**Joint**

**Capsule** - The capsule is a fibrous membrane that surrounds the joint and attaches to the articular eminence, the articular disc and the neck of the mandibular condyle (Sarachev, E., Usunov, N., 2001).

**Articular disc** - The articular disc is a fibrous extension of the capsule that runs between the two articular surfaces of the temporomandibular joint. The disc articulates with the mandibular fossa of the temporal bone above and the condyle of the mandible below. The disc divides the joint into two sections, each with its own synovial membrane. The disc is also attached to the condyle medially and laterally by the collateral ligaments. The anterior disc attaches to the joint capsule and the superior head of the lateral pterygoid. The posterior portion attaches to the mandibular fossa and is referred to as the retrodiscal tissue (Miloro, M. et al., 2004).

**Retrodiscal tissue** - Unlike the disc itself, the retrodiscal tissue is vascular and highly innervated. As a result, the retrodiscal tissue is often a major contributor to the pain of Temporomandibular Disorder (TMD), particularly when there is inflammation or compression within the joint (Langendoen, J. et al., 1997).

![The Temporomandibular Joint](image)

**The ligaments** give passive stability to the TMJ, temporomandibular ligament is the thickened lateral portion of the capsule, and it has two parts, an outer oblique portion and an inner horizontal portion; stylomandibular ligament runs from the styloid process to the angle...
of the mandible; sphenomandibular ligament runs from the spine of the sphenoid bone to the lingula of mandible.

The oto-mandibular ligaments are the discomalleolar ligament (DML), which arises from the malleus (one of the ossicles of the middle ear) and runs to the medial retrodiscal tissue of the TMJ, and the anterior malleolar ligament (AML), which arises from the malleus and connects with the lingula of the mandible via the sphenomandibular ligament (Loughner, B.A. et al, 1989; Rowicki, T.; Zakrzewska, J., 2006). The oto-mandibular ligaments may be implicated in tinnitus associated with TMD. A positive correlation has been found between tinnitus and ipsilateral TMJ disorder (Ren, YF; Isberg, A., 1995; Kuttila, S., et al., 2005). It has been proposed that a TMJ disorder may stretch the DML and AML, thereby affecting middle ear structure equilibrium (Eckerdal, O., 1991; Wright, E.F; Bifano, S.L., 1997; Cheynet, F. et al., 2003; Kim, H.J. et al., 2004). The otic symptoms (tinnitus, otalgia (ear pain), dizziness and hypoacusis) corresponding to altered ossicular spatial relationships (such as conductive middle ear pathologies) can also be produced from masticatory system pathologies (Ramirez, L.M. et al., 2009).
Muscles and Jaw Movement
The jaw can move forward and back, side to side and can open and close. Each of these movements are performed by a number of muscles working together to perform the movement while controlling the position of the condyle within the mandibular fossa. Chewing and talking require a combination of jaw movements in a number of directions (Saladin, K.S, 2005; Standring, S., 2008).

Opening— inferior head of lateral pterygoid, anterior digastric, mylohyoid. Opening is also controlled by eccentric contraction of the closing muscles against gravity. Opening is a complex movement consisting of an early rotary component in the first 2-3cm of movement with a forward glide towards the end of range. The articular disc moves forward with the condyle as it glides forward, effectively extending the superior articular surface of the mandibular fossa.

Closing— masseter, anterior and middle temporalis, medial pterygoid, superior head lateral pterygoid.

Protrusion— bilateral contraction of the lateral pterygoid.

Retrusion— middle and posterior temporalis, possibly helped by deep posterior portion of masseter.

Laterotrusion (side to side) – ipsilateral middle and posterior temporalis, contralateral inferior head lateral pterygoid.

2. Vascularization of TMJ
Predominant vessels are:
Superficial temporal artery – from the posterior;
Middle meningeal artery - from the anterior;
Internal maxillary artery - from the inferior.
Other important arteries are – the deep auricular, anterior tympanic and ascending pharyngeal arteries.
3. Innervation of TMJ

The TMJ is innervated by the **auriculotemporal** and **masseteric** branches of the mandibular nerve (CN V3).

4. Etiology, epidemiology and classification of TMJ disorders

   **Etiology**

   The etiology of the most common TMD is unknown. Two hypotheses, occlusal disharmony and psychological distress, have dominated the literature, but neither has been supported by the literature (Seligman D, Pullinger A., 1991). Research studying discrepancies between centric relation and centric occlusion, nonworking side interferences, and Angle’s classification has not shown a strong association in myofascial-pain patients when compared to controls (Solberg, W., 1972; Greene, C., Marbach, J., 1982; Mohl, N, Ohrbach, R., 1992). Studies of patients with myofascial pain and control subjects have failed to demonstrate significant differences in occlusion although there may be some cases in which occlusal problems are an initiating factor (Clark, N., 1982). A relationship between tooth loss and osteoarthrosis has been found in patient studies but has not been observed in nonpatient studies (Kopp, S., 1977) No difference between a symptomatic and control population was found when attempting to correlate incisal relationships, condylar position, and joint sounds. An observed relationship between severe overbite and TMD symptoms has been reported but has not been consistently observed. Alternatively, there is some experimental evidence to suggest that some observed occlusal changes could be produced by masticatory-muscle pain. Anterior open-bite malocclusion may result from severe TMJ involvement in patients with rheumatoid arthritis.

   The lack of a clear single cause has resulted in the proposal of a multifactorial etiology. These factors may contribute to the initiation, aggravation, and/or perpetuation of the disorder.

   Some of the factors proposed are the following (Blasberg, B., Greenberg, M.S., 2003):

   1. Parafunctional habits (eg, nocturnal bruxing, tooth clenching, lip or cheek biting)
2. Emotional distress
3. Acute trauma from blows or impacts
4. Trauma from hyperextension (eg, dental procedures, oral intubation for general anesthesia, yawning, hyperextension associated with cervical trauma)
5. Instability of maxillomandibular relationships
6. Laxity of the joint
7. Comorbidity of other rheumatic or musculoskeletal disorders
8. Poor general health and an unhealthy lifestyle

The frequency and the importance of these factors as causes are unknown.

Epidemiology: Between 65 and 85% of people in the United States experience some symptoms of TMD during their lives, and approximately 12% experience prolonged pain or disability that results in chronic symptoms (Dworkin Huggins, K.J, LeResche, L. et al., 1990). Although the prevalence of one or more signs of mandibular pain and dysfunction is high in the population, only about 5 to 7% have symptoms severe enough to need treatment. TMD patients are similar to headache and back pain patients with respect to disability, psychosocial profile, and pain intensity, chronicity, and frequency. The lower prevalence of TMD signs and symptoms in older age groups supports the probability that most TMD are self-limiting. Available evidence indicates that TMD are most prevalent between the ages of 20 and 40 years and predominantly affect women. The reason why women make up the majority of patients presenting for treatment is still unclear. In a community-based study, a greater likelihood of developing TMD was found if oral contraceptives were used and, in women over 40 years of age, if estrogen replacement was used.

While the prevalence of TMD is highest in the 20- to 40-year age range, signs and symptoms of masticatory-muscle and joint dysfunction are commonly observed in children. Belfer reported on a group of 40 children, aged 10 to 16 years, presenting with signs and symptoms of TMD; 14 (35%) of the 40 were diagnosed as having acute reactive depression (Belfer, M.,Kaban, L., 1982). In another study, arthrography and computed tomography were performed on 31 pediatric patients complaining of TMJ pain and dysfunction (Katzberg, R., Tallents, R.H,Hayakawa K, et al., 1985). Twelve were diagnosed with disk displacement with reduction, and 17 were found to have disk displacement without reduction. In 12 of the 29 patients with internal derangement, the problem was thought to be due to a previous injury. In a survey of 1,000 12-year-old children, 1% had a maximum mouth opening of less than 40 mm, and few children presented with clinical findings severe enough to warrant treatment (Mohlin B, Pilley J, Shaw W., 1991).

A number of studies have been performed to investigate a possible relationship between orthodontic treatment and the development of TMD, but the results do not support a causal relationship between orthodontic treatment and the subsequent development of TMD (Larson, E, Ronnerman, A., 1981; Sadowsky, C, Polson, A., 1984).

Classification: Due to the uncertainty about etiology, current diagnostic classifications of TMD are based on signs and symptoms. Earlier classifications characterized disorders as
intracapsular (TMJ) or extracapsular (muscle) disorders and were often not versatile enough to allow for multiple diagnoses of masticatory muscle and TMJ abnormalities.

In 1989, Clark and colleagues published a classification system that was useful to the practicing clinician (Blasberg, B., Greenberg, M.S., 2003).

<table>
<thead>
<tr>
<th>Diagnostic Category</th>
<th>Diagnoses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle and facial disorders</td>
<td>Myalgia; muscle contracture; splinting; hypertrophy; spasm; dyskinesia;</td>
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<tr>
<td></td>
<td>forceful jaw closure habit; myositis (bruxism)</td>
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<tr>
<td>TMJ disorders</td>
<td>Disk condyle incoordination; osteoarthritis; disk condyle restriction;</td>
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<tr>
<td></td>
<td>inflammatory polyarthritis; open dislocation; traumatic articular disease;</td>
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<td></td>
<td>arthralgia</td>
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<tr>
<td>Disorder of mandibular mobility</td>
<td>Ankylosis; adhesions (intracapsular); fibrosis of muscular tissue; coronoid</td>
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<td></td>
<td>elongation-hypermobility of TMJ</td>
</tr>
<tr>
<td>Disorders of maxillomandibular</td>
<td>Masticatory-muscle hypertrophy/atrophy; neoplasia (muscle, maxillomandibular or</td>
</tr>
<tr>
<td>growth</td>
<td>condylar); maxillomandibular or condylar hypoplasia/hyperplasia</td>
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The American Academy of Orofacial Pain (AAOP) has published a general classification of disorders that affect the cranial bones, temporomandibular joints, and masticatory muscles. This classification system is useful because it attempts to define the diagnostic terms and provide diagnostic criteria (Blasberg, B., Greenberg, M.S., 2003).

<table>
<thead>
<tr>
<th>Diagnostic Category</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Cranial bones (including the mandible)</td>
<td>Congenital and developmental disorders: aplasia, hypoplasia, hyperplasia, dysplasia (eg, 1st and 2nd branchial arch anomalies, hemifacial microsomia, Pierre Robin syndrome, Treacher Collins syndrome, condylar hyperplasia, prognathism, fibrous dysplasia). Acquired disorders (neoplasia, fracture)</td>
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</table>
| Temporomandibular joint disorders        | Deviation in form

Disk displacement (with reduction; without reduction)

Dislocation

Inflammatory conditions (synovitis, capsulitis) Arthritides (osteoarthritis, osteoarthrosis polyarthritides)

Ankylosis (fibrous, bony)

Neoplasia

Masticatory-muscle disorders               | Myofascial pain

Myositis

Spasm

Protective splinting

Contracture. |
The Research Diagnostic Criteria for Clinical Temporomandibular Disorder Conditions classification was developed for research purposes but is useful in clinical practice for the types of TMD most likely to present to a dentist. The classification does not include the conditions that are less common but still likely to present to clinicians. The RDC/TMD system allows for multiple diagnoses for each individual but only one muscle diagnosis and allows for each joint only one disk disorder diagnosis and one articular bone diagnosis. The terms used are clearly defined, and the criteria required to meet the diagnosis are detailed although the validation of these criteria and the classification system will have to await further research. To allow greater use in the research environment, the criteria do not include diagnostic imaging.

<table>
<thead>
<tr>
<th>Clinical Location</th>
<th>RDC/TMD Diagnoses</th>
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<tr>
<td>Muscle</td>
<td>Myofascial pain</td>
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<td>Myofascial pain with limited opening</td>
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<td>Disk displacement</td>
<td>Disk displacement with reduction</td>
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<td>Disk displacement without reduction, without limited opening</td>
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<td>Articular bone</td>
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<td></td>
<td>Osteoarthritis of the TMJ</td>
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<td>Osteoarthrosis of the TMJ</td>
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RDC/TMD = Research Diagnostic Criteria for temporomandibular disorders

The evaluation of the patient with temporomandibular pain, dysfunction, or both is like that in any other diagnostic workup. This evaluation should include a thorough history, a physical examination of the masticatory system, and problem-focused TMJ radiography.

Interview: The patient’s history may be the most important part of the evaluation because it furnishes clues for the diagnosis. The history begins with the chief complaint, which is a statement of the patient’s reasons for seeking consultation or treatment. The history of the present illness should be comprehensive, including an accurate description of the patient’s symptoms. The following attributes of symptoms should be explored during the interview: location, quality and severity, quantity, timing, setting in which symptoms occur, remitting or exacerbating factors, and associated manifestations (Bickley, L.S, Szilagyi, P.G., 2003).

It is often helpful to have the patient point to the exact location where the symptom occurs, especially if the symptom is pain. Determining the origin of pain is more reliable if the patient points to one specific location, for example, the joint capsule, rather than if the patient circles the entire left side of the face with a finger. Qualitative descriptors may also provide a clue as to the source of the symptom. For example, muscular pain is usually described as “dull” and “achy,” whereas acute joint pain may be “sharp” or “shooting.” The
use of a visual analog pain scale, rating the pain level on a scale from 1 to 10, may also help obtain an understanding of the patient’s perception of the severity of pain. The timing of the patient’s perceived pain is also helpful in determining a cause. Pain that occurs primarily in the morning may indicate a systemic arthritis such as rheumatoid arthritis or myofascial pain resulting from nocturnal bruxism. If pain only occurs toward the end of the day, osteoarthritis may be explored as a potential cause.

**The physical examination** consists of an evaluation of the entire masticatory system. The head and neck should be inspected for soft tissue asymmetry or evidence of muscular hypertrophy. The patient should be observed for signs of jaw clenching or other habits. The masticatory muscles should be examined systematically. The muscles should be palpated for the presence of tenderness, fasciculations, spasm, or trigger points.


The TMJs are examined for tenderness and noise. The location of the joint tenderness (e.g., lateral or posterior) should be noted. If the joint is more painful during different areas of the opening cycle or with different types of functions, this should be recorded. The most common forms of joint noise are clicking (a distinct sound) and crepitus (i.e., scraping or grating sounds). Many joint sounds can be easily heard without special instrumentation or can be felt during palpation of the joint; however, in some cases, auscultation with a stethoscope may allow less obvious joint sounds such as mild crepitus to be appreciated.

![Evaluation of temporomandibular joint for tenderness and noise. Joint is palpated laterally in closed position and open position (Nale, J.C., Tucker M.R.)](image)
The mandibular range of motion should be determined. Normal range of movement of an adult’s mandible is about 45 mm vertically (i.e., interincisally) and 10 mm protrusively and laterally. The normal movement is straight and symmetric. In some cases, tenderness in the joint or muscle areas may prevent opening. The clinician should attempt to ascertain not only the painless voluntary opening but also the maximum opening that can be achieved with gentle digital pressure. In some cases, the patient may appear to have a mechanical obstruction in the joint causing limited opening but with gentle pressure may actually be able to achieve near normal opening. This may suggest muscular rather than intracapsular problems.

The dental evaluation is also important. Odontogenic sources of pain should be eliminated. Teeth should be examined for wear facets, soreness, and mobility, which may be evidence of bruxism. Although the significance of occlusal abnormalities is controversial, the occlusal relationship should be evaluated and documented. Missing teeth should be noted, and dental and skeletal classification should be determined. The clinician should note any centric relation and centric occlusion discrepancy or significant posturing by the patient. The examination findings can be summarized on a TMD evaluation form and included in the patient’s chart (Nale, J.C., Tucker M.R.).

**Radiographic evaluation**

Radiographs of the TMJ are helpful in the diagnosis of intra-articular, osseous, and soft tissue pathologic conditions. The use of radiographs in the evaluation of the patient with TMD should be based on the patient’s signs and symptoms instead of routinely ordering a standard set of radiographs. In many cases, the panoramic radiograph provides adequate information as a screening radiograph in evaluation of TMD. A variety of other radiographic techniques that are available may provide useful information in certain cases.

**Panoramic radiography.** One of the best overall radiographs for screening evaluation of the TMJs is the panoramic radiograph. This technique allows visualization of both TMJs on the same film. Because a panoramic technique provides a tomographic-type view of the TMJ, this can frequently provide a clear assessment of the bony anatomy of the articulating surfaces of the mandibular condyle and glenoid fossa; and other areas such as the coronoid...
process can also be visualized (Blaschke, D.D, White, S.C, 1980). Many machines are equipped to provide special views of the mandible, focusing primarily on the area of the TMJs. These radiographs can often be completed in the open and closed positions.


**Tomograms.** The tomographic technique allows a more detailed view of the TMJ (Blair, G.S., Chalmers, I.M, Leggat T.G, et al., 1973). This technique allows radiographic sectioning of the joint at different levels of the condyle and fossa complex, which provides individual views visualizing the joint in “slices” from the medial pole to the lateral pole. These views eliminate bony superimposition and overlap and provide a relatively clear picture of the bony anatomy of the joint.

Corrected lateral (sagittal) tomograms. A represents lateral image slice, B represents a medial image slice of the same joint. Condyle appears centered in the lateral image and retruded in the medial image. C, Open view showing the degree of condyle translation during mandibular opening.

**Temporomandibular joint arthrography.** This imaging method was the first technique available that allowed visualization (indirect) of the intra-articular disk. Arthrography involves the injection of contrast material into the inferior or superior spaces of a joint, after which the joint is radiographed (Dolwick, M.F., Katzberg, R.W, Helms, C.A., et al., 1979). Evaluation of the configuration of the dye in the joint spaces allows evaluation of the position and morphology of the articular disk. This technique also demonstrates the presence of perforations and adhesions of the disk or its attachments. With the availability of more advanced, less invasive techniques, arthrography is rarely used.
Arthrogram shows dye in inferior and superior joint spaces. Anatomy and location of disk is indirectly interpreted from dye pattern observed after injection of joint spaces above and below disk. This arthrogram demonstrates anterior disk displacement without reduction. Closed position. Open position (Nale, J.C., Tucker M.R.).

**Indications for TMJ Arthrography:**
- Position and function of disk–pain and disfunction – long standing;
- History of locking-persistent;
- Perforations of the disk and retrodiskal tissue;
- Joint dynamics;
- Disk displacement-ant (anteromedial).

**Therapeutic:**
- To delineate loose bodies in the joint spaces;
- Diagnostic aspiration of joint fluid;
- Intraarticular injections of steroids.

**Contraindications:**
- Infections in the preauricular region;
- Patients allergic to contrast media;
- Patients with bleeding disorders and on anticoagulant therapy.

Contrast media – non ionic agents such as iopamidol-370, iohexol-350
**Computed tomography.** Computed tomography (CT) provides a combination of tomographic views of the joint, combined with computer enhancement of hard and soft tissue images. This technique allows evaluation of a variety of hard and soft tissue pathologic conditions in the joint. CT images provide the most accurate radiographic assessment of the bony components of the joint. CT reconstruction capabilities allow images obtained in one plane of space to be reconstructed so that the images can be evaluated from a different view. Thus, evaluation of the joint from a variety of perspectives can be made from a single radiation exposure.

*Computed tomography. Coronal images illustrate normal architecture of the right (R) condyle with alteration of the left condyle resulting from a history of trauma. Axial views depict the altered condylar anatomy referenced against the contralateral joint (Nale, J.C., Tucker M.R.).*

**Cone beam computed tomography.** Cone beam computed tomography (CBCT) has recently become a popular diagnostic tool among dentists and oral-maxillofacial surgeons, mostly because of its convenience, accuracy, and reduced cost. CBCTs are office-based scanners that are capable of providing tomographic views with three-dimensional reconstructions of the mandibular condyle and articular eminence. When evaluating bony structures, it has the diagnostic accuracy of conventional CT scanners but requires much less radiation exposure to patients (Hashimoto, K., Arai, Y., Iwai, K., et al., 2003.; Hintze, H., Wiese, M., Wenzel, A., 2007). The major limitation to the CBCT is that it does not provide diagnostic images of soft tissue structures.

*Cone beam computed tomography (CBCT). CBCT scanner.; Three-dimensional image of a remodeled condyle as a result of a childhood fracture (Nale, J.C., Tucker M.R.).*
**Magnetic resonance imaging.** The most effective diagnostic imaging technique to evaluate TMJ soft tissues is magnetic resonance imaging (MRI) (Manzione, J.V, Katzberg, R.W, Tallents, R.H, et al., 1986). This technique allows excellent images of intra-articular soft tissue, making MRI a valuable technique for evaluating disk morphology and position. MRI images can be obtained showing dynamic joint function in a cinematic fashion, providing valuable information about the anatomic components of the joint during function. The fact that this technique does not use ionizing radiation is a significant advantage.

![Magnetic resonance image. Normal positioning of the articular disk between the articular eminence and condyle during translation. Right image demonstrates anterior disk displacement without reduction, limiting range of motion (Nale, J.C., Tucker M.R.).](image)

**Nuclear imaging.** Nuclear medicine studies involve intravenous injection of technetium-99, a $\gamma$-emitting isotope that is concentrated in areas of active bone metabolism. Approximately 3 hours after injection of the isotope, images are obtained using a gamma camera. Single-photon emission CT images can then be used to determine active areas of bone metabolism (Oesterreich, F.U, Jend-Rossmann, I., Jend, H.H, et al., 1987). This technique is extremely sensitive, the information obtained may be difficult to interpret. Because bone changes such as degeneration may appear identical to repair or regeneration, this technique must be evaluated cautiously and in combination with clinical findings.

![Single-photon emission computed tomography (bone scan). The area of increased activity is apparent in the right temporomandibular joint (Nale, J.C., Tucker M.R.).](image)
5. Arthritis

The term means inflammation of the articular surfaces of the joint:

Infectious arthritis

Infection of the temporomandibular joint (TMJ) may result from direct extension of adjacent infection or hematogenous spread of bloodborne organisms. The area is inflamed, and jaw movement is limited and painful. Local signs of infection associated with evidence of a systemic disease or with an adjacent infection suggest the diagnosis. X-ray results are negative in the early stages but may show bone destruction later. If suppurative arthritis is suspected, the joint is aspirated to confirm the diagnosis and to identify the causative organism. Diagnosis must be made rapidly to prevent permanent joint damage.

Differential diagnosis:
- Specific arthritis – most common are chronic, especially productive inflammation with presence of granulation tissue. Diagnosis is based on isolation of specific species.
- Rheumatoid arthritis - RA is a polyarthritis, typically bilaterally symmetrical.
- Haemarthrosis - bleeding into joint spaces. It is a common feature of Hemophilia (Ugrinov, R., 2006).

Treatment includes antibiotics, proper hydration, pain control, and motion restriction. Parenteral penicillin G is the drug of choice until a specific bacteriologic diagnosis can be made on the basis of culture and sensitivity testing. Categories of NSAIDs include propionic acid derivatives (ibuprofen, naproxen), salicylates (aspirin, difluinusal), and acetic acid compounds (indomethacin, sulindac). These medications can be effective in reducing inflammation in muscles and joints and, in most cases, provide satisfactory pain relief. These drugs are not associated with severe addiction problems, and their use as an analgesic is strongly preferred over narcotic medications. Dosing of NSAIDs is most effective when they are administered on a time-regulated schedule rather than on a pain-dependent schedule. Patients should be instructed to take the medicine regularly, obtaining an adequate blood level that should then be maintained for a minimum of 7 to 14 days. Analgesic medicines for patients with TMJ disorders may range from acetaminophen to potent narcotics. One important principle of treatment for all pain and dysfunction in patients is to remember that the problem may be chronic and that medication could produce long-term addiction. Because of the sedative and depressive effects of narcotics and their potential for addiction, these medications should be restricted to short-term use for episodes of severe, acute pain or in a postoperative setting. In such instances, medications such as acetaminophen with hydrocodone or oxycodone should be sufficient. This medication should not be used for longer than 10 days to 2 weeks, if possible (Nale, J.C., Tucker M.R.).

Muscle relaxants may provide significant improvement in jaw function and relief of masticatory pain through control of dystonia. However, muscle relaxants have a significant potential for depression and sedation and can produce long-term addiction. In many patients with acute pain or exacerbation of muscular hyperactivity, muscle relaxants can be considered.
for short periods such as 10 days to 2 weeks. The lowest effective dose should be used. Diazepam (Valium), carisoprodol (Soma), cyclobenzaprine (Flexeril), and tizanidine (Zanaflex) are examples of commonly used muscle relaxants. Pharmacologic therapy often provides adequate relief of muscular symptoms in patients with TMD.

Antidepressants, most commonly tricyclic antidepressants used in low doses, appear to be useful in the management of patients with chronic pain (Kreisberg, M.K., 1988; Plesh, O., Curtis, D., Levine, J., et al., 2000). Tricyclic antidepressants (Amitriptyline used in small doses 10 to 25 mg at bed time) prevent the reuptake of amine neurotransmitters such as serotonin and norepinephrine, causing an inhibition of pain transmission.

Suppressive infections are aspirated or incised. Once the infection is controlled, passive jaw-opening exercises help prevent scarring and limitation of motion.

6. **Degenerative Joint Disease (Arthrosis, Osteoarthritis)**

DJD includes a variety of anatomic findings, including irregular, perforated, or severely damaged disks.

The mechanisms of TMJ degenerative diseases are not clearly understood but are thought to be multifactorial. Current concepts of DJD incorporate three possible mechanisms of injury:

1. direct mechanical trauma,
2. hypoxia reperfusion injury, and

Mechanical trauma may result from significant and obvious trauma to the joint or much less obvious microtrauma such as excessive mechanical loading. The excessive stress produced in the joint can lead to molecular disruption and the generation of free radicals, with resulting oxidative stress and intracellular damage. Excess loading can also affect local cell populations and reduce the reparative capacity of the joint.

![Arthroscopic visualization of disk perforation with exposure of condyle in superior joint space.](image)

The hypoxia-reperfusion theory suggests that excessive intracapsular hydrostatic pressure within the TMJ may exceed the blood vessel perfusion pressure, resulting in hypoxia. This type of increased intracapsular pressure has been clearly demonstrated in patients during clenching and bruxing (Nitzan, D.W., 1994). When the pressure in the joint is decreased and perfusion is re-established, free radicals are formed. These free radicals may
interact with other substances in the joint (e.g., hemoglobin) to produce even more damage. Neurogenic inflammation results when a variety of substances are released from the peripheral neurons. It is hypothesized that in cases of disk displacement, the compression or stretching of the nerve-rich retrodiskal tissue may result in release of proinflammatory neuropeptides (Holmlund, A., Ekblom, A., Hansson, P., et al., 1991). The release of cytokines results in the release and activation of a variety of substances, including prostaglandins, leukotrienes, and matrix-degrading enzymes. These compounds not only have a role in the disease process but also may serve as biologic markers that may help to diagnose and eventually treat pathologic conditions of the joint (Quinn, J.H, Bazan, N.G., 1991; Israel, H.A, Saed-Nejad, R., Ratliffe, A., 1991). It must be emphasized that it is impossible to predict the progression of pathologic conditions of the joint. In our country Bakardjiev, A., 1992 explained that subjective complaints of degenerative joint disorders are similar to complaints of another TMJ disorders, objective symptoms of degenerative joint disease are characterized by sounds and pain on palpation. According to the same author common etiological factors of degenerative TMJ disorders are pathology in occlusion and dentition (Bakardjiev, A., 1992).

History: Unilateral joint pain that is aggravated by mandibular movements. The pain is often constant but often worsens in late afternoon or evening.

Clinical characteristics – limited mandibular opening, joint pain. Crapitations. TMJ radiographs – structural changes in the subarticular bone of the condyle or fossa – flattening, osteophytes, erosions.

Differential diagnosis:
1. Monoarticular presentation of rheumatoid arthritis;
2. Chronic non-specific arthritis – The mandible often deviates toward the affected joint, chronic inflammatory response, joint hypomobility, constriction to joint capsule.
3. Myofascial pain syndrome – pain disorder, in which unilateral pain is referred from the trigger points in myofascial structures to the muscles of head and neck.

Definitive therapy – decrease the joint loading, anterior positioning appliance therapy, if hyperactivity is suspected then – stabilization appliance.

Occlusal splints are generally considered a part of the reversible or conservative treatment phase in the management of patients with TMD. Splint designs vary; however, most splints can be classified into two distinct groups: (1) autorepositioning splints and (2) anterior repositioning splints.

**Autorepositioning splints.** The autorepositioning splints are also called anterior guidance splints, superior repositioning splints, or muscle splints. These splints are designed to provide a flat surface with even contact in all areas of the occlusion. The splint provides full-arch contact without working or balancing interferences and without ramps or deep
interdigitation that would force the mandible to function in one specific occlusal position (Nale, J.C., Tucker M.R.).

**Anterior repositioning splints.** Anterior repositioning splints are constructed so that an anterior ramping effect forces the mandible to function in a protruded position (Nale, J.C., Tucker M.R.).

Supportive therapy – patient education, fabrication of appliance, pain medication, anti-inflammatory agents, restrict the movements within the painless limits, passive muscle exercises within painless limits. Spray and stretch is an effective method for improving range of motion. The theory behind spray and stretch is the concept that significant superficial skin stimulation can produce an over-riding or distracting effect on pain input that originates in the muscles and joints (Travell, J.G., Simons, D.J., 1983) By spraying a vapocoolant material such as fluoromethane over the lateral surface of the face, the muscles of mastication can be passively or actively stretched with a reduced level of pain.

Friction massage involves the use of firm cutaneous pressure sufficient to produce a temporary degree of ischemia. This ischemia and the resultant hyperemia have been described as a method for inactivation of trigger points, which are areas responsible for pain referred to muscles in the head and neck area. More frequently, this technique may be useful in disrupting small fibrous connective tissue adhesions that may develop within the muscles during healing after surgery and injury or as a result of prolonged muscular shortening from restricted motion.


**Surgical management of arthrosis: meniscectomia** (Atanasov, D., 2011).

7. **Polyarthritides** – in represent a group of arthritis conditions that are less common and present with similar findings as osteoarthritis:
Traumatic arthritis

- Cause: trauma – lead to sudden loss of subarticular bone, which may lead to a change in the occlusal condition – avascular necrosis
- Definitive therapy – not indicated, prevention of future trauma – full mouth protector for sports.

Supportive therapy – rest, restricted use of jaw, soft diet (small bites and slow chewing), NSAID-reduce the inflammation (NSAIDs not only reduce inflammation but also have an excellent analgesic action (Nale, J.C., Tucker M.R.), moist heat, ultrasound (ultrasound is an effective way to produce tissue heating with ultrasonic waves, which alter blood flow and metabolic activity at a deeper level than that provided by simple surface moist heat applications, increase in tissue temperature, circulation, uptake of painful metabolic byproducts (Nale, J.C., Tucker M.R.), stabilization appliance if bruxism is present or if pain increases when teeth are occluded.

7.2. Rheumatoid arthritis

A variety of systemic arthritic conditions are known to affect the TMJ. The most common of these is rheumatoid arthritis. Other processes, such as systemic lupus erythematosus can also affect the TMJ. In these cases, symptoms are rarely isolated to the TMJs, and several other signs and symptoms of arthritis are usually present in other areas of the body. In the case of rheumatoid arthritis, an inflammatory process results in abnormal proliferation of synovial tissue in a so-called pannus formation (Nale, J.C., Tucker M.R.).

TMJ symptoms that result from rheumatoid arthritis may occur at an earlier age than those associated with DJD. As opposed to DJD, which is usually unilateral, rheumatoid arthritis (and other systemic conditions) usually affects the TMJs bilaterally. Radiographic findings of the TMJ initially show erosive changes in the anterior and posterior aspects of the condylar heads. These changes may progress to large eroded areas that leave the appearance of a small, pointed condyle in a large fossa. Eventually, the entire condyle and condylar neck may be destroyed. Destruction of the condyles bilaterally may result in loss of condylar-ramus height, resulting in premature contact of posterior teeth and an anterior open-bite malocclusion. Laboratory tests such as rheumatoid factor and erythrocyte sedimentation rate may be helpful in confirming the diagnosis of rheumatoid arthritis (Nale, J.C., Tucker M.R.).

Three-dimensional reconstruction of a cone beam computed tomography scan showing an anterior open-bite as the result of loss of condylar-ramus height caused by degenerative joint disease (Nale, J.C., Tucker M.R.).

Differential diagnosis:
Diseases such as acute mono- and polyarthritis, chronic polyarthritis:
1. Specific and nonspecific Arthritis;
2. Reiter’s syndrome - form of inflammatory arthritis develops in response to an infection in another part of the body;
3. Felty syndrome characterized by rheumatoid arthritis, hepatomegaly, splenomegaly, positive rheumatoid factor, high erythrocyte sedimentation rate, anemia, thrombocytopenia, leukopenia, neutropenia;
4. Still’s syndrome – juvenile polyarthritis, anemia, leukocytosis;
5. Behçet's disease;
6. Systemic connective tissue disease with joint changes;
7. Gout;
8. Rheumatism.

Definitive therapy – cause is unknown (no definitive therapy)
Supportive therapy – directed towards pain reduction, stabilization appliance sometimes is given – decrease the force on the articular surfaces. Arthrocentesis and arthroscopic procedures may be helpful.
7.3. Hyperuricemia (Gout) – is an arthritic condition in which an increase in the serum urate concentration precipitates urate crystals (i.e. monosodium urate monohydrate) in certain joints. A genetic factor appears to be involved.

Definitive therapy – is directed towards lowering of the serum uric acid levels. Most effective method is by eliminating certain foods from the diet.

Supportive therapy – no supportive therapy exists for gout.

7.4. Psoriatic arthritis – inflammatory condition, psoriatic skin lesions

Therapy – NSAIDs, Gentle physical therapy-to maintain joint mobility is important, moist heat, ultrasound.

7.5. Ankylosing spondylitis – a chronic inflammatory disease of unknown cause primarily affects the vertebral column. It is a painful condition with hypomobile joint with no history of trauma.

Definitive therapy – no definitive treatment.

Supportive treatment – because this is a systemic disorder, a rheumatologist should direct the major treatment. NSAIDs can be helpful, gentle physical therapy. Moist heat, ultrasound.

8. Dislocation and subluxation

In dislocation of the mandible, the condyle is positioned anterior to the articular eminence and can not return to it's normal position without assistance. This disorder contrasts with subluxation, in which the condyle moves anterior to the eminence during wide opening but is able to return to the resting position without manipulation. It has been demonstrated that subluxation is a variation of normal function and that the normal range of motion of the condyle is not limited to the fossa (Blasberg, B., Greenberg, M.S., 2003). Subluxation also called as hypomobility. Patient with subluxation repots of a locking sensation whenever the mouth is opened too widely. During the final stage of maximal mouth opening the condyle can be seen to suddenly jump forward with a “thud” sensation. This is not reported as a subtle clicking sensation.

Definitive treatment of subluxation includes eminectomy which reduces the steepness of the articular eminence and thus decreases the amount of posterior rotation of the disk on the condyle during full translation.

Supportive therapy of subluxation: patient education (restriction of mouth opening); occasionally an intraoral device can be given to restrict the movement. Wearing the device develops a myostatic contracture of the elevator muscles, thus limiting opening to the point of subluxation. The device is worn continuously for 2 months and removed, allowing the contracture to limit opening.

Dislocations of the mandible usually result from muscular incoordination in wide opening during eating or yawning and less commonly from trauma; they may be unilateral or bilateral. The typical complaints of the patient with dislocation are an inability to close the jaws and pain related to muscle spasm. On clinical examination, a deep depression may be
observed in the pretragus region corresponding to the condyle being positioned anterior to the eminence. The condyle can usually be repositioned without the use of muscle relaxants or general anesthetics. If muscle spasms are severe and reduction is difficult, the use of intravenous diazepam (approximately 10 mg) can be beneficial. The practitioner who is repositioning the mandible should stand in front of the seated patient and place his or her thumbs lateral to the mandibular molars on the buccal shelf of bone; the remaining fingers of each hand should be placed under the chin. The condyle is repositioned by a downward and backward movement. This is achieved by simultaneously pressing down on the posterior part of the mandible while raising the chin. As the condyle reaches the height of the eminence, it can usually be guided posteriorly to its normal position.

Postreduction recommendations consist of a decrease in mandibular movement and the use of aspirin or nonsteroidal anti-inflammatory medications to lessen inflammation. The patient should be cautioned not to open wide when eating or yawning because recurrence is common, especially during the period initially after repositioning. Long periods of immobilization are not advised due to the risk of fibrous ankylosis.

Chronic recurring dislocations have been treated with surgical and non-surgical approaches. Injections of sclerosing solutions are not used as often now because of difficulty in controlling the extent of fibrosis and condylar limitation. Various surgical procedures have been advocated for treating recurrent dislocations of the mandible; these include bone grafting to the eminence, lateral pterygoid myotomy, eminence reduction, eminence augmentation with implants, shortening the temporalis tendon by intraoral scarification, plication of the joint capsule, and repositioning of the zygomatic arch.

The position of the mandibular condyle in dislocation, forward of the articular eminence and rotated
Facial asymmetry produced by the dislocation of the left TMJ (Caniniti, M.F., Weinberg, S. 1998).

X-Ray showing a bilateral dislocation of the mandible

Reducing a dislocated jaw
9. Contracture

According to etiology contracture of lower jaw is divided into several types (Ugrinov, R., 2006):

1. Inflammatory – they are more frequently. Arise from inflammatory processes of the tissues surrounding the lower jaw (abscesses, phlegmons, pericoronitis); infection around the temporalis muscle may also produce contracture (extracapsular ankylosis). Patients initially have limitation of opening and deviation to the affected side. In these cases, complete restriction of opening is rare, and limited lateral and protrusive movements can usually be performed (Nale, J.C., Tucker M.R.).

2. Postinjectional – it is due to muscle spasm which is caused when the injection is given into the medial and lateral pterygoid muscles causing tearing of the muscle fibers. The treatment includes hematoma compression, active and passive exercises, therapy with ultrasound, helium-neon laser, hydrocortisone.


4. Jaw and face fractures – trauma to the zygomatic arch area or zygomaticomaxillary complex, fusion of the coronoid process to the zygomatic arch, bony fusion of the upper and lower jaw is a rare. Management – surgical.


6. Tumors of the temporomandibular joint (TMJ), the most common types benign tumors are osteomas. Rarely can be observed osteoid osteoma, chondroblastoma, chondromyxoid fibroma, giant cell lesions. Malignant tumors – osteosarcoma,

7. Others.

Muscle contracture: the clinical shortening of the resting length of a muscle without interfering in its ability to contract further.

Myostatic contracture: it results when a muscle is kept from fully relaxing (i.e. stretching) for a prolonged time. The restriction may be because the full relaxation causes pain in an associated structure. For example, if the mouth can open only 25 mm without pain in the TMJ, the elevator muscles will protectively restrict movement to within this range.

History: the patient reports a long history of restricted jaw movement. It may have begun secondary to pain condition that has now resolved.

Clinical characteristics: Myostatic contracture is characterized by painless limitation of mouth opening.

Definitive treatment: The original cause to be identified and eliminated before effective treatment of the contracture can result. Gradual lengthening of the involved muscles done over a period of many weeks by – passive stretching, resistant opening.

Supportive therapy: When symptoms due occur, analgesics can be helpful and should accompany a decrease in the intensity of exercise program. Thermotherapy and ultrasound are also helpful.

Myofibrotic contracture – it occurs as a result of tissue adhesions within the muscle or its sheath. It commonly follows as myostatic condition or trauma to the muscle.

History: the history for myofibrotic contracture reveals a previous muscle injury or a long restriction in the range of movement. There is no pain complaint. Sometimes the patient will not even be aware of the limited range of opening because it has been present for so long.

Clinical characteristics: It is characterized by painless limitation of mouth opening. Lateral condylar movement is unaffected. Thus if the diagnosis is difficult, radiographs showing limited condylar movement during opening but normal movement during lateral excursions may help. There is no acute malocclusion.

Definitive treatment: The muscle tissue in this condition can relax but the muscle length does not increase. It is therefore permanent. Some elongation of the muscle can be accomplished by continuous elastic traction. This is done by linear growth of the muscle and is slow and limited by the muscle tissue health and adaptability. In general, surgical detachment and reattachment of muscles is done.

Supportive therapy: Rarely.

10. Ankylosis
Ankylosis of the Temporomandibular joint refers to restricted mandibular movements with to deviation to the affected side. This is the pathological fusion of parts of a joint.
Classifications:
Bilateral and unilateral ankylosis;
Fibrous and bony ankylosis;
Intracapsular and extracapsular ankylosis (contracture);
Complete and partial ankylosis;
True or false ankylosis.

Etiology:
Trauma – At birth with forceps, blow to the chin, condylar fracture;
Infections and inflammatory – Rheumatoid arthritis, septic arthritis, otitis media, mastoiditis, parotitis, osteoarthritis;
Systemic disease – Small pox, ankylosing spondylitis, syphilis, typhoid fever, scarlet fever;
Others – Malignancies, post radiology, post surgery, prolonged trismus.

Pathophysiology
Trauma-Extravasation of blood into the joint space-haemarthrosis-Calcification and obliteration of the joint space-Intracapsular and Extracapsular ankylosis

Intracapsular ankylosis.
Intracapsular ankylosis, or fusion of the joint, leads to reduced mandibular opening that ranges from partial reduction in function to complete immobility of the jaw. Intracapsular ankylosis results from a fusion of the condyle, disk, and fossa complex as a result of the formation of fibrous tissue, bone fusion, or a combination of the two. The most common cause of ankylosis involves macrotrauma, most frequently associated with condylar fractures. Other causes of ankylosis include previous surgical treatment that resulted in scarring and, in rare cases, infections. Evaluation of the patient reveals severe restriction of maximal opening, deviation to the affected side, and decreased lateral excursions to the contralateral side. If the ankylosis is the result primarily of fibrous tissue, jaw mobility will be greater than if the ankylosis is a result of bone fusion. Radiographic evaluation reveals irregular articular surfaces of the condyle and fossa, with varying degrees of calcified connection between these articulating surfaces.
Sequelae of TMJ ankylosis: Facial growth distortion; nutritional impairment; respiratory disorders; malocclusion; poor oral hygiene; multiple carious and impacted teeth.

*Differential diagnosis:*


*Treatment*: Purpose of surgical management is:

1. To release ankylosed mass and creation of a gap;
2. Creation of functional joint (improve patient's oral hygiene, nutrition and good speech);
3. To reconstruct the joint and restore the vertical height of the ramus;
4. To prevent recurrence;
5. To restore normal facial growth pattern.

Surgical procedures are:

a) Condylectomy
b) Gap arthroplasty
c) Interpositional arthroplasty

*Condylectomy:*

- Fibrous ankylosis;
- Preauricular incision is made;
- Cut at the level of the condylar neck;
- The condyle should be separated from the superior attachment carefully;
- The wound is then sutured in layers;
- The usually complications of this procedure is an ipsilateral deviation to the affected side and anterior open bite if the procedure was bilaterally.
Gap arthroplasty
- Extensive bony ankylosis;
- Two horizontal osteotomy, removal of bony wedges for creation of a gap between the roof of the glenoid fossa and the ramus of the mandible. The minimum gap should be 1 см to avoid re-ankylosis.

Interpositional arthroplasty – improvement (modification) on Gap arthroplasty.
Materials are used to interpose between the ramus of the mandible and base of the skull to avoid re-ankylosis. The procedure involves the creation of gap, a barrier is inserted between the two surfaces to avoid reoccurrence and to maintain the vertical height of the ramus.
**Materials used in interpositional arthroplasty:**

1. **Autogenous** – Temporalis muscles, temporalis fascia, fascia lata, cartilage graft (costochondral, metatarsal, sternoclavicular, auricular graft), dermis.

2. **Heterogenous** – Chromatised submucosa of pig's bladder, lyophilised bovine cartilage.

3. **Alloplastic** – Metallic (tantalum foil and plate, stainless steel, titanium, gold), nonmetallic (silastic, teflon, acrylic, nylon, ceramic).

Autografts, such as skin, temporalis muscle or fascia lata are considered the material of choice for interposition. Advantages of these flaps in TMJ reconstruction include close proximity to the TMJ without involving an additional surgical site.

**Complications of the surgical management of ankylosis**

1. **Intraoperative:**
   - Haemorrhage (damage of temporal vessels, transverse facial artery, etc);
   - Damage to the external auditory meatus;
   - Damage to the zygomatic and temporal branch of facial nerve;
   - Damage to the auriculotemporal nerve;
   - Damage to the parotid gland;
   - Damage to the teeth.

2. **Postoperative**
   - Infection;
   - Open bite.

**Recurrence of TMJ ankylosis**

- Inadequate gap created between the fragments;
- Fracture of the costochondral graft;
- Inadequate coverage of the glenoid fossa surface;
- Inadequate postoperative physiotherapy;
- Higher osteogenic potential and periostal osteogenic power is responsible for high rate of recurrence in children.
SALIVARY GLAND DISORDERS
(Petia Pechalova)

The salivary glands are divided into two groups: the major glands and the minor glands. All salivary glands develop from the embryonic oral cavity as buds of epithelium that extend into the underlying mesenchymal tissues. These epithelial ingrowths are apparent at 8 weeks’ gestation and then branch to form a primitive ductal system that eventually becomes canalized to provide a structural salivary gland unit for drainage of salivary secretions. The minor salivary glands begin to develop around the 40th day in utero, whereas the larger major glands begin to develop slightly earlier, at about the 35th day in utero. At around the seventh or eighth month in utero, secretory cells called acini begin to develop around the ductal system. The acinar cells of the salivary glands are classified as serous cells, which produce a thin, watery secretion, and mucous cells, which produce a thicker, more viscous mucous secretion. Minor salivary glands are well developed and functional in the newborn infant. The acini of the minor salivary glands produce primarily mucous secretions, although some are made up of serous cells as well, hence the classification of these minor glands as mixed. Between 800 and 1000 minor salivary glands are found throughout the portions of the oral cavity that are covered by mucous membranes, with a few exceptions, such as the anterior third of the hard palate, the attached gingiva, and the dorsal surface of the anterior third of the tongue. The minor salivary glands are located all over the oral mucosa—labial, buccal, palatine, tonsillar (Weber glands), retromolar (Carmalt glands), and lingual glands. The major salivary glands are paired structures and include the parotid, submandibular, and sublingual glands. The parotid glands contain primarily serous acini with few mucous cells. Serous cells are cuboidal cells with eosinophilic secretory granules and produce thin, watery secretions with a low viscosity. The sublingual glands are composed of mucous cells which produce a thick secretion with high viscosity. The submandibular glands are mixed glands, made up of approximately equal numbers of serous and mucous acini and therefore produce a secretion with an intermediate viscosity. The parotid glands, the largest salivary glands, lie superficial to the posterior aspect of the masseter muscles and the ascending rami of the mandible. Peripheral portions of the parotid glands extend to the mastoid process, along the anterior aspect of the sternocleidomastoid muscle, and around the posterior border of the mandible into the pterygomandibular space. The major branches of the facial nerve divide the parotid gland into a superficial lobe and a deep lobe while coursing anteriorly from their exit at the stylomastoid foramen to innervate the muscles of facial expression. Small ducts from various regions of the parotid gland coalesce at the anterosuperior aspect of the parotid gland to form the Stensen duct, which is the major duct of the parotid gland. The Stensen duct is about 1 to 3 mm in diameter and 6 cm in length. The duct traverses anteriorly from the gland hilum and is in a superficial position to the masseter muscle. At the location of the anterior edge of the masseter muscle, the Stensen duct turns sharply in a medial direction, and pierces through the fibers of the buccinator muscle. The Stensen duct opens into the oral cavity.
through the buccal mucosa in the maxillary posterior buccal vestibule, usually adjacent to the maxillary first or second molar. The submandibular glands are located in the submandibular triangle of the neck, which is formed by the anterior belly of the digastric muscle, the posterior belly of the digastric muscle, and the inferior border of the mandible. The posterosuperior portion of the gland curves upward around and above the posterior border of the mylohyoid muscle and gives rise at the hilum to the major duct of the submandibular gland known as the Wharton duct. This duct passes forward along the superior surface of the mylohyoid muscle in the sublingual space, adjacent to the lingual nerve. The Wharton duct is about 5 cm in length, and the diameter of the duct lumen is 2 to 4 mm. The Wharton duct opens into the floor of the mouth via a muscular punctum located close to the incisors at the most anterior aspect of the junction of the lingual frenum and the floor of the mouth. The punctum is a constricted portion of the duct, and it functions to limit retrograde flow of bacteria-laden oral fluids into the ductal system. The sublingual glands are located on the superior surface of the mylohyoid muscle, in the sublingual space, and are separated from the oral cavity by a thin layer of oral mucosa in the floor of the mouth. The main acinar ducts throughout the sublingual glands are called Bartholin ducts and in most instances coalesce to form 8 to 20 Rivinus ducts. These Rivinus ducts are short and small in diameter, and they open individually directly into the anterior floor of the mouth on a crest of mucosa, known as the plica sublingualis; or they open indirectly through connections to the submandibular duct and then into the oral cavity via the Wharton duct.

The functions of saliva are to provide lubrication for speech and mastication, produce enzymes for digestion, and produce compounds with antibacterial properties. The salivary glands produce approximately 1000 to 1500 milliliters of saliva daily, with the highest flow rates occurring during meal times. The relative contributions of each salivary gland to the total daily production varies, with the submandibular gland providing 70%, the parotid gland 25%, the sublingual gland 3% to 4%, and the minor salivary glands contributing only trace amounts of saliva. The saliva production begins to decrease gradually after the age of 20 years because of increased intraparenchymal fibrosis as well as decreased neural secretory stimulation.

Infections of the salivary glands (sialadenitis) could be acute or chronic. Sialadenitis affect more common major salivary glands. The clinical characteristics of acute bacterial salivary gland infections include rapid onset of swelling, with associated erythema and pain. Palpation of the involved gland reveals no flow or elicits a thick, purulent discharge from the orifice of the duct. The cause of acute suppurative sialadenitis of the parotid gland usually involves a change in fluid balance that is likely to occur in older, debilitated, malnourished, or dehydrated patients, or those with chronic illness or significant comorbid disease. In these cases, most gland infections are bilateral. Sialadenitis commonly, but not always, is related to obstructive disease, especially in the submandibular gland. Treatment of bacterial sialadenitis includes symptomatic and supportive care, including intravenous fluid hydration, antibiotics,
and analgesics. Initial empirical antibiotics should include a first-generation cephalosporins or semi-synthetic penicillin (oxacillin or dicloxacillin).

*Acute viral parotitis* (mumps) is an nonsuppurative contagious disease. The causative agent of mumps is specific virus (Myxovirus parotidis). Before the era of routine vaccination against the disease with MMR vaccine (against measles, mumps, and rubella), viral parotitis occurred in epidemics during the winter and spring seasons. It is is recommended to get two doses of MMR vaccine, starting with the first dose at 12 through 15 months of age, and the second dose at 4 through 6 years of age. The clinical differentiation of viral and bacterial salivary gland infections is important because viral infections require different treatment, not including antibiotic therapy. Mumps is characterized by a painful, nonerythematous swelling of one or both parotid glands that begins after incubation period of 2 to 3 weeks. This disease occurs most commonly in children. The signs and symptoms of mumps include preauricular pain and swelling, fever, chills, and headache. The quality of saliva was unchanged, the quantity could be increased. Viral parotitis usually resolves in 5 to 12 days after its onset. Treatment includes supportive care for fever, headache, and malaise with antipyretics, analgesics, and adequate hydration. Complications of the disease include meningitis, pancreatitis, nephritis, orchitis, testicular atrophy, and sterility in approximately 20% of young males affected.

There are two forms of chronic sialadenitis - parenchymatous and interstitial. Complains of the patients with chronic parenchymatous sialadenitis depends from the disease stage. In the beginning, only painless swelling of the involved area and bad breath could observed. The gland edema is a localised and well bordred. The disease usually affects one gland, and lasts by years, gives periodic aggravations. Palpation of the involved gland reveals decreased quantity of saliva with purulence or fibrin clots. The aggravation of the chronic parenchymatous parotitis appears in the autumn usually and is characterized by the stabbing pain during meal in the affected gland, enlargmenet of the edema, and general symptoms - body temperature increase till 38°C. The treatment is difficult even in the initial stage, because the changes of the gland parenchym are irreversible. The aim of treatment is to suspend the disease progressing and its aggravation. It provides with local antibiotics - by the duct, slowly, till the appearance of pressure sense followed by the massage of the gland for evacuation of antibiotics and inflamed contents until a clean saliva was observed, then antibiotic was applied and is left in gland. Such treatment provides daily until the clinical recovery. Another options for local treatment are use of the chymotrypsin solution (splits the fibrous clots, dilute the thickened saliva) and iodine (anti-inflammatory action). Orally could prescribe potassium-iodate solution (from 2% -3% to 10% solution, 1 tablespoon, 3 times per day during 2.5 months). Patients are encouraged to maintain ample salivary flow by using salivary stimulants such as citrus fruits, flavored candies, or at least glycerin swabs. Parenterally could use galantamine (0.5%, 1 ml) or pyrogenal (25 injection by individual scheme). Galvanization of a gland (daily, 30-40 days) or radiation therapy (total doze 5-8 Gr).
could apply in absence of improvement. Surgical treatment (partial or total extirpation) is a method of choice in rare cases, when other options fail.

In **chronic interstitial sialadenitis** interlobular tissues enlarge without destruction of glandular substance, hence the patients complains are from cosmetic discomfort from enlarged salivary gland. The disease affects both glands, without skin changes. During palpation the salivary gland is painless, with soft consistence. During the massage from salivary duct exudes decreased amount of clean saliva. At sialography narrow excretory ducts are observed, poor contoured. Treatment includes radiation therapy (0.6-0.9 Gr daily, total 6 - 8 Gr) and galantamine or surgery.

**Sialodochitis** is a dilation of the salivary duct resulting from epithelial atrophy as a result of repeated inflammatory or infectious processes, with irregular narrowing caused by reparative fibrosis.

Obstructive salivary gland disease (**sialolithisis**) is twice as common in men, with a peak incidence between 30 and 50 years of age. Traditional etiopathogenic factors associated with stone formation are obstruction, reduced salivary flow rate, dehydration, change in salivary pH associated with oropharyngeal inflammation, and impaired crystalloid solubility. Physiologically, microliths may be detected following precipitation in a supersaturated solution of mucous plugs or membrane phospholipids within redundant secretory vesicles. These microliths may become symptomatic and act as a nidus on which successive layers of organic and inorganic substances are deposited. Most recently, a retrograde theory of lithogenesis has been proposed; in this theory, a retrograde migration of food, bacteria, foreign bodies, and debris from the oral cavity enter the ductal system. These small particles then act as the nidus that leads to stone formation, and this process is facilitated by the sphincter actions of the ductal orifices that act to maintain the debris in the ductal system. The incidence of stone formation varies, depending on the specific gland involved. The submandibular gland is involved in 85% of cases, parotid gland in 10%, sublingual gland in 5%. A variety of factors contribute to the higher incidence of submandibular calculi. Salivary gland secretions contain water, electrolytes, urea, ammonia, glucose, fats, proteins, and other substances; in general, parotid secretions are more concentrated than those of the other salivary glands. The main exception is the concentration of calcium, which is about twice as abundant in submandibular saliva as in parotid saliva. The alkaline pH of submandibular saliva may further support stone formation. Several anatomic factors of the submandibular gland and duct are important in the pathogenesis of stone formation: 1) the Wharton duct is the longest salivary duct and saliva has a greater distance to travel before being emptied into the oral cavity; 2) the duct of the submandibular gland has two sharp curves in its course; 3) the punctum of the submandibular duct is smaller than the opening of the Stensen duct and may contribute to the reduction in salivary flow elimination from the Wharton duct. The clinical manifestations of the presence of submandibular stones become apparent when acute ductal obstruction occurs at mealtime, when saliva production is at its maximal and salivary flow is stimulated against a fixed obstruction. The resultant swelling is sudden and usually
very painful (salivary colic). Gradual reduction of the swelling follows, but swelling recurs repeatedly whenever salivary flow is stimulated. This process may continue until complete obstruction, infection, or a combination of both occurs. Obstruction may cause atrophy of the secretory cells of the involved gland. Bimanual palpation of the gland extraorally in the submandibular triangle and intraorally at the posterior floor of the mouth to stimulate salivary flow with simultaneous examination of the duct and its orifice may reveal complete absence of salivary flow or the presence of purulent material at the ductal orifice in cases of added inflammation. The diagnosis may be made clinically and confirmed radiographically by plain films, ultrasonography, CT or CBCT, sialography, or sialoendoscopy. The management of submandibular gland calculi depends on the duration of symptoms, the number of repeated episodes, the size of the stone, and the location of the stone. Submandibular stones are classified as anterior or posterior stones in relation to a transverse line drawn across the mandibular arch between the mandibular first molars. Stones that occur anterior to this line are generally well visualized on a mandibular occlusal radiograph and may be removed intraorally under local anesthesia. A suture is passed around the duct proximal to the stone to prevent propagation of the stone further toward the hilum of the gland. A lacrimal probe can be used to locate the ductal orifice and to estimate the direction of the duct in the floor of the mouth. Following exposure of the Wharton duct, a longitudinal incision is made in the duct directly over the palpable stone, the stone is retrieved, and the ductal lining is sutured to the mucosa of the floor of the mouth. Posterior stones may be located at the hilum of the gland or within the substance of the gland itself. The surgical treatment include removal of submandibular gland. The presence of a parotid stone at the hilum of the parotid gland or within the gland itself may necessitate an extraoral approach to remove the stone and possibly the superficial lobe of the parotid gland. Obstruction of the sublingual gland as a result of stone formation is unusual, but if it occurs, it is usually the result of obstruction of the Wharton duct on the same side of the oral cavity because of the intimate association between the Rivinus and Wharton ducts. Although stone formation is rare in the sublingual and minor salivary glands, the treatment is simple excision of the stone and associated gland. Shock wave lithotripsy is a better alternative to sialoadenectomy.

**Sjögren syndrome** has a variable presentation. The two types of Sjögren syndrome are (1) **primary Sjögren syndrome** (sicca syndrome, characterized by xerostomia (dry mouth) and keratoconjunctivitis sicca (dry eyes) and (2) **secondary Sjögren syndrome**, which is composed of primary Sjögren syndrome and an associated connective tissue disorder, most commonly rheumatoid arthritis. Although the cause of Sjögren syndrome is unknown, a strong autoimmune influence seems to be present. Sjögren syndrome shows a strongly female predilection (9 : 1) and the mean age of 50 years. The first symptoms to appear are arthritic complaints, followed by ocular symptoms, and, late in the disease process, salivary gland symptoms. The involvement of the salivary and lacrimal glands results from a lymphocytic replacement of the normal glandular elements. The xerostomia results from a decreased function of the major and minor salivary glands, with the parotid gland being the most
sensitive. The diagnosis of Sjögren syndrome is suggested by the patient’s complaints and by a variety of abnormal immunologic laboratory tests. The oral component of Sjögren syndrome may be diagnosed by using salivary flow rate studies and sialograms that can show typical acinar destruction. The use of a labial minor salivary gland biopsy is considered highly accurate in establishing the diagnosis of Sjögren syndrome, since the histopathologic changes seen in the minor glands are similar to those in the major glands (parotid). Keratoconjunctivitis sicca is suggested by the patient’s complaints and the Schirmer test for lacrimal flow can be performed by ophthalmologist to quantify the degree of lacrimal flow reduction. Usually, patients with Sjögren syndrome who have keratoconjunctivitis sicca are in the severe category. The treatment for Sjögren syndrome includes symptomatic care with artificial tears for the dry eye symptoms, and salivary substitutes for the dry mouth symptoms. Additionally, the cholinergic medication pilocarpine may be useful to stimulate salivary flow from the little remaining functional salivary gland tissue present.

**Mikulicz disease** is characterized with enlargement of salivary and lacrimal glands. The reasons of the disease are unclear. The proliferation of lymphoid tissue into the glands is observed. The disease begins with the painless swelling of salivary glands, more common the left and right parotid glands, then submandibular, sublingual salivary and lacrimal glands are involved. The disease develops slowly, for years, the edema achieves a significant sizes. The function of gland in the initial and clinical apparent stages is not affected. In early stages, during the palpation, glands are soft. In advance stages glands change the consistency and becomes solid. At sialogram the narrow excretory duct is observed. Treatment includes radiation therapy.

**Diagnostic modalities in salivary gland diseases:** The most important components of diagnosis in salivary gland disorders are anamnesis and clinical examination. Occasionally, the clinician could use imaging studies, functional studies, endoscopic procedures, and biopsy procedures.

1) The primary purpose of *plain radiographs* in the assessment of salivary gland disease is to identify salivary stones. A mandibular occlusal film (Simpson) is most useful for detecting sublingual gland and submandibular duct calculi.
2) An orthopantomogram is useful for visualizing stones in the parotid and submandibular glands.

![Orthopantomogram Image]

3) Sialography is the gold standard in diagnostic salivary glands. Indications include a detection of radiopaque (80%-85%) and radiolucent (15%-20%) stones, diagnosis of reactive-dystrophic processes, chronic inflammations, tumors, trauma. Sialography is contraindicated in acute sialadenitis because of a great risk of worsening of the inflammation and in patients with thyroid gland diseases. In addition, sialography may be used as a therapeutic maneuver because the ductal system is dilated during the study, and small mucous plugs or necrotic debris may be cleared during injection of contrast medium into the ductal system. The sialography technique can be performed under local anesthesia and includes the following steps: (1) cannulation of the salivary duct (Stensen or Wharton duct) with a plastic catheter; (2) injection of approximately 0.5 to 1 mL of contrast medium into the ductal system of the gland before the patient begins to experience pain from ductal distension and retrograde filling of the gland parenchyma; (3) acquisition of a series of radiographic images at various time points during this process. The two types of contrast media available for sialographic studies are water-soluble and oil-based solutions. Both types of contrast material contain relatively high concentrations (25%-40%) of iodine. Oil-based media are poorly eliminated from the ductal system and may cause iatrogenic ductal obstruction following the sialogram. Important information that can be obtained during the sialogram study includes the size, number, position, and mobility of the stones, as well as the diameter of the distal duct and presence of stenosis within the ductal system.

4) Ultrasonography can provide high-resolution images, is noninvasive, has a low cost, and is an easy to perform procedure that allows for accurate evaluation of the parotid and submandibular glands. Ultrasonography represents the most common examination method for nodular lesions and is useful to guide biopsies for diagnostic purposes, especially when clinical examination is limited because of the small sizes and locations of the nodules.

5) Computed tomography (CT) scanning can demonstrate salivary gland calculi, especially submandibular stones that are located posteriorly in the duct, at the hilum of the gland, or
in the substance of the gland itself and can assess the size of mass lesions of the salivary glands. The cone beam computed tomography (CBCT) technology has been evaluated with regard to the diagnosis of sialolithiasis in the major salivary glands.

6) **Magnetic resonance imaging (MRI)** is superior to CT in delineating soft tissue details of salivary gland lesions, specifically tumors or other mass lesions, with no radiation exposure to the patient or the necessity of contrast enhancement.

7) **Salivary scintigraphy (sialoscintigraphy)** uses a radioactive isotope, usually, technetium-99m, injected intravenously, which gets distributed throughout the body and is taken up preferentially by a variety of tissues with an active rate of turnover, including the salivary glands. The major disadvantages of sialoscintigraphy are the patient radiation exposure and the poor resolution of the images obtained. Salivary gland scintigraphy may demonstrate increased uptake of radioactive isotope in an acutely inflamed gland or decreased uptake in a chronically inflamed gland, as well as the presence of a mass lesion, either benign or malignant in nature. The most valuable application of sialoscintigraphy is in the diagnosis of patients with Sjögren syndrome.

8) **Salivary gland endoscopy (Sialoendoscopy)** is a specialized procedure that uses a small video camera (endoscope) with a light at the end of a flexible cannula, which is introduced into the ductal orifice. The endoscope can be used both diagnostically and therapeutically.
TRIGEMINAL NEURALGIA
(Petia Pechalova)

Pain is a complex human psychophysiologic unpleasant experience that influenced by past experiences, cultural behaviors, and emotional and medical states. Neuropathic pains arise from an injured pain transmission or modulation system. When a patient complains of burning or sharp shocklike pain in the face or mouth, pain of neuropathic origin should be included in the differential diagnosis. Trigeminal neuralgia (tic douloureux, painful tic) is a neuropathic facial pain arising from the trigeminal nerve that has specific inclusion criteria. The pain is intense, lasting for brief periods of seconds to minutes, followed by a refractory period during which the pain cannot be reinitiated. At times, a background aching or burning pain is present. Intraorally and extraorally trigger zones exist where nonintensive mechanical stimuli such as slight touch may provoke a painful attack. Common cutaneous trigger zones include the corner of the lips, cheek, ala of the nose, or lateral brow. Any intraoral site may also be a trigger zone for trigeminal neuralgia, including teeth, gingiva, or the tongue. Distribution of trigger zones in the second branch (maxillary nerve) and the third branch of the trigeminal nerve (mandibular nerve) are most common. The pain of trigeminal neuralgia differs from the somatic pains with the lack of a typical graded response to increasing stimulation. If light touch stimulation produces a pain response out of proportion to the stimulus, a neuropathic process should be considered. The pain of trigeminal neuralgia has a burning or electric shocklike quality. Sometimes a background aching pain accompanies trigeminal neuralgia, making it difficult to distinguish from the pain of acute pulpitis or, possibly, periapical periodontitis. Importantly, local anesthetic block of the trigger zone arrests the pain of trigeminal neuralgia for the duration of anesthesia and sometimes longer, which can lead the dentist to mistakenly ascribe a “dental” cause to the pain complaint. Gradual worsening of severity and frequency of attacks over the years are observed in patient with trigeminal neuralgia.

The typical clinical characteristics of neuralgic pain include: severe paroxysmal pain with unilateral location in almost all cases; predominantly affection of right side of the face; mild superficial stimulation provokes pain; most affected dermatomes are V₂ and V₃; patients are frequently pain free between attacks; no neurologic deficits were observed; no dentoalveolar causes were found; application of local anesthesia of trigger zone temporarily arrests pain.

https://www.swedish.org/services/neuroscience-institute/our-services/cerebrovascular-center/conditions-we-treat/trigeminal-neuralgia
The cause of trigeminal neuralgia is not entirely clear, but the consensus is that pressure on the root entry zone of the trigeminal nerve by a vascular loop leads to focal demyelination. This demyelination, in turn, precipitates ectopic or hyperactive discharge of the nerve. The site of demyelination determines the trigeminal division involved and, hence, the clinical presentation. Other diseases such as multiple sclerosis, tumors, and Lyme disease can produce pain similar to that produced by trigeminal neuralgia.

The treatment of trigeminal neuralgia is medical or surgical. Medical treatment is generally undertaken with anticonvulsants. The classic medication is carbamazepine (Tegretol, 400-1200 mg daily), but newer anticonvulsants as gabapentin (Neurontin, 600-3200 mg daily) and oxcarbazepine (Trileptal, 300-2400 mg daily) are commonly used. Tricyclic antidepressants as Amitriptyline (10–300 mg daily) and the antispastic baclofen (Lioresal, 15-80 mg daily) are prescribed also. Many of these medications have significant side effects. Surgical treatment includes microvascular decompression of the offending vascular loop (so-called Janetta procedure), GammaKnife radiosurgery, percutaneous needle thermal rhizotomy, or balloon compression of the root entry zone. Oral surgical procedures for management of trigeminal neuralgia with historical meaning only because of high rate of recurrences and incidence of development of malignancy after them, include neurotomy (cutting of the nerve after its exit from foramen of the facial skeleton), neurectomy (cutting of the nerve with removal of a part of nerve’s tissue after its exit from foramen of the facial skeleton) and neuroexceresi (after surgical exposure of the area of the foramen of the facial skeleton, the nerve was grabbed with hemostat, twisted and pulled for removing as major part of it as possible). For the dentist, the critical issue is recognizing trigeminal neuralgia so that unneeded dental treatment or extractions are avoided and providing an immediately referring to the neurologist.

As with trigeminal neuralgia, any of the cranial nerves with a sensory component appears capable of a neuralgic presentation. The most common of the other cranial nerves to present this way is the glossopharyngeal nerve producing glossopharyngeal neuralgia. The presenting symptom in glossopharyngeal neuralgia is typically sharp, electric shocklike pain on swallowing with a trigger zone in the oropharynx or the base of the tongue. Pain is usually experienced in the throat or tongue, but may be referred to the lower jaw. The facial nerve has a small somatic component on the anterior wall of the external auditory meatus in which shocklike pains are experienced.
FACIAL PARALYSIS
(Petia Pechalova)

The clinical sign of facial paralysis is full loss of facial movement because of facial nerve damage. Facial paresis is characterized with partial loss of facial movement. Usually, paresis is a condition that is easier for recovery compared to paralysis.

Anatomy: The facial nerve (cranial nerve VII) originates in the facial nucleus, which is located at the caudal pontine area. Corticobulbar fibers from the precentral gyrus (frontal lobe) project to the facial nucleus, with most crossing to the contralateral side. As a result, crossed and uncrossed fibers are found in the nucleus. The facial nucleus can be divided into two parts:

(1) the upper part, which receives corticobulbar projections bilaterally and later courses to the upper parts of the face, including the forehead

(2) the lower part, the predominantly crossed projections of which supply innervation to lower facial muscles (stylohyoid; posterior belly of digastric, buccinator, and platysma).

In terms of topography, the facial and intermedius nerves course from the posterior pontine area ventrally, passing through the facial canal together with the vestibulocochlear nerve. All three nerves are surrounded by pia mater through their subarachnoid course, with the pia mater thus becoming a common sheath at the internal auditory canal. The inferior anterior cerebellar artery and venous drainage enter the auditory canal together with the facial nerve. Within the parotid gland, the facial nerve terminates by bifurcating into five motor branches. These innervate the muscles of facial expression:

1. Temporal branch (innervates the frontalis, orbicularis oculi and corrugator supercilii)
2. Zygomatic branch (innervates the orbicularis oculi)
3. Buccal branch (innervates the orbicularis oris, buccinator and zygomaticus muscles)
4. Marginal mandibular branch (innervates the mentalis muscle)
5. Cervical branch (innervates the platysma)

Aik Kah T and Hanom Annuar, 2011
Two facial nerves, the right and the left, control all of the muscles in the face. The right facial nerve controls all of the muscles on the right side and the left facial nerve controls all of the muscles on the left side of the face. The facial nerves emerge from the middle of the brainstem (the pons) and carry motor fibers to the muscles of facial expression. These fibers come from the motor cortex of both cerebral hemispheres. From their origin in the motor strip of the cortex, they can be split into additional fibers that supply muscles in the upper face, including those controlling eye closure and forehead movement, and fibers that supply muscles in the lower face, including the mouth. The fibers that control the lower face travel from the cortex down to the brainstem. In the brainstem, these fibers cross over to the opposite, or contralateral, facial nerve. The fibers that control the upper face take a slightly different path. After travelling down to the brainstem, half of the fibers cross over to the contralateral facial nerve, and half remain on the same side and contribute to the ipsilateral facial nerve. Therefore, the eyes and forehead receive innervation from both hemispheres, while the lower face only receives innervation from the contralateral hemisphere.

The strictly contralateral innervation of the lower half of the face and dual innervation of the upper half of the face is critical when assessing facial weakness. Lesions that damage the motor cortex, such as acute ischemic strokes, will result in contralateral facial weakness of the lower face only, with preservation of the muscles of the upper face on both sides, due to the dual innervation of the upper face. Patients will have a weak smile, but will be able to close their eye tightly and wrinkle their forehead symmetrically. This pattern is often referred to as “central facial weakness,” because it’s caused by injury to the cerebral cortex, which is a part of the central nervous system.

Lesions that damage the facial nerve in the brainstem, or after it exits the brainstem, result in ipsilateral facial weakness involving both the upper and lower face. It doesn’t matter where the innervation is coming from; if the nerve is damaged, all the muscles on that side of the face are weak. These lesions are referred to as “peripheral lesions” because they affect the facial nerve as it exits the brainstem. Patients will be unable to wrinkle their forehead, tightly close their eye, or smile on the affected side. This distinction can aid in localizing the lesion to the appropriate place in the nervous system, thereby narrowing the differential diagnosis.

Facial nerve injury can be complete or partial. Generally, partial disruption of axonoplasmal flow reveals a greater chance of complete functional recovery. Loss of motor function can be observed immediately after facial nerve injury. Depending on the affected trunk and localization (proximal or distal), various patterns of motor function loss can be seen and used for primary diagnosis of the lesion site. Significant muscle fiber decay has been demonstrated when denervation has been present for more than 3 years. Early changes at the cellular level include chromatin changes and increased mitochondria number, deoxyribonucleic acid (DNA), and satellite cells, thus reflecting the plastic state of denervated muscle. In addition to clinical and histopathologic findings, it may also be found that parasympathetic functions such as salivation, lacrimation, and taste sensation are impaired.
**Etiology:** A reasons for development of facial paralysis could be idiopathic, infectious (viral and bacteriology), benign and malignant processes into and near to the facial nerve and trauma.

**Clinical course:** A common entity of facial nerve paralysis is Bell palsy, an acute peripheral facial nerve palsy of unknown etiology, causing rapid onset of facial weakness. Deficits accumulate over hours to days, and reach maximum severity within three weeks. The symptoms may also develop at night while the patient is sleeping, making them seem more acute. Facial weakness typically recovers—partially or fully—within six months. Although Bell’s palsy can affect patients of any age, the median age of onset is 40 years, and it’s more common in patients in their third to fifth decade. As Bell’s palsy affects the facial nerve, it causes facial weakness in a peripheral pattern—that is, weakness involving the mouth, eye and forehead. Specific clinical features include: weakness raising the eyebrow and furrowing the brow; difficulty or inability to close the eye; weakness in grimacing and smiling; and flattening of the nasolabial fold. Although the exact cause of Bell’s palsy is often unknown, infectious causes are thought to contribute in the majority of cases. A viral etiology (ie, herpes simplex virus) has been suspected as a precursor inciting factor. Bacterial infection also may lead to facial nerve paralysis, most often correlated to acute otitis media or externa. Infection with Borrelia burgdorferi via tick bites reveals another etiology of facial paralysis, thereby presenting along with all the symptoms of Lyme disease. Of patients affected with Lyme disease, 10% develop facial paralysis, with 25% of these patients presenting with bilateral palsy. Bell palsy normally has a sudden onset that is often preceded by facial dysesthesia, epiphora, pain, hyperacusis, dysgeusia, and decreased function of the lacrimal gland. Noninfectious causes of facial nerve palsy include head trauma affecting the intracranial intratemporal course of the facial nerve or, less commonly, the infratemporal course, as seen in facial blunt or sharp injury. Iatrogenic injury to the facial nerve most often is seen after surgery of the parotid gland, acoustic neuroma resection, or tumor resection at any point along the course of the facial nerve. Therefore, when facial paralysis occurs after surgery, operative exploration must follow if uncertainty exists concerning the intactness of the facial nerve. Due to topographic relations and/or tumor extension, the facial nerve occasionally must be sacrificed voluntarily as part of sound oncologic management. Tumor of the facial nerve (eg, hemangioma, neuroma) or tumors in the direct vicinity of the facial nerve often are concomitant with facial nerve palsy. In general, gradual onset of paralysis may lead to suspicion of a tumor as the cause. However, several authors have demonstrated a sudden onset of facial nerve palsy in patients with tumors (20-27%). Slow-onset facial nerve palsy is observed in patients with cholesteatoma (a noncancerous lesion with destructive and expanding growth, consisting of keratinizing squamous epithelium, that can develop in the middle ear and mastoid process, trapped within the skull base and that can erode and destroy important structures within the temporal bone).

Ramsay Hunt described a syndromic occurrence of facial paralysis, herpetiform vesicular eruptions, and vestibulocochlear dysfunction. Patients with Ramsay Hunt syndrome generally have a greater risk of hearing loss than do patients with Bell palsy, and the course of disease is more painful. Moreover, a lower recovery rate is observed in these patients. Medical treatment is equivalent to that for Bell palsy; most often, a combination of steroids and antiviral agents is used.
**Diagnosis:** Computed tomography scanning and magnetic resonance imaging are useful in the diagnosis of injury to intratemporal and intracranial affections of the facial nerve, as they may reveal temporal fracture patterns (vertical, transversal, mixed) and edema formation. Under certain circumstances, the facial nerve can be viewed, and swelling or disruption may be seen.

Electrophysiology can be useful to determine the extent of nerve disruption, possible outcome, and treatment options. Most frequently, the minimal and maximal stimulation test (MST) and electroneuronography (ENog) are used. These tests are performed with percutaneous stimulation of the facial nerve. ENog studies are required to determine timing and necessity of surgical intervention (decompression or microneurorrhaphy). Degeneration of 90% or more has been shown to predict poor prognosis without surgical intervention.

Histopathologic changes in the injured facial nerve include those in the distal part of the transected facial nerve and those found in the proximal part of the facial nerve. The distal stump undergoes Wallerian degeneration or anterograde degeneration: Schwann cells reveal massive proliferation, thus taking on a phagocytic role and removing myelin and axonal debris.

For suspected intracranial or infratemporal injury, always perform a Schirmer test of tearing to assess lacrimal gland function.

**Management:** Bell’s palsy is treated with a 10-day course of steroids. In some cases antiviral therapy may also be prescribed. Physical therapy has a special role in treatment plan. While some patients are left with permanent facial paralysis, the majority of patients with Bell’s Palsy experience a complete, or near complete, recovery.

Surgical treatment is limited in cases with loss of muscle tone on the affected side, influenced by severe contraction on the opposite, healthy side. The physician must inform the patient that his face will never be symmetrical or have a normal balance. Surgical options include direct coaptation, interposition nerve grafting, cross-face nerve grafting, and microneuromuscular free tissue transfer. If direct anastomosis of the facial nerve stumps is impossible, use an interposition nerve graft. Donor nerves for this procedure are the ansa hypoglossi, sural nerve, and medial cutaneous antebrachial nerve. Use of these nerves as donor nerves for either interposition grafting or cross-facial nerve grafting is described extensively in the literature. After active motion restoration, balancing and adjustment procedures are performed to give the face the final desired symmetry. These operations are static procedures, thus providing the face with more symmetry and balance at rest. Because of different patient opinions on further operations, these finishing steps should be made following mainly the patients' own desires of symmetry. Examples of these "touch-up" procedures are operations on the depressor anguli oris muscle group, enhancement of the nasolabial fold, and static eye procedures, such as upper eye lifting, static sling placement, and partial cervicofacial rhytidectomy.

Management of synkinesis and hyperkinesis can include botulinum toxin injection. This technique yields good results in the control of these sequelae of reinnervation procedures but must be repeated approximately every 3 months. Usually, 5-10 units are injected initially to control eyebrow spasm, and an additional 10-20 units are injected into the zygomaticus muscle and then repeated with an adapted dose as needed.
PARALYSIS OF HYPOGLOSSAL NERVE
(Petia Pechalova)

Hypoglossal nerve (cranial nerve XII) palsy is uncommon. Dysfunction of the hypoglossal nerve may be a consequence of supranuclear, nuclear, or infranuclear disease. The nuclear and infranuclear hypoglossal nerve can be divided into five segments: the medullary, cisternal, skull base, nasopharyngeal/oropharyngeal Carotid space, and sublingual segments. The hypoglossal nerve exits the medulla oblongata, extends through the skull base, and traverses the suprathyroid neck before ramifying to supply the tongue musculature. The fibers of the hypoglossal nerve arise in the hypoglossal nuclei, which extend through the medulla oblongata in a paramedian location (medullary segment). The fibers course anteriorly, lateral to the medial lemniscus, to exit the medulla in the preolivary sulcus. The rootlets of the hypoglossal nerve lie posterolateral to the vertebral artery within the premedullary cistern (cisternal segment). The rootlets then merge to form the hypoglossal nerve within the hypoglossal (anterior condylar) canal of the occipital bone (skull base segment). Emerging from the hypoglossal canal, the hypoglossal nerve enters the nasopharyngeal carotid space. At this point, the nerve lies deep to the internal jugular vein, internal carotid artery, and glossopharyngeal and vagus nerves. As the nerve passes inferiorly, it comes to lie between the internal carotid artery and internal jugular vein, superficial to the vagus nerve. At the level of the angle of the mandible, the nerve deviates from the path of these other lower cranial nerves. It loops anteriorly around the root of the occipital artery, lying inferior to the posterior belly of the digastric muscle, where it becomes superficial. At the level of the hyoid bone, the nerve crosses the lingual artery and curves anteriorly to run along the surface of the hyoglossus muscle, deep to the mylohyoid sling. This segment of the nerve lies within the sublingual space. As it passes anteriorly, the nerve lies on the surface of the genioglossus muscle before penetrating that muscle. The hypoglossal nerve supplies motor innervation to the intrinsic and extrinsic muscles of the tongue. The action of the hypoglossal nerve is entirely motor. The balanced action of both genioglossus muscles is necessary to protrude the tongue in the midline.

Disorders affecting the function of the hypoglossal nerve lead to imbalanced action of the genioglossus muscles, causing tongue deviation toward the weak side. Damage to this nerve produces characteristic clinical manifestations, of which unilateral atrophy of the tongue musculature is the most important. When left hypoglossal nerve is injured, the tip of the tongue is deviated to the left side – the saide of the lesion.

**Supranuclear disease** affecting the nerve results in paralysis of the tongue contralateral to the side of the lesion. Deviation of the tongue will occur away from the side of the lesion.
Fasciculation and atrophy of the tongue are absent. When disease affects the hypoglossal nerve at the nuclear or infranuclear level, the clinical signs and symptoms are ipsilateral (at the same side). There is deviation of the tongue toward the side of the lesion, with associated atrophy of the intrinsic and extrinsic tongue musculature and fasciculation of the tongue. This constellation may lead to dysarthric speech.

A large group of diseases and conditions of the head and neck region could lead to hypoglossal palsy. Hypoglossal nerve injury within the carotid space may result from a range of disorders, malignant disease being the most common. This includes both primary and nodal squamous cell carcinoma, lymphoma, salivary gland malignancies, and soft-tissue sarcomas. Extranodal metastatic disease from distant primary sites is also encountered, as are benign lesions such as lipomas, paragangliomas, and tumors of neural origin. Vascular disease in the neck can lead to dysfunction of the hypoglossal nerve. The nerve lies close to the vessels of the carotid space. As a result, ectasia and aneurysms of the carotid artery, as well as arterial dissection and jugular thrombosis, can compress the nerve and lead to palsy. Iatrogenic causes include jugular venous puncture and complicated carotid endarterectomy. Rarely, transient hypoglossal nerve palsy may complicate a traumatic delivery. Other abnormalities affecting the hypoglossal nerve within the carotid space include stab or gunshot wounds to the neck, head or neck radiation therapy, and infections originating from surrounding fascial spaces that may spread to involve this space. Malignant tumors are the most common cause of hypoglossal nerve paralysis in sublingual space. Locally invasive squamous cell carcinoma is most often seen, arising from the base or the oral portion of the tongue. Salivary gland tumors occur less commonly but may also lead to palsy of hypoglossal nerve. Odontogenic abscesses involving the sublingual space may also affect the distal portions of the nerve. Temporary palsy of the hypoglossal nerve after extraction of the third molar and after tonsillectomy has been reported.
DENTAL INJURIES
(Dimitar Atanasov)

Etiology: Dental injuries usually arise from an acute single injury - a direct impact on the tooth as a result of a fall, sports, playing, fighting, an injury caused by an animal or a transport accident (bicycle, car, wagon), biting (walnut), chewing hard foods, anesthesia (orotracheal intubation) and tooth extraction. In more rare cases, traumas arise from chronic trauma - in the event of abnormal teeth alignment, abnormalities of teeth and jaws (deep bite), errors in denture treatment (incorrectly selected abutment teeth or incorrect occlusion, high crowns, bridges, partial dentures), errors in endodontic treatment (high dental fillings), improperly designed or constructed dental braces, forced action of splints or braces, harmful habits (biting a pencil, tearing a thread, etc.).

Classification: Dental injuries can be classified as follows (Atanasov, D., 1992):

1. Dental concussion
2. Dental luxation
   - luxation without dislocation
   - luxation with dislocation in oral direction
   - luxation with dislocation in vestibular direction
   - luxation with extrusion
   - luxation with intrusion
   - lateral luxation
3. Dental avulsion
4. Tooth fracture
   - crown fracture
     - crown fracture involving enamel
     - crown fracture involving enamel and dentin, but without direct exposure of the pulp
     - crown fracture involving enamel and dentin, with direct exposure of the pulp
     - fracture of tooth neck
   - root fracture
     - in the cervical third of the root
     - in the middle third of the root
     - in the apical third of the root
5. Combined injuries - with involvement of teeth and jaw bones

Clinical presentation

Dental concussion. In mild trauma, hyperemia occurs in the vessels of periodontal ligament without affecting the pulp, and the condition is reversible. With more severe trauma, the hyperemia encompasses the vessels of the periodontal ligament and the pulp, which is
why blood circulation is disturbed. This is manifested in the pulp, which is located in a cavity with resistant walls, and in a number of cases, the hematoma formed in the apical part of the tooth contributes to that. The crown of the injured tooth changes its colour; at first it becomes pink due to the hemoglobin released. Further, the colour turns brownish or bluish (due to blood pigments hematoidin, methemoglobin and hemin). This indicates the progressive pulp necrosis and tooth devitalisation. In some cases, in the event of a severe injury, the vessels supplying pulp and the periodontal ligament are ruptured. Rapid pulp necrosis and tooth devitalisation occurs. This is clinically manifested with symptoms of traumatic periodontitis - the patient has the feeling that the tooth is elongated and it is painful upon pressure (eating) and percussion. There is mild tenderness upon palpation in the transitional fold of the site corresponding to the root of the injured tooth.

In some patients, the disease develops without pain, with pulp necrosis, formation of mucosal or cutaneous fistulas, or a change in the colour of the tooth - brown or bluish.

**Dental luxation.** Luxation is a permanent pathological displacement of the tooth relative to the tooth socket, accompanied by rupture of adjacent tissues (periodontal ligament, gingiva). The tooth is displaced from its normal position in different directions, most commonly in oral direction, and is partially removed from the tooth socket; more rarely the displacement is in vestibular direction. In some cases, the crown of the luxated tooth is below the level of the adjacent teeth - the tooth is intruded in the spongy part of the alveolar bone (Fig. 510). Patients with luxation complain of pain and change in the normal position of the tooth. The pain is most severe immediately after the injury and then gradually decreases; chewing is difficult. Tooth mobility (grade I, II and III) depends on the type of luxation and condition of the bone alveolar walls. The teeth displaced in vestibular or oral direction have sloped roots, they are mobile, very painful upon touch and percussion. Occlusion is impaired. Mucosa is lacerated and bleeding. The tooth intruded in the tooth socket is stationary, it is below the level of the adjacent teeth, and depending on the extent of intrusion, different part of the crown is visible above the gingiva. Sometimes the tooth is completely intruded in the tooth socket and looks as if it is in the process of eruption.

![Diagram of dental luxation](image)

**Dental avulsion.** It is characterized by a complete removal of the tooth from its socket. The tooth socket is empty and bleeding, or there is a blood clot in it (Fig. 508a). The injured person or his relatives usually carry the avulsed tooth in hands. In some patients, the tooth keeps its contact with the surrounding soft tissues and is positioned transversely to the alveolar ridge, showing the exposed tooth root. Lips and cheek injuries are often found.
Tooth fracture. Clinical symptoms depend on the localization of the fracture and the condition of the tooth - vital, devitalized. In fracture of the tooth crown involving only the enamel or fracture involving the enamel and dentin, but without exposure of the pulp, a crown defect with uneven surface is found. There are no subjective complaints, or patients report an increased sensitivity to mechanical and temperature irritation. In fracture of the tooth crown with exposure of the pulp, there is a strong spontaneous pain, becoming more severe upon minimal mechanical (probing), thermal (cold) and chemical impact.

Eating and speech are disturbed. There is pain upon percussion of the tooth and palpation of the gingiva around it. There is a defect in the crown that is pink in colour (because of the bleeding in it), or pink pulp is seen, looking like a bleeding point. In fracture of tooth neck, the crown is missing or highly mobile. There is tenderness due to irritation of the root pulp.

Root fractures may have different localization (in the cervical third of the root, in the middle third of the root, in the apical third of the root) and direction - transverse, longitudinal, oblique. In fractures in the cervical third of the root, the crown is missing and the root pulp is bleeding heavily, or the crown is supported only by soft tissues. In fractures in the middle third of the root and in the apical third of the root, tooth mobility is lower, often with the preserved vitality of the tooth. There is pain upon palpation and percussion. As the fracture approaches to the tip of the tooth, mobility and dislocation diminish. The apical root fragment does not change its position after the injury.
**Diagnosis:** Medical history should be focused on the circumstances in which the injury occurred - play, sport, falling from height, fighting, etc. Special attention should be paid to the fact whether the patient has lost consciousness, whether first aid has been given, and if yes - where, when, what type. During the objective clinical study, changes should be sought in the integrity of the tooth, its colour, mobility, displacement. When a crown defect is found, it should be specified whether it involves only hard dental tissue, or the pulp is also involved. The surrounding tissues - gingiva and alveolar mucosa - should also be examined. The mobility of the tooth should be investigated very carefully using dental tweezers, instruments or fingers, moving it in the labio-oral, mesio-distal and vertical directions. Clinical examination should be complemented with a paraclinical study, with radiography and electric pulp test (EPT) being of particular value. Intraoral dental x-ray image and, where appropriate, extraoral panoramic films should be obtained.

In dental **concussion**, there are no radiographic changes. In luxation, the following is found: shortened root with extended periodontal ligament space with the tooth displaced in vestibular or oral direction; empty upper part of the tooth socket and extended periodontal ligament space around the root is present when the tooth is partially removed from its socket; tooth in incorrect position and an extended periodontal ligament space is observed when the tooth is rotated around its axis: narrowed or absent periodontal ligament space and root seen far above the adjacent teeth is found when the tooth is intruded in the tooth socket; The tooth socket is empty in dental avulsion.

In **fractures**, radiography shows a fracture line located at different places in the tooth, depending on the location of the fracture. Electric pulp test (EPT) has a very important role for monitoring the condition of the dental pulp after mild injuries and absence of colour changes in the tooth crown. In tooth injuries, after the initial symptoms resolve (12-15 days after the injury), vitality of the tooth should be examined, and if excitability is reduced - 20-60 μA, follow-up should be performed at intervals of 5-6 days.

If the tooth does not respond above 100 μA, this means that the neurovascular bundle is broken, and endodontic treatment is necessary. In other cases, if after repeated studies, vitality is reduced or absent, endodontic treatment should be performed to prevent periapical changes.

**Differential diagnosis:** Differential diagnosis is made between:

- **Dental concussion and dental luxation**

  In concussion, there is pain upon pressure and percussion; the tooth does not change its position in the tooth socket and there are no radiographic changes. In luxation, in addition to the pain upon chewing and percussion, mobility and displacement of the tooth relative to the adjacent teeth are observed, and radiography shows extended periodontal ligament space.
- **Dental concussion and root fracture**
  Sometimes, is clinical presentation is similar, but in fracture, fracture line is present.

- **Dental luxation and root fracture**
  Clinical presentation is similar, with tooth movement, but in fracture radiography shows fracture line.

- **Luxation with intrusion, impacted tooth and dental avulsion**
  In impacted tooth, bone tissue can be seen, and in the case of an intruded tooth, there is no bone tissue above the crown. In avulsion, the tooth socket is empty, while the intruded tooth is located deep in the alveolar bone.

- **Dental luxation and periodontitis**
  Luxation occurs abruptly, while periodontitis has a long evolution. In luxation, one – in most cases frontal - tooth is affected, while periodontitis affects a group of teeth or the entire dentition. There are also radiographic changes.

- **Isolated and combined dental injury**
  In isolated injury, one or several teeth are mobile, but during the study, only the tooth we are examining moves. In a combined injury, when we examine one tooth, a group of teeth move, with bony edges and tenderness upon palpation in the affected bone area. Radiography shows bone fracture.

  *Treatment*: The choice of method of treatment depends on the type of injury and its extent.

  **In concussion** of a primary tooth, its opposing tooth should be reduced in order to ensure rest of the injured tooth. In concussion of a permanent tooth, rest should be provided by eating liquid and semi-liquid food, and temporary dressings (3-5 minutes) with chloramphenicol or tripaflavinum, and mouth washes with warm chamomile or brine (for faster resorption of the hematoma) should be prescribed. In concussion with break of neurovascular bundle and absence of vitality, endodontic treatment should be performed.

  **In luxation**, the tooth should be repositioned and fixed. If the tooth is significantly displaced and mobile, it should be repositioned under local anesthesia using fingers or tooth extraction pliers, while working with attention to prevent damage to the neurovascular bundle. In the case of an intruded tooth in the tooth socket, management depends on whether the tooth is primary or permanent. In primary teeth, treatment is not necessary until the age of 2 years, as the intruded tooth after forming its roots takes a normal position in the dentition. In children at the age of over 2 years and 6 months, with completely formed roots of the primary teeth, the injured tooth should be extracted in order not to damage the permanent tooth germ. After the extraction, a "space-keeper" should be placed. After the age of 5, the primary tooth should be extracted without placing a "space-keeper" (L. Z. Ilina-Malkosyan, 1954).

  When a permanent tooth is intruded in the tooth socket, no treatment is necessary - the tooth itself takes its initial position, and this is fastest when the root of the tooth is not completely formed.

  An intruded tooth should be extracted in the following cases (Chuprinina, N. M., 1978):
  - intruded away from the place of its normal position
- an intruded tooth with periapical changes present before the injury
- primary tooth, intruded in the follicle of a permanent tooth

Various ligatures and splints are used to fix luxated teeth. The basic principle of teeth fixation is to include teeth from two adjacent groups - for example, incisors and canines, canines and premolars, or pre-molars and molars (Roy, I., 1930).

The following splints are used for fixation:

- **Vestibular arch splint of composite resin**
  
  After the tooth (teeth) is (are) repositioned and thoroughly dried, composite resin should be applied to the vestibular surface of the teeth and cured using a light-curing lamp.

- **Interrupted ligature wire splint (Hirschfeld, I., 1940)**
  
  Ligature wire with thickness of 0.3-0.4 mm and length of 15-20 cm passes in vestibular or oral direction around a group of teeth. Separate wire ligatures are passed into the interdental spaces, with one end passing over, and the other end passing under the vestibular and oral arch of the splint. This is followed by turning and tightening the individual ligatures and fixation of the teeth.

- **Continuous ligature wire splint (Atanasov, D., 1986)**
  
  Using a ligature wire, 0.3-0.4 mm, a splint with 5 loops (2 cm long), spaced 1 cm apart from each other, is formed out of the mouth of the patient. After the last loop, the remaining wire is used to form the vestibular arc of the splint. The so shaped splint is inserted into the mouth from lingual (palatal) direction to vestibular direction, with each loop passing into one interdental space. The vestibular arch passes through the loops which should be turned and tightened.
8-shaped ligature wire splint

8-shaped ligature was used to treat luxated teeth in 1532 (Schott, J.), who used wax threads. Stainless ligature wire is used for this purpose today. The splint is made of ligature wire with a diameter of 0.3-0.4 mm and a length of 20-25 cm. After including the distal tooth from vestibular to oral direction, the two ends of the wire are passed into the interdental spaces, including the remaining teeth in vestibular and lingual direction in the form of eighth, fixing the luxated tooth.

Smooth vestibular wire splint

Steel or aluminum wire with a 1-2 mm section is used to form a vestibular arch, and then the teeth are fixed using ordinary wire ligatures, thus fixing the luxated tooth as well.

With a highly luxated tooth, after it has been repositioned, in order to hold to in place in the tooth socket, special wire ligatures are used (see treatment for avulsed teeth). The splints and ligatures remain on the teeth for about 8-12 weeks. During this period, electric pulp tests (EPT) shall be carried out in regular intervals and, in the absence of vitality, endodontic treatment should be performed.

In fracture, treatment depends on the localization and type of the fracture, the condition of the tooth before the injury (vital, devitalized), and the age of the patient. In the fracture of the enamel, sharp edges should be smoothed, and in children, fluoride paste should be rubbed in. In fracture of the crown limited to the dentin, without exposure of the pulp, paste should also be applied and a filling should be placed. In fracture of the crown near the pulp, but without exposure of the pulp, calcium hydroxide dressing and a temporary crown (8-12 months) should be placed until the formation of a secondary dentin or until the formation of the tooth root in children. After this period, an inlay or a permanent crown should be placed. In fracture of the tooth crown with exposure of the pulp, treatment depends on the age of the patient and the time elapsed since the injury. If the patient is under the age of 25 and several hours have elapsed after the injury, biological treatment should be performed. In older patients and in those who have sought a dentist after more than 12 hours, extirpation of the nerve should be performed, and in children with unformed root canals - pulpotomy.

In fracture of the root of a primary tooth, no treatment is necessary since in the physiological second dentition, its roots are absorbed. Its fracture in the cervical third of the root of a permanent tooth, pulp devitalisation should be performed irrespective of the condition of the pulp, followed by root canal treatment and recovery with a pin. In fractures in the middle third of the root and in the apical third of the root, without impairment of the pulp, splint shall be placed in order to ensure rest for 2-3 weeks. In a root fracture with pulp involvement, root canal treatment, filling with phosphate cement and a gutta-percha point is
indicated. In root fractures near the apex it is permissible to fill the canal to the fracture line, as the apical fragment does not cause inflammation in the periapical region. If an inflammation occurs, apicoectomy should be performed.

**In avulsion** of a tooth with a preserved alveolar bone plate, replantation should be performed. Up to the preparation of the tooth socket, the tooth should be kept in the vestibulum in order to be in constant contact with the saliva, or should be placed in milk (Blomlof, L. et al., 1987). Currently, it is recommended to carry out root canal treatment not before, but about 2 weeks after the replantation (Andreasen, J. O., Hjorting-Hansen. E, 1989, Atanasov, Д., 1992), not resecting the apex of the tooth. This, according to the authors, is a prerequisite for greater success rate due to less exposure of the tooth outside its socket, as well as minimal trauma to the periodontal ligament.

Under local anesthesia, curettage of the tooth socket should be performed, with visual inspection of its bone walls and saline wash, the tooth should be placed in its socket and should be fixed using vestibular splints of composite resin or wire ligatures.

For the selection of a splint, the following criteria should be met (Andreasen, J. O., Andreasen. F. M., 1994):

- shaping directly in the mouth should be possible
- the splint should stabilize the impaired tooth in its normal position
- the splint should ensure adequate fixation during the immobilization period
- the splint should not impair the gingiva and should not cause caries
- the splint should allow maintaining adequate oral hygiene
- the splint should not impair occlusion and articulation
- the splint should not interfere with endodontic treatment
- the splint should be aesthetically pleasing
- the splint should allow for secure mobility, supporting the periodontal ligament
- the splint should allow easy removal, without impairing the tooth

Presently, a method of choice for fixation of avulsed teeth is the use of a smooth wire splint with thickness of 1 mm and of composite resins.

When using a ligature to fix an avulsed tooth, a smooth vestibular wire splint should be positioned and fixed in advance. Only the replanted tooth should remain unfixed with ordinary ligatures. It should be fixed with steel wire ligature with a cross section of 0.3-0.4 mm. Shaping the ligature and its fixation by using various methods is different:

○ **Ligature by Limberg, A. A. (1957)**

Ligature wire with a length of 10-15 cm is folded in two and crossed at a distance of 0.5-1.0 cm from the curvature. The width of the curvature is formed to be equal to 1/2 of the width of the tooth masticatory surface. Then the lower edge of the ligature is bent at an angle of 150-160°. The ligature so formed is placed with the curvature on the incisive edge of the tooth, with the free ends of the wire passing from palatal (lingual) direction to vestibular direction in the interdental spaces, and go above the fastened vestibular splint (for the maxilla) and under the splint (for the mandible). Then one free end of the ligature is passed through the
curvature and is pulled until it lies tightly on the vestibular surface of the dental crown. After turning and tightening of the free wire ends, the tooth is fixed in its socket.

- **Ligature by Kolarov, G. A. (1976)**

  Ligature wire with length of 10-15 cm is passed around the splint in vestibular direction of the replanted tooth and is twisted 4-5 times. The two free ends of the wire are curved along the incisive edge of the tooth, one distally, and the other mesially, from vestibular to oral direction. Next, the wires are passed around the tooth neck from oral to vestibular direction above the splint and they are turned to the tooth.

- **Ligature by Sotirov, S. M. (1983)**

  The ligature is formed by two wires 10-15 cm long, which are placed perpendicular to each another, with their middle parts in contact. Then the wires folded and twisted 1-2 times, staying attached to each other. So prepared, the wires are placed on the tooth, so that the place of their contact is on the incisive edge. Orally, both ends of the wire pass laterally to the tooth and go over the wire vestibular splint. The vestibular arms lie on the vestibular surface of the tooth crown and over the splint, make a turn around it and stand at the ends of the first two arms.

Following replantation, follow-up radiography and antibiotic treatment should be prescribed. Instructions on personal oral hygiene should be given. On day 7-12, the sutures that fix the gingival tissue around the tooth should be removed. Endodontic treatment with follow-up radiography should be performed. In replantation of avulsed tooth in the first 2 hours after the injury, the splints should be removed in 14-20 days.
With proper diagnosis and timely treatment, the tooth can be saved after injury. In the case of an avulsed and replanted tooth, the results depend on the condition of the tooth (complete or incomplete root development), as well as on the time from the injury to the treatment. Thus, according to Andreasen, F. M, Jensen. J. D. (1992), teeth replanted in the first 10 minutes after the avulsion survive and the success rate is 95%, and in teeth replanted after 2 hours, success rate is only 5%. The replanted tooth, according to different authors, stays in its socket for 1 to 10 years, on average (Kadankov, D., 1973; Barrett, E. J., Kenny, D. J., 1997), with various complications - non-union of the tooth, root resorption, chronic periapical processes and loss of the tooth.
TRAUMATIC INJURIES IN THE MAXILLOFACIAL AREA
(Dimitar Atanasov)

Traumatic injuries accompany human civilization from the beginning of time. The ancient hunters and gatherers have been battling for their daily survival using primitive hunting tools and in that process they have been sustaining different injuries. The technological advance in modern days introduces even newer traumatic risks to the population. Since the first recorded car crash by Mr. Black in 1889 more people have been killed in traffic accidents than during both World Wars. Human population of the Earth grows with 200 000 each year, while 250 000 people get killed in traffic accidents. Trauma is the third most common cause for death after cardio-vascular pathologies and malign tumors. This is why it is very important that dentists receive knowledge about trauma in maxillofacial region, the systemic response of the organism and the management techniques.

Traumatology reviews and manages the damage to the organism caused by external kinetic, chemical and radiation factors. This pathology is revealed at several interconnected levels: Immediate localized tissue damage; General neurohumoral reaction of the organism that may include shock; Local inflammatory response that is crucial for the healing process.

Characteristics of trauma in maxillofacial region.
Several vital organs are located in the maxillofacial region – brain, trachea, larynx, eyes, nose. Very often traumatic injuries will affect all of them, which would determine the clinical presentation.

Anatomical characteristics
Features of the mandible
The lower jaw is protruding forward, poorly protected, has significant mobility via TMJ, has a number of anatomical weakspots (midline, canines, third molar, mental foramen, neck of the condylar process). This determines the greater incidence of mandibular fractures.

The location of the mandibular canal varies, in some cases it passes low between the roots of the teeth. Its lowest point is in the area of the first and second molar, less than 1 cm from the inferior margin of the mandible. In the saggital plane, the canal is closer to the lingual cortical plate - an average of 3 mm. This is relevant for the diagnosis and treatment of mandibular fractures.

The facial artery along with the facial vein curve over the body of the mandible (deep to platysma), as the anteroinferior angle of the masseter. The maxillary artery is close to the neck of the condylar process. The proximity of those structures to the mandible increases the probability of them getting damaged during traumatic injury or surgical intervention.

The muscles of mastication can influence and guide the displacement of bone fragments and therefore determine the clinical findings and treatment options for mandibular fractures. The digastric (biventer), geniohyoid and mylohyoid muscles pull the mandible downward (open the mouth). The fragments are pulled upward and laterally by the temporal and masseter muscles, while the medial pterygoid muscle pulls them upward and medially. The
lateral pterygoid muscle pulls the mandibular fragments in anterior and medial direction. For example, if a fracture of the mandibular angle occurs, the smaller fragment would be pulled upward and medially due to the prevailing influence of the pterygoid muscles, masseter and temporal muscle, while the larger fragment would be displaced downward by its weight and the pull by the digastric, mylohyoid and geniohyoid muscles.

The close proximity of the mandible to other important anatomical structures such as the tongue, salivary glands, large blood vessels and nerves of the neck, facial nerve can render the treatment of these fractures even more challenging.

Features of the maxilla

Several bones (maxilla, zygoma, orbital bone, lacrimal bone, vomer, palatine bone) make up the midface region, as well as the base of the skull (pterygoid process of sphenoid bone, zygomatic process of frontal bone, ethmoid bone). The maxilla connects via sutures to the adjacent bones of the zygomaticomaxillary complex and base of skull. This is why the maxilla rarely sustains isolated fracture, but is usually damaged together with its neighboring bone structures such as base of skull, which further diversifies the clinical findings in these patients.

The maxilla may be viewed as fulcrum point during mastication and is subjected to considerable occlusal pressure, which determines the formation of specific regions of increased strength (buttresses). The maxillary sinus reduces the strength of the maxilla but also acts as a buffer, protecting the base of the skull in case of severe midfacial fractures. These features as well as the varying distribution of cortical and cancellous bone determine to some degree the different patterns of maxillary fractures (fracture lines).

Maxillary fractures are almost always accompanied by lacerations of the mucosal membrane of nasal cavity, sinus, oral cavity and thus should be reviewed as infected.

Physiological characteristics

Trauma to the maxillofacial region will often affect eating, chewing, swallowing, speech and breathing, as well as CNS. The maxillary sinus and nasal cavity also may have a considerable impact on the clinical symptoms of traumatic injury. Gross fractures often create communications between the nasal cavity, maxillary sinus and intercranial space (for example Le Fort III), which enables infection dissemination to the brain and possible meningitis and meningoencephalitis.

The sensory organs of vision and hearing may be directly or indirectly affected by traumatic injuries, possibly causing severe functional disabilities.

The maxillofacial region has rich innervation (through trigeminal, hypoglossal and facial nerves) and blood perfusion, and is also located in close proximity to the carotid artery and the brain, which renders injuries to this area very painful, with severe neurogenic reactions, possibly including traumatic shock. Damage to the nerves may manifest as intense pain, paresthesia, anesthesia, paresis, paralysis, hyper- and hyposalivation, etc. However, the abundance of nerves enables faster healing, adaptation and compensation of the alterations. The rich perfusion of maxillofacial organs may cause severe bleedings but also is the reason
for faster regeneration processes. Lacerations to the lingual, facial, external carotid, maxillary and sublingual arteries produce life-threatening primary and secondary haemorrhage and possible haemorrhagic shock due to considerable loss of blood over short period of time.

The dentoalveolar system is unique to the maxillofacial region and determines some specific features of traumatic injuries. The presence of teeth (roots) will reduce the strength of the bone and will act as a medium between the bone and the environment, as well as enabling different odontogenic infections (especially from non-vital teeth and teeth that were denuded/luxated in the line of fracture). These aspects impair the healing process. Prosthetic crowns and other constructions, pieces of broken teeth, tartar when aspirated, may produce occlusion of the airways or pneumonia. However, very often teeth can complement the diagnostics and treatment of fractures to the jaws. Missing teeth and changes to the bite (dental occlusion) make the detection of a fracture easier and also helps with its localization.

The teeth serve as supports for the placement and fixation of splints, support the reduction (reposition) and immobilization of the fragments.

The salivary glands are another unique feature of the maxillofacial region. Traumatic injury and laceration of the skin can lead to fistula (sinus tract) formation. Saliva however, has certain antibacterial properties and can support the immune response.

The thin mucosal membranes in the oral cavity are easily ruptured in case of trauma. In maxillofacial fractures, communication with the oral or external environment may result from mucosal tears, perforation through the gingival sulcus and periodontal ligament, communication with sinus linings, and lacerations in the overlying skin. By definition, any jaw fracture within a tooth-bearing segment is an open or compound fracture. This type of fractures, as well as other injuries of the soft tissues and the tongue are always associated with difficulties eating and drinking.

Maxillofacial region’s features such as bountiful blood supply and innervation postulate the enhanced viability and regeneration potential of the tissues here, hence the conservative approach to surgical treatment of trauma (even in gunshot wounds) and extended indications for primary suturing.

Very often trauma to the maxillofacial region is associated with increased accumulation of dental plaque due to difficulties in maintaining good oral hygiene. Additionally, different fermentation processes take place intraorally, fueled and supported by the retention of food debris, which causes halitosis and further contributes to the patient distress.

Gross trauma may produce severe aesthetic defects to the patient, contributing to temporary or permanent psychological disorders and avoidance of social contacts.

**Classification of trauma.**

Depending on the circumstances (etiology) and the environment where trauma is received, the following classification is in order:

- Industrial trauma (factories, agriculture, mining)
- Non-industrial trauma (traffic accidents, sport accidents, domestic accidents)
- Deliberate trauma (military and war-time, surgical, self-inflicted, birth trauma, suicide)

Depending on the affected tissues:
- Soft tissue trauma (open or closed injuries)
- Bone fracture is the maxillofacial area (open or closed fractures)

Closed injuries to the soft tissues (contusions) can be further divided in ecchymosis, suffusion, haematoma, while open injuries (wounds) can be abrasions, lacerated, contused, lacerated, crushed, incised, penetrating and punctured, gunshot or bite wounds.

Basic principles of treatment of maxillofacial injuries include preservation of life and treatment of life-threatening pathology, maintenance of function and restitution of esthetics.

**Emergency medical service or first aid to trauma patients** aims at supplying the brain and heart with oxygenated blood and providing air ventilation to the lungs. This is achieved by:
- Securing passability of airways
- Diagnosing and if possible controlling external bleeding
- Prevention of shock
- Temporary wound dressing
- Temporary immobilization in case of bone fractures
- Transportation of the patient to specialized healthcare facility

Securing passability of airways

Aspiration of blood, mucus, vomit is the most common reason for breathing problems. In maxillofacial trauma and fractures to the mandible the tongue may fall back and obstruct the airflow to the trachea. In such cases occlusion happens in the upper respiratory tract and is relatively easy to treat. Clinician removes the foreign bodies manually from the oral cavity with his hand wrapped in patient’s clothes or sterile gauze. Further inspection and prompt cleanup of the blood, mucus or debris obstructing the nasopharynx is in order, as well as pulling the tongue out and securing it via suture to the patient’s collar if necessary. Patient’s body position is of critical importance for prevention of aspiration of blood, vomit or foreign material. The patient should be placed sideways or prone and not supine, unless trauma to the chest is present. In case of respiration impairment, the mandible should firmly be thrust forward (Esmarch grip) and artificial ventilation (mouth to mouth or mouth to tube) should be provided.

The aforementioned actions may not yield success in case of lower respiratory tract obstruction and cricothyrotomy (also called crike or inferior laryngotomy) is often the last resort to saving the patient’s life. An incision is then made through the skin and cricothyroid membrane to establish a patent airway and a small tube is inserted in the trachea. Cricothyrotomy may also be performed with several large needles placed in the cricothyroid membrane without incision of the skin, but this approach provides limited airflow.

Control and stopping of bleeding

Bleeding or haemorrhaging is the effusion of blood through damaged vascular wall.
Maxillofacial trauma can be associated with primary or secondary bleeding. Primary haemorrhaging occurs immediately after the injury, whereas secondary haemorrhage can happen later because of damage to blood vessels due to sharp fragments, dissolution of thrombus (blood clot), formation of haematoma, etc. Blood escaping the body is designated external bleeding and leakage to internal organs or cavities in cranium, abdomen is deemed internal bleeding.

Depending on the type of affected blood vessels, bleeding can have arterial, venous or capillary source. Blood from arterial source is usually bright red and flows in dense, pulsating stream. These are the most dangerous and are associated with up to six times more intense blood loss than venous bleedings. Reactive vasospasm limits the blood loss from fully severed arteries, whereas partial rupture to the arterial wall facilitates gaping and therefore greater haemorrhage.

Venous bleeding occurs in weaker, non-pulsating stream of darker colour, gradually filling up the wound. Haemorrhage from capillary source presents itself as small droplets of blood covering the wound. It is associated with superficial cutaneous abrasions and trauma to parenchymatous organs such as liver, spleen, kidney.

Control of external hemorrhage is vital to trauma patients. Hemostasis can be spontaneous or artificially achieved. Spontaneous hemostasis occurs naturally.

- The innermost layer (tunica intima) is peeled away and turned inwards. The muscles in the middle layer (tunica media) contract and reduce the lumen of the vessel. The outer layer (tunica adventitia) is drawn in front of the opening and further reduces the lumen of the vessel.
- Blood loss and fall in blood pressure further limits the bleeding.
- Hemostasis is further accelerated by the increase of thrombokinase, which derives from the damaged platelet cells at the site of injury.

Artificial hemostasis can be temporary and permanent. Temporary hemostasis can be achieved by compression dressing of the wound and is effective for bleedings of arterial and venous source. If blood sips through the dressing, additional layers of gauze or cotton should be secured with bandage. Once placed, compression dressing should not be removed before delivery of the patient to the hospital. Hemostatic tamponade is effective in case of small arterial, venous or capillary bleeding. Tamponade can be maintained for up to 48h after which the risk of infection increases. Digital compression can be applied directly to the wound or proximal to it. Proximal digital compression is performed at a distance from the wound and aims for compressing an artery against solid base. Digital compression of the facial artery is applied on the body of the mandible, in front of the masseter muscle; superficial temporal artery can be compressed at the zygomatic arch anteriorly to the ear; the frontal artery - at the supraorbital rim, at supraorbital foramen; common carotid artery can be compressed with 4 fingers at the carotid triangle against the transverse process of 6\(^{th}\) cervical vertebra.

Permanent hemostasis is achieved in the medical establishment through ligature, suture, electrocoagulation, angiotorsio or chemical topical agents.
**Immobilization of the fractured bone**

Temporary immobilization aims at preventing additional displacement of the fragments in a fracture, thus preventing secondary bleeding, pain and potential traumatic shock. Immobilization in case of mandibular fracture can be achieved using a triangular bandage.

**Transportation of the patient**

This part of the first aid procedure complex is vital as it can quite often determine the care provided to the patient subsequently.

**First help in the medical establishment**

Specialized care for the patient is provided by trained medical specialists using specific armamentarium and medication. Definitive wound treatment is performed, as well as hemostasis, blood transfusion if necessary. Cricothyrotomy is replaced with tracheostomy and the patient also receives subcutaneous tetanus booster shot (only for already vaccinated patients) – 1ml for adults, 0.5ml for children.

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**Soft tissue injuries**

**Closed injuries to soft tissues (contusions)**

*Aetiology*: Closed injuries to soft tissues represents specific kind of injury, occurring without laceration of the skin or mucosa. Contusions are usually caused by blunt force – pressure or hits by blunt objects such as wooden stick, stones, etc. The affected region of tissue varies between the dermal papillae, various subdermal structures and the periosteum depending on the force and direction of impact, shape of the object as well as the anatomical region.

*Clinical signs*: Closed soft tissue injuries make up for about 70% of trauma to the face and also about 28% of torso trauma in children.

*Ecchymosis* is represented by bluish, rounded spots or swellings caused by small extravasation of blood in the subdermal tissue. They are soon (up to 1-2 hours) or later (1-2 days) after the trauma.

*Hematoma* is represented by a larger subdermal hemorrhage caused by more significant amount of blood leaving the damaged vessel and creating a cavity.

The following symptoms of soft tissue trauma can be diagnosed:

- Hemorrhage is mandatory symptom for soft tissue injury. Extravasal blood is imbibed the traumatized and neighboring tissues nearby forming large bruises. Larger hemorrhage can split the tissues, blood then travels through the fascial spaces or creates hematomas. Hematomas are either subjected to resorption, or encapsulated by connective tissue, thus forming a traumatic cyst. Sometimes a hematoma may undergo ossification. Hematoma is initially dark red and changes its color over time to blue, green, yellow as resorption progresses.
- Swelling is better defined in regions with lots of loose connective tissue, such as the face. Swelling is most often observed in infraorbital and buccal spaces.
- Moderate pain at rest is enhanced by palpation and pressure and is usually present but passes quickly in small contusions. Mauling of tissues and massive bleeding elicit long-lasting pain.
- Depending on localization and pain the function of affected organ can be impaired.

**Diagnosis:** Diagnosis is based on history of trauma and the clinical signs.

**Differential diagnosis** should be made between hematoma and:
- Subperiosteal fracture. Common findings are hemorrhage, swelling, pain and impaired function. Differential can be established in radiography.
- Traumatic aneurysm of the facial artery. Common findings are hemorrhage, swelling, pain and impaired function. Differential is systolic pulsations of the swelling and burning pain across the face.

**Treatment:** Treatment in the dental office aims at reducing the internal hemorrhage or resorption of the hematoma. Packs of ice are applied over 20-30 min intervals during the first 24 hours. Application of heat in form of warm compress or ultra-high frequency procedures can accelerate the resorption after 48th hour. Hospital treatment is indicated for sever trauma patients with substantial damage to the soft tissues and possible blunt force trauma to internal organs or cranium.

Drainage is performed only if the hematoma fails to resorb after 7-10 of unsuccessful conservative treatment. After the removal of blood and blood clots antibiotic is administrated locally and compression bandage is placed. Suppuration calls for incision to the affected area and antibiotic treatment.

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**Open injuries to soft tissues (wounds)**

**Aetiology:** There are numerous types of open injuries to soft tissues depending on the circumstances and the surroundings – accidental, war-time, deliberate trauma.

**Classification:** Wounds in the oral cavity and on the face can be divided according to:
1. Surroundings and circumstances in which the injury is sustained.
   - Accidental
   - War-time
   - Iatrogenic during surgery
2. Type of injury and mechanism of damaging factor
   - Incised and slashed wounds
   - Penetrating and punctured wounds
   - Lacerated wounds
   - Contused lacerated wounds
- Gunshot injuries
- Bite wounds

All wounds excluding the iatrogenic intra-operative surgical incisions should be deemed infected.

Clinical signs: The clinical signs of every wound include:
- Pain of variable characteristics and intensity depending on the amount of damaged superficial, dermal or deeper nerve fibers. Superficial pain is sharp and lasts for a short time.
- Gaping of the wound depends of its shape, size, depth in relation to the elastic fibers of the dermis, which together with the mimic muscles determine the Langer lines. Injuries that are perpendicular to the Langer lines and the muscles will gape the most.
- Bleeding is affected by the type and size of severed blood vessels.

The clinical representation of any wound is determined by its type, borders, amount of damaged tissues and surrounding tissues.

Incised wounds have smooth borders, sharp angles and are usually linear. They can be inflicted by sharp objects, which cut the tissues without much pressure, this is why concussion or damage to the surrounding tissues is not present. Incised and slashed wounds gape and bleed profusely. Sharp objects cutting through the tissues tangentially (obliquely) will produce a flap instead of gaping wound. Surgical incisions are type of incised wounds that are deemed sterile.

Penetrating and punctured wounds appear small from the outside and can have minute diameter but reach deeply below the skin surface, possibly damaging internal organs and large vessels, which potentially causes lethal bleeding. The canal left by the penetrating object heals quickly, which can obstruct the drainage of inflammation exudation. Infection material left inside the tissues may cause suppuration. Penetrating and punctured wounds can be inflicted by needles, spear heads, nails, spikes, etc.

Lacerated wounds have irregular shape, lacerated and uneven margins and do not bleed profusely. This type of injuries result from excessive mechanical forces and sharp objects (The patient may step on a nail and dragging his/her sole, creating a tearing in the tissues. Dog bites can also be explained with the same mechanism).

Contused lacerated wounds are a combination between contused and lacerated wounds featuring linear or squashed margins, variable depth, bleeding in relation to the direction and amount of force. Such wounds are usually significantly contaminated and infected. Contused tissues surround the area determining its blueish or pale color, necrotic material may be visible at the borders. Severely contused tissues bleed scarcely because of crushed vessels, but are highly susceptible to infection.

Open crushed wounds (conquasatio aperta) lack bleeding because of crushed thrombosed vessels, but increase the risk of traumatic shock or gangrene.

Bite wounds feature lacerated and crushed borders, as well as hemorrhagic areas in the surrounding tissues while external bleeding is scarce. Such wounds may display the clinical
signs of incised, punctured, lacerated or contused lacerated wound. Bite wounds are considered highly contaminated and the potential for infection is substantial.

- Insect bite wounds (vulnus morsum insectibus) – type of punctured wound, externally representing a small dot. The pain is moderate to strong and there also is risk of allergic reaction.
- Snake bite wounds (vulnus morsum serpentibus) cause swelling, pain and internal bleeding.
- Horse bite wounds (vulnus morsum quinus) represent variant of contused lacerated wound.
- Pig bite wounds (vulnus morsum suillus) may look like lacerated wound but some tissue may be ingested by the animal
- Dog bite wounds (vulnus morsum caninus) may be deemed lacerated or contused lacerated wounds
- Human bite wounds (vulnus morsum humanus) – unfortunately this kind of injury is seen rather often in the recent years.

**Gunshot wounds** is similar to punctured and penetrating injury but can also display the characteristics of contused lacerated, contused or perforating wound (the projectile produces another exit wound). Bleeding is scarce apart from the occasions when a major blood vessel is damaged. Secondary arterial hemorrhage occur often and are potentially dangerous. Gunshot wounds feature 3 areas:

a) Area of destruction represented by the canal, which the projectile created in the tissue. It often contains necrotic material, blood and blood clots, foreign bodies, parts of the projectile.

b) Area of irreversible damage in which the tissue necrotize shortly after the trauma. Infection and foreign bodies are usually present here too.

c) Area of molecular concussion due to the kinetic energy transferred to surrounding tissues. Multiple capillary hemorrhages are present here but the alteration is reversible if infection is prevented.

Injuries from explosives are associated with excessive damage and avulsion of tissues and organs.

**Diagnosis:** Determining the diagnosis is usually easy.

**Differential diagnosis:** Differentiation between wounds and open fractures is done based on the clinical and radiographic findings.

**Treatment** of the wounds depends on the type and localization of injury, general status of the patient. Surgical management of wounds aims at reestablishment of form and function, as well as optimal esthetic result.

- Deep wounds that reach subdermal adipose tissue and muscles

These injuries can be treated in the dental office. Insect bite wounds are complemented with i.v. antihistamine agent such as Allergosan or 10% Calcium gluconate solution and
corticosteroids such as Urbason, Hydrocortisone. Snake bite calls for anti snake venom, where as dog bites require rabies vaccine. All other patients should receive tetanus booster shot.

Superficial abrasions are anesthetized and irrigated with 3% hydrogen peroxide or Ethacridine lactate (Rivanol). Surrounding skins is then cleansed with ethanol and the wound is bandaged. Deeper wounds should also be cleansed and debrided so that bacterial contamination, foreign bodies and unviable tissues are limited and removed. Diluted local anesthetic solutions of 0.5-1% are injected through the wound, which prevents penetration and damage to unaffected tissues nearby. Surgical management of wound is immediate (first 24h), early (up to 48h post injury) or late (after 48th hour), depending on when the patient receives medical attention.

Gunshot wounds should be treated by a maxillofacial specialist in a single and definitive procedure in the first 24h post injury or up to 4th day. Dead tissues are carefully and frugally excised in consideration to the greater regeneration potential of maxillofacial region supported by the rich blood supply and innervation. Foreign bodies are removed if they are visible inside the wound.

- Soft tissue wounds in combination with damage to nerve and large blood vessels should be treated in hospital by maxillofacial surgeon.
- Concomitant wounds with trauma to internal organs, brain and bone fractures should be immediately addressed in hospital with alleviating the life-threatening symptoms (profuse bleeding, shock) as a main concern.

**Fractures of the mandible**

Fractures of the mandible affect patients of various age, but predominantly males (males:females=5:1). Such fractures are prevalent in young male population aged 20-29, followed by patients of both genders aged 30-39.

**Aetiology:** Fractures in maxillofacial region make for up to 3% of total fracture morbidity and mandibular fractures are the most common of them (70-75%). Patients can suffer such trauma in various environment:

- Industry (9%) – manufacturing, agriculture, mining
- Non-industrial (89%) – domestic, traffic accidents, sports
- Deliberate (2%) – suicide, war-time, traffic accidents, sports

Mandibular fractures in Bulgaria are mostly caused be domestic violence (68.07%), followed by traffic accidents (13.19%) and falls (8.57%). 45% of all cases are concomitant to alcohol use.

**Classification:** Classification can be done according to large number of criteria.

1. Depending on aetiological factor
- Traumatic external force – kinetic, thermal, chemical, multi-factorial force
- Spontaneous (pathological) – caused by previously available pathology such as tumor, cyst, osteomyelitis

2. Depending on mechanism
- Directly in the place where force is applied. Such fractures can have linear shape and are caused by kick, baseball bat, etc. Crushed fractures happen when substantial force is applied to larger area (fall from height). Penetrating fractures may be caused by metal objects such as high velocity projectiles.
- Indirect fractures, which are manifested away from the place of application of the external force. They result from vertical compression, traction, rotation, bending. A good example for such fracture would be a blow to the mental region, which would cause condylar fracture.

3. Depending on the number of fragments and character of fracture.
- incomplete – only affecting the cortical bone plate
- simple (single) – has two segments
- comminuted (multiple) – the fractured bone is left in at least three separate segments
- fragmented – multiple fragments are identified

4. Depending on the dislocation of the fragments and the muscle pull proximal and distal to the fracture. In a favorable fracture, the fracture line and the muscle pull resist displacement of the fracture. In an unfavorable fracture, the muscle pull results in displacement of the fractured segments. The fragments may be dislocated at length (which produce shortening or lengthening), at an angle, sideways, or rotated.

5. Depending on the relation of the fracture to the adjacent soft tissues
- closed fractures – without violation of integrity of the soft tissues
- open fractures (compound fractures) – presented by damage to the adjacent soft tissues, such as skin or mucosal membrane. Such fractures may also be avulsed – with missing tissues.

6. Depending on the occlusion the fractures may or may not violate the occlusion of the dental arches. Special consideration should be advised to edentulous jaws.

7. Combined fractures to both jawbones or fractures to one jaw and another bone in maxillofacial region – mandible and zygoma.

8. Traumatic brain injury and co-occurring jawbone fracture.

9. Depending on localization in the mandible, fractures are designated as occurring in the condylar, ramus, angle, body, symphyseal, alveolar, and coronoid process areas.

Clinical signs: Multitude of symptoms can be observed in patients with mandibular fractures. The symptoms can be divided in two groups:

- Pathognomonic (main) symptoms are exhibited mandatorily by all patients with fractures. Such symptoms are violations of integrity of the bone, pathological mobility of bone/bone fragments, bone crepitus or crunching
Secondary symptoms are exhibited facultatively in some patients. Such symptoms may be pain, facial deformity, swelling, occlusal violations, lost or impaired function.

Patients may complain of swelling or bruise in maxillofacial region, pain, oral or nasal bleeding, reduced opening of the mouth, difficulties eating, impaired speech, breathing or vision. Recollections for loss of consciousness and received medical help deserve special attention. Subjective complaints vary according to localization of the fracture and damage to adjacent tissues and organs.

Clinical evaluation aims at determining main and secondary symptoms, with emphasis on configuration of the face, presence of contusions and wounds. Intraoral evaluation begins with inspection of mouth opening, dental occlusion and the status of intraoral tissues and organs. The main symptoms of violation of bone integrity and crepitus are demonstrated via palpation, whereas bimanual examination helps to determine pathological mobility.

The presence of main symptoms positively concurs jawbone fracture, whereas secondary symptom prove to insufficient for such diagnosis, because they can be seen in association with other pathology. Inflammatory and tumorous processes can display pain and swelling, and tooth mobility is diagnosed in number of conditions such as osteomyelitis, periodontitis, cysts, tumors, etc. Establishing both main and secondary symptoms together supports the right diagnosis.

Diagnosis of bone fracture is based on patient’s history and proper clinical evaluation, complemented by paraclinical tests.

Anamnestic data is vital for diagnostics and includes information about the time and place of trauma, as well as patient’s subjective complaints, which however can be exaggerated or played down. The physician should interrogate the patient about important details and not allow him to overlook something. For example, the parents of pediatric patient with trauma may be overly concerned with a wound on the mentum and may overlook essential information about impaired movement of the lower jaw. Reduced mouth opening in such patient may be indicative of condylar fracture, which, if left untreated, can produce ankylosis.

The physician should assess the presence and characteristics of pain – intensity, localization, duration, propagation, as well as nausea or vomiting. Other clinical findings may include bleeding from the nose, ears or mouth, occlusal defects. Auditory or visual complaints deserve special attention.

It should be determined whether the patient had received first aid previously, and if so by whom, where and when. Temporary immobilization of the fragments would help mitigating with pain mitigation and dislocation of fragments and would also prevent secondary bleeding. The general status of the patient should also be established given the patient’s history and medical journals. State of consciousness, respiration function and hemorrhage are of utmost importance.

Visual inspection of the mucosal membranes, teeth and alveolar ridges can help identify secondary symptoms of fracture, such as

- Facial deformities, swelling, change in normal symmetry
- Edema
- Hematomas and ecchymosis
- Abrasions, lacerations and other possible wounds
- Reduced and painful opening of the mouth
- Step deformity in dental arch or any form of malocclusion such as premature occlusion or open bite.
- Dentoalveolar injuries, luxated or avulsed teeth, fractures of crowns, etc.
- Paresthesia or anesthesia of inferior alveolar nerve
- Deviation in TMJ

After the inspection clinical evaluation utilizes palpation, which is vital for determining the main symptoms of fracture. Extraoral palpation is performed on both facial sides with the thumbs of both hands. Inferior margin of the mandible, the body, the angle, the symphysal area and the alveolar process should be felt. Any step deformity, bone crepitus or violation of the integrity of the jaw should alert the physician. Bimanual palpation can prove fractures in the body of the mandible. The thumbs of both hands are place on the occlusal surface of mandibular molars, while the palms and the rest of the fingers hold the mandible extraorally. Opposing forces are applied in different regions (1-1.5cm apart) across the entire palpable length of the mandible, starting from left retromolar region and finishing in the left one. Initially bimanual palpation is performed gently, but the force is gradually increased. One should be careful not to dislocate the fragments even further, but should check for additional fracture lines/fragments apart from the first one. Rough, unmeasured movements may cause secondary bleeding and increase the pain, which is most severe at the line of fracture.

Condylar fractures are established by extraoral palpation of the condylar process. The thumb is placed anteriorly to the tragus and the fifth finger is in the external auditory meatus. The patient is then asked to open and close his mouth and also move it side to side. Pain, inconsistency in bilateral finding, lack of movement on one side indicates a fracture. Luxation fracture to the condyle dislocates it away from its normal position, which can be clinically assessed by failure to feel the condyle in its designated palce anteriorly to the tragus.

Fractures in the area of the dental arch are established visually and manually based on step deformity between the teeth, lacerations and hematoma in the mucosa. Alveolar ridge fractures can be diagnosed by holding the ridge between the thumb and the index finger at the cervical level of the teeth and applying bucco-lingual pressure.

Fractures in the ramus are diagnosed through extraoral bimanual palpation. One hand palpates the ramus, while the other hand holds the mandible in the mental region and applies pressure upwards and downwards.

Additionally but mandatory to reaching a conclusive diagnosis, several radiographs in different projections can complement the clinical evaluation of the patient. Orthopantomography and computer tomography can reveal important information about the anatomy of the mandible an the teeth of the patient.
Differential diagnosis: Mandibular fractures don not represent a diagnostic difficulty, apart from TMJ luxation, which should be differentiated from condylar fracture.

Treatment: Treatment of the mandibular fractures aims at restoring the anatomical integrity and function of the bone. This goal is achieved by following the principles of reduction, fixation and immobilization of the fragments.

Reduction or reposition of the fragments to their original anatomical position is achieved by closed or open technique. Manual closed reduction is possible for recent fracture, in which the fragments are mobile and the dislocation is small. Reduction by elastic traction achieves gradual alignment over time of fractures with greater dislocation but mobile fragments. Open or surgical reduction is indicated for older fractures with immobile fragments when the closed technique is contraindicative. This includes cases of cyst or osteomyelitis in the line of fracture, overbite, edentulous jaw, avulsion fracture, etc.

Reliable fixation of the fragments and immobilization provide the prerequisites for proper healing, such as solid contact between the fragments and lack of mobility. Intermaxillary fixation (IMF) anchors both jaws together after proper occlusion has been established. Single-jaw rigid fixation allows the opening of the mouth, whereas IMF prevents it for some time.

Reduction, fixation and immobilization can be achieved by orthopedic, orthopedic-surgical and surgical methods.

First aid in the dental office dictates for temporary immobilization devices such as Gilmer’s ligature, Ivy loop or Ernst wiring.

Orthopedic means for treatment of fractures

This is the most often used group of methods, in which dental ligatures or standard arch bars are utilized.

Indications: There should be a number of teeth that are stable enough for fixation and immobilization. These methods are applicable for cases of small or no dislocation between mobile fragments, that allow for manual reduction or gradual traction reduction to be utilized.

Contraindications: Older fractures, significant dislocation between the fragments, mobile teeth (usually with periodontitis and 3rd degree mobility).
Different splints are used as orthopedic means for treatment of mandibular fractures: custom made, standard, laboratory made splints for single-jaw or inter-maxillary fixation. Such splints can be aluminum, steel, acrylic or reinforced acrylic and can be secured on the teeth or on edentulous alveolar ridges. IMF splints have hooks, in which elastic bands are placed.

**Dental splints**

They are used in cases when there are enough stable teeth present.

- Ligatures like these for temporary immobilization can be anchored to several teeth of both jaws (at least 3-4 ligatures are required)
- Single-jaw smooth wire splint made of aluminum or steel 0.8 to 1.3mm in thickness can be used for fractures of the alveolar process and the body of mandible, close to the teeth. These fractures should not have any significant dislocation. This splint is applicable for adults, as well as for children with mixed dentition.

1. Custom made dental wiring.
   These splints are indicated for cases of minimal dislocation, manual reduction and inter-maxillary fixation and immobilization. The presence of enough number of stable teeth on both maxilla and mandible are mandatory.
   - Risdon’s wiring – It requires two pieces of wire that are 0.4mm thick and 25 cm long. The wire is looped around the most distal tooth in left mandible and exits buccally. Both ends are twisted together to form a solid base buccally. Same is performed on the other side. Both twisted wires are connected together in the midline thus creating single structure. This wire is anchored at the cervical region of each tooth by ligating. After tying the ligatures, they form small hooks (0.6-0.8cm long), which are angled in such fashion, so that they can receive elastics for IMF.
   - Chavdarov’s wiring – This is a modification to Risdon’s technique, utilizing one longer piece of wire (45-50cm) instead of the two shorter ones, which creates the buccal base. The separate teeth are secured to the base with ligatures as already described.
   - Roy-Stout’s wiring – Single piece of 70-80 cm long and 0.4mm thick wire is adapted and secured distally to the last tooth on one side of the jaw, as described previously. The shorter part of the wire is then placed buccally to the dental arch, creating the buccal base for the wiring and small plastic or rubber tube is placed along the buccal base. The longer piece of the wire is then traversed in lingual-buccal direction through each interdental space over the tube and the buccal base and is brought back from the lingual side, creating loops. The tube is removed and the loops are twisted, thus firmly anchoring the buccal base to the teeth. The twisted loops create hooks, which are angled in a fashion, which is appropriate for IMF.
   - Baronov’s wiring – A single piece of 0.4mm thick 70-80cm long wire is formed into separate loops or hooks that are then driven from the lingual to the buccal side using a special beaked instrument. The hooks are then tied together in pairs on the buccal side,
so that they create larger hooks for IMF. The final hooks are at every tooth instead of in the interdental space and the buccal aspect is free from base wire, which allow for better oral hygiene.

- Atanasov’s wiring – This is further development of Roy-Stout’s wiring, which is created extraorally. 12-14 loops of wire, which are about 4mm long and 8-10mm apart are made on part of steel wire. The rest of the wire should not have loops and should be about 20cm long. The loops are then inserted in the interdental spaces from the lingual side toward the buccal sulcus, while the smooth part of the wire curves around the last molar and then goes through the loops to form the buccal base. The loops are then tightened and bend to make hooks for IMF and the buccal based is firmly anchored to the last molar of the other side. This wiring can be reduced to only 3-4 loops, which are placed around the molars and premolars, thus allowing for better oral hygiene.

2. Standard dental splints

These are represented by commercially available arch bars, used for inter-maxillary fixation and immobilization. There are different designs of arch bars such as Erich-Vassilev or Kavlakov-Atanasov-Spiridonov, which has the same advantages of Schuchardt’s arch bar.

![by Erich-Vassilev](image1.png) ![by Kavlakov-Atanasov-Spiridonov](image2.png)

Dimac ligature is a system that consists of pieces of wires (0.5mm thick and 5-6 cm long) that have plastic head with a hexagonal opening in one end and screw-styled thread in the other. The threaded end enters the interdental space and exits on the buccal side. The plastic head is then screwed with a special wrench, thus tightening the ligature. Several ligatures of this type should be placed to allow IMF by securing both jaws together with an elastic bands placed around the plastic heads. This method is easy to perform and interferes minimally with the oral hygiene, but is only indicated for minimal dislocation of the fragments.

Pre-made, commercially available orthodontic braces can sometimes be utilized for treatment of jawbone fractures, but find very limited use in oral and maxillofacial surgery.

3. Laboratory-made splints

- Schuchardt’s splint is made from thick piece of wire after previous impression of the dental arches. The splint has a base, arching on the buccal side, and a number of
small elements (seats), that are soldered perpendicular to it. Each ‘seat’ is 3-4mm long and has two ends – buccal end serves as a hook for IMF and the lingual (occlusal) end rests on the occlusal surface of the teeth, when the splint is first introduced in the mouth. The splint is secured to the teeth with simple ligatures and the occlusal end of each seat is cut and removed. Fixation to the teeth is enhanced by self-curing polymer. This splint is very stable and does not damage the free gingival margin.

- Dento-gingival splints such as Weber’s, Hofer-Pichler’s and Gunning-Port-Limberg’s designs are placed on the edentulous or partially edentulous alveolar ridge.

Weber’s splint is intended for primary and mixed dentition, as well as for partially edentulous adults. This acrylic construction is made in laboratory after initial impression. It lies on the alveolar ridge and around the teeth, to which it is secured with self-curing polymer. Hooks for IMF can also be modelled. The biggest disadvantage of this splint is its unreliable fixation.

Hofer-Pichler’s splint is another laboratory-made splint, created after initial impression. It rests on the lingual aspect of the teeth and the alveolar ridge in this region. Pre-modelled plastic beams, placed in the interdental spaces, are drilled and receive wire ligature, which secures the splint to the dental arch. It is indicated for bucco-lingual dislocation of fragments in patients of all ages.

Gunning-Port-Limberg’s splint is manufactured after initial impression and is intended for edentulous jaws. Maxillary and mandibular construction are secured together with self-curing polymer and the entire splint is anchored to the jaws with special grooves, and screws or peralveolar and circummandibular wiring.

- Occlusal splints are mostly intended for pediatric patients with mandibular fractures. They can be made out of cellulose (Pomerantseva-Urbanska’s design), metal (Theudorescu’s design), polymer (McLennan’s design), rubber (Berteux’). One common disadvantage of all occlusal splints is the fact that they rest on the occlusal aspects of the teeth, which can hinder the accurate reduction of the fragments.

Self-curing polymer occlusal splint by Pfaiffer is indicated for dentoalveolar and alveolar crest fractures. The polymer is applied on the teeth after reduction has already been done and the child is guided to centric position.

Polymer occlusal splint by McLennon is made from self-curing polymer after initial impression. The bone fragments are replicated and reduced at the gypseous model and the splint itself can be designed with hooks for IMF. The construction is fixed on the teeth using seld-curing adhesive.

Occlusal splint by Frigoff represents a hybrid between occlusal splint from self-curing polymer and eight-shaped ligature wiring. The metal wire ligature reduces the fragments and the SCP is added for fixation. This construction is indicative for fractures in the frontal area of the dental arch, IMF can also be achieved using hooks and elastic bands.
Orthopedic-surgical means for treatment of fractures

This group of methods are used when closed manual reduction of the fragments is feasible, but there are not enough stable teeth for fixation of arch bars.

Circummandibular wiring procedure (cerclage) can enhance the fixation of and arch bar splint when the patient is missing teeth or his teeth are mobile. The wire is passed around the body of the mandible using a special instrument or a needle with large lumen. One end of the wire is first introduced at the lingual side of the jaw and the other comes at the buccal side. Single point of entry below the inferior border of the mandible is utilized. The wire is tightened around a plastic plate or the patient’s denture.

Direct suspension (suspension fixation) from the piriform aperture or anterior nasal spine to the patient’s dentures may be used for edentulous or pediatric patients.

Surgical means for treatment of fractures

These methods utilize various types of osteosynthesis, in order to achieve contact and unification between the bone fragments.

- Edentulous jaws
- Edentulous fragment of fracture in the body of mandible
- Unstable teeth present in case of fracture in the body of mandible
- Oblique fractures of the body with gross dislocation (unfavorable fractures of the body)
- Unfavorable fractures of the condylar process
- Cases, when open surgical reposition is required
- Open fractures
- Fractures in regions of cyst, osteomyelitis
- Cases of patients with mental disorders
- Aged fractures and traumatic osteomyelitis

Contraindications for surgical treatment may be
- Acute infection
- Life-threatening complications in other regions (skull base fracture, rupture of lungs, etc.)
- Substantial loss of blood
- Shock
- Febrile state secondary to infection in the area of trauma

The contemporary treatment of fractures to the jawbones abides by the following principles
- Accurate reduction of the fragments
- Dense and firm contact between the fragments
- Firm fixation of the reduced fragments during the entire healing period, aimed at excluding any pathological mobility
- Gradual introduction to function after reliable fixation of the fragments

Surgical methods for treatment of mandibular fractures can be classified into:
- Closed osteosynthesis – including intraosseous transfixation by Kirschner wire and supraosseous cerclage wiring
- Open osteosynthesis – suture wiring, miniplate osteosynthesis and screw osteosynthesis
- Suspension fixation

**Closed osteosynthesis with Kirschner wire**

This method was intended for fractures of the mentum, condylar process and angle of the mandible. The wire is driven through the bone, thus connecting the fragments. One significant disadvantage of the method is the fact that rotation around Kirschner’s wire is possible, which can alter the centric position of the jaws and delay healing. Another problem is that the end of the wire is left outside the tissues, which increases the risk of infection. Today this method has become obsolete.

**Closed osteosynthesis with circummandibular ligature wiring (cerclage)**

Cerclage is applied in edentulous and pediatric patients. The contemporary technique does not require incision to the skin. Large lumen injection needle is used to guide the steel wire (0.4mm width). The skin in the canine region below the inferior border of the mandible is cleaned and local anesthesia is applies. The needle containing the wire in its lumen is inserted in this region from the buccal side. It exits intraorally through the buccal sulcus, while the needle is moved in contact with bone. One end of the wire is then pulled and the needle goes back in the tissues. Without exiting the initial insertion site the needle is then traversed to the lingual side of mandible where the other end of the wire is pulled away. The wire is then used to fix an acrylic plate or denture to the alveolar ridge. This method achieves
single-jaw fixation appropriate for manual reduction of oblique fractures of the mandible with minimal dislocation and there is no impairment to mandibular function.

Open osteosynthesis by means of suture wiring

Initially, incision to the skin is made and the bone fragments are exposed and reduced. Small holes are drilled in the bone 0.8-1cm from the fracture line between two fragments. Steel piece of wire (thickness of 0.5-0.8cm) is then driven through 2, 3, 4 openings in X-, Z, or N-shaped suture and tied firmly. Some fractures in the angle allow for suture wiring without extraoral incision to the skin.

Open compression osteosynthesis by metal plates

Standard plates for osteosynthesis may not always ensure firm and dense contact between the fragments. Titanium compression plates are used to alleviate this problem, thus allowing for optimal healing. Inter-maxillary fixation and immobilization is unnecessary because this advanced treatment method provides enough stability during the function of osteosynhetized single jaw. The plates have 2, 4, 6 openings and they anchor to the fragments with special screws, which allows for gradual tightening over time, so that the firm contact between the fragments is maintained. Compression osteosynthesis is intended for treatment of fractures with gross dislocation of the fragments when suture wiring cannot provide reliable fixation. Such cases include fracture lines in cystic lesion, avulsion fractures or when suspension fixation is contraindicated.

Open osteosynthesis by miniplates

This is the contemporary method of choice for treatment of the majority of mandibular fractures. It often favors intraoral over extraoral incision and also provides reliable single-jaw fixation. The fragments should be surgically reduced to their anatomical locations and centric position of the mandible should be achieved. Miniplates are then fastened to the fragments using screws for osteosynthesis. This method requires additional surgical intervention after 4-6 months for the removal of the plates.
Suspension fixation

Indications for this type of treatment is localization of the fracture line beyond the dental arch, in the angle, ramus or condylar process of the mandible, as well as multiple and fragmented fractures that require IMF. Various types of suspension fixation designs are proposed for adults and pediatric patients. Thoma’s design utilizes fixation to the lateral wall of the piriform aperture bilaterally. Broadbent combines circummandibular ligature wiring with suspension fixation at anterior nasal spine.

Panagopoulos achieves solid inter-maxillary fixation by bilateral circummandibular ligature wiring and bilateral suspension at the lateral walls of piriform aperture.

Dal Pont proposes the use of 4 hooks anchored to the mandible and to the piriform aperture. Mektubjian modifies this method by changing the design of the hooks and implementing IMF by ligature wire. He later changes the mandibular hooks with circummandibular ligature wire.

Atanasov implements transalveolar inter-maxillary fixation with ligature wiring through drilled holes in the maxillary alveolar process, which are then secured to patient’s denture or occlusal plate together with the circummandibular ligature wire.
Berardo uses suspension fixation with intraosseous screws anchored to the bone and ligature wiring for IMF. This method is implemented in Bulgaria by Vladimirov in 2006.

Any suspension fixation technique has the drawbacks of IMF. Furthermore, it provides conditions for rotation and micromobility of the fragments because it uses one or two fulcrum points per jaw.

**Fractures of the maxilla**

*Aetiology:* Maxillary fractures make for about 3% of total fractures in the body. Such fractures are 3.3-4% of fractures of the facial skeleton and jawbones, with most patients aged 21-40.

Cause for fractures of the maxilla can be gunshot or non-gunshot trauma, such as domestic-, industrial-, sport- or traffic accidents. Motor vehicle accidents are the most common reason for this traumatism in Bulgaria.

*Classification:* The maxilla is connected to the rest of the mid-facial skeleton with sutures, this is why the fracture lines traverse through anatomical weakspots and not through the sutures. There are many classifications proposed for maxillary fractures, including:

1. Depending on aetiology:
   - Gunshot and non-gunshot
2. Depending on changes to occlusion
   - With or without changes to occlusion
3. Depending on the localization of fracture line(s)

**Partial fractures**

Alveolar ridge fractures; sagittal unilateral fractures by Ombredan; by Nastev

These are the most commonly observed fractures in the anterior maxilla. Alveolar ridge fragment usually carries single or a few teeth and hangs loosely on soft tissues. Fracture line may be arched, trapezoid or conical and may be located at various level across the alveolar ridge, sometimes communicating with the maxillary sinus. The shape of fracture line depends of the anatomical features of the maxilla and roots of teeth. The alveolar process reaches the body of the maxilla with the root apices deep inside it, thus creating conditions for fracture at various level and of different shape.

Sagittal fractures by Ombredan are observed unilaterally after a powerful blow to anterior maxilla. Sagittal fracture line splits the hard palate, while vertical fracture line reaches nasal septum. Cranially, this fracture can reach floor of maxillary sinus, frontal process of maxilla or nasal bones. Nastev describes sagittal unilateral fracture with internal displacement of the small fragment.
Total fractures including Le Fort I, Le Fort II, Le Fort III, Wassmund I, Wassmund II

Le Fort I fracture is also described as transversal by Dingman, inferior horizontal fracture by Erich, or Guerin’s fracture. The fracture line progresses along the inferior horizontal weak line at the lower rim of piriform aperture, floor of maxillary sinus, inferior third of pterygoid process of sphenoid. Sometimes the alveolus of third maxillary molar is also fractured. The fragment consists of bilateral alveolar ridge, hard palate, nasal and maxillary sinus floor. The fragment is usually mobile and the lateral and medial pterygoid muscles, as well as the superior pharyngeal constrictor affect its displacement.

Le Fort II fracture is also known as Erich’s pyramidal fracture. The fracture line passes along the lateral oblique weak line – nasal root, nasal septum, ethmoid labyrinth, inferomedial orbital angle, zygomaticomaxillary suture, maxillary sinus, middle third of pterygoid processes of sphenoid. Medial and lateral pterygoid muscles can further displace the fragment backward and downward, which usually causes anterior open bite and premature occlusion of the molars.

Le Fort III fracture is called Erich’s transverse facial fracture, transorbital fracture, Rowe’s suprazygomatic fracture, or craniofacial disjunction. The fracture line progresses along the superior horizontal weak line – nasal root, orbital flor, lateral orbital rim, zygomatic arch and cranial third of the pterygoid processes of sphenoid. Masseter muscle, along with the pterygoid muscles, take part in displacement of the fragments in posterior and caudal direction, which causes anterior open bite and premature occlusion of the molars.

Wassmund’s fractures are characterized by the fracture line progressing along the medial oblique weak line. Wassmund I is a variant of Le Fort II with intact nasal bones. The fracture line starts above the piriform aperture, continues in the medial orbital angle and later repeats the typical Le Fort II line. Wassmund II is another type of craniofacial disjunction or Le Fort III fracture with intact nasal bones. The fracture line begins above piriform aperture,
travels along the medial oblique weak line toward the medial orbital angle and orbital floor, after which progresses along the typical Le Fort III line.

Clinical signs.
Various clinical symptoms can be asserted depending on localization of the fracture:
- Pathognomonic or main symptoms - violations of integrity of the bone, pathological mobility of bone/bone fragments, bone crepitus or crunching
- Secondary symptoms are exhibited facultatively by some patients. Such symptoms may be pain, facial deformity, swelling, occlusal violations, lost or impaired function.

Partial fractures
- Alveolar ridge fractures. Pain is typical complaint and bleeding tears to the mucosa of the alveolar ridge are present. Upper lip is traumatized and can be swollen, painful, contused and of blueish colour. The mouth is half-open with blood and saliva seeping through. Occlusion is very painful and can be distorted in case of palatal dislocation of the fragments, which causes posterior open bite and edge-to-edge anterior bite. The fragment may also be displaced in anterior direction. Dentoalveolar injury is often present.
- Sagittal fractures. Typical findings include bleeding from the mouth or nose, ruptures in gingiva, nasal mucosa or maxillary sinus membrane. Facial disproportion is caused by hematoma and swelling, abrasions and wounds to the skin, as well as displacement of the fragment. A step deformity is always present, additionally open bite on the affected side can be observed.

Total fractures
- Le Fort I fractures. This fracture is characterized by intense pain along the fracture line, with tears to the intraoral mucosa, which reveals the maxillary sinuses and piriform aperture. Blood and saliva flows out of the mouth. Epistaxis or nosebleed is also present. Upper lip is contused and swollen. Mobility of fragment allows for elongation of the face, restriction to breathing, painful mastication.
- Le Fort II fractures. Intense pain is one of this fracture’s signature features. Gross edema and swelling of the face spreads around the upper lips, infraorbital regions, inferior eyelids and cheeks (Moon face or ballooning). Epistaxis, as well as bleeding from the half-opened mouth is present. Bilateral circumorbital oedema and ecchymosis (Black eye) spread outside the orbicular ocular muscle and are manifested soon after the trauma. Downward displacement of the fragment causes premature occlusion of the molars (Monkey face), while backward displacement may provoke gagging reflex. Paraesthesia of infraorbital region and mobility of the fragment is also present. This type of fracture often co-exists with traumatic brain injury. Similar findings are also reported for Wassmund I fractures. The fragment in this case excludes nasal and lacrimal bones and frontal process of maxilla.
- Le Fort III fractures. Also called cerebrofacial disjunction, the fragment carries the entire maxilla, nasal and lacrimal bones, orbital floor and zygomatical complex.
Pathological mobility is present in the entire mid-facial region and the oculus, which follows the orbital floor.

The face is elongated and the midfacial skeleton hangs on the skin and other soft tissues. Gross edema and swelling of the face is at hand, as well as bilateral circumorbital ecchymosis (Raccoon eyes). Retroocular hematoma may cause unilateral or bilateral exophthalmos. Damage to optic (blindness) and ophthalmic nerves (cross-eye), ethmoid bone, bleeding from various divisions of the maxillary artery are quite common. Cerebro-spinal fluid seeps from the nose or ears as result from meningeal damage and potential fracture of the cranial base. Paresis and paralysis of facial nerve, loss of hearing and brain concussion syndrome complement the clinical findings in some patients.

Clinical examination aims at establishing main and secondary symptoms with emphasis on facial symmetry and traumatic brain injury. After extraoral inspection the opening of mouth, occlusion, intraoral structures is assessed. Bimanual palpation will reveal the main symptoms of fracture - loss of bone integrity, pathological mobility of bone/bone fragments, bone crepitus.

Diagnosis is based on the anamnesis and clinical evaluation. Patient’s complaint may include facial swelling, pain, bleeding from nose, mouth or ears, difficulties in opening of mouth, mastication, impaired speech, breathing or vision. Loss of consciousness is very important finding and the clinician should gather detailed information about it, such as time, duration, location where it occurred. The patient may experience variety of symptoms, depending on localization of the fracture and the collateral damage to soft tissues.

Clinical evaluation begins with inspection, which provides information about the contours of the face. Total fractures will often produce “panda bear face”. Additionally, secondary symptoms are also established:

- Characteristic “dish face” deformity, typical for type II and III Le Fort fractures
- Emphysema
- Ecchymosis in the buccal sulcus of the maxilla
- Occlusion deformities
- Pain
- Abrasions or other wounds, including lacerations and tears of skin and mucosa. Total fractures with lacerated oral mucosa may reveal the nasal cavity or maxillary sinus
- Paraesthesia of the cheek and/or nose due to damage to the infraorbital nerve
- Periorbital hematoma in type II and III Le Fort fractures are commonly accompanied by epistaxis because of damage to the nasal mucosa and ethmoid arteries
- Subconjunctival hematomas (echimoma)
- Rhinorrhea of cerebro-spinal fluid from the nose is established in some type II and all type III Le Fort fractures and indicates cranial base fracture, complicated by traumatic brain injury.

Pathognomonic symptoms (loss of bone integrity, pathological mobility of bone/bone fragments, bone crepitus) are established by the means of symmetrical bimanual palpation.
Palpation follows the typical fracture lines and begins at both sides of the nasal alas. The fingers then move to zygomaticomaxillary suture, nasal root, infraorbital regions, lateral orbital walls and zygomatic arches. In Le Fort I fractures with displacement the maxillary fragment hangs on soft tissues and is readily mobile during palpation or opening of mouth. Intense pain is present in palpation at the maxillary tuberosities and laterally from nasal alas. The most painful region under palpation in case of type II and III Le Fort fractures is at the nasal and lacrimal bones. Type II fractures are painful at the anterior walls of maxillary sinus and the tuberosities, while type III fractures – at the lateral orbital rims and zygomatic arches. The mobility of the fragments can be verified by bimanual examination.

In patients with Le Fort I fractures the fingers of the right hand grasp the anterior teeth and alveolar ridge of maxilla and try to apply up-down “rocking” movements. The fingers of the other hand are initially placed laterally to the nasal alas and the palm is resting on the forehead. The fingers are later moved toward zygomaticoalveolar crest and the tuberosities. Mobility can be sensed while rocking the fragment.

In patients with Le Fort II fractures the right hand is placed the same way. Palm of the left hand rests on the forehead, thumb and index finger are placed at the nasal root and apply gentle pressure to nasal and lacrimal bones. The fingers are then moved on the infraorbital rim. Mobility should be sensed along the line of fracture. Step deformities may be present in these areas. In Wassmund I fractures the nasal and lacrimal bones will remain intact and mobility will be felt only at the infraorbital rim.

In patients with Le Fort III fractures the right hand is placed as described previously. Palm of the left hand rests high on the forehead, thumb and index finger are carefully placed at the nasal root, at lateral orbital rims and at the zygomatic arches in an attempt to sense pathological mobility in these areas during the “rocking” motions. Wassmund II fracture lines will exclude the nasal and lacrimal bones, frontal maxillary process and ethmoidal labyrinth.
The diagnosis should be further supported by panoramic radiography or axial tomography. Smell and vision sensations and organs require additional tests. The patient is asked to follow the clinician finger, as it is moved up, down and sideways. The patient may also report blindness or double vision (diplopia).

*Differential diagnosis* is unnecessary in patients with maxillary fractures.

*Treatment*: Treatment of maxillary fractures aims at restoring the anatomical continuity and function of the midfacial skeleton, as well as the facial symmetry and esthetics. This is possible if proper reduction, fixation and immobilization is established. The fragments are usually fixated and immobilized in relation to superiorly positioned intact facial or cranial bone structures, while reestablishing dental occlusion and central position of mandible.

Patients with maxillary fractures require special attention because co-existing life-threatening complications can sometimes be overlooked. Traumatic brain injury, occlusion of upper airways and substantial loss of blood present the greatest risk. Such complicated cases should be offered first aid and then referred to specialized treatment in hospital.

**Alveolar ridge fractures.** First aid and temporary immobilization with standard arch bars is necessary. Ombredan and Nastev type of fractures should be immobilized with or without occlusal plate and submental bandage. Specialized treatment is provided in hospital.

Reduction of fragments to their anatomical position is usually achieved with the closed manual technique. Immobilization and fixation needs to protect the teeth on the fragment. Cases with minimal dislocation are treated with standard arch bars and IMF or single-jaw splints. All alveolar fractures are considered open and therefore infected, which necessitates application of antibiotic treatment.

**Total maxillary fractures** are treated by means of orthopedic, surgical or orthopedic-surgical methods.

*Orthopedic methods* require specially designed orthopedic devices, comprised of two functional parts – one part is reducing the fragments to their anatomical position and restore occlusion, while the other one provides fixation to intact structures of the cranium. Mandibular arch bar can be used as reducing part. The other portion of the device comprises of three main elements – elements that secure it to the cranium, elements that secure it to the maxilla, connecting rods and wires. One such apparatus was implemented by Ferdinand von Gräfe but nowadays such devices are deemed obsolete because of the large extraoral elements, poor hygiene and general discomfort of the patient.

*Orthopedic-surgical methods* require orthopedic devices and surgical methods in order to achieve fixation.

Adams’ approach was originally purely surgical method for reduction of maxillary fractures. Fixation is achieved at superiorly positioned bone structures. Such structures may be the zygoma, infraorbital and supraorbital rims. Le fort I fractures are suspended by the intact zygoma (zygomaticoalveolar crest), type II fractures are attached to the infraorbital rim and the supraorbital rim is used as fulcrum for type III fractures. However Le fort I and II fractures may be suspended at all intact structures above them.
The procedure requires general anaesthesia. Skin incision is placed in the distal part of the eyebrow, so that the operator can gain direct access to the supraorbital rim. A hole is drilled above the zygomaticofrontal suture and 0.5mm thick ligature wire is traversed through it. Special kind of needle guides the wire under the zygomatic arch in the buccal sulcus, close to the first maxillary molar, which serves as a fulcrum point. Later the author recognized the potential damage to the first molar and implemented arch bar splint for the maxillary dental arch instead. Two wires are then fastened to the splint bilaterally. Elastic bands attached to the mandibular splint put into effect the IMF.

Lesney’s approach implements bilateral circumferential ligature wiring around the intact zygomatic arch for treatment of type I and II Le Fort fractures. Both ligatures are attached to maxillary splint. IMF is secured by elastic bands between both jaws. In case of edentulous jaws the author secures the dentures with circummandibular and peralveolar wiring and then attaches them via the abovementioned ligatures to the zygoma.

Klisarov proposes an upgrade for Adams’ approach. After the wire passes through the hole above zygomaticofrontal suture it is guided around the zygomatic arch, then underneath the skin of the forehead and the same procedure is done on the other facial half. Both wires are then fastened to the maxillary splint and IMF is set.

Hardin recommends the suspension fixation to openings in the frontal sinus. He considers that the suspension fixation of molars at a fulcrum point that is posterior to the face brings about possibility for internal rotation and micromobility. Bilateral wires are guided
subcutaneously through the holes in the frontal sinus, then lateral to the nose and into buccal sulcus, where they are fastened to the maxillary splint.

Kufner and Mektubjian implement other methods of suspension fixation to the frontal bone via special screws or hooks, thus preventing rotation around posteriorly positioned axis.

Surgical methods
Direct open osteosynthesis by means of suture wiring or metal plates allow for rigid single-jaw fixation in the area of fracture line – lateral or inferior orbital rim, piriform aperture, zygomatic arch, etc.

Closed osteosynthesis with Kirschner wires achieves fixation of Le Fort II fracture to intact zygomatic complex.

The surgical and the orthopedic-surgical methods outclass the classic orthopedic approach to treatment of maxillary fractures in many ways. They bring about better, more accurate and more predictable results, thus providing faster restitution to normal function.

Fractures of zygomatic bone and zygomatic arch

Surgical anatomy. The zygomatic bone is one of the paired bones in the facial skeleton. Its position is the superolateral area of the face. The zygoma sets the width of the face and some of facial contours, which may vary across genders, races and individuals.

The zygoma resembles a rectangular plate with lateral, temporal and orbital surfaces. The orbital surface is the weakest part of the bone but it is well protected by the adipose contents of the orbit and the infraorbital rim, which can often deflect the fracture line.

The maxillary process of zygoma fuses to the maxilla in the zygomaticomaxillary suture. The temporal process of zygoma fuses with the temporal bone in the
zygomaticotemporal suture, thus bringing about the zygomatic arch, from which the masseter muscle is suspended. Other muscles (major and minor zygomatic muscles, m. levator anguli oris), start at the zygomatic bone as well, which affects the displacement of fragments pulled by them. Zygomaticofrontal suture marks the fusion with the frontal bone.

The divisions of facial and trigeminal nerves are located close to the zygoma, which explains why they get easily damaged by trauma or surgical procedures.

The weakspots of the zygoma are actually its sutures with the adjacent bones.

**Aetiology and classification:** Fractures of zygomatic bone and arch often happen together with total fractures of the maxilla, while separate fractures are only second to mandibular fractures and make for 9.9-25% of all facial fractures. Such trauma is caused by substantial force of direct impact or compression. These fractures can be classified as gunshot injuries and non-gunshot injuries. Most common reason is motor vehicle accidents. Several different types of fracture can be distinguished depending on the localization:

- Fractures of the zygomatic bone – with displacement; without displacement; with damage to maxillary sinus;
- Fractures of zygomatic arch – with displacement; without dislocation;
- Fractures of zygomatic bone and arch – with displacement; without displacement; with damage to maxillary sinus;

Zygomatic fractures can also be complicated, open or closed, unilateral or bilateral. Bernadskiy describes fresh and aged (after 10th day) fractures.

**Clinical signs** of zygomatic fractures can vary, depending on the damage to adjacent soft tissues, bones, nerves, blood vessels and sensory organs. Establishment of main symptoms (loss of bone integrity, pathological mobility of bone/bone fragments, bone crepitus) sometimes may be difficult because of substantial hematoma and edema. Secondary symptoms of particular interest are:

- Facial deformity and esthetic disbalance
- Restrictions to the mandibular excursion
- Sensory abnormalities of the infraorbital nerve
- Ocular symptoms
- Pain
- Hematoma and edema
- Abrasions and wounds

**Facial deformities** become visible in the first few hours after the trauma. Hematoma and edema in the region can mask the dislocation or avulsion of bone fragments. The width of the face is changed and concavity in the area may become apparent. Open fractures and gunshot injuries are associated with extensive trauma to hard and soft tissues, such as lacerations of the eyelids and damage to oculus. Facial deformities may become visible only after hematoma and edema disappear.
Restriction to the mandibular excursion are experienced by many patients and are often caused by foreign bodies and displacement of bone fragments in inward, downward and backward direction toward the coronoid process. Hematoma or damage to mastication muscles may bring about limitations to the opening of the mouth.

Sensory dysfunctions of the infraorbital nerve may be caused by dislocation of fragments and damage to the nerve. Reduced or missing sensitivity is reported. Other complaints may include traumatic neuritis or rarely neuralgia. Numbness of incisors and premolars is indicative of fracture of anterior sinus wall. Sensory dysfunctions are usually transitory and go away after 2 weeks, but if they persist, the physician should consider greater displacement of the fragments or step deformity at the inferior orbital rim. Zygomatic arch fractures are not associated with sensory dysfunctions.

Ocular symptoms are brought about in some cases of fractures of the zygomatic bone. Periorbital ecchymosis and edema, diplopia, drooping, subconjunctive hemorrhagia, enophthalmos, exophthalmos, cross-eye and sometimes blindness may be diagnosed.

Periorbital hematoma manifests soon after the trauma and can spread beyond the orbicular ocular muscle. The lower eyelid is usually affected unilaterally. Soon after edema presents itself. The swelling may be quite large, making examination of the orbit especially difficult.

Diplopia is associated with fracture of the zygomatic bone and orbital wall. It is caused by prolapse of orbital content into the maxillary sinus or damage to nerves and muscles. This symptom may persist for a while. Temporary diplopia is brought about by hematoma of orbital floor and manifests as double vision. Persisting diplopia diagnosed after 3-4 months.

Epistaxis is indicative of fracture of maxillary sinus wall secondary to fracture of zygomatic bone. There is rarely profuse bleeding but hemosinus can be observed on radiographs. After a week the blood is resorbed, which can be diagnosed with another radiograph.

Gross dislocation of zygomatic bone upward and backward may break the larger wing of sphenoid, as well as the superior orbital fissure, which results in damage to the extraocular nerves and muscles. Such complication will produce immobility of oculus and pupil, drooping of upper eyelid, retroocular pain, subconjunctive ecchymosis, exophthalmos.

Fracture of zygomatic arch is presented as depression orconcavity on the arch, which becomes apparent after the resorption of hematoma and edema.
**Diagnosis** is based upon patient’s history and complaints, clinical findings and radiographs. Symmetrical palpation will help establish the main symptoms of loss of bone integrity, pathological mobility of bone/bone fragments, bone crepitus. Clinical evaluation should reveal important secondary symptoms such as sinking or concavity of zygomatic bone or arch. Final diagnosis is supported by panoramic radiographs in different projections, as well as CT.

Zygomatic arch fractures shows radiographically as V-shaped concavity.

**Treatment:** Treatment of zygomatic fractures aims at re-establishing normal function of mandible and TMJ, eye, as well as normal contour of the face. This is achieved by reduction of the fragments and fixation when necessary. Fractures with minimal or no dislocation and no apparent esthetic or functional abnormalities do not require treatment. The patient is given advice about suitable diet and analgesics. Large periorbital hematomas and hemosinus may require physical therapy and antibiotics for prevention of complications.

Patient’s complaints (restricted mandibular movement, facial deformity, ocular and sensory symptoms) and gross displacement of fragments call for conservative or surgical treatment. Medical attention to fresh fractures (up to 10 days) yields better results than aged fractures. After the 20th day reduction is very difficult.

**Conservative (bloodless or non-surgical) reduction**
- **Muchin’s approach** is intended for fresh fractures of zygomatic bone. After application of local anesthesia the index finger of the right hand is placed intraorally at the maxillary tuberosity and pushed the fragment upward and outward. The other is placed on the face.

**Surgical reduction**
- **Lothrop’s intraoral approach.** This method is indicated for open, displaced fractures of the zygomatic bone, as well as comminuted fractures of zygoma and anterior wall of maxillary sinus. The anterior sinus wall is reached through incision in the mucosa starting from central incisor up to second molar. The fragments are reduced with special instrument inserted in a trepanation of the canine fossa. Prolapse of orbital content requires packing of the sinus with iodoform gauze. The gauze is then guided in inferior nasal meatus. The packing procedure may also be done with balloon instead. Suture is placed and the gauze drainage is pulled away after 7-14 days.

- **Keen-Vielage’s intraoral approach.** This method is intended for fragmented fractures of zygomatic bone and arch. 1cm long incision is placed posteriorly to zygomaticoalveolar crest under local anesthesia. A periosteal elevator or similar instrument is then driven through the incision toward the bone at depth of 2-3 cm. Zygomatic bone is pushed upward and reduced, so that clicking sound is heard. Later, analgesics and antibiotics are assigned.
- **Limberg’s extraoral approach.** This method is implemented for V-shaped fractures of the arch. A 0.5 long incision is placed on the skin above the concavity and special hook is introduced through it. The hook pulls and reduces the fragments, evident by the clicking sound. Finally, the skin is sutured.

- **Aronson’s extraoral approach.** Indications repeat Limberg’s method but this time surgical treatment is implemented, should the fragments become dislocated once again. The author uses osteosynthesis with suture wiring.

- **Davidov’s extraoral approach.** This method is intended for comminuted fractures of the arch with multiple small fragments. The fragments are exposed and reduced via incision in the skin. Fixation is achieved by acrylic plate, which is reinforced with metal, and the skin is sutured. After complete recovery the plate may be left inside, as it is biocompatible.

- **Fratini’s extraoral approach.** This technique is implemented for treatment of fragmented fractures of zygomatic bone and arch. Reduction is achieved by periosteal elevator or hook, inserted in 2cm long incision at the lateral part of the eyebrow. Aged fractures are osteosynthetized at zygomaticotemporal suture.

- **Gillie’s extraoral approach.** The author suggests osteosynthesis of multiple large fragments to the infraorbital rim. Transorbital infraorbital access is achieved by incision in the skin, placed 0.5cm from the lower eyelid. Orbicular ocular muscle is the reflected and the orbital rim is exposed. The periorbital is removed and suture wiring is applied. Fragmented fractures of the frontal process are osteosynthetized by suture wiring or metal plates, accessed by incision in the skin at the lateral part of the eyebrow.

- **Tessier’s extraoral approach.** This achieves osteosynthesis of displaced zygomatic bone at the infraorbital rim via transconjunctival access.

- **Osteosynthesis by miniplates.** This technique is indicated for gross dislocation of the fragments. Incision of the skin provides access to the fragments, which are then reduced. The miniplates offer stable fixation but need to be removed after bone reunion occurs (6-9 months).
Fractures of the nasal bone

*Surgical anatomy*: The bony skeleton of the nose is comprised of the paired nasal bones and frontal processes of the maxillae. The nasal bone resembles a rectangular plate, which have a thick and narrow upper portion and sharp, thin, uneven lower portion. The nasal spine of the frontal bone supports both nasal bones. The fractures of nasal bone usually occurs between its upper and lower portion.

Two paired and single unpaired cartilage bring about the nose. Anteroinferior portion of the nasal septum is comprised of the septal cartilage, which resembles a rectangular plate. Posteriorly it fuses with the vomer and superiorly it connects with the perpendicular lamina of ethmoid bone. The lower portion of the cartilage is slightly mobile and lies on the nasal crest, which allows for its displacement in case of fracture. The paired cartilages support the lateroinferior portions of the nose. The alar cartilage supports the tip of the nose. The paired cartilages comprise of medial and lateral cruras, which come together at the highest part of the nasal tip. The lateral cartilage is a triangular plate, attached to the mesial aspect of the frontal maxillary process and the nasal bones. The lateral cartilage attaches to the septal cartilage at the midline of the nose and caudally connects to the alar cartilage. Fractures of the nasal bones may cause sinking (concavity) of the nose, which may break the cartilages as well.

*Aetiology*: Nasal bone fractures make for 8-42.89% of all maxillofacial fractures. The fractures may be caused by direct impact. The nose protrudes anteriorly, which explains its susceptibility to trauma.

*Classification*: Kavrakirov’s classification follows:
- Fractures of nasal bones without displacement or avulsion – closed or open fractures of nasal bones
- Fractures of nasal bones with deformity of external nose and displacement – closed or open fractures
- Damage to nasal septum – luxation at the base of nasal septum; fracture of bony and/or cartilaginous portion of nasal septum
- Damage to bony and cartilaginous structures of the nose – with or without avulsion

The clinician should be aware of the thin nasal mucosa which gets torn apart even in relatively mild trauma.

*Clinical signs*: Clinical signs are in association with the force and the direction of impact. Heavy blow at straight angle might damage the orbit, maxillary sinus or ethmoid labyrinth, therefore base of cranium.

Isolated nasal trauma usually brings about complaints about severe, localized pain and headache. Even gentle touch to the nose may provoke intense pain and loss of consciousness.

*Epistaxis* and breathing difficulties are usually experienced. Nasal mucosa gets easily damaged even if no displacement of fragments is at hand. The large vessels of the ethmoid, when ruptured, may cause life-threatening bleeding. Unilateral bleeding may also be caused
by damage to the Kiesselbach plexus at the anteroinferior portion of nasal septum. Hypertension, hemorrhagic diathesis may provoke profuse bleedings after minimal trauma. Breathing difficulties are facilitated by bleeding, blood clots, edema of the mucosa, displacement of fragments.

Hypo- and anosmia are impairment of the sense of smell. Peripheral reasons for such impairment may be physical obstructions such as blood clots, mucus, edema of the mucosa, displacement of nasal fragments. All of these limit the airflow to superior nasal meatus and the olfactory fibers. Central impairment occurs in gross fractures of the ethmoid bone and damage to the olfactory fibers.

Periorbital hematoma and subconjunctival ecchymosis are often observed in patients with nasal fractures. These symptoms manifest soon after the trauma (up to 2-3 hours after it). The edema of the nasal region can spread to the check. Unilateral hematoma is reported seldom, typically associated with unilateral fractures of nasal bones and frontal process of maxilla.

Deformities of the nose are usually present when its bony or cartilaginous structure sustain damage. They depend on the direction and force of impact and are typically obvious in bilateral fractures of nasal bones, septum and frontal process of maxilla. Such deformities may manifest as displacement of nasal tip to the side or concavity.

The clinician should try to establish the pathognomonic symptoms. Bone crepitus is easily diagnosed and pathological mobility of fragments may be palpated immediately after the trauma or after secondary symptoms have been alleviated.

Diagnosis: Diagnosis is based on recollection of trauma, subjective complaints of the patient and pathognomonic symptoms. Bimanual examination is performed as the left hand of the physician immobilizes patient’s head and index finger and thumb of the right hand palpate the area around the potential fracture line. Establishing pathological mobility of fragments may be difficult due to substantial edema and hematomas. In such cases the clinician should assign several radiographs in different projections.

Treatment: Treatment aims at reestablishing anatomical form and function, while also considering the esthetics of the patient.

Management of bleeding is done after establishing its source. Bleeding septum is packed with gauze, which has been soaked in 3% hydrogen peroxide solution. Other way to stop such hemorrhage is by applying pressure at nasal alas toward the septum. Profuse bleeding is managed by anterior nasal packing with gauze, soaked in epinephrine, PABA, EACA). Posterior nasal packing is done when these methods are deemed unsuccessful. The packing is removed after 2-3 days because it may increase the risk of infection.

Displacement of fracture calls for reduction and fixation, preferably performed in the first 24 hours. Substantial hematoma and edema may defer this procedure for 2-3 days. After this period malunion of the fragments will occur and reduction is hampered.
Closed reduction and fixation of the fragments

Closed fractures of nasal bones call for reduction under local anesthesia in the first 1-2 days. Straight elevator or periosteum elevator is wrapped in gauze and inserted in superior nasal meatus. The fingers of the left hand are placed on the nose for better control.

After the reduction of the fragments rhinoscopy and inspection of the septum is performed. Submucosal hematoma is drained via horizontal incision. Displacement of the septum is alleviated with special instrument, tears in the mucosa are sutured excluding the cartilages, after which fixation dressing is applied. Separate gauze tampons are placed in both nostrils. Plastic tube is wrapped in gauze, which is then soaked in antibiotic unguent. These tampons support the reduced fragments and facilitate breathing through the tube. Externally, plaster or other dressing is applied. The internal tamponade is removed after a week and the external dressing remains for another 3-7 days.

Open reduction and fixation of the fragments

The injuries in the soft tissues are managed, nasal mucosa is then sutured, after which the fragments are reduced and fixated. At the end the skins is sutured.

Fragmented fractures require the use of special fixation devices, which consist of endonasal portion and externally fixated elements. Antibiotics and analgesics are assigned after the surgical procedure.
LIFE-THREATENING CONDITIONS IN MAXILLOFACIAL TRAUMA  
(Dimitar Atanasov)

ASPHYXIA

For the ancient Greeks asphyxia meant no heart activity, absence of pulse. Today, asphyxia is defined as a condition of severely deficient supply of oxygen to the tissues and choking. All conditions characterized by hypoxia (acute tissue oxygen deprivation) result in asphyxia. The end-stages of cessation of life, regardless of the cause, are characterized by oxygen starvation and asphyxia.

The factors underlying the development of asphyxia are $O_2$ and $H_2CO_3$. Normally, they are balanced in the body, and any disturbance of this balance (oxygen depletion or Carbonic acid accumulation) results in asphyxia. Carbonic acid is the strongest stimulus to the respiratory center.

Etiology: Asphyxia may be due to a reduction in oxygen partial pressure in the air or due to a pathological process localized in some of the units involved in the oxygen exchange in the body. So, choking may occur with airway obstruction or chest compression, in lung diseases (pneumonia, pulmonary edema, emphysema), blood poisoning (carbon monoxide, nitrates, aniline), heart diseases (congenital heart defect, heart failure), in impairment of neural regulatory mechanisms controlling the process of respiration (morphine poisoning, strychnine poisoning, tetanus), in injury of reflexogenic zones.

Classification: Depending on etiological factors, asphyxia can be subdivided into: mechanical, pulmonary, circulatory, tissue, blood, neurogenic, reflex.

For the dental practitioner, oral and maxillofacial surgeon, the mechanical type of asphyxia is essential. It, according to Ivashtenko. A. (1968) can be subdivided into:

Dislocation asphyxia – it occurs when the root of the tongue goes back and the trachea entrance is blocked. It is found in double, multiple, defective and splintered fractures of the mandible where, along with the dislocation of the fragments, muscles lose their insertion point and the root of the tongue is displaced backwards. A similar type of asphyxia can also occur with total maxillary fractures with heavy backward displacement and nasopharyngeal occlusion.

Obstructive asphyxia - occurs when the upper or middle airways are blocked by a foreign body (tooth, bone fragment, projectile, part of denture, coagulum). The foreign body may enter the larynx, the bifurcation of the trachea or the bronchi.

Stenotic asphyxia – develops in narrowing of the upper department of the larynx and the trachea due to external pressure (neck hematoma, emphysema due to injured trachea).

Valvular asphyxia - occurs when the trachea entrance is blocked by injured soft tissues of the soft palate or mesopharynx. When inhaling, the detached soft tissue adheres to the opening and blocks it partially; the exhalation is free.

Aspiration asphyxia - develops due to obstruction of the lower airways by fluid content (blood, vomited matter).
Clinical presentation: Clinical symptoms of asphyxia are various, occurring in 5 stages: pre-asphyxia, blue asphyxia, white asphyxia, terminal respiration, terminal automatic heartbeat.

Management: To save the life of a patient with breathing difficulties due to trauma in the maxillofacial area, proper management by the dental practitioner is of utmost importance; the patient shall be placed in adequate position and should be timely referred to specialized hospital treatment. In asphyxia due to injuries in the maxillofacial area, the following activities should be carried out in the specified sequence:

- Placing the injured person in position of pronation or semipronation. When the chest is injured, the patient should be placed in supine position, with his head turned to the side. This prevents blood, as well as possible vomited matter, from entering the trachea and the bronchi.
- Cleaning of the oral cavity – using instruments or with fingers wrapped in gauze, the oral cavity should be cleaned in the event of obstructive asphyxia.
- Pulling and fixing the tongue - in dislocation asphyxia.
- Adjustment and suturing soft tissue wounds on the soft palate or mesopharynx in valvular asphyxia,
- In the absence of respiration, artificial respiration - mouth-to-tube or AMBU.
- temporary immobilization of fractured jaw fragments
- coniotomy or tracheotomy by an ENT specialist
- intubation (orotracheal or nasotracheal) by an anesthesiologist

HEMORRHAGE

Hemorrhage is bleeding from a blood vessel when the integrity of its wall is impaired.

Classification: In injuries in the maxillofacial area and the oral cavity, different types of bleeding occur:

- primary - occurs as an immediate result from the injury
• secondary – results from additional impairment of blood vessels by sharp edges of bone fragments, breakdown of thrombus or hematoma
• early secondary - develops 2-5 days after the injury
• late secondary - appears 10-12 days after the injury (purulent destruction of thrombus, vascular wall necrosis)
• arterial - blood flows as a strong pulsating jet and is of bright red colour. They are extremely dangerous because blood runs out at a rate 6 times that of venous bleeding. When the vessel is totally interrupted, bleeding is weak (the vessel shrinks), and if it is injured, bleeding is heavy due to leakage from the vessel.
• venous - the blood flows in a weak spray, pulsation is absent and blood colour is dark. The leaking blood does not spray, but fills the wound like a lake.
• capillary - appears in the form of small droplets and covers the injured area. It occurs in superficial skin damage.

Clinical presentation: The dental practitioner can detect and diagnose external bleeding only. Depending on the type and size of the injured vessel, the rate and amount of blood loss, the age and general condition of the patient, clinical symptoms are different in individual patients. Thus, in adult patients, slow bleeding and significant amounts of blood loss (15-20%) do not result in significant changes in the body's condition. In children, even small amounts of blood loss (150-200 mL) may be fatal. With rapid blood loss and blood loss over 20-25%, life threatening changes occur in the body (see hemorrhagic shock).

Management: It is necessary to quickly diagnose external bleeding and rapidly control it, which is achieved by artificial hemostasis, which is:

- Temporary. In the absence of experience and impossibility to achieve definitive control.
  - Wound swab
    It stops small arterial, venous and capillary bleeding. The swab can stay for up to 48 hours due to the risk of wound infection.
  - Compressive dressing.
    It stops small arterial and venous bleeding.
  - Digital compression

Depending on the size of the injured vessel, it is performed with a finger directly in the wound (venous and small arterial bleeding) or finger pressing the artery supplying the respective area at a distance from the bleeding (proximal). So, there are certain places for the different arteries of the face: Common carotid artery – pressing using 4 fingers in carotid triangle on transverse process of cervical vertebra 6; facial artery - pressing on the edge of the mandible in front of the insertion point of masseter muscle; Superficial temporal artery - pressing in front of the ear on the zygomatic arch; frontal artery - pressing on supraorbital foramen.
Definitive

This should be performed in a health care institution with available instruments and relevant indications.

- mechanical or medication effect on the wound (capillary, arterial or venous bleeding) - gauze, Gelaspon, Penga, Hemarcol, hydrogen peroxide, RAMBA
- electrocoagulation - in arterial and venous vessels with small diameters
- smashing of the vessel - in small arterial and venous vessels
- torsion of the vessel - small arterial and venous vessels
- ligature - stopping bleeding with a hemostatic tool and ligating arterial and venous vessels of medium diameter
- suture – used in an uninfected wound and a vessel with a large diameter (carotid artery, internal jugular vein). Lateral or circular suture should be used.

TRAUMATIC (HEMORRHAGIC) SHOCK

Shock is a complex of severe disorders of the body’s most important systems: blood circulation, respiration and metabolism.

Etiology and pathogenesis: In maxillofacial trauma, shock may occur due to severe irritation of the nervous system (pain) or to blood loss (hemorrhage) in muscle, soft tissue and jaw injuries. In the first case, shock is called traumatic, and in the second case - hemorrhagic (hypovolemic) shock.

Impaired microcirculation processes, such as distribution of blood in the blood pools (liver, spleen, muscles), underlie traumatic shock. In hemorrhagic shock caused by acute blood loss (over 20-25% of circulating blood), the volume of circulating blood decreases. In both cases, the resulting changes cause a reduction in the volume of circulating blood and hypovolemia. The body compensates the reduced amount of blood entering the heart (reduced venous return) with increasing tachycardia. The inability to maintain proper blood circulation with increased heart rate is compensated by peripheral vascular spasm. Hypovolemia stimulates the sympathoadrenal system and adrenaline and noradrenaline are released. This results in a peripheral vascular spasm as a compensatory mechanism supporting arterial blood pressure and sending blood to vital organs (brain, heart, liver and kidneys). This condition is defined as "centralization of blood circulation”. Peripheral vascular spasm causes paleness of the skin and appearance of cold drops of sweat. Elevated catecholamine levels reduce renal circulation and lead to oliguria. Hyovolemia results in reduced flow of oxygen to tissues and, thus, in hypoxia. Hypovolemia results in changes in the capillary walls, increased blood clotting and viscosity, and intravascular aggregation (adherence) of formed blood elements occurs.

Hypovolaemia and hypoxia cause disturbance in cell gas exchange and accumulation of large amounts of underoxidized products (histamine, lactic acid) and development of acidosis.
They, in turn, increase the permeability of blood vessels and aggravate the existing hypovolemia. Continuous hypovolemia results in decompensation of vessels and stasis (blood congestion). Hypovolemia, hypoxia and stasis result in the development of hypercoagulation. Thus, the insufficient blood supply of tissues is exacerbated, and if urgent measures are not taken, processes become irreversible.

Clinical presentation: Clinical development of traumatic (hemorrhagic) shock includes 2 periods:

**Erectile phase** - not always perceptible. It occurs immediately after the trauma and is characterized by agitation (motor and speech), anxiety, fear, rapid respiratory movements (rhythmic and regular), accelerated and rhythmic heart activity, elevated blood pressure (by 15-20 mm). The duration of this period is from several seconds to several minutes.

**Apathetic (torpid) phase** - excitement turns into retention, arterial blood pressure drops (by 30 mm from the normal level for the patient), heart rate accelerates. The patient is conscious but is flaccid and answers to the questions asked with difficulty. Patient’s skin is pale with cold drops of sweat on the face, patient’s eyes are sunken, and pupils are enlarged. Arterial blood pressure drops (60/30 mm Hg and less), heart rate is increased (130-140/min). Respiration becomes superficial and rapid (30-40/min). This condition lasts for up to 1 hour, and if rapid action is not taken, consciousness is altered and temporarily absent. Depending on the occurring changes, we distinguish 4 classes of hypovolemic shock (Table 31).

Shock changes last for more than 30 minutes and affect:

- **cardiovascular system** - low blood pressure (30 mm less than baseline), accelerated or filiform heart rate
- **skin** - pale, cyanotic, with cold drops of sweat on the face
- **CNS** - flaccid, hyposthenic, indifferent sight
- **kidneys** - reduced diuresis (oligo- to anuria)
- **blood** - presence of lactic acid above the upper limit of the normal (0.96-1.79 mmol/L).
### Classes of Hypovolemic Shock by de La Fontaine, P. Unger, P. F. (1991)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
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<tr>
<td><strong>Blood loss</strong></td>
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<td></td>
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</tr>
<tr>
<td>in %</td>
<td>&lt; 15</td>
<td>15 - 30</td>
<td>30 - 40</td>
<td>&gt; 40</td>
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<td>in mL</td>
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<td>570 - 1500</td>
<td>1500 - 2000</td>
<td>&gt; 2000</td>
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<td>=</td>
<td>=</td>
<td>↓</td>
<td>↓↓</td>
</tr>
<tr>
<td>diastolic</td>
<td>=</td>
<td>orthostatic ↓↑</td>
<td>↓</td>
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<tr>
<td><strong>Heart rate</strong></td>
<td></td>
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<tr>
<td>systolic</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>diastolic</td>
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<td></td>
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<td></td>
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<tr>
<td><strong>Respiratory rate</strong></td>
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<td>&gt; 20/min</td>
<td>&gt; 30/min</td>
<td>&gt; 35/min</td>
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<td><strong>Urine flow rate (mL/h)</strong></td>
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<td>20 - 30</td>
<td>10 - 20</td>
<td>0 - 10</td>
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<td><strong>Limbs</strong></td>
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<td>pale</td>
<td>pale</td>
<td>pale and cold</td>
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<td><strong>Skin colour</strong></td>
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<td>pale</td>
<td>waxy</td>
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<td>restless, aggressive, depressed</td>
<td>depressed unconscious</td>
</tr>
</tbody>
</table>

**Diagnosis:** Diagnosis is based on evidence from:
- medical history and clinical presentation
- central venous pressure (normal limits: 5-12 mm water column)
- diuresis (depends on the volume of kidney blood flow) - urinary catheterisation should be performed in each patient in shock. Normal limits 25-50 mL/h. In shock, due to impaired hemodynamics, 2-10 mL/h of urine is released or urine is absent (anuria).

**Management:** Urgent care in hypovolemic shock aims:
- immediate detection and stopping of external bleeding (compression dressing, manual compression)
- placing the injured person in the right position - pronation or semipronation
- transportation to a hospital

**Treatment:** Treatment in shock aims:
- Improving microcirculation processes and maintenance of oncotic pressure in the plasma
- prevention of intravascular aggregation and thrombosis and including the accumulated blood in the active blood flow
- maintaining the water-electrolyte and alkaline-acid balance in the blood
- detoxifying action
- increasing the volume and improving the rheological properties of the blood

Treatment of shock should **begin immediately with i. v. infusion of saline** and hemotransfusion solutions with hemodynamic and rheological effect (Ringer's lactate...
solution, Periston, Macrodex, Hemodez, Dextran, Rheopolyglukin, 4% albumin, gelatin - Physiogel).

According to the American College of Surgeons Committee on Trauma, 1997, treatment should be chosen based on hemodynamic parameters, and in 70 kg individuals, the following is recommended:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>blood loss (mL)</td>
<td>up to 750</td>
<td>750 - 1500</td>
<td>1500 - 2000</td>
<td>&gt; 2000</td>
</tr>
<tr>
<td>blood loss (% vol)</td>
<td>up to 15</td>
<td>15 - 30</td>
<td>30 - 40</td>
<td>&gt; 40</td>
</tr>
<tr>
<td>heart rate</td>
<td>&lt; 100</td>
<td>&gt; 100</td>
<td>&gt; 120</td>
<td>&gt; 140</td>
</tr>
<tr>
<td>arterial blood pressure</td>
<td>normal</td>
<td>normal</td>
<td>reduced</td>
<td>reduced</td>
</tr>
<tr>
<td>pulse pressure</td>
<td>normal or elevated</td>
<td>reduced</td>
<td>reduced</td>
<td>reduced</td>
</tr>
<tr>
<td>respiratory rate</td>
<td>14 - 20</td>
<td>20 - 30</td>
<td>30 - 40</td>
<td>&gt; 35</td>
</tr>
<tr>
<td>urine released (mL/h)</td>
<td>&gt; 30</td>
<td>20 - 30</td>
<td>5 - 15</td>
<td>negligible</td>
</tr>
<tr>
<td>mental status</td>
<td>mild anxiety</td>
<td>moderate anxiety</td>
<td>anxious, confused</td>
<td>confused, lethargic</td>
</tr>
<tr>
<td>fluid replacement*</td>
<td>crystalloids</td>
<td>crystalloids</td>
<td>crystalloids and blood</td>
<td>crystalloids and blood</td>
</tr>
</tbody>
</table>

* 300 mL electrolyte solution per each 100 mL of blood loss.

Blood transfusion in traumatic (hemorrhagic shock) should be administered only in blood loss over 20% of circulating blood or if hematocrit is below 30%, but one should never start with blood transfusions. Blood increases the intravascular aggregation of formed elements and can deepen the shock. Treatment is a difficult and responsible task and is conducted in hospital setting. However, it is important to know how to give first aid in order to save the life of the injured person.
CLOSED CRANIOCEREBRAL TRAUMAS
(Dimitar Atanasov)

Etiology: Closed cranioencebral lesions are found in various types of injuries (occupational accidents, non-occupational accidents, wartime injuries), and 70% of the cases are due to road accidents. Cranioencebral traumas accounts for 10-40% of all injuries, with mortality of 5-20% (Popov, V. L., 1998).

Clinical presentation: Clinical presentation of some cranioencebral traumas (CCT) was first described by Hippocrates in his work "On Wounds in the Head", where he suggest the term "commotio cerebri". The subdivision of closed CCT into concussion, compression and contusion was suggested by Boriel (1677) and Littre (1705).

Nowadays, the clinical symptoms of cranioencebral traumas can be subdivided into 5 groups:

1. General surgical symptoms - different types of lesions of the soft and hard tissues of the head (bruising, hematoma, wound, fracture) found during visual inspection and clinical examination (palpation).

2. General cerebral symptoms - The objective study finds the following:
   ♦ Changes in consciousness - quantitative and qualitative changes are observed
     • obnubilation - Announcement - verbal contact is maintained but the patient does not remember what happened, is absent-minded, has difficulties to concentrate, easily gets mentally tired
     • somnolence - verbal contact with the present, but responses are short, the patient falls asleep easily, awakens easily under the influence of external stimuli
     • sopor sopor - the highest level of altered consciousness; the injured person is in a state of deep sleep and responds only to strong pain stimuli. Verbal contact is impossible. Stereotyped automatic movements observed, such as scratching, removing a dressing, covering with a blanket. Pelvic reservoirs cannot be controlled. Sometimes the patient is excited.
     • coma - the patient is unconscious, secondary contact is impossible. There are three levels of coma - shallow, deep and coma dépassé.
   ♦ Memory disorders
     • retrograde amnesia - no memory of what happened
     • anterograde amnesia - no memory after the trauma
   ♦ Impairment of instinctive reflexes and vegetative functions
     • nausea, vomiting, headache, vertigo, fainting
     • impaired pupil and eyelid reflexes, impaired respiratory functions, blood circulation, temperature and metabolism

3. Focal neurological symptoms - their presence is always indicative of organic brain damage. These are present in the event of impairment of the functions of various cranial
nerves, impairment of motor, sensory or speech functions, changes in pupillary response, presence of neck stiffness and symptoms of meningradicular irritation.

4. **Radiological changes** - skull fracture and intracranial hematoma.

5. **Changes in the cerebrospinal fluid** - blood in the cerebrospinal fluid, elevated pressure of the cerebrospinal fluid.

Glasgow Coma Scale is used to determine the neurological status of the injured person (Tesdale, G. Jennett, B., 1974):

**Glasgow Coma Scale**

<table>
<thead>
<tr>
<th>clinical indicators</th>
<th>scoring</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Eye response</td>
<td></td>
</tr>
<tr>
<td>• Opens eyes spontaneously</td>
<td>4</td>
</tr>
<tr>
<td>• Opens eyes in response to voice</td>
<td>3</td>
</tr>
<tr>
<td>• Opens eyes in response to painful stimuli</td>
<td>2</td>
</tr>
<tr>
<td>• Does not open eyes</td>
<td>1</td>
</tr>
<tr>
<td>2. Motor response</td>
<td></td>
</tr>
<tr>
<td>• Obeys commands</td>
<td>6</td>
</tr>
<tr>
<td>• Localizes to painful stimul</td>
<td>5</td>
</tr>
<tr>
<td>• Flexion/Withdrawal to painful stimuli</td>
<td>4</td>
</tr>
<tr>
<td>• Abnormal flexion to painful stimuli</td>
<td>3</td>
</tr>
<tr>
<td>• Extension to painful stimuli</td>
<td>2</td>
</tr>
<tr>
<td>• No motor response</td>
<td>1</td>
</tr>
<tr>
<td>3. Verbal response</td>
<td></td>
</tr>
<tr>
<td>• Oriented</td>
<td>5</td>
</tr>
<tr>
<td>• Disoriented</td>
<td>4</td>
</tr>
<tr>
<td>• Incoherent words</td>
<td>3</td>
</tr>
<tr>
<td>• Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>• No verbal response</td>
<td>1</td>
</tr>
</tbody>
</table>

Patient score determines the degree of neurological changes:

15 = normal; 13-14 = mild impairment;

12 = moderate impairment; 3-8 = severe impairment

**CEREBRAL CONCUSSION**

Only brain dysfunction is found in concussion of the brain; no focal neurological symptoms, no radiographic changes and no changes in the cerebrospinal fluid. It is characterized by a brief loss of consciousness - from seconds to minutes or hours. Only general brain symptoms are present:

- **Changes in consciousness** - they are mild and are in the range of mild altering of consciousness to shallow coma. The most important clinical presentation is loss of consciousness. It is a protective mechanism against disorders in blood and cerebral
fluid circulation. The underlying process is suppression that protects ganglion cells in the cerebral cortex and the brainstem against severe exhaustion. After regaining consciousness, somnolence, mental and physical fatigue, reduced interest, and emotional lability are observed; these are transient and soon resolve.

- **Memory impairment** - retrograde (patients do not remember anything they did before the trauma) and anterograde amnesia are not always present, but if they occur, they have short duration (up to 1-2 hours)

- **Impairment of instinctive reflexes** – headache is almost always present, a symptom found in more than 80% of patients. In the majority of patients the headache is generalized, and in more rare cases localised at the site of the trauma. It becomes more severe in an upright position, and upon physical or mental overload. Dizziness is found – it occurs in 30-90% of the patients and is one of the pathognomonic symptoms of concussion of the brain. There are symptoms of nausea and vomiting in the first hours and days after the trauma, which are not related to food intake but are related with the headache. Patients often have complaints of eyebrow pain and dizziness.

- **Autonomic manifestations** - changes in heart rate (moderate bradycardia and rarely tachycardia), vasomotor lability with a tendency to orthostatic collapse, occasionally hyperhydrosis, are observed. Symptoms depend on whether the sympathetic nervous system (tachycardia and elevated arterial blood pressure) or parasympathetic nervous system (bradycardia and hypotension) prevail.

CEREBRAL CONTUSION

Functional and morphological (necrosis, hemorrhage, edema) changes in the brain are observed, and the 5 groups of changes are manifested:

- **General surgical symptoms** - there are wounds or skull fractures, and the presence of cerebral detritus or leakage of cerebrospinal fluid is always indicative of cerebral contusion.

- **General cerebral symptoms** - more pronounced than in cerebral concussion, and are the result of developing diffuse cerebral edema and intracranial hypertension. Memory impairment is common - retrograde or anterograde amnesia encompassing a longer period of time. Significant changes in breathing, hemodynamics and body temperature regulation are observed. Consciousness is changed to varying degrees - from mildly altered to deep coma. After the recovery of consciousness, patients have long-standing complaints of headache, nausea and vomiting, mental fatigue (the so-called cerebrostenic syndrome develops).

- **Focal neurological symptoms** – they are extremely diverse - from isolated neurological symptoms (involvement of cranial nerves) to neurological syndromes (motor, sensory, meningeal changes).

- **Radiological** - about two-thirds of patients with cerebral contusion have skull fractures.

- **Changes in cerebrospinal fluid** - a pathognomonic sign of cerebral contusion is the
presence of blood in the cerebrospinal fluid. Elevated pressure of cerebrospinal fluid is observed.

False positive result may be obtained due to the puncture needle. Therefore, testing cerebrospinal fluid for presence of blood should be carried out approximately three hours after the injury, following centrifugation.

CEREBRAL COMPRESSION

Cerebral compression is mainly due to intracranial hemorrhage - epidural, subdural, intracerebral. Clinical recognition of the type of hemorrhage is extremely difficult and impossible, and all types have four major syndromes:

• **Traumatic injury syndrome** - only the presence of a direct injury of the head (wound, hematoma) is found.

• **Primary craniocerebral trauma syndrome** - there is evidence of a cerebral concussion or cerebral contusion, and only in rare cases the presence of primary CML is debatable. The clinical presentation of intracranial hemorrhage is superimposed on cerebral concussion or compression.

• **Syndrome of increasing intracranial pressure** (hypertensive) - this is the major syndrome and it includes general cerebral symptoms. Changes in level of consciousness are typical.

• **With an atypical lucid interval** - without evidence of changes in consciousness immediately after the injury. In different periods of time (minutes, hours, days, weeks, months), the patients become sleepy, confused, and coma, impairment of vital functions and death occur gradually or quickly.

  ✓ **With clinical lucid interval** - the short-lasting loss of consciousness after the injury resolves quickly and after a different period of time, signs of impairment of consciousness appear.

  ✓ **With semi-lucid interval** - the consciousness lost after the injury is restored not completely, but partially (e. g. from coma to somnolence). After a certain period of improvement, patient’s condition worsens again

• **With a progressive deepening of the initially impaired consciousness**, without lucid intervals. This condition is characterized by a rapid evolution and poor prognosis.

In case of hematoma, the impairment of vital functions in the initial phases depends on the severity of the craniocerebral trauma. When it is mild, compression is typically accompanied by the presence of bradycardia (40/min) and elevated arterial blood pressure. This is the so called vagotonic period or brainstem compensation - reflex increase in arterial blood pressure overcomes compression in brain vessels and provides satisfactory perfusion pressure. If the patient is not treated, the so-called vasoparalysis period (brainstem decompensation) occurs, with fulminant drop in blood pressure, tachycardia, breathing abnormalities (Cheyne–Stokes respiration).
**Cerebral compression syndrome** - two types of focal neurological symptoms are observed:

- **local** – of hemi type, with or without sensory changes
- **general** – up to respiratory arrest and cardiac arrest

The exact location and size of the hematoma can be found by radiography (CT).

Analysis of cerebrospinal fluid shows presence of blood, with elevated pressure of cerebrospinal fluid.

**Epidural Hematoma** - blood is accumulated between the bone and the tough outer membrane of the brain. It occurs in fractures of the temporal bone and laceration of the branches of the middle meningeal artery or the venous vessels on the inside of the tough outer membrane of the brain. A limited hematoma is formed, which gradually increases.

The lucid interval is short - from hours to days, and in severe injuries it may be absent and is superimposed on the contusion syndrome.

Patients complain of headache, nausea, vomiting - as a result of increased intracranial pressure. Early-onset focal symptoms such as monoplegia, hemiplegia, seizures also occur. Symptoms of impairment of the brainstem - paralysis of cranial nerves II, IV, and VI on the hematoma side develop rapidly. There are no changes in the cerebrospinal fluid.

**Subdural Hematoma** - blood is accumulated between the tough outer membrane of the brain and the arachnoid membrane due to laceration of venous vessels in this area.

It develops slowly and there is a lucid interval, depending on which subdural hematoma is divided into:

- acute subdural hematoma - a lucid interval of up to 1 week
- subacute subdural hematoma - a lucid interval of 2 to 3 weeks
- chronic subdural hematoma - a lucid interval of over 3 weeks

**Intracerebellar Hematoma** - due to laceration of vessels in the brain matter (arteries or veins). It develops with a short lucid interval or lucid interval is absent. Focal symptoms appear before general cerebral symptoms.

**Fracture of the skull base**

Fracture of the skull base may be in the region of the anterior, middle or posterior cranial fossa:

- **Anterior cranial fossa** – clinical presentation is bleeding from the nose or the nasopharynx without any direct injury of the nose; bleeding in the eye sockets - occurs unilaterally or bilaterally, several hours (rarely several days) after the injury, with more intense colouring in the periphery; presence of cerebrospinal rhinorrhea or cerebral detritus in the nasal cavity or nasopharynx; impaired olfaction – lacerated fila olfacioria; blindness or diplopia (impaired visual nerve or affected motor nerves of the eye).
- **Middle cranial fossa** - bleeding or cerebrospinal fluid leakage from the external auditory meatus. With preserved integrity of the tympanic membrane, the cerebrospinal fluid may leak along the Eustachian tube into the nose and deceive the
physician that there is a fracture of the anterior cranial fossa; impairment of the facial nerve; reduced hearing; impaired equilibrium; affecting the trigeminal branches

- **Posterior cranial fossa** - presence of bruising of the skin in the area of mastoid process, paresis and paralysis of cranial nerves.

*Diagnosis:* Diagnosis of closed craniocerebral trauma is based on patient’s history, objective clinical study, paraclinical tests (cerebrospinal fluid and radiographic study) and consultation with a neurosurgeon.

*Differential diagnosis:* Differential diagnosis is made between:

- **Cerebral concussion, contusion and compression**

  In the first two conditions, the symptoms are most pronounced at the time of injury and immediately after it, and then they regress. In compression, at first there is a concussion syndrome, and after it resolves, symptoms of increasing intracranial pressure are added gradually. Hematomas, prior to loss of consciousness, are characterized by local pain upon percussion of the skull on the side of the hematoma. The headache is also on the side of the hematoma. Mydriasis is present.

  *Treatment:* The dental practitioner should be familiar with closed craniocerebral traumas, and should be able to diagnose such traumas; final diagnosis and treatment is performed by a specialist neurosurgeon.

  **In cerebral concussion,** complete bed rest for 1-3 weeks is indicated, and alcohol, coffee and cigarettes should not be consumed. Restriction of external irritation - noise, music, bright and shimmering light (TV). Analgesic (non-steroidal anti-inflammatory) agents should be prescribed.

  **In cerebral contusion,** depending on the severity of the injury, the options are rest for at least 3 weeks, conservative intensive treatment or surgical treatment (removal of necrotic foci).

  **In cerebral compression,** the only option is neurosurgical treatment (trepanation of the skull and evacuation of the hematoma).

  **In skull base fractures,** treatment depends on the concomitant cerebral complications (concussion, contusion, compression).
PRENEOPLASTIC DISORDERS
(Petia Pechalova)

The concept of ‘precancer’ begins in 1805 with a suggestion given by a European panel of physicians that there are benign diseases which will always develop into invasive malignancy if followed for a long time. The term „precancer“ was first coined in 1875 by Victor Babes, a Romanian physician. This concept later widened to include a number of diseases in various organ systems. Junctional nevi and xeroderma pigmentosa of the skin, leukoplakia and papillomas of the urinary bladder and larynx, polyps of the colon and solitary adenomas of the thyroid are some of the lesions thought to be precancerous. Oral precancer, in particular, have a rich and fascinating literature extending as far back as 1870s, when Sir James Paget, proposed that „leukokeratosis“ or „smokers patch“ of the hard palate (nicotine palatinus) or the tongue in innervate pipe smokers carried an increased risk of eventual cancer transformation. Subsequently in the literature, various terminologies appeared in relation to the “precancer” concept like “premalignant”, „preneoplastic“, „carcinoma prone“, „intra-epithelial neoplasia“ etc. Recently proposed term is “potentially malignant disorders” for the individuals with no known predisposing disorders or any clinically evident lesion but oral mucosa may be susceptible to cancer.

There are two types of potentially malignant disorders:

- a “precancerous lesion“ - a morphologically altered tissue in which cancer is more likely to occur than in its apparently normal counterpart (leukoplakia, erythroplakia)
- a “precancerous condition“ - a generalized state associated with a significantly increased risk of cancer (lichen planus, actinic cheilitis, discoid lupus erythematosus, oral submucous fibrosis, sideropenic dysphagia or Plummer-Vinson syndrome, melanoplakia, keratoacanthoma, cornu cutaneum)

The etiology of precancerous lesions of oral mucosa is not well-known. Some risk factors such as tobacco chewing, tobacco smoking, and alcohol play an important role in development of potentially malignant oral conditions. The clinical concept of oral epithelial premalignancy ismore than 150 years old. Sir James Paget first described an association between an oral lesion (“ichthyosis”) and the subsequent development of tongue carcinoma. Nineteen years earlier, Paget had found that white oral mucosal patches in pipe smokers bore a risk of transforming into cancer. Seven years after Paget’s report, Schwimmer also described white lesions of the tongue, which developed into cancer in patients with tertiary syphilis, which he termed “leukoplakia”.

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Leukoplakia is defined as “a white plaque that cannot be removed easily or recognized as another clinicopathological entity.” Oral leukoplakia presents as white, thick patches on the oral mucosa due to hyperkeratosis of the epithelium. The prevalence of oral leukoplakia varies between 1.1% and 11.7%, with a mean value of 2.9%.

Although leukoplakia can occur at any age, it often occurs in individuals under the age of 40.

Leukoplakia showed hyperkeratosis and/or acanthosis without or with evidence of dysplasia.

Leukoplakia is seen six times more among smokers than among non-smokers. Clinically, leukoplakia may affect any part of the oral and oropharyngeal cavity and can be divided into two subtypes including homogeneous and non-homogeneous types. Homogenous lesions are characterized by uniformly flat, thin, uniformly white in colour and shows shallow cracks of the surface keratin. Non-homogenous lesions have been defined as a white and red lesion (known as erythroleukoplakia) that may be either irregularly flat (speckled) or nodular. Verrucous leukoplakia is yet another type of non-homogenous leukoplakia.

Leukoplakia is strictly a clinical term. Its use as a microscopic diagnosis is inappropriate. This clinical entity shows a constellation of microscopic findings, some of which fall in the dysplastic or even invasive realm. Leukoplakia can show a range of pathological changes histologically, and the only way to assess the seriousness of the lesions is by performing a biopsy to determine the presence or absence of dysplasia, and if it’s present, its severity. Leukoplakia can receive malignant transformation. Risk factors of malignant transformation of leukoplakia are

1) Female gender
2) Long duration of leukoplakia
3) Leukoplakia in non-smokers
4) Location on the tongue and/or floor of the mouth
5) Size > 200 mm²
6) Non-homogenous type
7) Presence of epithelial dysplasia

The differential diagnosis of leukoplakia includes aspirin burn, chemical injury, oral pseudomembranous and hyperplastic candidiasis, frictional lesions, oral hairy leukoplakia, leukoedema, linea alba, lupus erythematosus, morsicatio buccarum, papilloma and allied lesions, mucous patches in secondary syphilis, tobacco-induced lesions, smoker’s palate (nicotinic stomatitis), stuff-induced lesion, white sponge nevus, oral lichen planus, and lichenoid reaction.

Diagnosis and treatment: Oral leukoplakia should be confirmed by mucosal biopsy at first. The treatment is full removal of the lesion. The most commonly preferred treatment options are surgical excision or CO₂ laser therapy. In widespread lesions, photodynamic therapy may be considered. Cryotherapy is another preferred destructive method. Non-surgical treatment modalities might be considered in selected patients. Carotenoids (β-carotene, lycopene), vitamins [L-ascorbic acid (vitamin C), α-tocoferol (vitamin E), retinoic acid (vitamin A), and fenretinide], and bleomycin may be used in patients with oral leukoplakia.

Erythroplakia is defined as “a fiery red patch that cannot be characterized clinically or pathologically as any other definable disease”. Clinical appearance is characterized by flat or even depressed erythematos change of the mucosa without a patch lesion. Prevalence of erythroplakia varies between 0.02% and 0.83%. It mainly occurs in the middle aged and the elderly. Male gender is most frequently affected. A red lesion of the glans penis in a syphilitic man that evolved into cancer was designated “erythroplasia” by Queyrat. In 1924, Darier first described a red oral mucosal lesion with the potential to turn into cancer and designated this the “erythroplasia of Queyrat”.

Oral erythroplakia presents as a solitary lesion of the mucosa of the oral cavity. The most commonly affected areas are the soft palate, the floor of the mouth, and the buccal mucosa. Clinically, typical lesion of oral erythroplakia is less than 1.5 cm in diameter. Histopathologically, moderate or severe dysplasia was usually seen in lesion with erythroplakia. Malignant transformation rates is very high (vary from 14% to 50%).

Owing to the high malignant transformation rate, early effective treatment is mandatory. Surgery, either by cold knife or by laser, is the recommended therapy. Surgical excision may be used in lesions with severe epithelial dysplasia or carcinoma in situ.
Oral erythroplakia should be diminished from any disease which clinically appears red colour in oral cavity. Oral candidiasis, oral histoplasmosis, oral tuberculosis, atrophic oral lichen planus, lupus erythematosus, pemphigus, pemphigoids, amelanotic melanoma, haemangioma, telangiectasia, lingual varices, Kaposi’s sarcoma, early squamous cell carcinoma, local irritation, mucositis, drug reaction, median rhomboid glossitis, and oral purpura may be confused with oral erythroplakia.

Lichen planus was first described by Erasmus Wilson in 1869. The disease is a chronic, autoimmune, inflammatory disease which may affect skin, oral mucosa, genital mucosa, scalp, and nails. Prevalence of oral lichen planus varies from 0.5% to 3%. It mainly occurs among female gender and the age of onset is usually between third and sixth decade. Although it is believed that oral lichen planus is a T-cell mediated autoimmune disease, its cause is partially understood in most cases. Several factors have been proposed for the etiology including genetic background, dental materials (amalgam, metals, gold, and composite restorations), drugs (especially antimalarials, cardiovascular agents, gold salts, non-steroidal anti-inflammatory drugs, hypoglisemics), infectious agents (herpes simplex virus, Epstein-Barr virus, cytomegalovirus, herpes virus-6, hepatitis-C virus, and human papilloma virus), autoimmunity, immunodeficiency, food allergies, stress, habits, trauma, diabetes and hypertension, malignant neoplasms, and bowel disease. Oral lichen planus may affect any part of the oral mucosa, most commonly affected areas are dorsum of the tongue, buccal mucosa, and gingiva. Clinically, oral lichen planus may be seen as six types including:

1. papular,
2. reticular,
3. plaque-like,
4. atrophic,
5. erosive, bullous type.

The most common type is the reticular pattern which present as fine white striae known as “Wickham’s striae”. Typically, lesions present symmetrically and bilaterally, and usually asymptomatic.

Atrophic pattern presents as a red lesion. Erosive pattern is usually seen as irregular erosion or ulceration covered with a fibrinous plaque or pseudomembrane. Both atrophic and erosive pattern are generally associated with a burning sensation and pain that exacerbated by trauma and hot, spicy or acidic foods.
The papular pattern, which is rarely seen, is characterized by small, white, raised papules with fine white striation at the periphery of the lesion.

Plaque type clinically resembles leukoplakia because of its homogenous white nature. The dorsum of the tongue and buccal mucosa are the most affected areas in the oral cavity of patients with plaque type oral lichen planus. Multifocal plaque type lesions may be seen. This subtype is more common among tobacco smokers.

The first case of oral lichen planus-related oral carcinoma was reported by François Henri Hallopeau in 1910. Malignant transformation ratio has been reported in 0% to 10% of patients. Increased malignant transformation risk occurs greater in erosive and atrophic forms and in cases of lesions of lateral border of the tongue.

*Diagnosis:* If Wickham’s striae are observed, the diagnosis is easy and can be made clinically, especially reticular pattern of oral lichen planus. But erosive or atrophic pattern need to be confirmed by biopsy in order to make the correct diagnosis. Direct immunofluorescence may be useful to distinguish from some bullous diseases such as pemphigus vulgaris, benign mucous membrane pemphigoid, and linear immunoglobulin A (IgA) bullous dermatitis.

*Treatment:* Patients with reticular and other asymptomatic oral lichen planus can be followed without treatment. But if there are any symptoms and/or potential malignant risk, lesions should be treated. Both topical and systemic treatment modalities have been reported for oral lichen planus: (1) topical treatments, (2) systemic treatments, (3) surgery. For topical treatment appropriate medications include corticosteroids (triamcinolone, fluocinolone acetonide, fluocinonide, clobetasol, fluticasone propionate, betamethasone sodium phosphate, mometasone furoate); cyclosporin; tacrolimus and pimekrolimus; retinoids (tretinoin, isotretinoin); Aloe vera; hyaluronic acid 0.2% gel. Surgical interventions are aimed to destruction of the lesion by excision, cryotherapy or CO\textsubscript{2} laser.

*Actinic cheilitis* is a potentially malignant disease of the lip caused by exposure solar radiation. It is commonly seen the surface area of the lower lip due to the anatomic proximity. In addition to solar rays, tobacco use, lip irritation, poor oral hygiene, and ill-fitting dentures may play a role in the development of actinic cheilitis. The disease predominantly occurs in men compared to the women. The actinic cheilitis shows erythema and edema in the early stages of the disease. Diffuse scaling, thickened epithelium with small greyish-white plaques, inflammatory areas, and linear fissures may present in the late stages of the disease.

Malignant transformation rate has been estimated ranging from 1.4% to 36% at an interval of 1 to 30 years. Diagnosis should be confirmed by biopsy to evaluate the degree of dysplasia. In treatment, 5-fluorouracil, scalpel vermilionectomy, chemical peel,
electrosurgery, cryosurgery, CO₂ laser, imiquimod, photodynamic treatment, diclofenac 0.3% gel can be preferred.

**Cutaneous horn** (cornu cutaneum) is unusual keratinous skin lesion with conical appearance. They are small in size, localized. The treatment is surgical excision or cryo-/CO₂ laser therapy.

**Keratoacanthoma** is a low-grade abnormal cell growth, which originates from the neckline of the hair follicles on the skin or the pilosebaceous glands and pathologically and clinically resembles the high-grade squamous cell carcinoma. When left untreated, Keratoacanthoma can grow as squamous cell carcinoma.

Keratoacanthoma growth is mainly found on skin that receive heavy exposure to sunlight and it affects the face, hands and forearms. The growth is characterized by dome shape, symmetrical structure and is surrounded by an inflamed skin with smooth wall. The skin is capped with debris and it has keratinized scales. One peculiar characteristic with this low-grade tumor is that it grows rapidly and can reach a large size in just a few days or weeks. Surgical excision is necessary.

**Melanoplakia:** Oral pigmentation is a relatively common condition that may involve any portion of the oral cavity. Multiple causes are known, and they may range from simple iatrogenic mechanisms, such as implantation of dental amalgam, to complex medical disorders, such as Peutz-Jeghers syndrome. Local irritants, such as smoking, may also result in melanosis of varying degrees. Oral pigmented lesions result from cellular hyperplasia that can range from benign nevi to fatal oral melanoma. Patients with oral malignant melanoma often recall having an existing oral pigmentation months to years before diagnosis, and the condition may even have elicited prior comment from physicians or dentists. Pigmented entities may arise from intrinsic and extrinsic sources. The color may range from light brown to blue-black. The color depends on the source of the pigment and the depth of the pigment from which the color is derived. Melanin is brown, yet it imparts a blue, green, or brown color to the eye. This effect is due to the physical properties of light absorption and reflection described by the Tyndall light phenomenon or effect.

Sometimes hyperpigmentation of the gingiva could be racial sign. Other causes of hyperpigmentation arre endocrine diseases.

Peutz–Jeghers syndrome is an autosomal dominant genetic disorder characterized by the development of benign hamartomatous polyps in the gastrointestinal tract and hyperpigmented macules on the lips and oral mucosa.
Nevus of Ota (nevus fuscoceruleus ophthalmomaxillaris) is a dermal melanocytic hamartoma that demonstrates bluish hyperpigmentation along the ophthalmic and maxillary divisions of the trigeminal nerve. Nevus of Ota is a benign melanocytic pigmentary disorder with rare malignant transformation potential. It can be either congenital or acquired during adolescence. Most patients have no positive family history.
PROPHYLAXIS AND EARLY DIAGNOSIS OF ORAL CANCER
(Petia Pechalova)

Oral cancer remains a highly lethal disease and is one of the most debilitating and disfiguring of all malignancies. It presents the whole dental team with important obligations, challenges and a real opportunity to save a life. Squamous cell carcinoma of the oral cavity (oral cancer) accounts for approximately 3% of all cancers worldwide. Oral cancer includes cancers of the lip, tongue and rest of the oral cavity, but not cancers of the major salivary glands. Those of the tonsil and oropharynx are included as oropharyngeal cancers. The highest incidence rates occur in three developing countries (Pakistan, Brazil and India) and one developed country (France). Incidence is higher in men than in women, in older compared with younger age groups and varies from region to region. Although the oral cavity is a potentially accessible site for examination, up to 50% of oral cancers are not detected until the disease is well advanced.

Recognised risk factors are tobacco smoking and alcohol consumption. Betel quid and smokeless tobacco chewing are also important risk factors in some populations, and human papillomavirus (HPV) infection appears to be a risk factor for younger populations. Other risk factors are age above 55 years, exposure to sunlight for long periods of time, an immune system that has been weakened by certain medications, kin disease as lichen planus, graft-versus-host disease or certain inherited conditions of the blood.

Oral cancer has a various clinical presentations including presens of white or red patches on lips, gum, tongue or mouth lining, a lump (mass) which can be felt inside the mouth or on the neck, pain or difficulty chewing, swallowing or speaking, hoarseness lasting a long time, numbness or pain in any area of the mouth that doesn’t heal, swelling of the jaw, loosening of teeth, difficulty wearing dentures, bleeding in the mouth, a sore (ulcer) on the lips or in the mouth that doesn’t heal. The problem is not simply that the number of new oral cancer cases is rising, as people continue to put themselves at risk through smoking and excessive drinking, but also that these cancers are being detected at an advanced stage. No other cancers have shown such significant increases in their incidence. Furthermore, treatment of many cancers is showing impressive improvement in survival, but oral cancer continues to have high death rates.

The first priority in management of oral cancer is primary prevention (prophylaxis). Secondary prevention means catching cancers early (early diagnosis). Tertiary prevention is to stopping recurrence and spread of cancer. Important part of dental practice is to provide special oral care for patients with other forms of cancer.

Incidence of oral cancer is increasing. Mortality is high: five year survival is around 50%. Dentists should identify those patients with a risk factor for oral cancer and supply a short information about the potential disease. All patients attending for routine care should receive an opportunistic oral soft tissue examination. Prophylaxis of oral cancer aims to
change behaviours (lifestyle) known to be associated with oral cancer including smoking cessation, reduces alcohol consumption, the benefits of good nutrition.

**Early diagnosis** of oral cancer has a key role in the treatment of the disease. The earlier the lesions are found, the greater the chance of a cure and of a good quality of life and function. A major problem is that more than half of all oral cancer cases have already metastasised to regional or distant structures at the time of detection, which decreases the five year survival rate.

**Screening** is defined as the application of a test or tests (including a clinical examination) to identify individuals who probably have a disease, in order to separate them from those who probably do not. A screening examination is not a diagnostic examination, but aims to identify abnormalities that should be referred for further investigation, diagnosis and management. Oral cancer screening is the process by which a practitioner evaluates an asymptomatic patient to determine if he is likely or unlikely to have a potentially-malignant or malignant lesion.

Some people who screen positive might, on further investigation, be found not to have the disease (*false positives*) while others might have a negative screen, but go on to develop the disease (*false negatives*). The aim is to keep false negatives and positives as low as possible - that is, to develop a test with high sensitivity and specificity. An oral mucosal examination looks for pre-symptomatic cancers or precancerous lesions which can be treated early to prevent progression of the disease.

Different types of screening could be apply for detecting the oral cancer - ‘**population based screening**’, when a population is assessed specifically for the purpose of detecting oral cancer, ‘**opportunistic screening**’, when patients who are attending a health care provider for another purpose are examined for signs of oral cancer or precancer, or ‘**targeted screening**’, when high risk individuals are selected for screening. In any of these contexts, along with a visual and tactile examination of the oral mucosa, the practitioner should ask the patient about their health history including tobacco and alcohol use. The risk of oral cancer is increased with age, alcohol and tobacco use and a history of upper aerodigestive tract cancer.

Detection of lesions may be enhanced by the use of adjunctive aids such as Toluidine blue, Light-based detection systems, Chemiluminescence (*ViziLite Plus; Microlux/DL, Orascoptic-DK*), Tissue fluorescence imaging (*VELscope*), Tissue fluorescence spectroscopy.

**Toluidine blue** staining is claimed to be a simple, inexpensive and sensitive adjunct tool for identifying early oral squamous cell carcinoma and high-grade dysplasias. Although toluidine blue has been found to be highly sensitive and moderately specific for malignant lesions, it is far less sensitive for premalignant lesions with false negative rates of up to 58% for identifying mild-to-moderate dysplasia. Toluidine blue has also been demonstrated to help assess the status of margins around oral cancer at the time of resection. Although toluidine blue test is helpful in identifying oral cancers, it should not be viewed as a substitute for biopsy, and a negative test does not preclude the presence of dysplasia or even oral cancer.
A protocol of usage of Toluidine blue for detection of oral cancer

The patient rinses the mouth consequently as follow:

1) with 1% acetic acid for 20 seconds to clean the area;
2) with plain water for 20 seconds;
3) with 1% aqueous toluidine blue for 60 seconds;
4) with 1% acetic acid for 20 seconds;
5) with plain water for 20 seconds.

Immediately after the end of rinses, dentist observes the patient and looking for dark blue lesions were considered as „positive“ results.

Diffused white light: Dentists routinely perform visual inspections of the oral cavity under regular white light. Healthy tissue is naturally more fluorescent and reflective when exposed to light. Mucosal abnormalities cause the cells to be non-reflective. Diffused white light enhances normal tissue’s natural fluorescence, creating a sharp contrast with the diseased tissue (abnormal tissue has a diffused vasculature), even in very early stages.

Autofluorescence and chemiluminescence have been studied as non-invasive in-vivo tools for the detection of (pre-)malignant tissue alterations. Autofluorescence of tissues under excitation with light is produced by several endogenous fluorophores. It was reported that autofluorescence spectroscopy may provide valuable information for diagnosis.

VELscope is a tool designed to assist in the discovery of soft tissue abnormalities in the mouth based on the loss of tissues autofluorescence. The blue light emitted from the VELscope causes the tissue to fluoresce. Normal tissue fluoresces in a particular pattern, and abnormal tissue shows up darker.

Chemiluminescent illumination with kit ViziLite: The patients were instructed to rinse their mouth with the ViziLite rinse recommended (1% acetic acid solution). They were asked to swish the rinse all over the mouth for 1 min. and expectorate the contents. The examination room was dimmed to minimize ambient light. The ViziLite capsule was activated and assembled with the ViziLite retractor. The oral cavity was re-examined using the illumination from the ViziLite assembly. The observations were recorded accordingly and duly photographed. The ViziLite device was discarded. The presence of "acetowhite" lesion after one minute rinse with 1% acetic acid solution was considered as a "positive" test.

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The term “benign” is used to describe both medical conditions and tumors and usually refers to a process that's not especially dangerous. The term “malignant” is often used synonymously with the word “dangerous” in medicine.

Benign tumors of soft tissue are more common than benign tumors of bone. They can occur at almost any site, both within and between muscles, ligaments, nerves, and blood vessels. These tumors vary widely in appearance and behavior. Among the most common tumors which can be classified as benign soft tissue tumors are lipoma, angiolipoma, fibroma, benign fibrous histiocytoma, neurofibroma, schwannoma, neurilemmona, hemangioma, giant cell tumor of tendon sheath, and myxoma.

A similarity between the malignant and benign tumors exists, including:
1) Both can grow quite large: Size alone does not make the distinction between these types of tumors.
2) Both can be dangerous at times: While benign tumors tend to be more of a discomfort, they can, in some cases, be life-threatening. An example is benign brain tumors. When these tumors grow in the enclosed space of the brain, they can put pressure on, and destroy other brain structures, resulting in paralysis, speech problems, seizures, and even death. Some benign tumors, such as pheochromocytomas, secrete hormones that can cause life-threatening symptoms as well.
3) Both can recur locally: If cells are left over after surgery, both benign and malignant tumors may later recur near the region of the original tumor.

A main differences between benign and malignant tumors are:
1) Rate of growth: In general, malignant tumors grow much more rapidly than benign tumors, but there are exceptions.
2) Ability to metastasize: Benign tumors expand locally, whereas malignant tumors can spread (metastasize) to other parts of the body by way of the bloodstream and lymphatic channels.
3) Site of recurrence: While benign tumors may recur locally (near the site of the original tumor), malignant tumors may recur at distant sites, such as the brain, lungs, bones and liver, depending on the type of cancer.
4) "Stickiness": The cells in benign tumors manufacture chemicals (adhesion molecules) that cause them to stick together. Malignant tumor cells do not produce these molecules and can break off and “float away” to other regions of the body.
5) Tissue invasion: In general, malignant tumors tend to invade nearby tissues, whereas benign tumors do not (though they may grow large and cause damage to nearby
organisms by creating pressure on them). A very simplistic way of thinking about this is to envision a benign tumor as having a wall or boundary (literally, a fibrous sheath surrounding the tumor). This boundary allows the tumor to expand and displace nearby tissues aside but does not allow the tumor to penetrate nearby tissues. In contrast, cancer has “fingers” that can reach into nearby tissues. In fact, the Latin word “cancer” derives from the word crab, used to describe the crablike, or fingerlike, projections of cancerous tumors.

6) Cellular appearance: Under a microscope, cells that are benign often look much different from those that are malignant. One of these differences is that the cell nucleus of cancer cells is often larger and appears darker due to an abundance of DNA.

7) Effective treatments: Benign tumors can usually be removed with surgery alone, while malignant tumors will often require chemotherapy, radiation therapy, targeted therapies, or immunotherapy medications. These additional treatments are needed to attempt to reach cancer cells that have spread beyond the region of the tumor or are left after surgery for a tumor.

8) Likelihood of recurrence: Benign tumors rarely recur after surgery, whereas malignant tumors recur much more commonly. Surgery to remove a malignant tumor is more difficult than surgery for a benign tumor. Using the fingerlike analogy, it is much easier to remove a tumor that has a clear fibrous boundary than a tumor that has penetrated nearby tissues with these fingerlike projections. If cells are left over from these fingers, the tumor is more likely to come back.

9) Systemic effects: Malignant tumors are more likely to have systemic, or total body, effects than benign tumors. Due to the nature of these tumors, symptoms such as fatigue and weight loss are common. Several types of malignant tumors also secrete substances that cause effects on the body beyond those caused by the original tumor. An example of this is the paraneoplastic syndrome caused by some cancers, resulting in a wide array of physical symptoms from hypercalcemia to Cushing's syndrome.

Some benign tumors can become malignant tumors over time. Some benign tumors very rarely become malignant tumors, whereas other benign tumors frequently transform into malignant tumors.
The squamous papilloma derived from stratified squamous epithelium and presents as a papillary or verruciform mass. This tumor is induced by Human papillomavirus (HPV). HPV comprises a large family (more than 100 types). A squamous papilloma is a common tumor (3% of all oral lesion submitted for histopathological evaluation). Histopathologically squamous papilloma is characterized with growth of keratinized squamous epithelium, well vascularized stroma and inflammatory cell infiltration.

The squamous papilloma occurs with equal frequency in both men and women, most often in persons between 30 and 50 years of age. Sites of predilection include the soft palate, tongue, lips, and cheek, but any mucosal or skin surface may be affected. The squamous papilloma is a soft, painless, usually pedunculated (with stalk) or sessile (without stalk), exophytic nodule with numerous finger-like surface projections that impart a “califlower” appearance. The tumor may be pale, slightly red, or normal in color, depending on the amount of surface keratinization. The papilloma is usually solitary and enlarged rapidly to a maximum size of about 0.5 cm. However, tumors more than 3.0 cm in diameter have been reported.
A surgical excision, including the base of the lesion, is adequate treatment ensured absence of recurrences. Another options in the management of squamous papilloma include cryotherapy, laser, electrosurgery.

**Fibromas** are tumors of fibrous (connective) tissue that can grow in any organ. Fibroma typically appears as a smooth-surfaced pink nodule that is similar in color to the surrounding mucosa. In black patients, the mass may demonstrate grayish brown pigmentation. In some cases, the surface may appear white as a result of hyperkeratosis from continued irritation. Most fibromas are sessile, although some are pedunculated. They range in size from tiny lesions that are only a couple of millimeters in diameter to large masses that are several centimeters. Most fibromas in oral cavity are up to 1-1.5 cm. The tumors are symptomless. Treatment is surgical excision, cryotherapy, laser, or electrosurgery.

**Lipomas** are slow growing benign mesenchymal neoplasms composed of mature adipocytes, commonly surrounded by a thin fibrous capsule that originate in mature fat cells. Most oral lipomas are composed of mature fat cells that do not differ microscopically from normal fat cells; however, differ metabolically from normal fat cells. Although lipoma can occur in any part of the body that contains fat tissue, it is not commonly found in the oral cavity. Lipomas are painless, soft on palpation, sessile tumors with or without capsule. Treatment is surgical excision.

**Madelung's disease** is a disorder of fat metabolism and lipid storage that results in an unusual accumulation of fat deposits around the neck and shoulder areas. It is characterized by massive lipoma located symmetrically around the neck, and on the shoulders, upper arms and upper trunk. These abnormal fat deposits may grow rapidly over the course of months or more slowly over a period of years. The rest of the body may be lean in contrast to the affected parts. The exact cause of Madelung’s disease is not known. The body’s inability to properly metabolize fat indicates that it may be an endocrine disorder. Some scientists believe a predisposition to the disorder may be inherited. Madelung’s disease most frequently affects middle-aged males. The condition is most common in those who abuse alcohol. However, this disease is also found in women and persons who do not consume alcohol. For reasons that are unclear, the disorder appears to be more prevalent in Europe than in the United States. Treatment consists of surgical removal of the fatty deposits from the areas around the head, neck, shoulders and trunk. Liposuction has been used successfully to remove single fatty tumors.

**Myomas** are tumors that grow from muscle. *Leiomyomas* grow from smooth muscle. They can start in the walls of blood vessels. A rare benign tumor of skeletal muscle is *rhabdomyoma*. These tumors are solid and painless on palpation. In oral cavity, myomas of the tongue are more common. The growing of the tumor may worsen the function of the organ where it developed and if it affected tongue, could be a reason of difficulty in swallowing and speaking. Treatment of myoma is surgical removal.
Historically benign vascular tumors were classified as follow:

I. According to the type of fluid they contained as:
   1. Hemangioma
   2. Lymphangioma

II. According to the size of the vascular channels as:
   1. Capillary
   2. Cavernous

A hemangioma is an abnormal buildup of blood vessels in the skin or internal organs. Two types of hemangiomas exist:

1) Hemangioblastoma: These tumors are slow-growing, and well defined. They arise from cells in the linings of blood vessels.

2) Hemangiopericytoma: These are rare tumors. They appear to originate in the cells surrounding the blood vessels in the meninges. They are more aggressive compared to hemangioblastoma.

A hemangioma is a birthmark that most commonly appears as a rubbery, bright red nodule of extra blood vessels in the skin. A hemangioma grows during the first year of life, and then recedes over time. A child who had a hemangioma during infancy usually has little visible trace of the growth by age 10. A hemangioma can occur anywhere on the body, but most commonly appears on the face, scalp, chest or back. In the oral cavity hemangioma could affect soft tissues and bones. Treatment of a soft tissue hemangioma usually isn’t needed, unless the nodule interferes with vision or breathing.

Several treatment modalities have been recommended and selection is largely dependent on the size of the lesion, the age of the patient, and any anticipated complications. Sclerosing agents such as absolute ethanol may be injected into the lesion, thereby inducing an inflammatory response within the endothelium, resulting in fibrosis and obliteration of the vessels. Irradiation is suggested if the lesion is large or surgically inaccessible. While it may cause regression in some lesions, it is not curative, and recurrences have been reported. Radiation also possesses high-risk side effects such as damage to growth centers in developing patients as well as possible induction of neoplastic transformation. Cryotherapy has been reported to have some measure of success in small lesions, but tends to have undesirable effects on adjacent tissues, including loss of innervation. Embolization of large vessels feeding the lesion is often recommended. Embolizing agents such as silicone pellets or isobutyl cyanoacrylate are injected into large vessels under fluoroscopic control. This technique carries potentially severe complications such as embolization of pulmonary or cerebral vessels. Embolization has been shown to be temporary, with the development of collateral vessels restoring the lesion to pre-procedure size. Therefore, its primary use is adjunctive to surgery to reduce intraoperative bleeding.

Lymphangiomas are congenital malformations of the lymphatic system that involve the skin and subcutaneous tissues. Lymphangiomas are rare. They account for 4% of all
vascular tumors and approximately 25% of all benign vascular tumors in children. Lymphangioma can become evident at any age, but the greatest incidence occurs at birth or early in life. About 50% of lymphangiomas are seen at birth, and most lymphangiomas are evident by the time the patient is aged 5 years. The classification of lymphangiomas lacks a standard clear definition and universal application, in part because of the nature of lymphangiomas, which represent a clinicopathologic continuum. Lymphangiomas are classified microscopically into four categories:

1. **Lymphangioma simplex**: composed of small, thin walled lymphatics.
2. **Cavernous lymphangioma**, comprised of dilated lymphatic vessels with surrounding adventitia.
3. **Cystic lymphangioma**, consisting of huge, macroscopic lymphatic spaces with surrounding fibrovascular tissues and smooth muscle.
4. **In benign lymphangioendothelioma**, lymphatic channel dissect through dense collagen bundles.

Lymphangiomas can occur anywhere in the skin and the mucous membranes. The most common sites are the head and the neck, followed by the proximal extremities, the buttocks, and the trunk. However, they sometimes can be found in the intestines, the pancreas, and the mesentery. Their skin involvement ranges from small, well-demarcated areas to large, diffuse regions with unclear borders.

In the case of **lymphangioma simplex**, the underlying lesions constitute abnormal dilated lymph vessels involving the upper part of the dermis. The site of predilection is oral cavity, especially the tongue. Lymphangioma simplex can occur in conjunction with cavernous lymphangioma and cystic lymphangioma. Surgical excision is the usual treatment of lymphangioma simplex. Lymphangioma simplex has a high recurrence rate after excision because of its deep component.

**Cavernous lymphangioma** usually arise during infancy. The most common sites are the head and neck areas and, less frequently, the extremities. These lesions are seated deep in the dermis, forming a painless swelling or thickening of the skin, mucous membranes, and subcutaneous tissue. Unlike lymphangioma simplex, the overlying skin usually is uninvolved. Occasionally, patients report pain when the involved area is pressed. The affected area may be 1 cm, it may be as large as several centimeters in diameter, or it may involve an entire organ. Upon examination and palpation, lipomas or cysts can be mistaken for these lesions.

Some authors categorize **cystic lymphangioma** as an independent entity. Many authors agree that cystic lymphangioma is a form of cavernous lymphangioma in which the degree of involvement and character is determined by its location. These congenital lesions are deeply seated in areas of loose connective tissue. They appear early in life as large soft-tissue masses, usually on the axilla, neck, or groin. These lesions are soft, vary in size and shape, and tend to
grow extensively if not surgically excised. Typical lesions are multilocular cysts filled with clear, yellow in color lymph fluid. Usually, cystic lymphangioma is diagnosed clinically with its large size, location, and translucence.

A schwannoma is a benign nerve sheath tumor composed of Schwann cells, which normally produce the insulating myelin sheath covering peripheral nerves. Schwannomas are homogeneous tumors, consisting only of Schwann cells. The tumor cells always stay on the outside of the nerve, but the tumor itself may either push the nerve aside and/or up against a bony structure (thereby possibly causing damage). Schwannomas of the head and neck are a fairly common occurrence and can be found incidentally in 3-4% of patients at autopsy. Treatment is surgical excision. Schwannomas can be associated with neurofibromatosis type II.

Neurofibromatosis is a group of three conditions in which tumors grow in the nervous system:

- **Type I (von Recklinghausen's disease):** the nerve tissue grows neurofibromas that may cause serious damage by compressing nerves and other tissues. The cause is a genetic mutation in certain genes. Neurofibromatosis is an autosomal dominant disorder, which means only one copy of the affected gene is needed for the disorder to develop. Therefore, if only one parent has neurofibromatosis, his or her children have a 50% chance of developing the condition as well.

- **Type II:** bilateral acoustic neuromas (tumors of the vestibulocochlear nerve or cranial nerve VIII) develop, often leading to hearing loss.

- **Schwannomatosis:** painful schwannomas develop on spinal and peripheral nerves.
Odontogenic tumors of the jaws are a relatively rare and heterogeneous group of neoplasms, hamartomas, and other bone-related lesions that demonstrate great variability in etiology, biologic behavior, and clinical significance. Odontogenic tumors derived from tooth-related tissues and originate only in the maxilla or mandible. The majority of odontogenic tumors are benign. Purely defined, odontogenic refers to derivation from a tooth-related apparatus. Tooth formation is a complex process that involves both connective tissues and epithelium. Three major tissues are involved in odontogenesis including the enamel organ, the dental follicle, and the dental papilla. The enamel organ is an epithelial structure that is derived from oral ectoderm. The dental follicle and dental papilla are considered ectomesenchymal in nature because they are in part derived from neural crest cells. Odontogenic tumors are typically subclassified by their tissues of origin (WHO histological classification of odontogenic tumors, 2005):

I. Benign tumors of odontogenic epithelium:
   1) Ameloblastoma: a) solid/multicystic type
      b) extraosseous/peripheral type
      c) desmoplastic type
      d) unicystic type
   2) Squamous odontogenic tumor
   3) Calcifying epithelial odontogenic tumors
   4) Adenomatoid odontogenic tumor
   5) Keratocystic odontogenic tumor

II. Benign tumors of odontogenic epithelium with odontogenic ectomesenchyme with or without dental hard tissue formation:
   1) Ameloblastic fibroma
   2) Ameloblastic fibro-dentinoma
   3) Ameloblastic fibro-odontoma
   4) Odontoma: a) complex type
      b) compound type
   5) Odontoameloblastoma
   6) Calcifying cystic odontogenic tumor
   7) Dentinogenic ghost cell tumor

III. Benign tumors of odontogenic ectomesenchyme with or without odontogenic epithelium:
   1) Odontogenic fibroma
   2) Odontogenic myxoma
   3) Cementoblastoma

The ameloblastoma is a true neoplasm of odontogenic epithelial origin. The etiology of ameloblastoma is not known. Excluding odontomas, ameloblastomas are the most common
odontogenic neoplasm and account for approximately 10% of all tumors that arise in the mandible and maxilla. Presenting symptoms may include a slow-growing submucosal mass, loose teeth, malocclusion, paresthesia, and pain. As many as 35% of patients are completely asymptomatic and the lesions are discovered as incidental findings on routine dental radiographs. The median age at presentation is 35 years and there is no gender predilection. Eighty percent of ameloblastomas arise in the mandible, usually in the ramus region, and are often associated with molar teeth. Frequency of different types of ameloblastomas is as follow: solid/multicystic (92%), unicystic (6%), peripheral type (2%).

Solid/multicystic ameloblastoma presents as locally aggressive tumors that demonstrate inherent neoplastic cellular proliferation, are associated with high recurrence rates when inadequately treated, and represent the most clinically significant odontogenic tumor based on potential morbidity and prevalence. If left untreated, these tumors can grow to extreme sizes. A painless expansion of the jaws is the most common clinical presentation of solid/multicystic ameloblastoma. Neurosensory changes are uncommon, even with large tumors. The most common radiographic feature is that of a multilocular radiolucency. Buccal and lingual cortical expansion is common, frequently to the point of perforation. Resorption of adjacent tooth roots is common.

The term “conservative treatment” is often used in surgery to describe a heterogeneous group of therapeutic modalities excluding segmental resection, including enucleation and curettage, excision with peripheral ostectomy, excision with margin and adjuvant treatment such as cryotherapy. Enucleation and curettage has been associated with a recurrence rate of 60–80%. So, enucleation and curettage alone is inadequate for definitive treatment of solid ameloblastoma. Since ameloblastoma is a benign disease, many surgeons are reluctant to perform radical surgery on these patients. This understandable reserve has led to a number of other techniques including enucleation with peripheral ostectomy and/or adjuvant treatment with liquid nitrogen cryotherapy. Neoplastic cells have been shown to be present several millimeters from the radiographic margin of the tumor, which has led to the recommendation that resection be performed with care to maintain 1 cm tumor-free margins. This so-called radical resection generally results in local control rates greater than 90%. Resection is the gold standard and currently offers the most predictable recurrence-free treatment for solid ameloblastoma.

Unicystic ameloblastomas are most commonly seen in young patients. The average age of patients with unicystic ameloblastomas has been reported as 22 years, compared with 40 years for the solid or multicystic variant. More than 90% of these tumors are found in the mandible, usually in the molar/ramus region. A unilocular radiolucency, mimicking a dentigerous cyst, is the most common radiographic presentation for the unicystic ameloblastoma. Most, if not all, unicystic ameloblastomas are unilocular radiolucencies.

The treatment of an unicystic ameloblastoma is enucleation and curettage. In a collective sense, the “recurrence” rate of all unicystic ameloblastomas has been reported as 10 to 20% following enucleation and curettage. This is significantly lower than that of
enucleation and curettage of the solid or multicystic ameloblastoma. Resection is an option in cases of the recurrent unicystic ameloblastoma, the mural (histological subtype which is more aggressive) ameloblastoma, and in very large tumors with significant expansion such that an enucleation and curettage surgery would effectively result in a resection of the involved jaw.

The peripheral or extraosseous ameloblastoma is the most rare variant of the ameloblastoma. Clinically, these tumors present as nonulcerated sessile or pedunculated gingival lesions. Most examples are less than 1.5 cm and usually occur over a wide age range, with an average reported age of 52 years. Although these tumors do not infiltrate bone, they may be seen to “cup out” bone in the jaws. The peripheral ameloblastoma is most appropriately treated with a wide local excision. When surgical margins are negative for tumor, cure is the likely consequence.

The desmoplastic variant of ameloblastoma was first described by Eversole et al. in 1984 and probably represents between 0.9% and 12.1% of all ameloblastomas. The mean age at initial presentation is 41 years and there appears to be equal distribution between the maxilla and mandible. Desmoplastic ameloblastomas often present as an asymptomatic, slowly enlarging bony or soft tissue mass in the maxilla or mandible. Unlike other ameloblastomas, the radiographic appearance is suggestive of fibro-osseous lesions, having been described as poorly defined radiolucent/radiopaque lesions, with or without loculations. Treatment outcomes seem to be similar to those of other ameloblastoma variants: simple enucleation results in unacceptably high rates of local recurrence and radical resection is curative.

The ameloblastic fibroma is considered to be a true tumor in which the epithelial and mesenchymal tissues are both neoplastic. This is in distinction to the ameloblastic fibro-odontoma and odontoma that represent developmental stages of the same hamartomatous lesion. The ameloblastic fibroma tends to occur in young patients in the first two decades of life. The posterior mandible is affected in 70% of cases. Radiographically, either a unilocular or multilocular lesion is observed. The ameloblastic fibroma is localized more common in posterior region of the mandible and often is associated with unerupted tooth. The tumor is well defined and corticated. It presents as a small, unilocular or large, multilocular lesion. Displacement of adjacent teeth could be observed, as well as vestibular or lingual/palatal expansion. The ameloblastic fibroma is recognized as an indolent tumor that is effectively treated by an enucleation and curettage surgery. Although recurrence is rare under the
circumstances, resection should be reserved for recurrent lesions. Approximately 45% of ameloblastic fibrosarcomas develop in the setting of a recurrent ameloblastic fibroma.

The ameloblastic fibro-odontoma probably represents a hamartoma. A hamartoma (from Greek hamartia “fault, defect” and -oma, denoting a tumor or neoplasm) is a benign tumorlike malformation made up of an abnormal mixture of cells and tissues found in areas of the body where growth occurs. It is considered a developmental error and can occur at a number of sites. Moreover, some investigators believe that this lesion is only a stage in the development of an odontoma and does not represent a separate entity. Ameloblastic fibro-odontoma occurs more frequently in the posterior regions of the jaws. This lesion is commonly asymptomatic and is discovered serendipitously or when radiographs are exposed to provide a diagnosis for asymmetric eruption of the dentition in children. These lesions are distinctly well circumscribed and appear as mixed radiopaque/ radiolucent masses. The ameloblastic fibro-odontoma is treated effectively with an enucleation and curettage surgery. Recurrence after this approach is very rare. Malignant transformation of ameloblastic fibro-odontoma is rare.

Odontomas are the most common of the odontogenic tumors, represent a benign hamartoma rather than a true neoplasm, and generally have a benign clinical course. Odontomas are described in two types: complex and compound. The difference between the two forms is that compound odontomas contain recognizable enamel, denin, and sometimes cementum, shaped in toothlike structures; whereas complex odontomas are composed of irregular masses of dentin and enamel and have no anatomic resemblance to a tooth.

Complex odontomas are typically found in the posterior maxilla or mandible.

The compound odontomas are predominately located in the anterior maxilla. They are twice as common as the complex odontomas. They appear as well circumscribed, radiopaque masses, often surrounded by what appears to be a periodontal ligament. The masses are treated with simple enucleation and curettage, which is virtually always curative.

Odontoameloblastoma “combines the clinical and histological features of ameloblastoma with those of an odontoma”. This is a rare neoplasm (fewer than 50 cases have been reported) that occurs in the maxilla and mandible with equal prevalence, usually in the first 3 decades of life. Clinical signs and symptoms, as well as biological behavior, may be identical to those of a solid ameloblastoma. The radiographic appearance of
odontoameloblastoma differs from that of ameloblastoma by varying amounts of radiopaque material, which generally represents displaced or unerupted teeth. Similar to conventional ameloblastoma in its biological behavior, odontoameloblastoma is locally aggressive and requires surgical excision with 0.5–1.0 cm tumor-free margins. The same treatment-related controversies that surround optimal treatment of ameloblastoma are presumably shared by this rare neoplasm.

The odontogenic fibroma is a very rare neoplasm that is defined by the WHO as containing “varying amounts of inactive-looking odontogenic epithelium embedded in a mature fibrous stroma”. Two histological subtypes have been described: epithelial poor (formerly termed simple type) and epithelial rich (formerly termed complex type). The OF occurs as a radiolucent lesion involving the jaws without respect to gender, age, or site (maxilla or mandible). The radiographic appearance is that of a radiolucent lesion that may be unilocular or multilocular and cause cortical expansion. Although aggressive variants have been described, neither subtype appears to behave in an aggressive fashion and both respond well to enucleation and curettage.

The odontogenic myxoma is relatively common amongst odontogenic tumors and may comprise between 3 and 20% of all such lesions. It is the second or third most common odontogenic tumor. The term “myxofibroma” is used when a relatively greater amount of collagen is evident. Two thirds of myxomas are located in the mandible, commonly in the molar regions, and typically present in the second to fourth decade of life without gender predilection. The myxoma is a benign but locally aggressive tumor that is probably less aggressive than ameloblastoma, but can result in progressive growth with skull base involvement if left untreated. The overall recurrence rate is between 10% and 33%. The approach to smaller lesions is typically enucleation with peripheral ostectomy. Larger lesions that exhibit aggressive biological behavior, such as cortical erosion or recurrence following treatment, should be excised with 0.5–1.0 cm tumor-free margins.

Cementoblastoma is a rare lesion that comprises less than 1% of all odontogenic tumors. It is characterized by the formation of a radiopaque cementum-like mass intimately associated with the root of a tooth (usually a lower second or third molar). The tooth typically remains vital and symptoms include varying degrees of cortical expansion and pain.

Classically, the tumorous mass is surrounded by a radiolucent ring that represents the periodontal ligament. Treatment is otherwise by excision with peripheral ostectomy for all but the largest tumors. Recurrence is possible if incompletely excised.
BENIGN NON-ODONTOGENIC BONE TUMORS

(Petia Pechalova)

Non-odontogenic bone tumors develop from the epithelium or mesenchyme of a wide variety of tissues in the body, often originate in non-tooth-bearing facial bones, and may develop in other sites outside of the head and neck.

Classification:

I. Osseous producing:
   1) Osteoma
   2) Osteoid osteoma
   3) Osteoblastoma

II. Cartilage (chondroid) producing:
   1) Enchondroma
   2) Osteochondroma
   3) Periosteal chondroma
   4) Chondroblastoma
   5) Chondromyxofibroma

III. Fibrous producing:
   1) Nonossifying fibroma
   2) Fibrous dysplasia
   3) Desmoplastic fibroma
   4) Benign fibrous histiocyteoma

IV. Non matrix producing: Eosinophilic Granuloma

V. From vascular origin:
   1) Hemangioma
   2) Lymphangioma

VI. Benign Giant Cell tumors:
   1) Giant cell tumor
   2) Aneurysmal bone cyst

VII. Others:
   1) Lipoma
   2) Schwannoma
   3) Ganglion
   4) Pediatric bone tumors

Osteoma is a rare benign bone tumor, composed of mature osseous tissue. It presents as a protruding mass of dense periosteal intramembranous bone, usually on surface of host bone. Osteomas may arise on the surface of bone (periosteal/ peripheral osteomas) or centrally within the bone (endosteal/ central osteomas). It is abnormally dense but is normal bone formed in the periosteum. Almost exclusively affect skull, parasal sinuses and facial bones. Radiographically presentations as sharply defined, radiopaque smooth, homogeneous bone protruding mass from the surface of a bone; almost appears as a localized thickening of the bone.
Osteomas are most commonly discovered during the second and fifth decades of life, although they have been noted in all age groups. Males appear to be affected more frequently than females. They are often discovered as asymptomatic radiopacities. Treatment of osteomas is surgical excision, recurrences are very rare.

Osteoblastoma and osteoid osteoma are generally felt to be variants of the same lesion and are related to fibro-osseous disease. Cementoblastoma and gigantiform cementoma are the equivalent cemental lesions and are associated with teeth. The alternative name for the osteoblastoma is giant osteoid osteoma, and it is generally felt to represent a larger version of the osteoid osteoma.

The osteoblastoma occurs primarily in the vertebrae and long bones, but it has been described in the jaws. Clinically it often grows rapidly and the predominant clinical feature is pain, which is generally localized to the lesion itself.

Radiographic features are variable, usually consisting of a combination of radiolucency and radiopacity. The designation osteoblastoma is normally reserved for lesions more than 2 cm in diameter. The osteoid osteomas usually are less than 2 cm. They are well circumscribed radiographically with a thin radiolucency surrounding the variably calcified contents. A sunray pattern of new bone formation similar to that described in malignant bone tumors may be evident. Treatment of the osteoblastoma is generally confined to conservative surgical excision either with curettage or local excision. Recurrences are rare but have been reported and may necessitate more aggressive treatment such as en bloc resection. Rare examples of malignant transformation have been reported, but some of these may be related to an incorrect initial diagnosis.

A chondroma is a benign tumor of mature cartilage. The occurrence of these lesions in the jaws is extremely rare; in fact, whether they ever occur in the jaws or whether they are usually described as chondromyxomas or chondromyxoid fibromas has been questioned. Most
reports concern the mandibular condyle, suggesting that these lesions may arise from cartilaginous remnants. The chondroma presents as a painless slowly progressive swelling, which may result in mucosal ulceration. The gender distribution is equal, and most tumors occur under the age of 50 years. Radiographically they present as irregular radiolucent lesions, although foci of calcification may occasionally be present. Resorption of tooth roots has been reported.

Treatment of the chondroma is localized, and conservative surgical excision is normally recommended. Because of the doubtful nature of these lesions and the always-present possibility of a lesion representing a low-grade chondrosarcoma, some authorities have suggested wide excision for all of these lesions.

**Hemangiomas in an intraosseous location**, especially of the jaws, are rarely reported entities. Patients often experience a firm, painless swelling of the bone which may or may not cause facial asymmetry. Other reported symptoms are pressure or discomfort, oozing or pulsatile bleeding from the gingiva of teeth in the region of the lesion, a bluish discoloration of the gingiva, mobile teeth, and accelerated exfoliation of teeth. In lesions with high vascular pressure, patients often report a sensation of pulsation, and large lesions extending into adjacent soft tissues may have audible bruits. Paresthesia in the region is not uncommon. It is crucial to report that patients may not demonstrate any signs or symptoms. Failure to consider and recognize the presence of an intraosseous hemangioma before surgical therapy may lead to significant hemorrhage and even death.

Intraosseous hemangiomas possess a varied radiographic appearance and thus cannot be accurately diagnosed on plain films. In general, they can present with an osteolytic pattern possessing a multi-locular “soap bubble” appearance with irregular, poorly defined margins. A differential diagnosis would include central giant cell lesion, ameloblastoma, and odontogenic myxoma. As such, angiography can play a crucial role in diagnosis as it can confirm the suspicion of the vascular lesion, delineate its margins, and indicate feeder vessels. Definitive diagnosis of an intraosseous hemangioma cannot be made without histologic examination, but due to the risk of severe hemorrhage, needle aspiration should precede biopsy of any suspicious lesion. The presence of easily aspirated blood with significant volume and brisk hemorrhage from the puncture site should preclude biopsy. In the presence of clinical signs and symptoms, radiographic appearance, positive aspiration, and suggestive arteriograms, biopsy may be deferred and treatment should be initiated with a presumptive diagnosis of intraosseous hemangioma. Surgical treatment of bone hemangiomas is generally accepted as the definitive treatment, with en bloc resection the recommended procedure. Extension beyond the lesion reduces the risk of disruption and may reduce the risk of catastrophic hemorrhage. Ligation of feeder vessels should precede the removal of the lesion. The potential for anatomic variation emphasizes the importance of angiography for proper identification of vessels associated with the primary lesion. Even with proper identification and pre-operative planning, significant bleeding is to be expected intra-operatively, and clinicians should be prepared for rapid transfusion. Upon successful surgical removal of the lesion, appropriate reconstruction measures may be taken to restore the patient to optimum function.
MALIGNANT TUMORS IN ORAL AND MAXILLOFACIAL AREA
(DIMITAR ATANASOV)

Malignant neoplasms of the soft tissues and organs in the oral cavity and the maxillofacial area

Squamous cell Carcinoma is the most common malignant neoplasm of the oral cavity (90-95%). It forms 4% of all malignant neoplasms in males and 2% of malignancies in females.

Clinical presentation: There are three different periods in the clinical appearance of the squamous cell cancer: initiative period, clinical manifestation and terminal period.

I. Primary (initiative) period: The initiation of the disease is asymptomatic and it often mimics benign conditions such as erythroplakia; leukoplakia; a non-healing ulcer, that develops slowly or rather fast and does not respond to the applied treatment, an induration of the mucosa, with clearly visible borders and normal color; papillary formations that grow fast. These initial symptoms do not go together with any subjective complaints and patients seek medical help when these lesions (tumors) have grown to a significant size.

Squamous cell carcinoma develops mainly in men (50-70 years old) and women (60-80 years old), who abuse alcohol and cigarettes consumption. In these patients the tumors have three specific intraoral localizations - the floor of the oral cavity, the ventrolateral surfaces of the tongue and the complex area that includes the hard palate, the tonsils and the retro molar area (Mashberg, A., Samit, A., 1995).

II. Period of clinical appearance: During this period certain symptoms are present: pain that has different intensity (this symptom may not be present even in tumors with great size) and irradiation, bleeding, ulceration, increased release of saliva (hypersalivation), halitosis (fetor ex ore). Clinically, during this stage the cancer comes into two types:

1. Exophytic with two subtypes:
   ✅ Papillary – the tumor looks like a cabbage or like a sponge, it has well distinguished borders and superficial development.

   ✅ Ulcerative – superficially developed ulcer that has thick margins.
(2) Endophytic with two subtypes:

- Ulcerative-infiltrative: There is an ulcer that has thick and uprising margins which has developed on an infiltrated fundament, comprised of tumor cells. This infiltrate does not have well distinguished borders and spreads deep inside surrounding tissues. The ulcer has a fissure-like appearance.

- Infiltrative: The tumor spreads deep inside tissues, while the covering mucosa remains intact for a long time.

The exophytic types have better prognoses than the endophytic ones, which are characterized by a diffused growth and greater malignant potential. Carcinomas that are located in the posterior areas of the oral cavity tend to be more malignant than the ones that develop in the anterior ones.

III. The clinical manifestation of the squamous cell carcinoma depends on it’s localization in the oral cavity, the histology and the stage of the tumor. In the initial stage of the development of the cancer the malignant metastases are a rare finding, while in the stage of the clinical manifestation metastases in the regional lymph nodes or in distant organs can be found in 53% of the patients.

Diagnose: It is based on the data from the medical history (anamnesis), the clinical examination and the histopathological finding.

The squamous cell carcinoma in the anterior areas of the oral cavity grows slower and has better histological differentiation in comparison to the carcinoma that grows in the posterior areas, which grows faster and more aggressive and is less differentiated.
Cancer of the lips

The cancer of the lips occurs in 98% of the cases on the lower lip and in 2% of the cases on the upper lip. It affects more commonly males and peasants. It makes 0.2% of all malignancies in the human body and 25-30% of malignancies in the oral cavity. The incidence of this cancer is 2.7 in 100000 men and 0.3 in 100000 women. Data from Bulgarian patients suggests that the incidence of this cancer in Bulgaria is 7.3 in 100 000 men and 1.61 in 100 000 women (1994).

Clinical presentation: Squamous cell cancer of the lips develops in an asymptomatic manner, there is an ulceration which is hardly distinguished from a non-malignant syphilitic or herpetic ulcer. It develops from the mucosa next to the vermilion of the lips and is located sideways to the midline of the lip and is rarely located at the angle of the lip (commissure). It typically develops from long lasting non-healing wounds, that are covered by crusts and their margins are infiltrated. This means that every ulcer, that does not heal for two weeks should be biopsied.

Cancer of the lips comes in two different types:

1) Exophytic, with two subtypes:

- Papillary: It often develops from a papilloma, that grows slowly, and its margins insensibly become thick. As the disease progresses, the papilloma becomes soft and then disintegrates, the lips becomes infiltrated and the ulcer develops.

- Verruca-like: It develops on a field of diffuse diskeratosis, there are multiple growths on the lips, which merge with time and the lesion looks like cabbage. This type has slow evolution that includes deep infiltration of the underlying tissues and consecutive disintegration of the tumor.
(2) Endophytic, with two subtypes:

- **Ulcerative:** It has the appearance of an ulcer, which has irregular shape and uneven bottom. The margins of the ulcer are elevated above the surrounding tissues of the lip, twisted and infiltrated. The ulcer is painful, its base and borders are dense.

- **Ulcerative-infiltrative:** The ulcer is located on an infiltrated and dense base. The infiltrate has dense, wood like consistency on palpation.

The lip cancer metastasizes in the regional lymph nodes, particularly the submandibular lymph nodes. When there is lip cancer of the lower lip metastases can be observed in the submandibular, submental, superficial and deep cervical lymph nodes, and when the cancer affects the upper lip metastases can be observed in the submandibular, auricular and deep cervical lymph nodes.

**Diagnose:** The results from the medical history (anamnesis) and the clinical examination indicate performing a biopsy. The definite diagnose can be determined after checking the results from the histopathology. The origin of the cancer determines its histological appearance – in 96.8% of the cases it’s a squamous cell carcinoma. In 80-95% of the times it is keratinised. The non-keratinized carcinoma grows fast with ulcerations and metastasizes in the lymph nodes rather early.

**Differential diagnose:**
1) Herpetic heilitis
2) Ulcus durum
3) Tuberculous ulcer
4) Traumatic or inflammatory ulcer
5) Disintegrated syphilitic gumma

**Treatment modalities:** Two different modalities are being applied:

1) Surgical treatment: This modality is preferred when treating younger patients, because of the better cosmetic results and prevents the development of the so called radiation induced tumors.
(2) Radiation therapy: It’s a treatment option for the lip cancer, mainly administered extra orally. In older patients, who find it difficult to visit the radiologist every day, radiation therapy can be administered intraorally, by implanting radioactive “pearls”. The dosage and route of administration of the radiation therapy are determined by the stage of the cancer and different methods are:

Tis, T1 – near distance radiation therapy(72G) or interstitial(implantation) brachytherapy(70G).
T2, T3 – interstitial(implantation) brachytherapy(70G).
T4 percutaneous radiation therapy (70G) or preoperative radiation therapy(30-40G), followed by widened en block resection and postoperative radiation therapy (70G)

In case of absence of metastases only radiation therapy is administered, otherwise it is combined with cervical dissection (by Wanah). The five year survival rate is achieved in 60-90% of the patients.

**Cancer of the tongue**
(Carcinoma lingue)

Cancer of the tongue represents 50% of oral carcinomas and 13% of all malignancies of the respiratory system. The incidence in Bulgaria is 2.52 in 100 000 men and 0.18 in 100 000 women(NOC 1994). It is mainly located on the middle third of the lateral border of the tongue (62%) and the radix of the tongue (28%) and it is seldom located on the dorsal surface (7%) or the tip of the tongue (3%).

**Clinical presentation:** It has two clinical types:

(1) Exophytic with two subtypes:

- Papillary: A widespread formation, which has uneven surface and looks like cabbage.

- Ulcerative: An ulcer with irregular shape and dense elevated margins. This subtype demonstrates insignificant infiltration into the underlying tissues and metastasizes in the regional lymph nodes rather late.
(2) Endophytic with two subtypes:
  ✓ Ulcerative-infiltrative: An ulcer, which has dense, elevated margins and develops on a thick tumor mass. The infiltrate has no recognizable borders and spreads deep into the underlying tissues.

  ✓ Infiltrative: The infiltrate spreads deep into the surrounding tissues and quickly affects the muscles of the tongue, forming firm lump. It metastasizes the regional lymph nodes rather early, it frequently recurs after radiation therapy and has bad prognosis.

Diagnose: The histological appearance of the cancer of the tongue shows squamous cell, keratinised carcinoma. It shows moderate aplasy levels. The parenchyma of the tumor invades the underlying muscle tissue of the tongue.

Differential diagnose:
1) Papilloma
2) Decubital ulcer
3) Actinomycosis
4) Tuberculous ulcer
5) Ulcus durum

Treatment modalities: A biopsy is obligatory before initiation of the treatment. An incisional is preferred in most cases as excisional is accepted only when dealing with benign tumors or oral lesions that are less than 1 cm in size. Treatment modalities for tongue cancer include surgery, radiation or complex treatment, depending on the localization and stage of development of the tumor.

  Radiation therapy is a treatment modality, administered in one of the following ways:

T1, T2 – surgical excision when the tumor is located on the tip or the anterior aspect of the tongue( well differentiated, exophytic).

T2 – extra oral radiation therapy (50Gy) or interstitial curative therapy (70Gy), (endophytic, poorly differentiated).

T3 – percutaneous radiation (70Gy) or combined radiation (40G).
  – percutaneous radiation plus overdosing up to 70Gy by interstitial brachytherapy

T4 – percutaneous radiation therapy or radical surgical excision, followed by postsurgical radiation therapy (60Gy)
In tumors T1 and T2 stage it is recommended to do a combined treatment - intraoral radiation therapy (21-24Gy) and extraoral radiation therapy (50-55Gy) for the original tumor and additional extraoral radiation (60Gy) for the first order lymph nodes.

In cases of large tumors of the tongue certain authors recommend total glossectomy, combined with laryngotomy and postoperative radiation therapy up to 66Gy.

Some recommend in cases of tongue cancer no matter the clinical stage to do perform radiation therapy of the primary tumor and cervical dissection of the regional lymph nodes.

The five year survival rate is between 37 and 100%, depending on the clinical stage and the treatment modality.

Cancer of the floor of the mouth
(Carcinoma fundi cavi oris)

Cancer of the floor of the mouth is responsible for 7.8% of all malignancies of the superior aspect of the respiratory system. It is mainly located on the anterior aspects of the region, mainly sideways to the midline, but it can spread on tissues from both sides or on the distal areas of the region.

Clinical presentation: The disease develops for a long time unnoticed, and has two clinical types:

(1) Exophytic with two subtypes – papillary and ulcerative.

(2) Endophytic with two subtypes – ulcerative and ulcerative-infiltrative and these two forms infiltrate the adjacent oral tissues –tongue, muscles of the floor of the mouth, mandible even in the early stages. This leads to disturbances in the mobility of the tongue and the mandible, the speaking ability, and also halitosis. Severe pain starts and irradiates towards the root of the tongue and the peritonsillar area.

Cancer of the floor of the mouth metastasizes in the submandibular and the superficial cervical lymph nodes, more often bilaterally. In T3 and T4 clinical stage metastases can be observed in 80-90% of the cases, and in T1N0 and T2N0 clinical stages occult metastases are present in 10-15% of the cases.

Diagnose: The results from the histology remove all difficulties in determining the diagnose, especially when we can see the typical irregular nests, that comprise of the so called “cancerous pearls”.

Differential diagnose:
1) Decubital ulcer
2) Ulcus durum
3) Tuberculous ulcer
4) Actinomycosis
The treatment modalities include surgical therapy, radiation therapy and complex therapy, depending on the clinical stage of the cancer:

T1 – interstitial brachytherapy (70Gy)
  – percutaneous radiation (70Gy)
  – electric excision of the tumor
T2, T3 – percutaneous radiation therapy (70Gy)
  – electric excision in combination with en block resection and postoperative extraoral radiation therapy up to 60Gy
T4 – percutaneous radiation therapy (70Gy), which in most cases happens to be only a palliative method.

In early clinical stages (T1, T2) the cancer of the floor of the mouth should be treated in a complex way – intraoral (21-24Gy) and extra oral (50-55Gy) radiation therapy for the primary tumor (the combined maximum dose should not exceed 72-75Gy) and additional extra oral radiation therapy for the first order lymph nodes.

The five year survival rate is 41-95%, depending on the clinical stage of the cancer and the specific treatment method.

Cancer of the buccal mucosa
(Carcinoma buccae)

Buccal cancer is about 9.8% of all oral and maxillofacial cancer. It is mainly located on the commissures or the occlusal line of the cheek.

Clinical presentation: Buccal cancer has two clinical types – exophytic, which has the appearance of a cabbage and develops superficially and minimally infiltrates the underlying tissues, endophytic - ulcerative-infiltrative, which looks like a gap and quickly infiltrates the buccal muscles, and short after that the masticatory muscles too (m. masseter, m. pterygoideus medialis,), which leads to trismus and difficulties in chewing, swallowing, speaking. As it develops, the tumor might also affect the skin of the cheek. This type metastasizes into the submandibular and cervical lymph nodes rather fast and is much more malignant.
Diagnose: The buccal cancer may be well differentiated or poorly differentiated, depending on the clinical stage and the time that has passed before the patient seeks medical help.

The TNM classification is applied to this cancer the same way it is applied to the cancer of the tongue.

Differential diagnosis:
1) Decubital ulcer
2) Ulcus durum
3) Tuberculuous ulcer
4) Actinomycosis

Treatment modalities comprise of surgical therapy, radiation therapy and complex therapy, depending on the clinical stage of the tumor:
- T1, T2 – interstitial brachytherapy (70Gy)
  - percutaneous radiation therapy (70Gy)
  - electric excision and postoperative radiation (50-60Gy)
- T3 – preoperative percutaneous radiation therapy (40Gy), followed by radical electric excision or en block resection in combination with postoperative percutaneous radiation therapy up to 60Gy
- T4 – percutaneous radiation therapy up to 70Gy
  - complex surgical and radiation therapy as in T3

Cancer of the hard palate
(Carcinoma palatum durum)

Clinical presentation: Squamous cell cancer of the hard palate is a rare finding and has two clinical types – exophytic and endophytic, which both ulcerate rather fast and infiltrate the maxillary bone, nasal and paranasal cavities. After the ulcer if formed, patients start having some unpleasant sensations, pain with different severity and irradiation. Metastases in regional lymph nodes can be observed mainly in advanced tumors, affecting the submandibular and cervical lymph nodes.
Diagnose: Histologically this carcinoma has the same typical appearance as the other squamous cell carcinomas in the oral cavity, having mainly extra- and intracellular keratinization and the typical “cancer pearls”.

Differential diagnose:
1) Decubital ulcer
2) Ulcus durum
3) Tuberculous ulcer
4) Actinomycosis

Treatment modalities comprise of surgical therapy, radiation therapy or a complex therapy – surgery and radiation:
- T1, T2, T3 – partial or total resection of the maxilla, followed by a postoperative extra oral radiation therapy up to 60-70Gy
  - percutaneous radiation treatment (70Gy)
- T4 – percutaneous radiation therapy up to 40Gy, followed by total radical resection of the upper jaw and postoperative radiation therapy (70Gy)

Cancer of the gingiva

Gingival cancer is approximately 11.3% of the malignancies of the upper aspects of the respiratory system. The oral cancer is located in 15.3% of the cases on the lower jaw and in 12.4% of the cases on the upper jaw.

Clinical presentation: Gingival cancer has the same clinical types as all other types of oral cancer.

(1) The exophytic type grows superficially, has uneven surface, looks like cabbage and infiltrates the underlying bony tissues rather late.

(2) The endophytic type spreads into the alveoli of the teeth, which causes pain in intact teeth. During this early stage teeth may be extracted without actual indications due to this pain and this leads to the additional rapid spreading of the cancer within the bone. During these initial clinical stages there are no radiographic changes in the jaws and it takes some months before some changes can be observed.
Diagnose: Histologically this carcinoma has the same typical appearance as the other squamous cell carcinomas in the oral cavity, having mainly extra- and intracellular keratinization and the typical “cancer pearls”. Both subepithelial and deep layers that form the tumor nests show significant atypism.

Differential diagnose:
1) Decubital ulcer
2) Ulcus durum
3) Tuberculous ulcer
4) Actinomycosis

Treatment modalities comprise of surgical therapy, radiation therapy or a complex therapy – surgery and radiation:
T1, T2, T3 – partial or total resection of the maxilla, followed by a postoperative extra oral radiation therapy up to 60-70Gy
– percutaneous radiation treatment (70Gy)
T4 – percutaneous radiation therapy up to 40Gy, followed by total radical resection of the upper jaw and postoperative radiation therapy (70Gy)
The five year survival rate is 30-47%.

In all patients, that suffer from oral squamous cell cancer in IV clinical stage and undergo the traditional treatment the mortality rate is 70% in the first two years. In such patients some authors recommend the application of a complex therapy – chemotherapy and radiation therapy – cisplatin 60mg/m, 2,5 fluorouracil 700mg/m, up to 70Gy radiation. The survival rate after three years was 52%.

TNM system in epithelial malignancies (carcinomas) of the tissues and organs in the oral cavity

TNM system is a clinical classification of the stage of development of the tumors and is based on the values of three different parameters:
T – size of the primary tumor in cm
N – the presence or absence and the size of the metastases in the regional lymph nodes in cm
M – presence or absence of distant metastases
The clinical classification is based on the data received from the examination of the patient before treatment and is comprised of:
Primary tumor criteria
Tis – carcinoma in situ
T0 – there is no primary tumor
T1 – the tumor is no bigger than 2cm in diameter
T2 – the tumor is more than 2cm but less than 4 cm in diameter
T3 – the tumor is more than 4 cm in diameter
T4 – the tumor infiltrates the underlying tissues, for example the lower lip cancer can infiltrate the cortical layer of the mandible, the floor of the mouth, the skin of the neck; the oral floor cancer can infiltrate the mandibular bone, the muscles, the skin of the neck
Tx – it is impossible to fully determine the exact borders of the primary tumor

Regional lymph nodes metastases criteria:
N0 – the regional lymph nodes are not affected
N1 – it is possible to palpate a metastasis in a regional lymph node, which is mobile and is located on the same half of the body as the primary tumor (homo lateral), not more than 3cm in diameter
N2a – a metastasis in a single homo lateral lymph node, which is more than 3cm and less than 6 cm in diameter
N2b – metastases in multiple homo lateral lymph nodes, more than 3cm and less than 6 cm in diameter.
N2c – metastases in multiple bilatearal and contralateral lymph nodes, less than 6 cm in diameter
N3 – a metastasis in a lymph node, that is more than 6 cm in diameter
Nx – it is impossible to palpate the lymph nodes and determine their condition
M – distant metastases criteria:
M0 – there are no distant metastases
M1 – there is a distant metastasis
Mx – it is impossible to find distant metastases

Clinical stages, based on T, N and M values:
Stage 0 – TisN0M0
Stage I – T1N0M0
Stage II – T2N0M0
Stage III – T3N0M0, T1N1M0, T2N1M0, T3N1M0
Stage IV – T4N0M0; T4N1M0; T1, 2, 3, 4 N1 M0; T1, 2, 3, 4 N1, 2, 3 M1

Malignant melanoma
(Melanoma malignum)

The melanoma is a malignant tumor, that develops from pigment producing cells. It was given its name by Corwell, R., and the first description of a melanoma located in the oral cavity was made by Weber, C., O.,

The malignant melanoma is approximately 1.3% of all malignancies in the human body and 0.2-8% of it is located in the oral cavity. It affects both men and women from 6 to 95
years of age, but its peak is in the 6th or 7th decade. In Bulgaria the incidence of this tumor is 2.3 in 100 000 men and 2.4 in 100 000 women.

Clinical presentation: When it develops in the oral cavity, the melanoma is mainly located on the palate (40%) and the gingiva (34%) and rarely affects the lips, the cheeks, the tongue and the floor of the mouth.

The malignant melanoma is asymptomatic, on the mucosa appears a dark, pigmented stain, slightly elevated over the surrounding mucosa. It looks like a sponge, papilloma or a widespread formation. It can have a different shape – round, oval or polygonal. On palpation its consistency is either soft or thicker.

A lot of the patients report a lump that has dark color and have no other complaints. Later on the mucosa swells, so that if the patient has dentures, they become unstable. After a while bleeding can be observed, along with ulcerations, expansive growth of the tumor, increased mobility of some teeth and pain of different severity. These symptoms persuade patients to seek medical help.

Between 25 and 30% of melanomas metastasize rather fast by lymph or blood circulation and affect lungs, brain, heart, liver, digestive system. Metastases can be observed in the cervical lymph nodes, while the localization of the primary tumor is unclear. The metastases can be homo lateral or contra lateral. The presence of metastases indicates the continuous spreading of the cancer.

Diagnose: The diagnose is helped by the information acquired from the medical history of the patient, describing the appearance of a dark stain, which is elevated above the surrounding tissues and grows fast. It is confirmed by the histological results of the biopsy. According to Batsakis the preoperative incisional biopsy does not increase the risk of furthermore spreading of the tumor, this making the prognosis poorer.

Histologically, the pathognomonic mark of this tumor is the presence of large amount of melanin in the cytoplasm of large ovoid or polygonal cells, attached tightly to each other.

Treatment: A treatment modality of choice is the radical electric excision, 3cm out of the borders of the tumor. According to Bernadskii complex – radiation and surgical therapy has positive outcomes. The preoperative radiation therapy is administered in doses from 80 Gy to 350 Gy and is then followed by surgical therapy.
Anatomical – topographic specifics of the oral and maxillofacial region do not always allow the administration of radical surgical therapy. In such cases it is recommended to perform complex immune, radiation and chemotherapy. The immune therapy is active and non-specific – Interferon 3x10 IU daily, radiation therapy is in doses of 30Gy, and the chemotherapy includes Dacarbazine 200mg, Nimusine 100mg, Vincristine 1mg.

The five year survival rate is 4.5 – 29%. That is why it is of paramount significance that the dentist and the oral surgeon should always perform thorough examinations and do early biopsies when indicated. This way the early diagnose, the best treatment and the most favorable outcome can be expected.

Epithelial malignancies of the upper jaw (bone tumors)

The epithelial malignancies of the upper jaw can be primary, secondary and metastatic.

Primary intra osseous maxillary carcinoma

It originates from the epithelial embryonic residues of Malassez and develops deep into the jaw or it can originate from odontogenic cyst which eventually underwent malignant transformation.

Clinical presentation: The tumor develops rather fast, with the appearance of a swelling around the maxilla. As it goes on the bone is destructed and the tumor grows towards the oral cavity or the maxillary sinus. If there are teeth amongst the affected bone the first symptom might be pain from intact teeth, followed by increased mobility of the teeth. After exodontia is performed, the exposed alveoli do not heal, but are filled with some kind of soft tissue instead. In some cases there is absolutely no pain, it’s just the teeth that become mobile and fall out of the mouth by themselves.
Diagnose: It is based on the medical history (anamnesis) the clinical examination and the radiographic findings, and the last ones happen to be: destruction of bone tissue - homogenous radiolucency with uneven borders, the cortical bone is destructed later on, not in the beginning. The definite diagnose is determined after having the results from the biopsy.

Differential diagnose:
1) Osteoblastoclastoma
2) Adamantinoma
3) Sarcoma

Treatment: Partial or total resection of the affected jaw can be performed, depending on the clinical type and stage of the tumor.

Secondary carcinoma of the maxilla

Cancer of the alveolar mucosa

Clinical presentation: This tumor is in most cases squamous cell carcinoma. It has fast evolution, patients experience tooth pain, can also be increased tooth mobility and spontaneous tooth loss, ulceration on the mucosa. When a tooth is extracted with no indication, this leads to further spreading of the tumor and bloody-purulent exudation from the extraction socket. The painful sensation is constant.

In some patients the only symptom might be the ulceration on the mucosa, without any preceding symptoms. In the beginning the lesion is located and bleeds when irritated. In few months it infiltrates the alveolar bone, and may also affect the cheek, the palate or the nasal cavity. It has the two clinical types, that are typical for all squamous cell carcinomas – exophytic and endophytic. As the lesion develops, the alveolar bone is destructed, which leads to constant tooth pain and tooth mobility.

In the early stages of the evolution of the tumor there are no symptoms from the nose, and there is no headache. In the later stages such symptoms may appear.

Diagnose: It is based on the data from the medical history, the clinical examination and the biopsy.
**Differential diagnose:**
1) Decubital ulcer
2) Ulcus durum
3) Tuberculous ulcer
4) Actinomicosis

**Treatment:** The treatment plan depends on the clinical stage of the tumor and the amount of adjacent anatomical structures involved. The treatment modalities comprise of surgery, radiation therapy and complex therapy:

- T1, T2, T3 – Partial or total resection of the upper jaw, followed by postoperative radiation therapy up to 50-60Gy
- Percutaneous radiation therapy (70Gy)
- T4 – Percutaneous radiation up to 40Gy, followed by radical total resection of the maxilla and postoperative percutaneous radiation therapy up to 60Gy

**Cancer of the hard palate (Carcinoma palatum durum)**

*Clinical presentation:* It is less common than the cancer of the alveolar mucosa. It begins as a dense pale area on the palatal mucosa, 2-3 cm in diameter. It grows fast, there can be seen some hyperemic areas within it, its center softens and ulcerates, its borders become infiltrated. This happens fast and causes some unpleasant sensations or pain. Patients do not hesitate to seek medical help.

The so called adenocarcinomas, which originate from the minor salivary glands develop more often on the palatal mucosa. The lesion remains encapsulated for some time, it may even reach great size. Patients’ single complaint in these cases is the swelling on the palate. As it develops, it ulcerates, which leads to painful sensation, the hard palate is destructed, the tumor invades the maxilla.

*Diagnose:* It is based on the data from the medical history, the clinical examination and the biopsy.

**Differential diagnose:**
1) Decubital ulcer
2) Ulcus durum
3) Tuberculous ulcer
4) Actinomicosis

**Treatment:** The treatment plan depends on the clinical stage of the tumor and the amount of adjacent anatomical structures involved. The treatment modalities comprise of surgery, radiation therapy and complex therapy:

- T1, T2, T3 – Partial or total resection of the upper jaw, followed by postoperative radiation therapy up to 50-60Gy
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T4 – Percutaneous radiation up to 40Gy, followed by radical total resection of the maxilla and postoperative percutaneous radiation therapy up to 60Gy

Cancer of the maxillary sinus

The maxillary sinus tumors are 0.2-8% of all human malignancies, and their incidence is 0.3 in 100,000 people. The malignant ones are predominant as 45-80% of them are squamous cell carcinomas, 4-15% of them are salivary gland carcinomas and 4-6% are sarcomas.

The anatomical association between the nose and the paranasal cavities lead to the fact that these tumors infiltrate the adjacent anatomical structures and that is why some authors name them sinonasal tumors. There is great variety of nasal and paranasal tumors was systematized by the WHO histological classification:

- Epithelial tumors (carcinomas)
  - Sinonasal carcinoma
    - Squamous cell carcinoma
    - Cilindric cell carcinoma
  - Verrucous squamous cell carcinoma
  - Spindle cell carcinoma
  - Adenocarcinoma
  - Papillary adenocarcinoma
  - Interstitial type adenocarcinoma
  - Acine cell carcinoma
  - Muco-epidermoid carcinoma
  - Adenoid-cystic carcinoma
  - Polymorphous highly differentiated carcinoma
  - Carcinoma inside pleomorphous adenoma
  - Malignant myoepithelioma
  - Epithelial-myoepithelial carcinoma
  - Light cell carcinoma
  - Adenoid-squamous cell carcinoma
  - Carcinoïd tumor
  - Atypical carcinoid tumor
  - Small cell carcinoma
  - Lymphoid-epithelial carcinoma

This classification makes it evident, that all of the tumor described before can develop inside the maxillary sinus.

The malignant tumors of the maxillary sinuses begin their evolution quite discretely. They have long lasting latent period, which continues while the tumor is still in the maxillary sinus. In some of the patients the initial symptom might be the mucous-purulent exudate nasal
exudate, from one nostril only, which can sometimes also contain blood. In others the exudate may have brown color and bad smell. The breathing capacity from this nostril is progressively reduced, and as the tumor grows it may even become impossible, there can be epistaxis. Patients may start having pain in the jaw or a tooth pain in intact teeth (especially during the night). These teeth may become mobile and even fall out by themselves painlessly. Facial asymmetry can be observed, sometimes also changes in the eye bulb – exophthalmus, diplopia, non-moving eye bulb.

Mainly epithelial malignant tumors develop in the maxillary sinuses, connective tissue tumors are less likely to develop (4:1). From all carcinomas, squamous cell cancer is the most common (45-85%), followed by adenocarcinoma (4-15%).

**Squamous cell carcinoma of the maxillary sinus (carcinoma planocellulare)**

*Clinical presentation:* There is plethora of early symptoms, depending on the clinical stage and the localization of the tumor. The most common ones are:

- Dull pain in the maxilla (bone or teeth), which aggravate when the body is in horizontal position (33%)
- A swelling or lump in the maxillofacial area, with no other complaints (22.42%)
- Nasal symptoms (29.07%), that can be serous-purulent exudation (52.01%), serous-bloody exudation (20.71%), reduced or no breathing from one of the nostrils (28.57%). In certain cases headache, teardrops, pain in the eye, selling of the eyelids, pain in the ears or enlarged lymph nodes can be observed.

Some specific symptoms are associated with specific localization of the tumor. The clinical classification of the maxillary sinuses carcinomas, proposed by Ohngren in 1933, was proved to be extremely valuable for the clinical practice. He divided the maxillary sinus into four quadrants and there are specific symptoms for tumors, located in each quadrant. According to Ohngren a line that starts from the interior angle of the eye and ends at the angle of the mandible separates the maxillary sinus into two aspects superior-posterior and inferior-anterior. The mesial line divides them in two mesial and lateral aspect.

So if the tumor is located at the inferior-anterior mesial quadrant, destruction of the alveolar bone, the hard palate or the anterior of the sinus can be observed. If the tumor destroys the alveolar process or the hard palate, it can infiltrate the oral mucosa. Nasal symptoms can also manifest – exudation or no breathing, as well as pain in the jaws and in the teeth, asymptomatic tooth mobility and tooth loss, the tumor grows inside the non-healing tooth socket. There can also be facial asymmetry, a swelling at the frontal wall of the sinus, the nasal-labial fold disappears, if the tumor infiltrates the infraorbital and alveolar nerves the pain is severe.

If the tumor is located at the inferior-anterior-lateral quadrant the main symptom is facial asymmetry, due to swelling at the distal areas of the upper jaw. These tumors have the tendency to infiltrate the ramus of the mandible and the TMJ, which leads to decrease of
opening of the mouth. Sometimes tumors infiltrate the exterior carotid artery or its branches, causing severe hemorrhages. This type of tumor metastasizes the lymph nodes rather early.

When the tumor locates at the superior-posterior mesial quadrant, symptoms from the nose, orbit and ethmoid labyrinth can be observed. This quadrant is not easily accessible and that is why it is very difficult to recognize these tumors. The main symptoms are determined by the direction of the infiltration of the tumor. There can be bloody exudate from the nose, sometimes purulent exudate, that has dreadful smell, reduced breathing capacity, but only one of the nostrils is affected. Teardrops, hypoesthesia in the inner corner of the eye exophthalmos, diplopy, constant headache, pain that radiates through the branches of the maxillary nerve and disturbed skin perception may also be present. If the tumor grows inside the nasopharynx a pain in the temporal area may manifest.

If the tumor is located at the superior-posterior-lateral quadrant the main symptom is going to be stubborn pain in the jaws and teeth, as well as long lasting headache, exophthalmos and tendency of the tumor to grow towards the orbit and the pterygopalatine fossa.

When tumors have advanced significantly, the initial localization of the tumor is impossible to be determined.

In conclusion, the primary clinical symptoms of the cancer of the maxillary sinus are similar to the ones of the sinusitis, and the definite diagnose can be determined only after having the results from the biopsy. In some cases the data from the medical history, the clinical examination and the radiographic examination (CT) suggests that the diagnose should be chronic sinusitis, but after surgery is performed, the results from the biopsy show squamous cell carcinoma, which indicates secondary surgical procedure – resection of the maxilla, followed by radiation therapy. This fact shows the great importance of the histological examination for the definite diagnose, and the adequate treatment.

Diagnose: It is based on the data from the medical history, the clinical examination and the radiographic findings. The definite diagnose is determined after considering the results from the biopsy.

When the tumor is located at the inferior-anterior-mesial quadrant the CT will show destruction of the alveolar process, the hard palate and the frontal wall of the sinus.

If the tumor locates at the superior-posterior-mesial quadrant the CT will show total shadowing of the sinus, and destruction of the adjacent bone walls, and infiltration towards the nasal cavity, ethmoid labyrinth, orbit.

When the tumor is located at the superior-posterior-lateral quadrant the CT shows destruction of the walls of the sinus and spreading of the tumor within the adjacent structures.

Differential diagnose:
1) Chronic sinusitis
2) Osteoma
3) Fibroma
4) Odontogenic cysts
Application of the TNM system for the tumors of the maxillary sinus

Primary tumor criteria
Tis – carcinoma in situ
T0 – there is no primary tumor
T1 – the tumor is still within the mucosa of the sinus and there is no erosion or destruction of bone
T2 – the tumor has eroded or destructed the bone, including the hard palate and the mesial nasal meatus
T3 – the tumor infiltrates: the skin of the cheek, the posterior wall of the maxillary sinus, the floor or the mesial wall of the orbit, the anterior ethmoid sinus
T4 – the tumor infiltrates the orbital cavity or some of the following structures: lamina cribiformis, posterior ethmoid sinus, sphenoid sinus, nasopharynx, hard palate, pterygomandibular space, temporal fossa, base of the cranium.

Regional lymph nodes metastases criteria:
N0 – the regional lymph nodes are not affected
N1 – it is possible to palpate a metastasis in a regional lymph node, which is mobile and is located on the same half of the body as the primary tumor (homo lateral), not more than 3cm in diameter
N2a – a metastasis in a single homo lateral lymph node, which is more than 3cm and less than 6 cm in diameter
N2b – metastases in multiple homo lateral lymph nodes, more than 3cm and less than 6 cm in diameter.
N2c – metastases in multiple bilateral and contra lateral lymph nodes, less than 6 cm in diameter
N3 – a metastasis in a lymph node, that is more than 6 cm in diameter
Nx – it is impossible to palpate the lymph nodes and determine their condition
M – distant metastases criteria:
M0 – there are no distant metastases
M1 – there is a distant metastasis
Mx – it is impossible to find distant metastases

Clinical stages, based on T,N and M values:
Stage 0 – TisN0M0
Stage I – T1N0M0
Stage II – T2N0M0
Stage III – T3N0M0, T1N1M0, T2N1M0, T3N1M0
Stage IV – T4N0M0; T4N1M0; T1,2,3,4N2M0; T1,2,3,4N1,2,3M1

Treatment: The first choice therapy in these tumors is the complex therapy (surgery and radiation). The specific method depends on the clinical stage of the tumor, the age and systemic condition of the patient.
In clinical stages I, II, III the treatment of choice includes surgical resection of the upper jaw and postoperative radiation therapy. In clinical stages I and II, when the tumor is located at the alveolar process or the inferior aspect of the sinus, subtotal resection of the maxilla can be performed (preservation of the floor of the orbit). In clinical stage III, when the tumor is located at the mesial aspect of the sinus, a total resection of the maxilla, with or without removal of the structures in the orbital cavity is performed. In these cases it is also possible to administer preoperative radiation therapy (30-40Gy), followed by resection of the upper jaw and postoperative radiation therapy (60Gy).

When tumors are too advanced, treatment measures have only palliative manner – combined radiation and medical therapy are proposed (Bleomycin, Vincristin, Methotrexate, Cyclophosphamide)

The five year survival rate when complex therapy is performed is 35%.

Metastatic carcinomas of the upper jaw

The metastatic tumors are a rare occasion in the oral cavity and the jaw bones. They are 1-3.2% of all malignancies with this localization. Metastases in the jaws can rarely be observed, but are more common in the mandible. The men to women ratio is 1:1.1, while for oral mucosa metastases the ratio is 1.6:1. Metastases in the jaws are more common than the ones in the oral mucosa (2.5:1). The typical age of the patients is between 40 and 70 years of age.

The metastatic tumors of the jaws originate from lung cancer tumors, mammal gland tumors, kidney tumors, liver tumors, suprarenal gland tumors, thyroid gland tumors, testicle tumors, ovaries tumors, prostate gland tumors, tibia tumors, digestive system tumors. In men the oral metastases originate from tumors of the lungs, kidneys, prostate gland, bones, skin. In women the most common original tumors are the ones of the mammal glands, female sexual organs, colorectum, bones, and kidneys.

Clinical presentation: The clinical manifestation of the malignant metastases has great variety and individuality. Some cases can be totally asymptomatic and is some the sole clinical symptom might be pain in the pain in the jaws or in the teeth, hyperesthesia, hypoesthesia, anesthesia, trismus, TMJ pain, jaw bone deformities, tooth mobility, hardly or non-healing extraction wounds.

In cases when due to tooth pain and tooth mobility unneeded exodontia is performed, the extraction wounds do not heal, but are filled with bleeding granulations. The biopsy then indicates seeking the primary tumor. The radiographic findings and the lymph node biopsy may confirm the diagnose, which in this case would be pulmonary carcinoma, and the patient should be referred to an oncological facility (hospital).

Diagnose: It is based on the data from the medical history, the clinical examination and the biopsy.

Differential diagnose:
1) Primary intraosseous carcinoma
2) Malignant ameloblastoma
3) Central malignant tumor of the salivary glands
4) Sarcomas of the jaws

*Treatment:* Systemic treatment of the primary tumor is performed, and the therapeutic measures for the metastasis include palliative radiation and chemotherapy, in order to prevent bleeding and ulceration of the oral mucosa and gingiva.

The patients that have metastatic tumors in the upper jaw die within a year after the appearance of the metastasis, which is why we should be familiar with these conditions, in order to treat our patients properly.

**Malignant connective tissue tumors of the maxilla**

According to the international histological classification, the malignant connective tissue tumors (sarcomas) can be:

1. Bone forming tumors
   - Conventional central osteosarcoma
   - Teleangiectical osteosarcoma
   - Intraosseous well differentiated osteosarcoma
   - Round cell osteosarcoma
   - Juxtacortical osteosarcoma
   - Periosteal osteosarcoma
   - High degree superficial osteosarcoma

2. Cartilage forming tumors
   - Chondrosarcoma
   - Juxtacortical chondrosarcoma
   - Mesenchymal chondrosarcoma
   - Light cell chondrosarcoma
   - Malignant chondroblastoma

3. Bone marrow tumors
   - Ewing sarcoma
   - Primary neuroectodermal bone tumor
   - Malignant bone lymphoma
   - Myeloma

4. Vasal tumors
   - Angiosarcoma
   - Malignant hemangiopericytoma
   - Fibrosarcoma
   - Malignant fibrous histiocytoma
   - Liposarcoma
   - Leyomyosarcoma
   - Undifferentiated sarcoma
Osteogenic sarcoma

This tumor is more common in younger patients (5-58 years). It originates from cortical or cancellous bone of the alveolar process. In some cases it grows slowly (months, years) and to significant size 8-27cm, and in other cases it grows fast. When located on the upper jaw, it takes months or years to develop, but it sometimes develops really fast. As it develops, the tumor causes bone deformities, facial asymmetry, as well as difficulties in eating, talking, breathing, disturbed vision. In the beginning of its development the tumor has round shape and clear borders, it grows inside the bone and pushing it forms a capsule of a kind. It rarely infiltrates the adjacent tissues, the skin and the mucosa expand and become thinner, but the tumor does not infiltrate them, its surface is round, sometimes uneven. It is firm, with “bony” consistency on palpation. If the mucosa above the tumor is traumatized, it can ulcerate.

This tumor grows expansively, pushing the surrounding tissues aside, it rarely infiltrates and destroys them. If the tumor grows next to teeth it pushes them aside, they do not fall out and remain within the tumor instead. The skin and mucosa above the tumor are extended, but remain intact and preserve their original color and turgor. This tumor does not disintegrate and does not cause bleeding, but causes severe pain. It rarely metastasizes the lymph nodes and the lungs, it does not lead to any systemic symptoms, even in late stages.

Diagnose: The radiographic appearance depends on the type of the sarcoma. The osteolytic type shows radiolucency – intensive destruction of all layers of the affected bone. The reactive bone formation from the periosteum have needle-like appearance and is slightly present. This type grows into the adjacent structures (cheek) rather soon.

In the perosteal type during the initial stages there are no radiographic changes. As the tumor develops, a periosteal defect that keeps enlarging becomes visible, its margins are uneven, ragged, the cancellous bone is intact. In the late stages of development of the tumor, reactive changes in the periosteum and existence of radiantly positioned formations can be observed.

In the central type there is radiolucency, which has irregular shape, unclear, ragged borders. Within the tumor itself there are small bone formations, which resemble cotton pellets.

Diagnose: The definite diagnose requires a histological examination.

Differential diagnose:
1) Subacute osteomyelitis
2) Carcinomas
3) Complex odontoma
4) Osteoma

Treatment: The treatment of choice is the radical surgical therapy. No recurrences have been observed in five year period.
**Chondrosarcoma**

Chondrosarcoma is a rarely seen tumor, located at the jaws. It can be observed in young and middle aged patients, but in old ones it’s not very often. The initial localizations of the tumor are the upper and central aspects of the maxilla, mainly the midline of the alveolar ridge.

*Clinical presentation:* When the tumor is located in the upper jaw it causes facial asymmetry, that goes together with some functional disturbances - nose obstruction, epistaxis, exophthalmos, trismus. In the late stages chondrosarcoma has the typical clinical appearance of the malignant tumors – infiltrative growth, bone destruction, pain at rest and on palpation, development of distant metastases, mainly pulmonary.

This disease often begins and develops with severe pain, which cannot be coped even with opioid analgesics. The tumor has dense or elastic consistency and its borders are not well distinguished. Chondrosarcoma shows aggressive, infiltrative growth, it rapidly invades the maxillary sinus and the ethmoid bone. In the late stages it ulcerates and undergoes necrosis. It often metastasizes the lungs trough the blood circulation, but also sometimes metastasizes the regional cervical lymph nodes.

*Diagnose:* It is based on the data from the medical history, the clinical examination, the radiographic findings and the biopsy.

*Differential diagnose:*
1. Chondroma
2. Carcinoma

*Treatment:* The treatment of choice is the radical surgical therapy, but there can often be recurrences.

**Ewing’s sarcoma**

The first description of the disease was given by James Ewing (1921), who named it diffuse epithelial myeloma. It is 4-15% of all primary bone tumors and 1% of all malignancies in children. It is a rare finding in the maxillofacial area (0.74%) and is mainly located at the mandible. It is observed mainly in children at the age of 13.

*Clinical presentation:* The beginning of the disease is asymptomatic or starts with dull pain at night, warm or burning sensation. The pain is located in the jaws. These complaints are soon followed by tooth mobility, swelling of the soft tissues, adjacent to the bone. Increased body temperature up to 39-40 C. As it evolves, the disease causes general weakness and changes in the blood count – leukocytosis, increased erythrocyte sedimentation rate, secondary anemia.

In a lot of the pediatric patients, changes in the soft tissues (hyperemia, increased temperature) adjacent to the tumor may become evident. There can also be a sensation, that resembles fluctuation. After a while, the symptoms go away for some time, there is no pain, teeth are no longer mobile, but after that all the symptoms come back. The tumor grows and then reduces its size many times. In its initial phase, this tumor resembles the clinical presentation of acute or subacute osteomyelitis.
Ewing’s sarcoma metastasizes distant organs such as vertebrae, skull bones, ribs, rather early. Multiple metastases can be observed in the first months of development of the disease. There have never been described metastases in the regional lymph nodes.

**Diagnose:** It is based on the data from the medical history, the clinical examination, the radiographic findings and the biopsy.

The radiographic examination shows radiolucency in the jaw – destruction that looks similar to other bone tumors (eosinophilic granuloma, osteogenic sarcoma, jaw bone metastases) and is not a pathognomonic one. In the long bones of the body Ewing’s sarcoma shows periosteal bone formation, and in the jaws it does not.

Histologically, the tumor is made of abundant non-differentiated cells (round or oval, equal in size, containing a large hyperchromatic nucleus). The cells are equally dispersed, in a layer-like manner and can resemble non-differentiated carcinoma. Some necrotic fields can be visible.

**Differential diagnose:**
1) Acute and subacute odontogenic osteomyelitis
2) Eosinophilic granuloma (central type)
3) Odontogenic sarcoma or reticulosarcoma in children

**Histological differential diagnose:**
1) Malignant lymphomas
2) Metastatic neuroblastoma
3) Rhabdomyosarcoma
4) Small cell osteosarcoma
5) Primary neuroectodermal tumors

**Treatment:** The most common treatment modality is the radiation therapy, because this tumor is extremely radiosensitive. After the radiation therapy the pain is relieved, the size of the tumor decreases, the destructed bone is replaced by newly formed bone tissue. The general condition of the patient gets better. Other treatment modalities are surgery and chemotherapy.

**Multiple myeloma (Myeloma multiple, Plasmocytoma, Rustitski-Kohler’s disease).**

This disease has unknown etiology. It is a monoclonal gamapathy. All the pathologic cell are monoclonal and are capable of expressing only one type of protein. In most of the cases it is immunoglobulin G, immunoglobulin A is less frequent, and immunoglobulins D and E are extremely rare findings.

**Clinical presentation:** This disease develops slowly, main symptoms happen to be general weakness, pain in the bones. There sometimes are increased erythrocyte sedimentation rate, unexplained proteins in the urine, and anemia that that does not heal after treatment.

In certain cases coagulopathies develop, due to thrombocytopenia and damage to the thrombocytes, caused by the para-proteins and the pathologic complexes, comprised of the pathologic proteins and some coagulation factors (II, V, VIII, VII).
Anemia is due to the suppression of the normal hematopoiesis by the growing myeloma in the bone marrow, the short life of the erythrocytes and the blood loss due to the coagulopathies.

Diagnose: It is based on the specific triad – osteolytic changes in the bones, myeloma cells in the bone marrow, pathologic proteins in the blood or the urine. The radiographic changes appear as multiple osteolytic lesions (skull, vertebrae, ribs, jaws), lack of reactive processes around these lesions, they look like bitten by a moth.

When a blood sample is taken, the results show increased number of proteins, the so-called M gradient (a specific protein), increased levels of Calcium and urinal acid.

The M gradient can be observed in the urine too, and is a specific mark, more specific than the Ben-Jones’ protein.

There can also be normochromic anemia, normal values of leukocytes and thrombocytes, increased erythrocyte sedimentation rate.

Differential diagnosis:
1) Plasmocytosis in the bone marrow in hepatitis, liver cirrhosis, chronic infections, Hodgkin’s disease.
2) Metastatic carcinomas in the bones
3) Hyperthyroid lesions in the bones

Treatment: The treatment is performed in a hematology clinic, infusions are administered in order to correct the hypercalcemia, hyperurikemia, dehydration. Prednisolone and Alopurinole are infused. After that cytostatic drugs are administered – Sarcolisine, Cyclophosphamide. This way the proliferation of the plasma cells is suppressed, the pain in the bones is relieved.

The mean survival period is 3 years.

Fibrosarcoma

This tumor can have two types – central and peripheral, it can either originate from fibrous elements of the subperiosteal layer of the bone, or from fibrous polyps in the maxillary sinus.

Clinical presentation: Fibrosarcoma of the jaws develops rapidly (3-4 months) chief complaints are severe tooth pain, tooth mobility and tooth loss. The further symptoms depend on the localization and the spreading of the tumor. For example, if it grows into the maxillary sinus or the nasal cavity, there will be obstruction of the nose. The fibrosarcoma has irregular shape and is often made of separate nodes that merge, and are covered by eroded mucosa. The color of these nodes is dark blue. Tumor’s consistency is dense or elastic, it rarely metastasizes.
**Diagnose:** It is based on the data from the medical history, the clinical examination, the radiographic changes (destruction of the bone with unclear borders) and the biopsy.

**Differential diagnose:** Carcinomas

**Treatment:** The treatment of choice is the radical surgical therapy. It often recurs.

**Myxosarcoma**

This tumor is very rare, and also affects mainly young people and children. In the beginning of its development, this tumor has no specific symptoms. As it develops, it causes destruction of bone, which leads to deformities of the bones and tooth mobility. It rarely metastasizes. Diagnose is based on the biopsy. It does not recur after radical surgical therapy.

**Lymphoma of the maxilla**

Lymphomas in the oral cavity are 3.5% of all tumors in this region and are mainly located at the upper jaw (77%). It originates from lymphoid elements, situated in the submucous layer of the maxillary sinus or in the cancellous bone of the maxilla. This tumor has no specific symptoms, and deforms the jaw and grows into the adjacent structures, its borders are uneven. If the tumor grows into the oral cavity it often ulcerates. The symptoms depend on the localization of the tumor.

Radiographic findings show bone destruction with unclear borders. The definite diagnose is determined after having the results from the biopsy. This tumor is very radiosensitive. The treatment of choice is complex – radiation and chemotherapy. If the tumor is diagnosed during the early stages, the prognosis is better. The five year survival rate is 28-73%

**Malignant osteoblastoclastoma (Osteoblastoclastoma malignum)**

This malignant type of the tumor was first described by Cooper, W.H., Trawers, H. W., 1818. It may either be primary malignant or it can begin as a benign type.

**Clinical presentation:** It develops rapidly and asymptotically, there are facial asymmetry and bone deformation. The variable symptoms depend on the localization of the tumor and may be: tooth pain and mobility in intact teeth, movement of teeth, symptoms from the maxillary symptoms, exophthalmos, diplopia.
**Diagnose:** The radiographic images show bone destruction with uneven contours, which suggests a malignancy. This tumor has three specific histological characteristics: well-expressed bone destruction, cell pleocytosis, cellular atypism.

**Treatment:** The treatment of choice is complex – radical surgical excision of the tumor, followed by postoperative radiation therapy.

**Malignant epithelial tumors of the mandible**

These tumors can be divided into three groups, depending on their origin – primary, secondary and metastatic. The primary tumors originate from remaining epithelial cells inside the jaw or from radicular cysts and benign bone tumors (adamantinoma, myxoma, Ameloblastic fibroma). Secondary tumors originate from primary tumors of the gingiva and the floor of the mouth, that infiltrate the mandible. Metastatic tumors have variable origin – tumors of the thyroid gland, lungs, prostate gland etc.

**Primary intraosseous carcinoma (carcinoma primaria intraosale)**

This tumor develops from the epithelial residues of Malassez and spreads either deep inside the bone or under the periosteum. It affects both genders, but tumors in women are more often (3:2) and is mainly located at the posterior aspects of the mandible.

**Clinical presentation:** The tumor can develop asymptptomatically for a long period of time, especially when located at the molar region. Symptoms that emerge may be from the lower lip, the lymph nodes and the oral mucosa. The initial symptom is swelling of the oral mucosa in 80.6% of the cases, pain that irradiates through the branches of the trigeminal nerve.
(73.5%), metastases in the regional lymph nodes in 73.5% of the cases, disrupted sensitivity of the lower lip (60%).

Pain comes without any reason and have striking manner, irradiate and suggest pulpitis. After the inferior alveolar nerve is compressed and then destructed, the lower lip is numb.

In several cases the only symptom is the increased tooth mobility, which leads to exodontia, but the extraction wounds do not heal, and are filled with granulation-like tissue instead. Others may have increased tooth mobility on recession of the gingiva.

In asymptomatic patients, when the tumor has destroyed the cortical layer and grows inside the oral cavity, main symptoms are swelling of the mucosa and facial asymmetry. This tumor metastasizes the regional submandibular lymph nodes rather early.

WHO has not made clinical classification of this tumor, but Paches and col., have proposed one, which has clinical importance:

- **T1** – a tumor, which has affected only one anatomical area
- **T2** – a tumor, which affects not more than two anatomical areas
- **T3** – a tumor, which affects more than two anatomical areas
- **T4** – a tumor, which also affects other body systems.

The N and M criteria match the ones we previously described.

**Diagnose:** It is based on the data from the medical history, the clinical examination, the radiographic changes. The biopsy determines the definite diagnose.

The radiographic images demonstrate bone destruction, no reactive or reparative processes in the bone or the periosteum, teeth within the tumor are not resorbed. The other possible radiographic finding is destruction of bone and diffuse infiltrative growth and sharpened borders of the tumor.

**Treatment:** The choice of a treatment modality depends on the morphologic type of the tumor, its localization and spreading, the systemic condition and age of the patient. The surgical therapy may be partial or total resection of the mandible-hemi exarticulation, without lymph dissection. In clinical stage IV cases the treatment is palliative – radiation and chemotherapy.

**Malignant ameloblastoma (Ameloblastoma malignum)**

This tumor can either develop as primary malignant or can originate from primary benign adamantinoma.
Clinical presentation: Tumor’s clinical appearance is similar to the one of the benign ameloblastoma and the diagnose is determined after the histological examination. In other patients tumors grow rapidly, there is tooth pain and increased tooth mobility, tooth loss, the tumor infiltrates the adjacent tissues, metastasizes. This type of cancer metastasizes mainly the lungs, and less commonly the lymph nodes, bones, cranium.

Diagnose: The radiographic examination demonstrates destruction of bone with unclear borders, which suggests a malignant lesion. The definite diagnose is determined after considering the results from the biopsy.

Treatment: The therapy of choice is the surgical one, which, depending on the clinical stage of the tumor and its size can vary from partial to total resection of the mandible, without lymph nodes dissection. If there are distant metastases, the treatment is palliative – radiation and chemotherapy.

Secondary carcinoma

Secondary tumors of the mandible develop from already existing tumors of the gingiva and the floor of the mouth, which infiltrate the mandibular bone.

The anamnesis provides information about a persisting ulcer or swelling of the gingiva or the mucosa of the floor of the mouth, which gradually invades the mandibular bone. The disease remains asymptomatic and unnoticed for a long time. If an X-ray was done, radiolucency (destruction of bone) can be observed.

The treatment of choice is resection of the mandible – continuous, partial or total, depending on the size of the tumor.

Metastatic tumors

These tumors are extremely rare finding in the oral cavity – 1% of all malignancies in this region.

The metastatic tumors can originate from primary tumors of the lungs, mammal glands, liver, suprarenal glands, testicles, ovaries, prostate gland, pleura, peritoneum, digestive system.
Metastases can be located at the gingiva, buccal and palatal mucosa, the tongue, floor of the mouth, mandibular bone. The main localization is the mandibular bone (95%) and develops from mammal gland cancer.

Clinical presentation: The clinical symptoms may vary and are strictly individual. The metastases may happen to be the reason for the discovery of the primary tumor, or they can develop some time after the treatment of the initial cancer.

The changes in the oral cavity are completely asymptomatic and often resemble benign tumor or tumorous conditions. When searching for the reason for the existing general weakness, loss of weight, loss of appetite, clinicians sometimes find the primary tumor. The radiographic examination proves the existence of the primary tumor in lungs, thyroid gland, kidneys.

In some patients metastases in the jaw cause pain and resemble exacerbated odontogenic cyst, which suggest a surgical cystectomy, but when a sample is sent for biopsy the results show a metastasis of a malignant histiocyto ma. The patient is then referred to oncologic facility for further treatment.

Diagnose: Metastases in the mandible sometimes make it difficult to find the location of the primary lesion. The results significantly depend on the effective collaboration between a clinician and a pathologist.

Differential diagnose:
1) Primary intraosseous carcinoma
2) Odontogenic malignant tumors
3) Lower jaw sarcomas
4) Inflammatory odontogenic cyst
5) Osteomyelitis

The radiographic examination demonstrates the existence of a lesion of the bone with non-defined borders. In approximately 5% of the cases there are no radiographic changes, which does not mean that there is no malignancy. That is why the diagnose should be supported by the results from the histology.

Treatment: The metastases in the mandible suggest a widespread disease and the treatment should be palliative.

The prognosis in these cases is poor and patients die in few months after the emergence of the metastasis.

Connective tissue malignancies of the mandible

Osteosarcoma

This tumor is extremely malignant and is 4-6.5% of all sarcoma in people.

Clinical presentation: Osteosarcoma has a variety of clinical signs and symptoms, depending on the localization and size of the tumor. In the mandible this tumor develops rapidly, causing pain in intact teeth, mobility and movement of teeth, fetor ex ore,
paraesthesia or unpleasant sensations in the lower lip, a swelling with variable size and density. In some cases tumors grow to significant size, but there is no painful sensation. As tumors grow, patients show signs of intoxication – high body temperature (38-39°C), increased erythrocyte sedimentation rate, fatigue, loss of appetite, etc.

*Diagnose:* Osteosarcoma has three radiographic-morphologic types: osteolytic, osteoplastic and mixed. Osteolytic type is more common. Its radiographic appearance demonstrates osteolytic lesion, which has irregular shape, unclear borders, widened periodontal spaces and resorbed roots of the teeth. The osteoplastic type appears as non-structural, thickened bone, sometimes a needle-like shape can be observed, positioned perpendicularly to the cortical bone.

* Differential diagnose:*
  1) Subacute osteomyelitis
  2) Carcinomas
  3) Complex odontoma
  4) Osteoma

*Treatment:* It is complex and comprises of surgical therapy (resection), radiation therapy and chemotherapy.

The five year survival rate is 45.2%.

**Chondrosarcoma**

It is a rare tumor, located at the jaws. In the lower jaw it locates at the area of the premolars, molars and the symphysis of the mandible.

*Clinical presentation:* The clinical symptoms are determined by the localization of the tumor. If it affects the mandible, it manifests as asymptomatic swelling of the gingiva, which leads to increased mobility of the teeth, anesthesia of the lower lip. Some of the patients complain from pain in the jaw or in the teeth, stubborn headache. If a CT is made during this initial stage, a malignant destruction with unclear borders will be observed.
Diagnose: The radiographic imaging demonstrates a soft tissue lesion with partial calcification, or bone destruction with uneven borders, sometimes root resorption, which is not typical and suggests malignant development. The definite diagnose is determined after considering the results from the biopsy, which show groups of hondrocytic cells and chondrus matrix.

Treatment: The choice of the appropriate treatment modality is based on the size and localization of the tumor. Treatment modalities include surgical therapy, radiation therapy and complex therapy.

Resection of the jaws is the surgical treatment and it is performed 2-3 cm into healthy tissue. When surgery is not an option a radiation therapy can be performed.

The five year survival rate is 67.5%.

Fibrosarcoma
This tumor is relatively rare finding in the jaws. It develops from immature connective tissue. It affects both jaws, as the peripheral type affects the maxilla and the central type affects the mandible.

Clinical presentation: It develops rapidly and asymptotically. The initial signs and symptoms may be: more common – swelling of the mucosa on the vestibular area and facial asymmetry, less common – pain, mobility and transition of teeth. As the disease develops, it causes severe pain, which irradiates towards the ear, TMJ, temporal area, neck.

Diagnose: The radiographic examination proves the existence of a radiolucent lesion (destruction of bone), which has without clear and sharp edges, a typical presentation of a malignant tumor.

Histologically, the tumor is made of spindle-shaped long cells, containing small cytoplasm. There can also be seen ovoid cells. Mitotic figures are not often. That is why their presence suggests malignant transformation.

Treatment: Surgical therapy is the treatment modality of choice.

Mandibular lymphoma
Lymphomas in the oral cavity and the jaws are rare – 0.1-5% of all lymphomas. They develop in people between 3 and 82 years of age, and are more common in men than in women (2:1).
Clinical presentation: The clinical signs and symptoms of the tumor are determined by the clinical stage and localization of the tumor and can vary from unexplained tooth pain, increased mobility of teeth, anesthesia, ulcerations and bleeding gingiva to a soft tissue, growing inside the post extraction alveolar sockets.

In some patients there can be neuralgic pain or swelling, which causes facial deformation. The radiographic findings are similar to the ones of the other malignant tumors in the jaws.

Diagnose: The diagnose is based on the radiographic (destruction of bone) and histologic (biopsy) examination.

Treatment: Treatment modalities comprise of surgical therapy, radiation therapy and chemotherapy. Complex treatment provides best results - radiation therapy 24-60Gy and chemotherapy (Adriamycin). Surgical therapy may be tumor resection, partial or total resection of the jaw.

The literature data suggests three year survival rate in clinical stage I of 90%, in clinical stage II – 47.6%. The five year survival rate is approximately 50%, and patients in clinical stage I live more than ten years.
SALIVARY GLAND TUMORS
(Cvetan Cvetanov)

The majority of salivary gland tumors (about 80%) arise in the parotid glands. The submandibular glands account for 10 to 15% of tumors, and the remaining tumors develop in the sublingual or minor salivary glands. Approximately 80% of parotid gland tumors and 50% of submandibular gland tumors are benign, more than 60% of tumors in the sublingual and minor salivary glands are malignant. According to Tonchev, TS. et al., 2016 parotid masses are benign in 77.9% of the cases. The risk of malignancy increases as the size of the tumor decreases. Over 85% of salivary gland tumors occur in adults. Salivary tumors in children are most often located in the parotid glands, and about 65% of all salivary tumors found in children are benign (Main, J.H.P. et al., 1976; Batsakis, J.G. et al., 1981; Everson, J.W., Cawson, R.A., 1985; Spiro, R.H. et al., 1986; Bailey, B.J., 1993; Eisele, D.W., Johns, M.E., 1993; Regazi, J., Sciubba, J., 1993; Carlson, E.R., 1995; Bakardjieva, A., Pechalova, P., 2003; Grisius, M.M., Fox, P.S., 2003).

1. Classification of salivary gland neoplasms. The 2005 World Health Organization (WHO) classification of SGTs is complex and comprises 10 benign and 23 malignant entities of epithelial origin.

1. A. Benign epithelial tumors:
   - Pleomorphic adenoma
   - Myoepithelioma
   - Basal cell adenoma
   - Warthin tumor
   - Oncocytoma
   - Canalicular adenoma
   - Sebaceous adenoma
   - Lymphadenoma
   - Ductal papilloma
   - Cystadenoma

1. B. Malignant epithelial tumors:
   - Acinic cell carcinoma
   - Mucoepidermoid carcinoma
   - Adenoid cystic carcinoma
   - Polymorphous low-grade adenocarcinoma
   - Epithelial-myoepithelial carcinoma
   - Clear cell carcinoma, not otherwise specified
   - Basal cell adenocarcinoma
   - Malignant sebaceous tumors
   - Cystadenocarcinoma
   - Low-grade cribriform cystadenocarcinoma
2. Benign tumors

2.1. Pleomorphic adenoma

Etiology: The pleomorphic adenoma is the most common tumor of the salivary glands; overall, it accounts for about 60% of all salivary gland tumors. It is often called a mixed tumor because it consists of both epithelial and mesenchymal elements. About 85% of these tumors are found in the parotid glands, 8% are found in the submandibular glands, and the remaining tumors are found in the sublingual and minor salivary glands. This tumor represents the most common neoplasm in each of the salivary glands and accounts for about 50% of salivary tumors in the minor salivary glands (Everson, J.W., Cawson, R.A., 1985; Carlson, E.R., 1995).

Pleomorphic adenomas may occur at any age, but the highest incidence is in the fourth to sixth decades of life. It also represents the most common salivary neoplasm in children. There is a slight predilection for female gender (Main, J.H.P. et al., 1976).

Clinical manifestation: On clinical examination, these tumors will appear as painless, firm, and mobile masses that rarely ulcerate the overlying skin or mucosa. In the parotid gland, these neoplasms are slow growing and usually occur in the posterior inferior aspect of the superficial lobe. Mixed tumors in the submandibular glands present as well-defined palpable masses. It is difficult to distinguish these tumors from malignant neoplasms and indurated lymph nodes. Intraorally, the mixed tumor most often occurs on the palate, followed by the upper lip and buccal mucosa. Pleomorphic adenomas can vary in size, depending on the gland in which they are located. In the parotid gland, the tumors are usually several centimeters in diameter but can reach much larger sizes if left untreated. When observed in situ, the tumors are encased in a pseudocapsule and exhibit a lobulated appearance (Main, J.H.P. et al., 1976; Eisele, D.W., Johns, M.E., 1993; Regazi, J., Sciubba, J., 1993).
Pathology: The gross appearance of pleomorphic adenoma is that of a firm smooth mass within a pseudocapsule. Histologically, the lesion demonstrates both epithelial and mesenchymal elements. The epithelial cells make up a trabecular pattern that is contained within a stroma. The stroma may be chondroid, myxoid, osteoid, or fibroid. The presence of these different elements accounts for the name pleomorphic tumor or mixed tumor. Myoepithelial cells are also present in this tumor and add to its histopathologic complexity. One characteristic of a pleomorphic adenoma is the presence of microscopic projections of tumor outside of the capsule. If these projections are not removed with the tumor, the lesion will recur.

Differential diagnosis:
- Malignant tumors of the salivary glands – characterized by faster growing, infiltration of surrounding structures, facial nerve involvement; diagnosis performed by histopathological examination.
- Between different types of benign tumors - diagnosis performed by histopathological examination.
- Cystic lesions of the salivary glands-consistency is typically soft and fluctuant, they are characterized by accumulation of mucoid material (Atanasov, D., 2011).

Treatment: The treatment of this lesion consists of surgical removal with adequate margins. Because of it's microscopic projections, this tumor requires a wide resection to avoid recurrence. In spite of the capsule, close excision should not be attempted. A superficial parotidectomy is sufficient for the majority of these lesions. A small tumor in the tail of the parotid gland may be removed with a wide margin of normal tissue, sparing the remainder of the superficial lobe. Lesions that occur in the submandibular gland are treated by the removal of the entire gland (Main, J.H.P. et al., 1976; Eisele, D.W., Johns, M.E., 1993; Regazi, J., Sciubba, J., 1993).

2. 2. Monomorphic adenoma

A monomorphic adenoma is a tumor that is composed predominantly of one cell type, as opposed to a mixed tumor (pleomorphic adenoma), in which different elements are present (Regazi, J., Sciubba, J., 1993; Grisius, M.M., Fox, P.S., 2003).
2.3. Papillary cystadenoma lymphomatosum

Papillary cystadenoma lymphomatosum, also known as Warthin’s tumor is the second most common benign tumor of the parotid gland. It represents about 6 to 10% of all parotid tumors and is almost always located in the parotid gland, most commonly in the inferior pole of the gland, posterior to the angle of the mandible. The tumor demonstrates a slight predilection toward males, and it usually occurs between the fifth and eighth decades. These tumors occur bilaterally in about 6 to 12% of patients (Main, J.H.P. et al., 1976; Batsakis, J.G. et al., 1981; Everson, J.W., Cawson, R.A., 1985; Carlson, E.R., 1995).

Clinical Presentation: This tumor presents as a well-defined slow growing mass in the tail of the parotid gland. It is usually painless unless it becomes superinfected.

Pathology: The gross appearance of this tumor is smooth, with a well-defined capsule. Cutting a specimen reveals cystic spaces filled with thick mucinous material. Histologically, the tumor consists of papillary projections lined with eosinophilic cells that project into cystic spaces. The projections are characterized by a lymphocytic infiltrate.

Differential diagnosis:
1. Oncocytic papillary cystadenoma (OPC) - rare tumor of the major salivary glands, often confused with Warthin’s tumor. OPC characterized by lowest level of the lymphoid components;
2. Lymphadenoma, lymphoepithelial cysts – absence of double layered epithelium (Stratiev, A., 2000).

Treatment: Papillary cystadenoma lymphomatosum is most often located in the tail of the parotid gland and is easily removed with a margin of normal tissue. Larger tumors that involve a significant amount of the superficial lobe of the parotid gland are best treated by a superficial parotidectomy. Recurrences and malignant degeneration of this tumor are rare (Batsakis, J.G. et al., 1981; Everson, J.W., Cawson, R.A., 1985; Carlson, E.R., 1995).

2.4. Oncocytoma

Oncocytomas are less common benign tumors that make up less than 1% of all salivary gland neoplasms. The name of the tumor is derived from the fact that it contains oncocyes, which are large granular acidophilic cells. This tumor occurs almost exclusively in the parotid
glands and is equally distributed in both men and women. The sixth decade is the most common time of presentation (Johns, M.E. et al., 1977; Spiro, R.H., Dubner, S., 1990; Spiro, R.H., 1995; Rice, D.H., 1999).

**Clinical Presentation:** Oncocytomas are usually solid round tumors that can be seen in any of the major salivary glands but that are extremely rare intraorally. These lesions can be found commonly in the superficial lobe of the parotid gland. Bilateral presentation of this tumor can occur, and it is the second most common salivary gland tumor that occurs bilaterally (after Warthin’s tumor).

**Pathology:** On gross examination, these tumors appear noncystic and firm. Histologically, they consist of brown granular eosinophilic cells. The oncocytes within this tumor concentrate technetium, and this tumor can be visualized by Tc-99m scintigraphy. Malignant oncocytomas can occur, and these are aggressive lesions.

**Differential diagnosis:**
1. Pleomorphic adenoma - it is biphasic and is characterized by mixture of polygonal epithelial and spindle-shaped myoepithelial elements in a variable background stroma;
2. Mucoepidermoid carcinoma - not encapsulated and is characterized by squamous cells, mucus-secreting cells, and intermediate cells;
3. Epithelial-myoepithelial carcinoma (EMC), acinic cell carcinoma (ACC), metastases of hypernephroid carcinoma - malignant tumors with infiltrative growth (Stratiev, A., 2000).

2. 5. Basal cell adenomas

Basal cell adenomas are slow growing and painless masses and account for approximately 1 to 2% of salivary gland adenomas. This lesion has a male predilection (the male to female ratio is 5:1). Seventy percent of basal cell adenomas occur in the parotid gland, and the upper lip is the most common site for basal cell adenomas of the minor salivary glands.

*Pathology:* Histologically, three varieties of basal cell adenomas exist: solid, trabecular-tubular, and membranous. The solid form consists of islands or sheets of basaloid cells. Nuclei have a normal size and are basophilic with minimal cytoplasmic material. The trabecular-tubular form consists of trabecular cords of epithelium. The membranous form is multilocular and 50% of the lesions are encapsulated. The membranous form tends to grow in clusters interspersed between normal salivary tissue.

![Tubular basal cell adenoma of submandibular salivary gland](image)

_Differential diagnosis:_

1. Pleomorphic adenoma – chondromyxoid substance;
2. Adenoid cystic carcinoma (ACC) - lowest level of the cribriform structure;

_Treatment:_ Lesions are removed with conservative surgical excision extending to normal tissue. In general, lesions do not recur; however, the membranous form has a higher recurrence rate (Spiro, R.H., Dubner, S., 1990).

2. 6. Canalicular adenoma

Canalicular adenomas predominantly occur in persons older than 50 years of age and occur mostly in women. Eighty percent of cases occur in the upper lip. The lesions are slow growing, movable, and asymptomatic.
**Pathology:** This lesion is composed of long strands of basaloid tissue, usually arranged in a double row. The supporting stroma is loose, fibrillar, and highly vascular.

**Differential diagnosis:**
1. Basal cell adenoma (trabecular type) – the stroma is more fibrous;
2. Adenoid cystic carcinoma (ACC) - malignant tumor with infiltrative and destructive growth (Stratiev, A., 2000).

**Treatment:** Treatment is surgical excision with a margin of normal tissue. Recurrence is rare, patients should be monitored periodically (Spiro, R.H., Dubner, S., 1990; Eisele, D.W., Johns, M.E., 1993; Regazi, J., Sciubba, J., 1993; Rice, D.H., 1999).

**2. 7. Myoepithelioma**

Most myoepitheliomas occur in the parotid gland; the palate is the most common intraoral site. No gender predeliction exists, and lesions tend to occur in adults, with the average age being 53 years. Lesions present as a well-circumscribed asymptomatic slow-growing mass.

**Pathology:** Myoepitheliomas consist of spindle-shaped cells, plasmacytoid cells, or a combination of the two. Diagnosis is based on the identification of myoepithelial cells. Growth patterns vary from a solid to a loose stroma formation with myoepithelial cells. This tumor is epithelial in origin; however, it functionally resembles smooth muscle and is demonstrated by immunohistochemical staining for actin, cytokeratin, and S-100 protein.

2. 8. Sebaceous adenoma

Sebaceous adenomas are rare. These lesions are derived from sebaceous glands located within salivary gland tissue. The parotid gland is the most commonly involved gland.

Pathology: Cells derived from sebaceous glands are present. Benign forms contain well-differentiated sebaceous cells whereas malignant forms consist of more poorly differentiated cells.


2. 9. Ductal papilloma

Ductal papillomas form a subset of benign salivary gland tumors that arise from the excretory ducts, predominantly of the minor salivary glands. The three forms of ductal papillomas are simple ductal papilloma (intraductal papilloma), inverted ductal papilloma, and sialadenoma papilliferum.

Simple Ductal Papilloma. The simple ductal papilloma presents as an exophytic lesion with a pedunculated base. The lesion often has a reddish color. Microscopic examination reveals epithelium-lined papillary fronds projecting into a cystic cavity without proliferating into the wall of the cyst. Local surgical excision is the recommended treatment. A minimal recurrence rate is reported (De Buck, R. et al., 1973; Fox, P.C. et al., 1986; Spiro, R.H., 1991; Marks, L., Turner, K., O’Sullivan, J., et al., 2001).

Inverted Ductal Papilloma. The inverted ductal papilloma occurs in the minor salivary glands. It presents clinically as a submucosal nodule that is similar to a fibroma or lipoma. The inverted ductal papilloma histologically resembles the sialadenoma. This form of ductal papilloma also consists of projections of ductal epithelium that proliferate into surrounding stromal tissue, forming clefts. The lesion is treated by surgical excision. A low recurrence rate is reported (Spiro, R.H., Dubner, S., 1990; Eisele, D.W., Johns, M.E., 1993; Regazi, J., Sciubba, J., 1993; Spiro, R.H., 1995; Stafford, N.D., Wilde, A., 1997; Calearo, C. et al., 1998; Rice, D.H., 1999).
Inverted ductal papilloma on the left buccal mucosa (Kato, H., et al., 2012)

Sialadenoma Papilliferum. The sialadenoma papilliferum form of ductal papilloma is analogous to the syringocystadenoma papilliferum of the skin. An adult male predilection exists, and most lesions occur between the fifth and eighth decades of life. This lesion occurs primarily on the palate and buccal mucosa and presents as a painless exophytic mass. Clinically, the lesion resembles a papilloma. Microscopic examination shows epithelium-lined papillary projections supported by fibrovascular connective tissue, forming a series of clefts within the lesion. Local surgical excision is the recommended treatment. Recurrence is rare (Spiro, R.H., Dubner, S., 1990; Eisele, D.W., Johns, M.E., 1993; Regazi, J., Sciubba, J., 1993; Spiro, R.H., 1995; Rice, D.H., 1999).

Sialadenoma papilliferum in the buccal mucosa (Miyamoto, S., et al., 2017)

3. Malignant tumors

3.1. Mucoepidermoid carcinoma

Mucoepidermoid carcinoma is the most common malignant tumor of the salivary glands. It is the most common malignant tumor of the parotid gland and the second most common malignant tumor of the submandibular gland, after adenoid cystic carcinoma.
Approximately 60 to 90% of these lesions occur in the parotid gland; the palate is the second most common site. Men and women are affected equally by this tumor, and the highest incidence occurs in the third to fifth decades of life. As its name suggests, mucoepidermoid carcinoma consists of both epidermal and mucous cells. The tumor is classified as of either a high grade or a low grade, depending on the ratio of epidermal cells to mucous cells (Grisius, M.M., Fox, P.S., 2003). The low-grade tumor has a higher ratio and is a less aggressive lesion. Although low-grade tumors have the ability for metastasis and local invasion, they behave more like benign tumors. The high-grade form is considered to be a more malignant tumor and has a poorer prognosis (Eisele, D.W., Johns, M.E., 1993; Regazi, J., Sciubba, J., 1993; Spiro, R.H., 1995.; Stafford, N.D., Wilde, A., 1997; Calearo, C. et al., 1998).

Clinical Presentation: The clinical course of this lesion depends on it's grade. It is not uncommon for low-grade tumors to undergo a long period of painless enlargement. In contrast, high-grade mucoepidermoid carcinomas often demonstrate rapid growth and a higher likelihood for metastasis. Pain and ulceration of overlying tissue are occasionally associated with this tumor. If the facial nerve is involved, the patient may exhibit a facial palsy.

Pathology: Macroscopically, low-grade mucoepidermoid carcinomas are usually small and partially encapsulated. The high-grade tumors are less likely to demonstrate a capsule because of the more rapid growth and local tissue invasion. After sectioning, the low-grade tumors usually demonstrate a mucinous fluid, but the high-grade lesions are usually solid in appearance. Microscopically, the low-grade lesions consist of regions of mucoid cells with interspersed epithelial strands. The high-grade tumors consist primarily of epithelial cells, with very few mucinous cells. In fact, special stains are necessary to differentiate between high-grade mucoepidermoid carcinoma and squamous cell carcinoma (Grisius, M.M., Fox, P.S., 2003).

Differential diagnosis:
- Benign tumors of the salivary glands - diagnosis performed by histopathological examination. Malignant tumors of the salivary glands characterized by faster
growing, infiltration of surrounding structures, facial nerve involvement.

- Between different types of malignant tumors - diagnosis performed by histopathological examination.
- Chronic sialadenitis – inflammation of the salivary glands, without facial nerve involvement, affecting lymph nodes with elastic consistency.
- Tuberculous sialadenitis - diagnosis performed by histopathological examination (Atanasov, D., 2011).

Treatment: A low-grade mucoepidermoid carcinoma can be treated with a superficial parotidectomy if it involves only the superficial lobe. Usually, the facial nerve can be spared. High-grade lesions should be treated aggressively to avoid recurrence. A total parotidectomy is performed, with facial nerve preservation if possible. If there is any possibility that the tumor involves the facial nerve, the nerve is resected with the tumor (Grisius, M.M., Fox, P.S., 2003). Immediate nerve reconstruction can be performed at the time of tumor extirpation. Neck dissections may be performed for lymph node removal and staging in high-grade lesions. Postoperative radiation therapy has been shown to be a useful adjunct in treating the high-grade tumor. With high-grade lesions, recurrence with metastases can occur in up to 60% of patients. The survival rate for patients with low-grade lesions is about 95% at 5 years; for patients with high-grade lesions, this rate drops to approximately 40% (Eisele, D.W., Johns, M.E., 1993; Regazi, J., Sciubba, J., 1993; Spiro, R.H., 1995.; Stafford, N.D., Wilde, A., 1997; Calearo, C. et al., 1998).

3. 2. Adenoid cystic carcinoma

Adenoid cystic carcinomas make up about 6% of all salivary gland tumors and are the most common malignant tumors of the submandibular and minor salivary glands. They make up 15 to 30% of submandibular gland tumors, 30% of minor salivary gland tumors, and 2 to 15% of parotid gland tumors. Approximately 50% of adenoid cystic carcinomas occur in the minor salivary glands. The tumor affects men and women equally and usually occurs in the fifth decade of life (Szanto, P. et al, 1984; Virkam, B. et al. 1984; Van der Waal, J.E., 1989; Eisele, D.W., Johns, M.E., 1993; Regazi, J., Sciubba, J., 1993; Spiro, R.H., 1995.; Stafford, N.D., Wilde, A., 1997).

Clinical Presentation: Adenoid cystic carcinoma usually presents as a firm unilobular mass in the gland. Occasionally, the tumor is painful, and parotid tumors may cause facial nerve paralysis in a small number of patients. This tumor has a propensity for perineural invasion; thus, tumor tissue often can extend far beyond the obvious tumor margin. The tumor’s slow growth may delay diagnosis for several years, allowing perineural invasion to be advanced at the time of surgical extirpation. An intraoral adenoid cystic carcinoma may exhibit mucosal ulceration, a feature that helps distinguish it from a benign mixed tumor. Radiographically, the tumor reveals extension into adjacent bone. Metastases into the lung are more common than regional lymph node metastasis.
Pathology: On gross examination, the tumor is unilobular and either partially encapsulated or non-encapsulated. There is often evidence of invasion into adjacent normal tissue. Microscopically, the individual cells are small and cuboidal. Nuclear atypia and mitotic figures are not seen, but chromatin aggregation is dense. Pseudocystic spaces filled with acellular material are a characteristic feature of this tumor, microscopic evidence of perineural or intraneural invasion is a distinguishing feature of adenoid cystic carcinoma.

Differential diagnosis:
- Pleomorphic adenoma – chondromyxoid substance, without infiltrative growing;
- Polymorphous low-grade adenocarcinoma (PLGA) - nuclear uniformity, most notably location in the palate;
- Epithelial-myoeptithelial carcinoma (EMC) – without cribriform pattern (Stratiev, A., 2000).

Treatment: Because of the ability of this lesion to spread along the nerve sheaths, radical surgical excision of the lesion is the appropriate treatment. Even with aggressive surgical margins, tumor cells can remain, leading to long-term recurrence. Frozen pathologic sections of the nerve sheath can help the surgeon achieve a clear margin. Some clinicians feel that a more conservative surgical resection and radiation therapy can provide adequate treatment. Neutron beam radiation has been shown to be more effective than photon beam therapy for the treatment of this tumor. Because of this tumor’s capability for long-term recurrence, patients need to be observed indefinitely. Factors affecting the long-term prognosis are the size of the primary lesion, it's anatomic location, the presence of metastases at the time of surgery, and facial nerve involvement (Szanto, P. et al, 1984; Virkam, B. et al. 1984; Van der Waal, J.E., 1989; Eisele, D.W., Johns, M.E., 1993; Regazi, J., Sciubba, J., 1993; Spiro, R.H., 1995.; Stafford, N.D., Wilde, A., 1997).

3.3. Acinic cell carcinoma

Acinic cell carcinoma represents about 1% of all salivary gland tumors. 90 to 95% of these tumors are found in the parotid gland; almost all of the remaining tumors are located in the submandibular gland. The distribution of acinic cell carcinoma reflects the location of
acinar cells within the different glands. This tumor occurs with a higher frequency in women and is usually found in the fifth decade of life. It is the second most common malignant salivary gland tumor in children, second only to mucoepidermoid carcinoma.

**Clinical Presentation:** These lesions often present as slow growing masses. Pain may be associated with the lesion but is not indicative of the prognosis. The superficial lobe and the inferior pole of the parotid gland are common sites of occurrence. Bilateral involvement of the parotid gland has been reported in approximately 3% of cases.

**Pathology:** The gross specimen is a well-defined mass that is often encapsulated. Microscopically, two types of cells are present; cells similar to acinar cells in the serous glands are seen adjacent to cells with a clear cytoplasm. These cells are positive on periodic acid–schiff (PAS) staining. Lymphocytic infiltration is often found.

**Treatment:** Acinic cell carcinomas initially undergo a relatively benign course, long-term survival is not as favorable, and the 20-year survival rate is about 50% (Grisius, M.M., Fox, P.S., 2003). Treatment consists of superficial parotidectomy, with facial nerve preservation if possible. When these tumors are found in the submandibular gland, total gland removal is the treatment of choice (Szanto, P. et al, 1984; Virkam, B. et al. 1984; Van der Waal, J.E., 1989; Mehle, M. et al, 1993).

3. 4. **Carcinoma ex pleomorphic adenoma**

Carcinoma ex pleomorphic adenoma is a malignant tumor that arises within a pre-existing pleomorphic adenoma (Ugrinov, R., 2006). The malignant cells in this tumor are epithelial in origin. This tumor represents 2 to 5% of all salivary gland tumors.

**Clinical Presentation:** These tumors are slow growing and have usually been present for 15 to 20 years before they suddenly increase in size and become clinically apparent. Carcinoma ex pleomorphic adenoma occurs more often in pleomorphic adenomas that have been left untreated for long periods of time (it is for this reason that early removal of pleomorphic adenomas is recommended) (Grisius, M.M., Fox, P.S., 2003).
Pathology: Macroscopically, these tumors are nodular or cystic, without encapsulation. The sectioned tumor appears similar to pleomorphic adenoma except for the presence of necrosis and hemorrhage associated with the malignant tumor. Histologically, the tumor appears as a squamous cell carcinoma, adenocarcinoma, or undifferentiated carcinoma located within a benign mixed tumor. It may appear as a small focus of malignancy within the pleomorphic adenoma, or the malignant cells can almost completely replace the mixed tumor, making its appearance similar to that of a primary malignant tumor. Destructive infiltrative growth is usually seen around the malignancy.

Treatment: This is a malignant salivary gland tumor that has an aggressive course and that carries a very poor prognosis. Local and distant metastases are common. Surgical removal with postoperative radiation therapy is the recommended treatment. Early removal of benign parotid gland tumors is recommended to avoid the development of this lesion (Eisele, D.W., Johns, M.E., 1993; Mehle, M. et al, 1993; Regazi, J., Sciubba, J., 1993; Spiro, R.H., 1995.; Stafford, N.D., Wilde, A., 1997).

3.5. Adenocarcinoma

Any tumors arising from salivary duct epithelium are, by definition, adenocarcinomas. This group of neoplasms has been divided into discrete entities based on structure and behavior. The term “adenocarcinoma” is often used as a catchphrase to refer to lesions that do not meet the specific criteria for other lesions (such as polymorphous low-grade adenocarcinoma, epimyoepithelial carcinoma, or salivary duct carcinoma). Clarification of the type of adenocarcinoma with a histologic description should be obtained in order to determine an appropriate treatment approach (Eisele, D.W., Johns, M.E., 1993; Mehle, M. et al, 1993; Regazi, J., Sciubba, J., 1993; Stafford, N.D., Wilde, A., 1997).

The parotid gland, hard palate, buccal mucosa and lips are common sites of occurrence (Stratiev, A., 2000).
Adenocarcinoma of the left buccal mucosa (Ettl, T. et al., 2008).

Clinical Presentation: Can present as thick nodule with surrounding structures involvement. Cancer cells have invaded into blood or lymphatic vessels. These tumors invade adjacent nerves. The most common distant metastatic site is bone and lungs (Stratieve, A., 2000).

Treatment: Extended total parotidectomy, immediate plastic of the facial nerve with anterior branch of N. auricularis magnus. Indications for cervical lymph node dissection - enlarged lymph nodes determined by palpation. 3 to 5 weeks after extended total parotidectomy should be done postoperative radiation therapy (Ugrinov, R., 2006).

3. 6. Lymphoma

By definition, “primary lymphoma” describes a situation in which a salivary gland is the first clinical manifestation of the disease. Primary lymphoma of the salivary glands probably arises from lymph tissue within the glands. However, primary lymphoma of the salivary glands is rare (Grisius, M.M., Fox, P.S., 2003). The major forms of lymphoma are non-Hodgkin’s lymphoma (NHL) and Hodgkin’s disease. NHL is less curable and is often disseminated at diagnosis. There is an increased incidence of NHL in patients with autoimmune disease. The parotid gland is the most commonly involved gland, followed by the submandibular gland.

Clinical Presentation: This lesion commonly presents as painless gland enlargement or adenopathy.

Treatment: Superficial parotidectomy is recommended for isolated asymptomatic parotid gland masses. A staging workup is required to determine treatment. An initial phase of observation is not uncommon for patients with asymptomatic low-grade disease. Appropriate treatment includes radiation therapy, chemotherapy, or a combination of the two, depending on the staging of the lymphoma (Mehle, M. et al., 1993., Moody, A.B., 1999).
4. TNM classification for cancer of major salivary glands (parotid, submandibular, and sublingual)

The TNM classification for cancer of the major salivary glands (parotid, submandibular, and sublingual) is provided below, along with anatomic staging.

4. 1. Primary tumor (T)
- **TX** – Primary tumor cannot be assessed
- **T0** – No evidence of primary tumor
- **Tis** – Carcinoma in situ
- **T1** – Tumor ≤2 cm in greatest dimension without extraparenchymal extension (clinical or macroscopic evidence of invasion of the soft tissues, not microscopic evidence)
- **T2** – Tumor >2 cm but not more than 4 cm in greatest dimension without extraparenchymal extension
- **T3** – Tumor >4 cm and/or tumor has extraparenchymal extension
- **T4a** – Moderately advanced disease; Tumor invades the skin, mandible, ear canal, and/or facial nerve
- **T4b** – Very advanced disease; Tumor invades skull base and/or pterygoid plates and/or encases carotid artery.

4. 2. Regional lymph nodes (N)
- **NX** – Regional nodes cannot be assessed
- **N0** – No regional lymph node metastasis
- **N1** – Metastasis in a single ipsilateral lymph node, ≤3 cm in greatest dimension
- **N2** – Metastasis in a single ipsilateral lymph node >3 cm but not more than 6 cm in greatest dimension; or in multiple ipsilateral lymph nodes, none >6 cm in greatest dimension; or in bilateral or contralateral lymph nodes, none > 6 cm in greatest dimension
- **N2a** – Metastasis in a single ipsilateral lymph node >3 cm but not more than 6 cm in greatest dimension
- **N2b** – Metastasis in multiple ipsilateral lymph nodes, none >6 cm in greatest dimension
- **N2c** – Metastasis in bilateral or contralateral lymph nodes, none >6 cm in greatest dimension
- **N3** – Metastasis in a lymph node >6 cm in greatest dimension

4. 3. Distant metastasis (M)
- **M0** – No distant metastasis
- **M1** – Distant metastasis
## Anatomic stage/prognostic groups

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### 5. Surgical treatment of tumors of the parotid gland

Surgery is the most common treatment for salivary gland cancer. The parotid glands are the largest salivary glands in humans and are frequently involved in disease processes. There are distinct variations of the parotidectomy:

- **Partial parotidectomy** – resection of parotid pathology with a margin of normal parotid tissue. Indications – benign pathology and low-grade malignancies.

- **Superficial parotidectomy** – resection of the superficial lobe of parotid gland. Indications – benign or low-grade tumor of the superficial lobe of parotid gland, metastases removal to parotid lymph nodes from skin cancer, melanoma (Sarachev, E., 1999) or from cancer of the external auditory meatus.

- **Total parotidectomy** – resection of the entire parotid gland, usually with preservation of the facial nerve.

- **Radical parotidectomy:** This procedure is a total parotidectomy, along with resection of the facial nerve.

- **Extended total parotidectomy** – removal of the superficial and deep part of the parotid gland with extension to involve adjacent structures, such as the temporal bone, skin of face overlying the parotid gland, muscles attached to the styloid process, posterior belly of the digastric muscle, muscles of mastication and part of the mandibular ramus (bloc resection) (Grisius, MM., Fox PS., 2003; Ugrinov, R., 2006).

Complications after parotidectomy:

1. Bleeding, including hematoma;
2. Seroma - collection of normal body fluid in the neck after removal of the drain;
3. Infection - presence of microbes that cause disease;
4. Sialocele - collection of saliva under the skin;
5. Sensory disturbance;
6. Frey’s syndrome - sweating on face during eating process or even think about eating;
7. Facial nerve injury – can be partial (if only some of the branches of the facial nerve are injured) or total (if the main trunk of the facial nerve or all branches of the facial nerve are injured). It can be temporary (if the nerve is just stretched) or permanent (if the nerve is cut).

6. Submandibular, sublingual, and minor salivary gland surgery

Tumors of the submandibular and sublingual glands are treated by total removal of the gland. The loss of salivary flow from a single submandibular gland is negligible, and patients tolerate this procedure well. The risks associated with the removal of the submandibular gland include hemorrhage, infection, and injury to the hypoglossal, lingual, or marginal mandibular nerves.

The treatment of tumors of the minor salivary glands depends on the location and extent of disease. Complete excision is usually sufficient for benign tumors. The treatment of malignant tumors may involve maxillectomy or composite resection (Grisius, MM., Fox PS., 2003).
LYMPHORETICULAR MALIGNANCIES
(Petia Pechalova)

The lymphoreticular system consists of organs (lymph nodes, spleen, thymus) and ill-defined tissues (mucosa-associated lymphoid tissue) that are concerned with the growth, development and deployment of white blood cells. The lymphoreticular system include the thymus, lymph nodes, spleen, tonsils, bone marrow, Peyer's patches (the stomach and intestines also have lymphoid tissue), avian bursa of Fabricius.

White blood cells are crucial for immune responses. The lymphoreticular system comprised of lymphocytes, monocytes, macrophages, and the stromal elements that support them. Lymphocytes are generated in the central lymphoid organs (thymus, bone marrow) and migrate to the peripheral or secondary lymphoid organs (spleen, lymph nodes, tonsils, Peyer's patches) where they are maintained and where adaptive immune responses are initiated.

The two major types of lymphocytes are B lymphocytes (B cells) and T lymphocytes (T cells). Normal B cells and T cells have different jobs. B cells help protect the body from germs (bacteria and viruses) by making proteins called antibodies. The antibodies attach to the germs, marking them for destruction by other parts of the immune system. Almost all cases of Hodgkin disease start in B lymphocytes. There are several types of T cells, and each has a special job. Some T cells directly destroy certain kinds of bacteria or cells infected with viruses or fungi. Other types of T cells play a role in either boosting or slowing the activity of other immune system cells.

The lymph nodes lie along the course of the lymphatics, receiving lymph from the tissues and destroying or mounting immune responses to foreign agents before they reach the bloodstream. Both benign and malignant disorders of the lymph nodes often manifest as lymph node enlargement (lymphadenopathy), and lymph node biopsy is necessary to determine the diagnosis and indicate further management. Of total 800 lymph nodes in the human body, 300 are in the neck.

The spleen receives blood from the arterial system. It functions as a filter by removing obsolescent red blood cells and particulate matter from the blood, and mounts immunological responses against foreign agents. Most benign and malignant disorders of the spleen manifest as splenic enlargement (splenomegaly). Rupture of the spleen is a potentially life-threatening condition that requires prompt management.

The thymus is an important component of the lymphoreticular system in fetal life, but probably has no significant role in adults. Failure of normal development of the thymus causes deficient immune responses and, in adults, thymic hyperplasia and thymic tumours may develop.

The bone marrow is the spongy tissue inside certain bones, which is where new white blood cells (including some lymphocytes), red blood cells, and platelets are made.

Lymphoma is the name for a group of malignant disorders that develop in the lymphatic system. The two main types are Hodgkin lymphoma and non-Hodgkin lymphoma (NHL).
**Hodgkin Lymphoma** is a lymphoreticular cancer. It is one of the most curable forms of cancer and is named for Dr. Thomas Hodgkin. The disease was called Hodgkin's disease until it was officially renamed Hodgkin lymphoma in the late 20th century. Hodgkin's lymphoma was first described in an 1832 report by Thomas Hodgkin, although Hodgkin noted that perhaps the earliest reference to the condition was provided by Marcello Malpighi in 1666. While occupied as museum curator at Guy's Hospital, London, Hodgkin studied seven patients with painless lymph node enlargement. Hodgkin's report on these seven patients was presented to the Medical and Chirurgical Society in London. Theodor Langhans and WS Greenfield first described the microscopic characteristics of Hodgkin's lymphoma several years later. In 1898 and 1902, respectively, Carl Sternberg and Dorothy Reed independently described the cytogenetic features of the malignant cells of Hodgkin's lymphoma, now called Reed–Sternberg cells. Tissue specimens from Hodgkin's seven patients remained at Guy's Hospital for a number of years. Nearly 100 years after Hodgkin's initial publication, histopathologic reexamination confirmed Hodgkin's lymphoma in only three of seven of these patients. The remaining cases included non-Hodgkin lymphoma, tuberculosis, and syphilis.

Hodgkin disease can start almost anywhere in the body. Most often it starts in lymph nodes in the upper part of the body. The most common sites are in the chest, in the neck, or under the arms. Hodgkin disease most often spreads through the lymph vessels in a stepwise fashion from lymph node to lymph node.

Rarely, and late in the disease, it can invade the bloodstream and spread to other parts of the body, including the liver, lungs, and/or bone marrow. All types of Hodgkin disease are malignant - as they grow they can invade and destroy normal tissue and spread to other tissues. Different types of Hodgkin disease are classified histopathologically. This is important because types of Hodgkin disease may grow and spread differently and may be treated differently. The two main types are:

I. **Classic Hodgkin disease (HD)** accounts for about 95% of all cases of Hodgkin disease. The cancer cells in classic HD are Reed-Sternberg cells. These cells are usually an abnormal type of B lymphocyte. Reed-Sternberg cells are much larger than normal lymphocytes and also look different from the cells of non-Hodgkin lymphomas and other cancers. The enlarged lymph nodes in classic HD usually have a small number of Reed-Sternberg cells and a large number of surrounding normal immune cells. It is mainly these other immune cells that account for the enlarged lymph nodes. Subtypes of classic Hodgkin disease are:

- Nodular sclerosis Hodgkin disease
- Mixed cellularity Hodgkin disease
- Lymphocyte-rich Hodgkin disease
- Lymphocyte-depleted Hodgkin disease

II. **Nodular lymphocyte predominant Hodgkin disease (NLPHD)** accounts for about 5% of Hodgkin disease. The cancer cells in NLPHD are large cells called popcorn cells
(because they look like popcorn), which are variants of Reed-Sternberg cells. NLPHD usually starts in lymph nodes in the neck and under the arm. It can occur in people of any age, and is more common in men than in women.

Risk factors for Hodgkin lymphoma are:

1) Age: between 15 and 30 and after 55.
2) A family history of lymphoma: having a close family member who has Hodgkin's lymphoma or non-Hodgkin's lymphoma increases the risk of Hodgkin's lymphoma.
3) Sex: males are slightly more likely to develop Hodgkin's lymphoma.
4) Past Epstein-Barr infection: People who have had illnesses caused by the Epstein-Barr virus, such as infectious mononucleosis, are more likely to develop Hodgkin's lymphoma than are people who haven't had Epstein-Barr infections.
5) A weakened immune system: a compromised immune system, such as from HIV/AIDS or from having an organ transplant requiring medications to suppress the immune response, increases the risk of Hodgkin's lymphoma.

At this time, there are no widely recommended screening tests for Hodgkin lymphoma. The best way to find Hodgkin disease early is to pay attention to possible symptoms. The most common symptom is enlargement of one or more lymph nodes, causing a lump or bump under the skin which is usually not painful. This is most often on the side of the neck, in the armpit, or in the groin. Other symptoms (minor symptoms, B-symptoms), which present bad prognostic factors, can include (1) fever, (2) drenching night sweats that often require changing bed sheets or night clothes, (3) unexplained weight loss (more than 10% for 6 months) with loss of appetite; (4) severe and constant itching; (5) persistent fatigue. One of early signs of HD is increased sensitivity to the effects of alcohol or pain in lymph nodes after drinking alcohol.

Classifying non-Hodgkin lymphoma (NHL) can be quite confusing because there are so many types and because several different systems have been used. The most recent system is the World Health Organization (WHO) classification. The WHO system groups lymphomas based on how they look under a microscope, the chromosome features of the lymphoma cells, and the presence of certain proteins on the surface of the cells.

The more common types of non-Hodgkin lymphoma according to whether they are B-cell (85%) or T-cell lymphomas (less than 15%) are:

I. B-cell lymphomas:
   1. Diffuse large B-cell lymphoma: a) Primary mediastinal B-cell lymphoma b) Intravascular large B-cell lymphoma
   2. Follicular lymphoma
   3. Chronic lymphocytic leukemia /small lymphocytic lymphom
   4. Mantle cell lymphoma
   5. Marginal zone lymphomas
   6. Burkitt lymphoma
   7. Lymphoplasmacytic lymphoma (Waldenstrom macroglobulinemia)
8. Hairy cell leukemia
9. Primary central nervous system (CNS) lymphoma

II. T-cell lymphomas:
1. Precursor T-lymphoblastic lymphoma/leukemia
2. Peripheral T-cell lymphomas:
   a) Cutaneous T-cell lymphomas (mycosis fungoides, Sezary syndrome, and others)
   b) Adult T-cell leukemia/lymphoma:
      ✓ smoldering subtype
      ✓ chronic subtype
      ✓ acute subtype
      ✓ lymphoma subtype
   c) Angioimmunoblastic T-cell lymphoma
   d) Extranodal natural killer/T-cell lymphoma, nasal type
   e) Enteropathy-associated intestinal T-cell lymphoma (EATL)
   f) Anaplastic large cell lymphoma (ALCL)
   g) Peripheral T-cell lymphoma, unspecified

**Burkitt lymphoma** makes up about 1% to 2% of all lymphomas. It is named after the doctor who first described this disease in African children and young adults. The cells are medium-sized. Another kind of lymphoma, Burkitt-like lymphoma, has slightly larger cells.

Because this second kind of lymphoma is hard to tell apart from Burkitt lymphoma, the WHO classification combines them. This is a very fast-growing lymphoma. In the African (or endemic) variety, it often starts as a tumor of the jaw or other facial bones. It is linked to infection with the Epstein-Barr virus (which can also cause infectious mononucleosis). The endemic type of Burkitt lymphoma is rare in the Europe and United States.

In the types seen more often in developed countries, Burkitt lymphoma usually starts in the abdomen, where it forms a large tumor mass. It can also start in the ovaries, testicles, or other organs, and can spread to the brain and spinal fluid. The type seen in the United States is usually not linked to Epstein-Barr viral infection. Close to 90% of patients are male, and the average age is about 30. Although this is a fast-growing lymphoma, more than half of patients can be cured by intensive chemotherapy.

For making a diagnosis of lymphoma, in addition to the physical examination, several tests could be used: blood tests, imaging tests, lymph node biopsy (extirpation of whole lymph node), bone marrow biopsy (during this procedure, a small amount of bone marrow, blood and bone are removed through a needle).

The Ann Arbor classification is named on Ann Arbor, Michigan, USA where the Committee on Hodgkin's Disease Staging Classification met in 1971. Stages of lymphomas (**Ann Arbor staging**) are:

**Stage I.** The lymphoma is limited to one lymph node region or a single organ.
**Stage II.** The lymphoma is in two lymph node regions or has invaded one organ and the nearby lymph nodes. But the cancer is still limited to a section of the body either above or below the diaphragm.

**Stage III.** The lymphoma involves the lymph nodes both above and below the diaphragm. Lymphoma may also be in one portion of tissue or an organ near the lymph node groups or in the spleen.

**Stage IV.** This is the most advanced stage of Hodgkin's lymphoma. Cancer cells are in several portions of one or more organs and tissues. Stage IV Hodgkin's lymphoma affects not only the lymph nodes but also other parts of the body, such as the liver, lungs or bones.

Additionally, the letters A and B indicates whether the patient is experiencing symptoms of Hodgkin's lymphoma: “A” means that he doesn't have any significant symptoms as a result of the cancer; ”B” indicates that have significant signs and symptoms, such as a persistent fever, unintended weight loss or severe night sweats.

Hematologists and oncologists are specialists who treat people who have Hodgkin lymphoma or other types of blood cancer. Hodgkin's lymphoma was one of the first cancers which could be treated using radiation therapy and, later, it was one of the first to be treated by combination chemotherapy. Which treatment options are appropriate for certain Hodgkin's lymphoma depends on the type and stage of the disease and the overall health. The goal of treatment is to destroy as many cancer cells as possible and bring the disease into remission. For classical Hodgkin's lymphoma, radiation therapy can be used alone, but it is often used after chemotherapy. Patients with early-stage lymphocyte-predominant Hodgkin's lymphoma typically undergo radiation therapy alone.

Treatment strategy of lymphomas could be present diagramatically as follow:
A stem cell transplant is a treatment to replace the diseased bone marrow with healthy stem cells that help to grow new bone marrow. A stem cell transplant may be an option if Hodgkin's lymphoma returns despite treatment. During a stem cell transplant, patient's own blood stem cells are removed, frozen and stored for later use. Next he receives high-dose chemotherapy and radiation therapy to destroy cancerous cells in his body. Finally the stem cells are thawed and injected into his body through the veins. The stem cells help build healthy bone marrow.

The **International Prognostic Index** (IPI) was first developed to help doctors determine the outlook for people with fast-growing lymphomas. However, it has proven useful for most other lymphomas as well.

The index depends on 5 factors: (1) the patient’s age; (2) the stage of the lymphoma; (3) whether or not the lymphoma is in organs outside the lymph system; (4) performance status (PS) – how well a person can complete normal daily activities; (5) the blood (serum) level of lactate dehydrogenase (LDH), which goes up with the amount of lymphoma in the body.

Each **poor** prognostic factor is assigned 1 point. People with no poor prognostic factors would have a score of 0, while those with all of the poor prognostic factors would have a score of 5. The index divides people with lymphomas into 4 risk groups:

- **Low** (0 or 1 poor prognostic factors)
  - **Low intermediate** (2 poor prognostic factors)
  - **High intermediate** (3 poor prognostic factors)
  - **High** (4 or 5 poor prognostic factors)

About 75% of people in the lowest risk group lived at least 5 years, whereas only about 30% of people in the highest risk group lived at least 5 years.
The incidence of oral squamous cell carcinoma, particularly in younger patients, is increasing in developed countries. Oral cancer is the eighth most common malignancy worldwide, although the prevalence varies from country to country. In countries such as India, oral malignancy accounts for 40% of the total, whereas in the UK around 4000 new cases present every year, accounting for around 3% of all new malignancies. Around 50% of these patients will die from their disease and overall survival rates have not improved over the last three decades, despite advances in surgical and oncological techniques. The roles of tobacco and alcohol are firmly established as risk factors for oral cancer. However, authors demonstrated that approximately 33% of oral squamous cell carcinoma patients were never smokers. An easy, but wrong, explanation for the increase in oral cancer rates with younger patients has been human papilloma virus (HPV). Based on the available studies there is a lack of reproducible molecular data supporting the role of HPV in the development of oral cancer. A multicenter case-control study of 1415 cases of oral cavity cancer detected HPV DNA in only 3.9% of biopsy specimens. This is in contrast to oropharyngeal carcinogenesis where HPV has a much stronger etiologic role.

To make a preoperative assessment, including staging and creation of treatment plan, several clinical and paraclinical tests are usable. The appointment starts with comprehensive history and physical examination, followed by histopathology. Systematic palpation of the neck should be performed on every patient at every visit, although the sensitivity of clinical examination to detect metastasis, even by expert surgeons, has been inadequate, with a low sensitivity rates (up to 50%). A panoramic radiograph should be performed in almost all cases of oral cavity carcinomas. Even if the carcinoma does not involve the maxilla or the mandible the dentition should be evaluated prior to surgery to determine whether extractions will be required at the time of surgical resection if postoperative radiation therapy is anticipated. Nasopharyngoscopy can be easily and painlessly performed with proper preparation of the nasopharyngeal membranes with local anesthetic and a topical vasoconstrictor. Nasopharyngoscopy can be used to evaluate the nasopharynx, base of tongue, and supraglottic structures. Sophisticated techniques such as computed tomography (CT) scans or magnetic resonance imaging (MRI) are used for assessment of both the primary and metastatic disease. Many authors favor a CT scan from the diaphragm to the base of the skull to identify evidence of second primaries in the aerodigestive tract, the state of the lungs, and the extent of the primary disease and metastasis in the neck. An MRI scan is slightly more sensitive and specific than a CT scan for soft tissue imaging, with sensitivity up to 83% and specificity of around 85%. Positron emission tomography (PET) scanning is emerging as a useful method for detecting tumours, and also recurrence in previously treated areas, with sensitivities and specificities reported as slightly more than 90%. It is poor at anatomically defining tumours.
but images can be combined and superimposed on CT or MRI scans to help determine resection margins. Unfortunately, despite improved resolution and software analysis, all imaging techniques (computed tomography, magnetic resonance imaging, positron emission tomography, ultrasound) are still insufficiently sensitive for detecting occult neck metastases.

Treatment methods of malignant oral and maxillofacial tumors include: (1) surgery, (2) radiotherapy, (3) chemotherapy, (4) immunotherapy, (5) gene therapy, (6) maintenance treatment. Treatment comprises surgery, radiotherapy or combined treatment (particularly for larger or more aggressive tumours). These treatments have been the mainstay of oral cancer treatment for the last 50 years.

**Principles of cancer surgery:** The aim of any cancer surgery are to remove the cancer with an adequate margin of normal tissue with minimal morbidity. Clear margins have an impact on local control. Margin requirements differ according to the origin of the tumour and the functional impact must be considered. Many solid tumours require removal of the draining lymph nodes for the purpose of staging or to achieve local control. Levels of prophylactic lymph nodes dissection vary according to tumour type and may increase surgical morbidity. Surgery in some tumours has become more conservative with the advent of sentinel node biopsy when lymph node metastases are not evident pre-operatively. Sentinel node biopsy is frequently used in melanoma. The aim of the sentinel node biopsy is to provide an assessment as a staging tool to predict prognosis and influence use of adjuvant therapies. Several types of oncologic surgery exist:

1) Prophylactic surgery (removal of precancer lesions)
2) Diagnostic surgery (biopsy)
3) Curative surgery (the aim is to eradicate the cancer)
4) Cytoreductive surgery (the aim is to decrease the size of the cancer, but there is a risk of metastasizing)
5) Palliative surgery (the aim is to relieve symptoms or improve quality of life in incurable cancer patients)
6) Supportive surgery
7) Reconstructive surgery

**Surgical treatment of tongue carcinoma:** the tumor infiltrates the parallel muscle fibers and there is no fascia to resist invasion. The rich lymphatic supply leads to early cervical metastasis. The lymphatic drainage of the lateral tongue, the most common portion of the tongue affected, is into the submandibular and jugulodigastric nodes. The base of tongue is embryologically, anatomically, and biologically distinct from the oral tongue. Surgery, with consideration of postoperative chemoradiotherapy, is the primary treatment for oral tongue carcinoma. The tongue, affected of carcinoma, should be resected simultaneously with neck dissection. If the primary tumor and neck lymph nodes are resected separately, the lymphatics, within the floor of mouth along the mandible, are at risk for metastasis following surgery. Surgical resections of tongue carcinomas are (Kirita, 2015):
(1) Partial glossectomy refers to the resection of a part or less than half of the oral tongue. Impairment of oral function after partial glossectomy is mild.

(2) Oral tongue hemiglossectomy refers to a resection to the lingual septum by a hemiglossectomy of only the oral tongue.

(3) Oral tongue subtotal-total glossectomy refers to a resection of over half (subtotal) or all of the oral tongue.

(4) Hemiglossectomy refers to a hemiglossectomy that includes the base of the tongue.

(5) Subtotal-total glossectomy refers to a resection of over half (subtotal) or all of the tongue including the base of the tongue.

“Pull-through operation” for tongue carcinoma is a surgical technique in which the primary lesion is resected en bloc with dissected cervical tissue and pulled out from the submandibular region through the floor of the mouth. However, when neck dissection is conducted concurrently because cervical metastasis is suspected upon imaging, the appropriateness of the pull-through operation becomes an issue. The majority opinion mostly supports conducting the pull-through operation, but there are also those who deem it unnecessary.

Surgical treatment of mandible carcinoma:

(1) Marginal mandibulectomy: a part of mandible is resected, but continuity of the mandibular arch is maintained. The bone defect does not necessitate any specific reconstruction except for soft tissue coverage, usually with a soft tissue flap or skin graft.
(2) **Segmental mandibulectomy:** involves resection of a full thickness of bone, less than half of the mandible without condyle, which creates a discontinuity.

(3) **Hemi-mandibulectomy:** resection of half of the mandible with one side condyle – from the midline of the symphisis to the condyle.

(4) **Subtotal mandibulectomy:** resection of more than half of the mandible with or without condyle.

(5) **Total mandibulectomy:** resection of the mandible.

**Surgical resection of maxillary carcinoma:**

(1) **Partial resection:** removal of alveolar process or part of the hard palate.

(2) **Subtotal maxillectomy:** removal of the maxilla from one side without infraorbital edge.

(3) **Total maxillectomy:** removal of the maxilla from one side with infraorbital edge with or without eye removal
Surgical treatment of lip cancer: During surgical removal of lip carcinoma, resection margins should be 1 cm. The primary function of the lower lip function is control of saliva and prevention of sialorrhea. The lower lip also functions for articulation. If possible symmetric coverage of the teeth should be maintained. Restoration of cosmesis and function is best achieved with the use of local flaps. The reconstructive plan must include the skin, lip, and buccal mucosa. Many methods are available to reconstruct the lips following resection.

Surgical treatment of regional metastasis (Neck dissection): neck dissection is a systematic approach to removing entire groups of lymph nodes from the neck. A neck dissection can be done as an elective neck dissection, which is the removal of the lymph nodes without any evidence that there is obvious cancer in the neck, or as a therapeutic neck dissection, which is the removal of lymph nodes in the neck with known cancerous lymph nodes in the region based on a biopsy or a high level of suspicion based on their appearance on imaging studies. An elective neck dissection will be considered if there is a high risk that there is microscopic (hidden or not clinically apparent) cancer in the lymph nodes (more than 20%) - occult metastasis. The rates of occult metastasis for oral cancer are very high and range from 20–45% for T1 tongue cancer for example. The extent on the neck dissection will depend on a number of factors. Perhaps most important is the site of the primary cancer. Interestingly, there is a pattern to which level certain cancers spread when they enter the lymphatic system. For example, cancers of the oral cavity are known to spread to Levels I, II and III; therefore, an elective neck dissection for a cancer of the oral cavity should include these lymph node groups on the side of the primary cancer. The general patterns include:

- Level I, II, III: oral cavity
- Level II, III, IV: oropharynx, hypopharynx, larynx
- Level V: scalp, facial skin
- Level VI: thyroid, larynx
- Level VII: thyroid

Radical neck dissection (Crile method) refers to the removal of lymph node groups I to V, as well as the sternocleidomastoid muscle, internal jugular vein and spinal accessory nerve. This used to be the standard neck dissection years ago but has been replaced with neck dissections that spare some or all of these structures.
An extended radical neck dissection includes all of these, plus removal of additional lymph node groups or non-lymphatic structures not accounted for in the radical neck dissection definition. This method must to perform unilaterally (in one side of the neck) only, because there is a risk of increasing of intracerebral pressure as a result of the internal jugular vein ligature.

Modified radical neck dissection is the removal of lymph node groups I to V, while sparing one or more of the three structures taken in the radical neck dissection (sternocleidomastoid muscle, internal jugular vein and spinal accessory nerve). In old nomenclature, depending on what structure was removed, surgeons would call them Type I, Type II or Type III modified radical neck dissections. These days, they should be described as a modified radical neck dissection with sacrifice of the internal jugular vein and sternocleidomastoid muscle (this implies that the spinal accessory nerve was preserved). A modified radical neck dissection that preserves all three structures is also called a comprehensive neck dissection, indicative of the removal of lymph nodes from Levels 1 through 5.

Selective neck dissection is the removal of a select group of lymph nodes in the neck, with or without sacrifice of additional non-lymphatic structures. Most neck dissections in current times are really selective neck dissections. Some common selective neck dissections are given names such as the following:

- Supraomohyoid neck dissection: This is the removal of lymph node Groups I, II and III.
- Lateral neck dissection: This is the removal of lymph node Groups II, III and IV.
- Posterolateral neck dissection: This is the removal of lymph node Groups II, III, IV and V.

Radiotherapy is given in divided doses, usually over a period of 6 weeks. One local regime is to give 66 Gray (Gy) in 33 fractions over a period of 6 and 1/2 weeks. Other forms of hyperfractionation can be given to reduce the length of treatment and increase the concentration of the radiotherapy effects. Brachytherapy (a subtype of radiotherapy), or implanted radiotherapy elements, to the local site may be used to deliver a high dose of radiotherapy to the index primary tumour while sparing the surrounding tissues (e.g. the salivary glands). Plastic tubes are looped through the tumour from the neck and, after volumetric planning has been carried out, can be loaded with radioactive wires. If the wires are too far apart then insufficient radiotherapy levels will be delivered to the tumour. Conversely, if the wires are too close then tissue necrosis will ensue. Brachytherapy is an important option for treating the tongue, particularly the posterior tongue, because it allows function to be preserved. The most common side-effect from brachytherapy treatment is pain at the tumour site. The role of radiation therapy in the management of oral cancer is adjuvant. Oral cavity carcinomas are poorly responsive to radiotherapy as primary treatment. Preoperative radiation therapy is not given for oral cavity carcinoma. In certain cases
preoperative radiation therapy, in the dose range of 50 Gy, is used for sinonasal or paranasal carcinomas to improve resectability. Preoperative radiation therapy is associated with wound healing complications. The surgical margins would not change following radiation therapy for oral cavity carcinoma. Possible indications for postoperative radiation therapy include positive or close margins, perineural invasion, extensive bone invasion, and neck involvement (strong indication for more than one lymph node involved or extracapsular spread). While these recommendations are commonly used, each case must be evaluated independently and there is not universal agreement on these indications. For patients with stage III and IV oral SCC, postoperative radiation therapy improves control of the carcinoma, particularly in the setting of positive margins. Timing of radiation therapy is critical and should be started as soon as possible following healing. Multiple studies have demonstrated that recurrence rates increase when there is a delay in starting radiation therapy. A delay of greater than 6 weeks after surgery leads to increased recurrence rates. Locoregional control of oral cavity cancer improves if the duration of all treatment is less than 100 days.

The side-effects of radiation therapy can be divided into:

- acute, but mostly reversible effects;
- late, more commonly irreversible conditions.

The acute side-effects almost always decline within 4–6 weeks after completion of radiation, whereas the late effects can develop after different periods of time and they will also almost always last for the rest of the patient’s life. Among the acute effects, an inflammatory reaction in the mucosa (mucositis) of the oral and pharyngeal mucosa is the most common. The appearance of the mucosa can vary from an increased redness, to an intense reddish appearance, to white necrotic lesions. The damage to the oral mucosa can also make it more susceptible to both bacterial and fungal infections. These conditions are almost always associated with varying degrees of pain, and may need relatively strong analgesics. The skin reactions can vary between a tan-like appearance to almost burn-like wounds. Softening and lubricating lotions are recommended for the affected skin. Males should be recommended to use an electrical shaver in order to spare the skin as well as not to use aftershave, since the alcohol may add to dryness of the skin. An increased production of sticky mucus can also be observed after radiation of cancers in the pharyngeal region. Both still and sparkling water seem to be effective in dissolving the mucus. The use of milk products should be minimized. Nutritional problems are frequently seen, and are almost always a result of local reactions in the mucosa as well as of the pain. A loss of appetite may also be a result of loss of, or change of, taste. A majority of the patients undergoing radiotherapy to the head and neck region need professional assistance with nutrition. Some people may need some type of gastric tube to be able to feed themselves. Tiredness and nausea are other side-effects which are frequently observed during radiotherapy.

Among the irreversible side effects observed after radiotherapy, xerostomia is the most common. The degree differs between different patients. A majority of all saliva is secreted by the large salivary glands, and frequently these glands are within the field of irradiation. A
secondary effect of increased oral dryness may be an altered taste and increased sensitivity to different tastes. Some patients need to avoid some type of spices, for instance pepper. The mucosa can also be more sensitive to different types of food, i.e. its consistency. Xerostomia carries an increased risk for development of dental caries and other dental problems. Radiation also decreases the elasticity of the tissues, which can result in both dysphagia and trismus. The negative effect of radiation on vessels is also well recognised; decreased vascularity can lead to necrosis of both the soft tissues and the bone within the irradiated area.

**Management of oral health during radiation:** Prior to the start of radiation therapy for treatment of head and neck cancers, it is of great importance to survey the oral health of the patient. A thorough oral examination, including radiographs, with special attention to marginal and apical periodontitis, calculus, and dental caries, is necessary. All infections should be treated, i.e. teeth may be either extracted or treated endodontically. Calculus should be removed, i.e. the teeth should be scaled and cleaned professionally. All caries should be excavated and the teeth should, at least, be supplied with temporary fillings. All amalgam fillings and metal restorations must be replaced, because the metal became a source of secondary radiation and will lead to damages. Daily rinsing with sodium fluoride solutions is recommended and prescribed to each patient as a complement to the use of fluoridated toothpaste. By taking impressions of both the upper and the lower jaw, the dental technician can make casts on which different devices can be produced. These devices, the mouth-opening device and the mandibular protection device, are used in order to reduce the effect of irradiation on, or to protect, the surrounding tissues not in need of treatment. The casts can also be used to produce trays to administer high-concentration (0.2%) sodium fluoride gel on a daily basis. The mouth-opening device can be designed with and without a tongue depressor. A device without a tongue depressor is used during the radiotherapy when the cancer is located in the floor of the mouth, in the submandibular gland, or in the chin. In contrast, a device supplied with a depressor is used when the cancer is located in the tongue, in the base of the tongue, in the maxillary sinus, in the nose, in the upper lip, in the palate, and sometimes also in the floor of the mouth. The mandibular protection device is used during interstitial radiotherapy to displace the tongue, the cheek or the lip away from the implanted radiation source. It also protects the mandibular bone. This device is used during interstitial radiation of carcinomas in the tongue, the base of the tongue, the tonsils, the floor of the mouth, and the lips. The mandibular protection device is made of approximately 6 mm thick plastic with 2 mm of lead included. The device can also be used to protect the bone of the maxilla, although it is most frequently used to protect the mandible. During the period of radiation, at least weekly professional cleaning of the teeth and mouth is recommended. As treatment continues, i.e. radiation accumulates within the tissues, the patient will have more discomfort, making it hard to maintain proper oral hygiene.

**Management of postradiation conditions:** To reduce the xerostomia the patient should be recommended frequent intake of small amounts of water. Today, there are also a number of saliva replacement products, artificial saliva, and saliva stimulatory products.
available on the market, which have many positive effects on oral well-being. The use of such products can also be of benefit for taste. Life-long use of additional sodium fluoride products, i.e. daily rinsing, should be recommended in order to prevent rapid development of dental caries. Frequent and scheduled visits to a general dentist are desirable.

Reduced mouth-opening capacity is another late side-effect of irradiation. Different training programs are available to prevent early development of this condition. Sometimes it can be of benefit to begin the training already prior to radiation, and continue it as soon as possible after termination of treatment. A very strict and frequent use of these training programs is desirable since the effect is quickly reduced and even eliminated if they are not followed.

Soft tissue and bone necrosis (osteoradionecrosis) are the side-effects that usually develop last. It is not possible to predict which patient is at risk for development of these conditions, though they rarely occur in tissues subjected to less than 55 Gy of radiation. When and if osteoradionecrosis has developed, it is important to maintain good dental hygiene, to prevent development of infections in the affected area. Some alternatives are available to reduce the speed of development, although controversy remains. Hyperbaric oxygen treatment has been used to reduce the development of, and also to treat, osteoradionecrosis. In later stages of osteoradionecrosis it may be necessary to carry out surgical resection, with or without adjunctive hyperbaric oxygen. Microvascular reconstruction, using free flap technology, after surgical resection of the affected area, is today a relatively common treatment in order to restore both function and esthetics in this group of patients.

To prevent the development of these conditions it is of benefit to treat dental problems immediately and conservatively, i.e. regular visits to a general dentist to prevent development of both periodontal disease and dental caries. Dental extractions should be avoided as long as possible. Instead, endodontic treatment should be the treatment of choice when apical periodontitis has developed. If extraction is the only alternative, it should be performed surgically with the aim of covering the area with soft tissue. Systemic use of antibiotics for 7–10 days is recommended.

Chemotherapy has a small role to play but, as new drugs are developed and the biology of the disease becomes more understood, its role may increase. The use of drugs in the treatment of head and neck cancer is still a matter of debate. Despite considerable research, there is no drug or combination of drugs available today that can cure solid cancers in the head and neck region. In general oncology, chemotherapy is given either to cure cancer, or to reduce the speed of progression, or to ameliorate the symptoms. Sometimes chemotherapy is also administered to reduce the risk of recurrences, so-called adjuvant treatment. Chemotherapy is generally used prior to (induction), or parallel (concomitant) to radiotherapy. The purpose of chemotherapy is to kill the cancer cells, or at least impede their growth. Delivery of chemotherapy is made either by intravenous or oral administration. Different drugs are normally combined in different ways depending on the type of cancer to be treated.
It is necessary to repeat the treatment since all cancer cells are not killed at the same time. During the treatment of cancer in the head and neck region, it is normal to administer the drugs two to three times prior to radiotherapy, where the third treatment frequently is concomitant to radiotherapy. Prior to a new administration of drugs, blood counts are always analyzed. After a series of administrations of drugs, an analysis of the treatment is performed, often by a CT scan. The drugs used in chemotherapy affect the cells in different ways. Some drugs are designed to affect the DNA. Other drugs interfere with the intracellular transporting systems. The aim of all drugs is, however, to result in apoptosis of the cancer cells. To achieve maximum effect, with as few side-effects as possible, different combinations of drugs are normally used.

The drugs are normally given every second to every fourth week. Drugs frequently used are cisplatin, 5-fluorouracil (5-FU), and methotrexate. Cisplatin is the one of most useful chemotherapeutic agent. Cisplatin is delivered intravenously over 1 to a few hours. It is typically prescribed in a dose of 100 mg/m2 of body surface area. Cisplatin was given three times during radiotherapy (days 1, 22, and 43). The primary complication is nephrotoxicity but is prevented with hydration before and after treatment.

Chemotherapy seems to be most effective when it is combined with radiotherapy concomitantly. Synchronous chemoradiotherapy seems to show some survival advantages as well as improved function in large posterior tumours. The addition of chemotherapy to radiation therapy has improved both locoregional tumor control and survival rates. High risk was defined as having any of the following: histologic evidence of invasion of two or more regional lymph nodes, extracapsular extension of nodal disease, and microscopically involved mucosal margins of resection. Most importantly, concurrent postoperative chemotherapy and radiotherapy significantly improved the rates of local and regional control and disease-free survival.

The positive neck is usually treated surgically with the addition of postoperative radiotherapy dependent on the extent of neck disease. If combined therapy is required then the surgery is carried out first in most cases. When dealing with oral and oropharyngeal tumours, it is critical that surgical margins should be clear of tumour by more than 10 mm on the pathological specimen, because this will have a considerable bearing on the outcome. It has been shown that survival rates are reduced by 10% on similar tumours, despite all other remedial treatment, if the tumour is not completely excised. Many factors seen on the pathological specimen will affect the outcome and a specialist pathology service is therefore mandatory. Tumour thickness, perineural invasion, vascular invasion, lymphatic invasion, differentiation of the tumour cells and whether the invasive front is cohesive are important in deciding further treatment. Tumour thickness has a bearing on the prognosis, with tumours less than 2 mm thick having a much better prognosis than those with a tumour thickness greater than 4 mm. If the invasive front is cohesive then the prognosis is better than that with a non-cohesive front, while perineural invasion has been associated with local recurrence. Not surprisingly, lymphatic invasion is associated with spread to the lymph nodes whereas vascular invasion increases the likelihood of distant metastases. When surgery is performed
then the decision to give postoperative radiotherapy may not be made until the final pathology is reviewed. An advantage with the use of chemotherapy is that the drug, as it is administered via the bloodstream, is able to affect cancer cells on their way to producing metastasis somewhere distant from the original tumor. A disadvantage with the use of chemotherapy is that none of today’s available drugs are able to distinguish between cancer cells and normal, healthy cells, which means they also have effects on normal tissues. Among the normal tissues most affected by chemotherapy are tissues with a high turnover rate, i.e. bone marrow, hair, and mucosa, resulting in decreased blood counts, loss of hair, and gastrointestinal problems (nausea and diarrhea). Fortunately, the normal and healthy cells have a great capacity for recovery and repair of the DNA, resulting in complete or almost complete remission of the side-effects after termination of chemotherapy. This is the reason for interruptions in the chemotherapy schedule.

The side-effects of chemotherapy therapy include:

1) Loss of hair is the most well known side effect of chemotherapy and is usually observed between 1–2 weeks after the treatment.

2) All mucous membranes are susceptible to chemotherapy due to the quick turnover of cells. Wounds and dryness of the mouth, nausea, and diarrhea may be the result of chemotherapy. Nausea is observed more often in women and younger people. The condition can, however, be reduced effectively with medications.

3) Loss of appetite is another side-effect, resulting in loss of weight. Professional nutritional counseling is frequently needed. It is, however, not unusual for a patient to gain weight during chemotherapy due to the fact that they may feel better with food in the stomach.

4) Another vital organ affected by chemotherapy is the bone marrow. The effect may be low blood counts for all types of blood cells, resulting in anemia, increased susceptibility to infections, and increased risk of spontaneous bleeding, e.g. nose bleeds and gingival bleeding. Transfusions may be necessary during chemotherapy.

5) Tiredness is also a frequently seen side-effect. Lack of energy and problems with concentration may be related to the tiredness. It is of great importance to rest when necessary, to eat as good and well balanced food as possible, and to exercise.

6) The most positive fact related to chemotherapy is, however, that all side-effects almost always subside after termination of the treatment.

7) Bisphosphonate-associated necrosis of the jaws: Bisphosphonates (BPs) are antiresorptive drugs that act specifically on osteoclasts, thereby maintaining bone density and strength. The drug is used for treatment of bone metastases, multiple myeloma, osteoporosis, hypercalcaemia, Paget’s disease. The first report describing osteonecrosis of the jaw (ONJ) in patients receiving bisphosphonates came 2003. The etiology of ONJ remains uncertain. Initially, when the condition was called bisphosphonate-related osteonecrosis of the jaw (ONJ) its similarities with radiation-induced osteonecrosis led to the assumption that the condition started with sterile necrosis of the jaw bone.
Therefore, the term osteonecrosis was used otherwise reserved for sterile bone death usually because of impaired blood supply. It is defined as an area of exposed bone in the maxillofacial region that does not heal within 8 weeks in a patient who is currently receiving bisphosphonate medication and has not had radiation to the head-neck region.

Local risk factors include anatomical features where protruding cortical bone with thin mucosal coverage like tori and exostoses implies greater risk for necrosis as well as periodontal disease, any surgical intervention which breaks the mucosal lining, especially tooth extractions. The optimal treatment strategy for ONJ is still to be established. Cessation of BP treatment will not be sufficient. A multidisciplinary team approach for evaluation and management of the conditions is recommended including a dentist, an oral-maxillofacial surgeon, and an oncologist. In early stages, surgical debridement and coverage has been successful. The Bisphosphonate-associated necrosis of the jaws is irreversible. Prevention is a cornerstone to reduce the incidence of ONJ and before starting BP therapy, the patient should be referred for thorough dental evaluation to identify and treat any potential source of infection. Start of BP therapy should be delayed by 4–6 weeks to allow appropriate bone healing.

Management of oral health during chemotherapy: It is important to thoroughly examine the oral health of patients who will receive chemotherapy. An individual treatment plan should be determined for each patient. The importance of maintaining good oral hygiene during the period of chemotherapy cannot be overstressed. Frequently, the patient may need professional help to achieve optimal oral hygiene. It is, if possible, desirable to manage the most acute oral problems prior to the start of chemotherapy, as for patients subjected to radiotherapy. However, this is often impossible since chemotherapy is initiated rather quickly after diagnosis. Management of oral health is then scheduled to be performed just before the beginning of the second or third cycle of chemotherapy. It is important to obtain a current blood count before any invasive dental treatment, to prevent development of infections, and any undesirable postoperative bleeding. A low leukocyte count – up to 0.5 Giga/l (neutropenia) is considered to represent a high risk of infection. A count of 0.5–1.0 G/l should be accompanied by antibiotic treatment for 7–10 days postoperatively, whereas a leukocyte count of >1.0 G/l only should be followed by a single dose of antibiotics prior to any kind of surgery or other invasive dental treatment, i.e. scaling or endodontics. Extractions and endodontic treatment should be performed at the latest 10 days before an expected leukocyte count of less than 0.5 G/l. Mucositis normally develops after 5–10 days of chemotherapy. At
later stages, ulcers can develop. The ulcers can be infected secondarily with bacteria, viruses or fungi. They can also be very painful, resulting in nutritional problems.

The multidisciplinary approach to the treatment of oral and maxillofacial cancer is now fundamental and all units treating this disease should have a team of specialists. The main team includes:

- A surgeon (maxillofacial surgeon, otolaryngology surgeon, plastic surgeon)
- A cytopathologist
- An oncologist with a specialist interest in head and neck malignancy is also mandatory (radiation and chemotherapy)

The supportive team includes:

- A specialist nurse
- A speech and language therapist
- A nutritionist
- A psychologist
- A social worker
PREPROSTHETIC SURGERY
(Petia Pechalova)

Preprosthetic surgery is a term for surgical procedures undertaken on the edentulous or partially dentate oral tissues before denture construction. Preprosthetic surgery include surgical procedures that aim to improve the condition of the oral tissues to enable a removable denture to rest on a sound base, free from marked bony protuberances or undercuts, with no interfering muscle attachments, flabby soft-tissue excess or hyperplastic oral mucosa.

The aim of preprosthetic surgery is to provide the ideal denture-bearing area for a prosthesis, having satisfactory stability and retention.

Preprosthetic surgery is therefore indicated when prosthodontic measures alone are insufficient. Preprosthetic surgery procedures are ideally undertaken in consultation with a prosthodontist. To achieve the best results, the skills of the oral surgeon and the prosthodontist are combined in a team approach.

Objectives of preprosthetic surgery are:

1) General factors affecting alveolar ridge resorption:
   • nutritional abnormalities
   • systemic bone diseases (osteoporosis)
   • endocrine dysfunction
   • other systemic condition that may affect bone metabolism

2) Local factors affecting alveolar ridge resorption:
   • alveoloplasty techniques used at the time of tooth removal
   • localized trauma associated with loss of alveolar bone.

Although many advances in dental health have been made over the past few decades, it is nevertheless rare for an individual to retain a full complement of natural teeth for life. Teeth are lost for various reasons, notably periodontal disease, dental caries, pathological conditions of the jaws and trauma. Prosthodontics aims to restore not only the function and aesthetics of the dentition after tooth loss but also the aesthetics of the facial form. A well-constructed removable prosthesis that replaces missing teeth will restore function and appearance. A removable prosthesis should be stable and have adequate retention and stability. To achieve this, the prosthesis should be seated onto well-shaped alveolar ridges with adequate basal bone and a healthy oral mucosa. There will ideally be no major vertical or horizontal skeletal discrepancy, which can compromise denture stability. Endosseous implants are commonly placed in suitable patients to improve the stability and retention of removable dentures as well as fixed prostheses. Implants may avoid the need for more complex surgery to improve an otherwise unsatisfactory edentulous ridge.
Denture wearing also may contribute to alveolar ridge resorption because of improper ridge adaptation of the denture or inadequate distribution of occlusal forces. Variations in facial structure may contribute to resorption patterns in two ways: (1) the actual volume of bone present in the alveolar ridges varies with facial form; (2) individuals with low mandibular plane angles and more acute gonial angles are capable of generating higher bite force thereby placing greater pressure on the alveolar ridge areas.

The long-term result of combined general and local factors is the loss of the bony alveolar ridge, increased interarch space, increased influence of surrounding soft tissue, decreased stability and retention of the prosthesis, and increased discomfort from improper prosthesis adaptation. In the most severe cases of resorption, a significant increase in the risk of spontaneous mandibular fracture exists.

The preprosthetic surgery aimed to create proper supporting structures for subsequent placement of prosthetic appliances. The best denture support has the following characteristics:

- No evidence of intraoral or extraoral pathologic conditions
- Proper interarch jaw relationship in the antero-posterior, transverse, and vertical dimensions
- Alveolar processes that are as large as possible and of the proper configuration
- No bony or soft tissue protuberances or undercuts
- Adequate palatal vault form
- Proper posterior tuberosity notching
- Adequate attached keratinized mucosa in the primary denture-bearing area
- Adequate vestibular depth for prosthesis extension
- Added strength where mandibular fracture may occur
- Protection of the neurovascular bundle
- Adequate bony support and attached soft tissue covering to facilitate implant placement when necessary
Evaluation of supporting tissue includes visual inspection, palpation, radiographic examination, evaluation of models.

Preprosthetic surgical procedures are:

I. Hard tissue recontouring

The abnormalities associated with hard tissues are classified into two categories: (1) acquired abnormalities (after extraction of the teeth - sharp spicules, bone edges); (2) congenital abnormalities (torus palatinus, torus mandibularis, multiple exostoses). **Alveoloplasty** is the surgical procedure performed to recontour the alveolar bone, aiming to facilitate the healing procedure as well as the successful placement of a future prosthetic restoration. **Removal of exostoses:** they are generally bony protuberances, which develop in various areas of the jaw. Some authors believe that exostoses are peripheral osteomas. Exostoses are classified into three types: (1) torus palatinus,(2) torus mandibularis and (3) multiple exostoses. **Tuberosity reduction** is necessary in cases of excessive growth, as well as **genial tubercle reduction.** The surgical technique for all bone procedures includes elevation of mucoperiosteal flap, removing of bone, smoothing, and suturing.

II. Soft tissue recontouring

**Fibrous hyperplasia** of the mucosa (formerly known as epulis fissuratum or inflammatory hyperplasia) is usually due to chronic trauma of the mucosa of the mucolabial or mucobuccal fold, due to ill-fitting complete or partial dentures. Fibrous hyperplasia is characterized by folds of hyperplastic fibrovascular connective tissue that develop in association with an ill-fitting denture. Treatment is surgical and consists of excision of the hyperplasia.

**The hypermobile gingival tissue** will not provide adequate base in denture-bearing area. Treatment is surgical and consists of segmental excision of the gingivae.
A frenum is a mucous membrane fold which contains muscle and connective tissue fibres that attach the lip and the cheek to the alveolar mucosa, the gingiva and the underlying periosteum. The aberrant frena can be treated by frenectomy or by frenotomy procedures. Frenectomy is the complete removal of the frenum, including its attachment to the underlying bone, while frenotomy is the incision and the relocation of the frenal attachment. Frenectomy can be accomplished either by the routine scalpel technique, electrosurgery or by using lasers. Since the conventional procedure of frenectomy was first proposed, a number of modifications of the various surgical techniques like the Miller's technique, V-Y plasty and Z-plasty have been developed to solve the problems which are caused by an abnormal labial frenum. The classical technique was introduced by Archer (1961) and Kruger (1964). This approach was advocated to ensure the removal of the muscle fibres which were supposedly connecting the orbicularis oris with the palatine papilla. The technique is an excision type frenectomy which includes the interdental tissues and the palatine papilla along with the frenulum. The frenum was engaged with a haemostat which was inserted into the depth of the vestibule and incisions were placed on the upper and the undersurface of the haemostat until the haemostat was free. The triangular resected portion of the frenum with the haemostat was removed. A blunt dissection was done on the bone to relieve the fibrous attachment. The edges of the diamond shaped wound were sutured. The Miller's technique was advocated by Miller in 1985 and consists of several steps: excision of the frenulum and exposure of the labial alveolar bone in the midline; a horizontal incision was made to separate the frenulum from the interdental papilla; a laterally positioned pedicle graft (split thickness) was obtained and it was sutured across the midline; a periodontal dressing was placed. Z-plasty technique is indicated when there is hypertrophy of the frenum with a low insertion and in cases of a short vestibule. The length of the frenum was incised with the scalpel and at each end, limbs at between 60° and 90° angulation, incisions were made in equal length to that of the band. By using fine tissue forceps, with care not to damage the apices of the flaps, the submucosal tissues were dissected beyond the base of each flap, into the loose non-attached tissue planes. Thus, double rotation flaps which were at least 1 cm long were obtained. The resultant flaps which were created were mobilized and transposed through 90° to close the vertical incisions horizontally. Sutures were placed, first through the apices of the flaps, to ascertain the adequacy of the flap repositioning and then they were evenly spaced along the edges of the flaps, to close the wound along the cut edges of the attached mucoperiosteum and the labial mucosa. V-Y plasty can be used for lengthening the localized area, like the broad frena in the premolar-molar area. The frenum was held with the haemostat and an incision was made in the form of V on the undersurface of the frenal attachment. The frenum was relocated at an apical position and the V shaped incision was converted into a Y, while it was sutured.

Vestibuloplasty is a sulcus deepening procedure, which is a selective method of ridge extension by deepening the vestibule without any addition of bone. Only the soft tissue attachments are shifted to a favorable zone in the jawbones so that more of denture
bearing area is available to increase the retention and stability of the denture. The procedure is indicated in cases of high muscle attachments, inadequate depth in labial and buccal vestibule, inadequate fixed tissue coverage in denture bearing areas. The goals of vestibuloplasty are to provide adequate depth for lateral and buccal area and adequate amount of fixed tissue to form denture seal. Different techniques of vestibuloplasty exist:

1. Submucous vestibuloplasty:
   a) Closed submucous vestibuloplasty
   b) Open view submucous vestibuloplasty

2. Secondary epithelialisation:
   a) Kazanjianaris technique – a mucosal flap pedicled from the alveolar ridge is elevated from the underlying tissue and sutured to the depth of the vestibule. The inner portion of the lip is allowed to heal by secondary epithelialization.
   b) Clarks technique – this technique uses mucous pedicled flap from the lip. Horizontal incision is performed from canine to canine between immobile gingiva and mobile gingiva. After supraperiosteal dissection the mucosa is sutured at the depth of the vestibule. The periosteum heals by secondary epithelialization.

3. Grafting vestibuloplasty
   a) Skin
   b) Mucosal

4. Lingual sulcoplasty
   a) Anterior lingual sulcoplasty
   b) Posterior lingual sulcoplasty
   Caldwell’s procedure
   Trauner’s procedure – Trauner described detaching the mylohyoid muscles from the mylohyoid ridge area and repositioning them inferiorly, effectively deepening the floor of the mouth and relieving the influence of the mylohyoid muscle on the denture.
   Obwegeser’s technique – vestibuloplasty described by Obwegeser is the method in which labial extension procedure provide maximal vestibular extension to both the buccal and lingual aspects of the mandible.
PERIODONTAL SURGERY
(Petia Pechalova)

Objectives of periodontal treatment are:
- To restore bone and soft tissues
- To reestablish the physiological contours necessary for preservation of periodontal health
- To prevent recurrence of disease
- To reduce tooth loss
- To eliminate pain, gingival inflammation and bleeding
- To reduce periodontal pockets
- To stop the destruction of soft tissue and bone
- To reduce the abnormal tooth mobility
- To establish optimal occlusal function

The periodontal surgical procedures could be divided in three main categories:
1. Resective Procedures.
2. New attachment procedures.
3. Regeneration procedures.

The proper method could be chosen by these criteria for method selection:
1) Characteristics of the pocket: depth, relation to bone, and configuration.
2) Accessibility to instrumentation, including presence of furcation involvements.
3) Existence of mucogingival problems.
4) Response to initial therapy.
5) Plaque control.
6) General health.
7) Diagnosis of the case and previous periodontal treatment.
8) Aesthetic consideration.

The goal of the resective procedures is to eliminate or reduce the pocket, by excising or amputating the tissue constricting the pocket wall. This group of periodontal procedures includes:

- Gingivectomy (excision of soft tissue wall of periodontal pocket)
- Gingivoplasty (restoration of the gingival contours by external bevel incision aimed to removal of excess gingiva; healing is by secondary intention)
- Apically positioned flap with or without osseous surgery (the aim is to move the gingival margin apically and without gingival incision)
- Root resection.
*New attachment procedures* present the reunion of connective tissue by formation of new cementum with inserting collagen fibers on root surface that has been deprived of its periodontal ligament. Healing occurs by the formation of long junctional epithelium. This group includes:

- Closed curettage (the aim is to remove the epithelium that lines the pocket wall; it is not applicable in case of deep pockets; disadvantage is limited vision)
- Excisional new attachment procedure, ENAP (a sharp removal of the epithelial lining of the pocket it is not applicable in case of deep pockets; disadvantage is limited vision)
- Open flap curettage and Modified Widman flap procedure (internal bevel incision for reflection of a flap; cleaning the area; repositioning of the flap back to its original site, a new attachment between tissue and root surface is created and a pocket is reduced)

*Regeneration procedures* are surgical procedures aimed at reconstruction of lost or injured periodontium by the restoration of the periodontium to the normal physiologic levels. After regeneration procedures new bone and periodontal ligament formation was occurred. This group of methods include:

- Grafts (bone grafts, soft tissue grafts)
- Guided tissue regeneration (the method aims to guide the right type of cells, e.x. periodontal ligament, to attach to the root surface, and trying to exclude undesirable cells, e.x. epithelium, from attaching to root surface)
- Coronal positioned flap
- Root surface demineralization (citric acid chemicals)
- Interdental denudation.

Factors influencing the success or failure of all regeneration techniques are plaque control, systemic status that affect the periodontium, traumatic injury to teeth and tissue, root preparation, wound closure, soft tissue approximation, post operative and long term maintenance.
FUNDAMENTAL PRINCIPLES OF PLASTIC AND RECONSTRUCTIVE SURGERY. FREE SKIN GRAFTING. OSTEOPLASTY.
(Dimitar Atanasov)

*Definition:* Surgical correction of congenital or acquired defects or deformities of external organs, constituting a functional or cosmetic defect, with the goal to achieve partial or complete restoration of the affected organ.

**Plastic and reconstructive surgery** deals with a variety of maxillofacial defects and deformities that hinder normal functions (chewing, swallowing, speech, and breathing) and affect the appearance of the patient.

- congenital defects and deformities (cleft lip and cleft palate, nose deformities, maxillofacial abnormalities)
- acquired defects and deformities as a result of:
  - mechanical injuries (firearm wounds and other injuries)
  - thermal injuries (burns, frostbites, electrocution injuries)
  - chemical injuries (alkalis, acids, poisons)
  - previous infections (osteomyelitis, syphilis)
  - radiation therapy (skin and mucosal lesions, radiation necrosis)

**Indications**
- biological - restoration of the shape and function of the organ (cleft lip and cleft palate, oroantral communication due to injuries or tumours, partially or completely missing nose, ear, etc.)
- cosmetic (facial wrinkles removal, nasal hump and saddle nose, floppy ears)

**Contraindications**
- local contraindications
  - inflammation of the area of the defect
  - purulent process near the defect
  - non-healing wounds due to syphilis
  - recent radiotherapy (up to one year)
  - signs of tumour recurrence after treatment
- general contraindications
  - age (due to a lag in the development in this field, cosmetic surgery is not performed on children under the age of 8 or 9)
  - cardiovascular diseases
  - hematopoietic system diseases

*Planning of the surgery:* When planning a surgery, an individual plan for each patient should be prepared. The plan should include:

- medical history - congenital or acquired diseases
• anatomical and topographic diagnosis - a precise analysis of the defect should be performed.
• psychological evaluation - it is necessary to determine what the patient's expectations and requirements are (reduced, normal, high or depraved).
• preparation of a surgical treatment plan
  o choice of method
  o location of graft harvesting (adjacent, distant, etc.).
  o technique and route for the relocation
  o type and number of the individual operations
  o time between the different stages
  o postoperative care and documentation

Classification of plastic and reconstructive surgery based on the material used:
• autotransplantation - transplantation of tissues, from one part of the body to another in the same person
• allotransplantation - transplantation of tissues from one individual to another
• xenotransplantation - transplantation of tissues from one species to another (from animal to human)
• explantation - transplantation of tissue from organic but not biological origin.

Skin grafting
In the practice of the plastic and reconstructive surgery, autotransplantation skin autotransplantation is used most commonly, while allotransplantation and xenotransplantation are used extremely rarely (for extensive skin burns and plastic surgery in the oral cavity).

Autotransplantation of skin can be performed by:
• free skin grafting
• non-free skin grafting
• local tissue flap (adjacent)
• flap on nutrient base (pedicled flap)
• tubed pedicle flap

Free skin grafting. Free skin grafting is associated with the name of the Swiss surgeon Reverdin, who in 1869 reported in Paris the transplantation of small epidermal grafts onto a granulating wound. In 1874, Thiersch reported, and later (1886) published 100 cases where dermo-epidermal graft was used. Later, this method became known as the Thiersch method.

A full-thickness skin graft was first applied by the Italian physician Baronio (1804), but it was Lowson (1870) and Krause (1893) who played the major role in the development and the introduction of this method into surgical practice, which later became known in the literature as the Krause method.

In 1929, Blair and Brown introduced the term "split-skin graft" or "thick Thiersch graft". In this method, the skin used has a thickness of 1/2 to 1/3 of the thickness of the skin.
In this method, grafts are taken using a special device designed by Humby, and later by Padgett Dermatome (1939). Nowadays, electric dermatomes and mesh-graft dermatomes are used.

The free skin grafts used in surgery are subdivided, depending on the thickness, into:

- **Thin skin graft**
  This is a dermo-epidermal surface graft or 1/3 graft (Reverdin, Ollier-Thiersch).

- **Split skin graft**
  This is a dermo-epidermal deep graft or 2/3 graft (Blair and Brown with ½ to 2/3 of the skin thickness, or with thickness of 0.3-0.4 mm, or 0.5-0.6 mm; Padgett with ¾ or up to 0.7 mm skin thickness).

- **Total skin graft**
  This is a skin graft containing all skin layers, with full thickness epidermis and derma (Lawson, Krause), 0.8-1.0 mm.

- **Lipodermal graft**
  This is a full thickness skin graft together with the adipose tissue beneath the skin.

In plastic and reconstructive surgery of the face, the jaw bones and oral cavity, the split skin autografts are most important. They contain part of the dermis of the donor area (sweat and sebaceous glands and hair follicles), which, under ointment dressing, allows healing in 2-3 weeks. This allows harvesting large grafts that are most close to the normal skin (except for total graft).

When choosing the type of a skin graft, it is very important to know the advantages and disadvantages of the different skin grafts:

<table>
<thead>
<tr>
<th>Thickness</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thin</td>
<td>Easy harvesting, smooth and easy healing; good survival; minimum scarring of the donor area.</td>
<td>Tendency to wrinkle and change colour; not so good esthetic appearance</td>
</tr>
<tr>
<td>Split-skin</td>
<td>Easy harvesting; smooth and easy healing of the donor area</td>
<td>The donor area may change its colour</td>
</tr>
<tr>
<td>Total</td>
<td>It does not change; it does not darken and it covers securely and thickly the spot on which is placed.</td>
<td>It survives more difficultly, it has limited indications and requires additional treatment of the donor area.</td>
</tr>
</tbody>
</table>

**Indications:**

- fresh or granulating wounds (burns)
- vestibuloplasty in preprosthetic and periodontal surgery
- scarring between the tongue, the floor of the mouth and the mandible
- fresh defects (wounds)
- removal of hyperpigmentation spots and keloids
- scar deformities of the gingiva and lips
• after tumour resection (maxilla, cheeks)
• arthroplasty
• facial hemiatrophy

*Technique:* Free skin grafting should be carried out in the following sequence:

• **Preparation of the recipient site**
  For the success of the surgery, it is important to prepare the recipient site so that to achieve a flat (smooth) surface and meticulous hemostasis. When placing a flap onto a granulating surface, it is important to eliminate any inflammation of the wound first.

• **Graft harvesting**
  The graft can be harvested using a dermatome from areas of the body with smooth and unimpaired skin. Most often, these are the areas of the thigh, abdomen, and gluteus. In facial plasty, best esthetic and functional results are achieved when using skin harvested from the area behind the ears.

• **Placing the graft on the recipient site and fixation of the graft**
  The graft must cover the wound surface well, without pulling, and should fit tightly, which means that it must have the same dimensions as the recipient site. After the graft has been sutured, a compression dressing should be applied in order to avoid formation of hematoma under the graft. The first 96 hours are critical for the success of the free skin grafting, and important factors for that are the following:
  - well-vascularized recipient site (rich in capillaries)
  - good contact between the graft and the recipient site
  - no hematoma (or serous fluid collection) between the graft and the recipient site
  - preventing injury of the graft during the healing period.

*Non-free skin grafting* Non-free skin grafting is divided into:

• adjacent flap (local tissue flap)
• flap on nutrient base (pedicled flap)
• tubed pedicle flap

* Adjacent flap.*
  *Indications:* It is used in all cases where there is a sufficient tissue reserve close to the defect.

  According to Limberg A. (1963), the adjacent flap plasty is the following types:

  • **resection of tissue and approximating the wound edges**
    Elliptical, triangular or rhomboid shapes, or Burow's triangles are used in this technique. The most commonly used shape is the ellipse and after resection, the tissues are dissected and the wound edges are brought close together. For wound surfaces with irregular shape, additional tissue should be resected on the purpose of achieving a suitable geometric shape and approximating the edges of the wound without the occurrence of deformities.

  • **resection of tissue and distancing the wound edges**
    Here, the C. Celsus method for covering defects of the skull, by making incisions at a distance from the defect, dissecting the tissues around the defect, pulling them over it and
suturing them, is applied. The surfaces of the new wounds epithelialize per secondary intention. Langenbeck's technique for palate plasty, Zange's technique for maxillary sinus perforations, and the use of the rhomboid shape in lingual frenulum surgeries are based on this principle.

- **plasty by reshaping and rearranging opposite triangular flaps - Z-plasty or N-plasty**

This is a method with wide variety of applications in plastic surgery, based on two or more opposing flaps, forming one shape created by incisions, making the flaps exchange their places. This technique was proposed by Horner (1837) and Sere (1842), and it was mathematically developed and proposed for use in plastic surgery by Limberg, A. (1939-1946). This technique is indicated for defects and deformities (scarring) in the oral cavity and the maxillofacial area without the need for tissue removal.

When performing Z-plasty or N-plasty, the following elements are outlined:

- a zigzag line consisting of three equal sections.
- two opposing triangular flaps forming an isosceles triangle
- four angles, two of which are open (defining the type of the shape) and two closed (0°) at the end points of the zigzag line
- two diagonals, the short one represents the midline of the incision, and the long one - the imaginary line connecting the two endpoints of the zigzag line

When rearranging the opposing triangular flaps, Limberg found certain dependencies:

1) the final shape represents a mirror image of the initial, rotated at the crossing angle of the diagonals
2) the crossing angle of the diagonals does not change
3) the point at which the diagonals cross does not change
4) the diagonals of the new shape switch their places, and the short one takes the place and the direction of the long one, and vice versa

In Z-plasty, the middle incision (the short diagonal) is aligned along a skin deficiency or a scar (frenulum), and the lateral two - in the surrounding skin (mucosa). After the triangles are rearranged, lengthening is implemented at the place of the middle incision (the short
diagonal). When aligning the incisions, symmetrical and asymmetrical shapes are obtained. In symmetrical shapes, the angles are the same, and in asymmetrical - they are different. When using symmetrical shapes and angles of 60°, an optimal distribution of the skin tension in the surgical field is obtained, and no cones are formed at the base of the triangles, in contrast to the result with smaller angles.

Using mathematical methods, Limberg determined that the greatest elongation occurs at angles of 90° - 2.24 cm, but this is of no practical value, because triangles formed in this way are difficult to move. What is important for the surgical practice is the 75% elongation with 60° angles, which means that if the middle incision is 1 cm long, then after the rearrangement the length will be 1.75 cm. With 45° angles, the elongation is 50%, or with 1 cm incision, the achieved elongation is 1.5 cm. According to the data provided by Limberg, angles above 75° and below 30° have no practical significance, as with the latter only a negligible elongation can be achieved.

<table>
<thead>
<tr>
<th>Angle</th>
<th>30°</th>
<th>45°</th>
<th>60°</th>
<th>75°</th>
<th>90°</th>
</tr>
</thead>
<tbody>
<tr>
<td>30°</td>
<td>1.24</td>
<td>1.34</td>
<td>1.45</td>
<td>1.47</td>
<td>1.50</td>
</tr>
<tr>
<td>45°</td>
<td>1.34</td>
<td>1.47</td>
<td>1.59</td>
<td>1.67</td>
<td>1.73</td>
</tr>
<tr>
<td>60°</td>
<td>1.45</td>
<td>1.59</td>
<td>1.73</td>
<td>1.87</td>
<td>1.93</td>
</tr>
<tr>
<td>75°</td>
<td>1.47</td>
<td>1.67</td>
<td>1.87</td>
<td>1.99</td>
<td>2.10</td>
</tr>
<tr>
<td>90°</td>
<td>1.50</td>
<td>1.73</td>
<td>1.93</td>
<td>2.10</td>
<td>2.24</td>
</tr>
</tbody>
</table>

**Advantages of adjacent flap plasty:**
- one-stage procedure
- minimal risk
- good cosmetic results due to the same colour, turgor and physiology of the tissue flaps used

**Skin flap on nutrient base (pedicled flap)**

*Indications*
- in cases where the esthetic results from free skin grafting are unacceptable
- in cases where there is not enough donor tissue in the adjacent area

*Technique*

Pedicled flap is either a one-step or a two-step method. In the one-step method, tissue close to the defect is used, while the two-step method uses distant tissue. In the two-step method, the first step involves raising a skin flap from three sides and fixing it on the recipient site. In the second step, the nutrient base is resected and the defect is covered. This takes place about 2-3 weeks after the first step. The two types of flaps that are commonly used are simple pedicled flap and arterialized flap.
Simple pedicled flap

In order to preserve the nutrition, the skin flap should be formed with a length-width ratio of 2:1 to 3:1. Here, it is relied on the rich capillary network of the skin to provide nutrition. Therefore, when raising the flap, it is necessary to keep an even layer of subcutaneous adipose tissue. After the flap has been moved to the recipient site, it must be sutured without tension.

The two-step flap was proposed by the Italian physician Talliacozzi (1597), who harvested skin from the inside of the brachium and moved it to the face (nose and face). Tubed pedicle flap is one of the techniques that belong to this group; it is formed with length-width ratio of 1:1.5 to 1:2. The tube can be formed on the arm or neck, and the free end can be sutured to the facial defect.

Arterialized pedicled skin flap

A nutrient vessel (artery and vein) is included in the skin flap, and the pedicle can be either one-sided or two-sided. In this way, the flap can be narrower and longer, for example, with length-width ratio of 1:4 to 1:5.

- Indian method of rhinoplasty (1794)
  A flap harvested from the forehead, with included supraorbital artery or its branch is used to cover a partial nose defect or for nose reconstruction.
- Lexer’s upper lip reconstruction technique (a flap harvested from the scalp with included superficial temporal artery).
- Abbe-Estlander technique for upper lip defects (a flap from the lower lip)

Tubed pedicle flap

The Russian physician Filatov was the first to use a skin flap with a circular tube, formed at a distant location from the defect. This technique was used on 9 September 1916 in a 62-year-old man with a carcinoma of the lower eyelid, and the information was published in the journal Ophthalmology in 1917. The same technique was also used by the English surgeon Gillis (1935).

Indications:
- covering large defects in maxillofacial area and in the oral cavity, for which it is not possible to use adjacent tissue flaps or free skin flaps
  - oroantral communication due to tumour treatment, specific processes or osteomyelitis, injuries
  - full thickness reconstruction of tissues and organs (nose, cheek, lower lip, chin)
Technique

The surgical procedure for forming a circular tube is performed in the following sequence:

- **Choosing a place to form a tube**

  It can be formed anywhere on the body, where there is scar-free skin which can be included within a fold with the required width. For the purpose of the reconstructive surgery of the face and oral cavity, a tube can be formed at any of the following locations: the humeral and thoracic area, the anterior and lateral surface of the chest, the back (below the scapula to the midline direction), or the abdomen.

- **Forming the tube**

  Two parallel skin incisions, which outline the tube, are made under local anesthesia. Their dimensions should be consistent with the length and width of the tube. In order to ensure good nutrition, length-width ratio should be 2:1 to 3:1. When forming the tube, the size of the defect that needs to be reconstructed should be taken into account. Thus, the width of the flap should be planned to be 2 times the width of the defect, and the flap length should be greater by 2-3 cm than the length of the defect. After the incisions are made, a skin flap (containing subcutaneous adipose tissue) is raised on two nutrient bases, and the tube is formed. Next, sutures are placed.

- **Care for the tube**

  Care for the tube should begin as early as the surgical procedure, during which, after the tube is formed, care should be taken to avoid contact with the skin. Gauze should be placed under and around the two nutrient bases (pedicles) of the tube. The use of compression dressing should be avoided. Complete rest should be ensured and the sutures should be removed in 10-12 days.

- **Training the tube**

  Training the tube should begin after the sutures are removed, by tightening the base of the pedicle which will be cut and moved. A rubber tube or a small tool is placed, and it should be tightened twice a day for 5-10 minutes in the beginning. The flap should be monitored for colour changes (becoming bluish), for swelling or changes in its temperature (getting cold). Daily training should be carried out with a gradual increase of time by 5-10 minutes until the period of 120 minutes twice a day is reached. If there are no changes, the flap is considered mature, and the physician may proceed to the next step.

- **Moving and spreading the tube**

  Moving the flap can be done in two ways, by using the so-called “crawling caterpillar” – “waltz dancing” techniques or by moving it towards the arm and then to the recipient site.
If the flap is to be used in the maxillofacial area, it is most often attached to the anatomic snuffbox, forming a circular reception site. After additional training and establishing that the flap is mature, it is moved and sutured to the recipient site. Then, after some more additional training and maturing, the flap is spread out and the organ is shaped.

**Osteoplasty**

First attempts to perform osteoplasty took place in the 19th century, and bone grafts were harvested together with skin-muscle flaps. In this way, Bardenhauer (1892) replaced a bone defect of the mandible after a sarcoma resection using a graft from the frontal bone. In the same year, Wildt used a pedicled bone flap from the clavicle to treat a mandible defect. The first free osteoplasty was performed by the Russian surgeon Zikov, V. M. (1900), who replaced a mandible defect due to osteomyelitis using a free bone graft harvested from the healthy half of the jaw.

Free osteoplasty continued to develop using tibia grafts (Holsted, 1904), rib grafts (McWilliams, 1912) and iliac bone grafts (Rauer, 1924).

Together with the use of autografts, the first attempts to use allografts were made. Lexer (1908) administered allografts from tibia (allograft sterilized by boiling or freshly harvested...
graft) after resection of the mandible due to a malignant tumour. One year later, McEven (1909) reported the replacement of a mandible defect with an allograft taken from the rib of another patient. Gradually, the use of allografts taken from frozen and lyophilized bone started. In 1947, the first tissue bank in the world (New York) was established.

Classifications of bone grafts
- autograft – tissue graft from the same individual
- isograft – tissue graft between two individuals who are genetically identical (i.e. twins)
- syngeneic graft – from parents to children
- homograft - from one individual to another of the same biological species (from human to human, from dog to dog)
- heterograft - from one individual to another, but from a different biological species (from calf to human, from rabbit to dog)
- fetal or embryonic grafts - grafts obtained from an embryo, a fetus or a newborn
- alloplasty - grafts of inorganic substance

Indications
- defects in the shape and the function of the jaw
- resection of the mandible due to tumours and other lesions
- chronic osteomyelitis
- defects caused by injuries (firearm wounds and other injuries)
- ankylosis of the mandibular joint
- pre-prosthetic surgery

Contraindications
- acute inflammation of the area of the defect
- absence of adequate amount of available soft tissue to cover the defect
- infectious diseases (up to 3-4 weeks after healing)
- general poor health
- severe injuries to other parts of the body

Technique: Before performing the surgery, it is necessary to carry out sanitation of the oral cavity (2-3 weeks before the surgery).

The surgery includes the following steps:

- Preparation of the recipient site
  Osteoplastic surgery of the mandible can be either primary or secondary, depending on the underlying disease (benign tumour, ankylosis, malignant tumour, or osteomyelitis). Primary osteoplasty is performed in cases of benign tumours and ankylosis, where osteoplasty is performed immediately after jaw resection. In these cases, the tissues of the recipient site are slightly altered and well vascularized, which is a good basis for the survival of the graft. In cases of firearms injuries, osteomyelitis and resection of malignant tumours, secondary osteoplasty or the so-called delayed grafting is performed.
An important prerequisite for the success of the surgery is that the prepared site does not contain any scar tissues and that good hemostasis has been achieved. When performing primary osteoplasty, it is important to maintain a good recipient periosteal bed.

- **Harvesting and fixation of the graft**

  In autotransplantations, a second surgical field is created - in the tibia, the iliac bone or the rib. The graft should be free of periosteum when placed in the well-shaped periosteal bed. If the amount of periosteum in the recipient site is not sufficient, a rib with periosteum can be harvested. The graft should be of the size of the jaw defect and should fit well and be firmly fixed to the defect – by bone suture or by mini plates. Rest should be ensured during the healing period.

- **Covering the wound**

  The graft placed in the recipient bed should be primarily covered and the soft tissue should be firmly fixed around the graft in order to avoid formation of hematoma. At the same time, unnecessary strain of the soft tissue should be avoided because of the risk of impairment of the blood supply and the risk of perforation of the oral mucosa.
CONGENITAL CLEFT DEFECTS IN THE MAXILLOFACIAL AREA

(Dimitar Atanasov)

Cleft lip and cleft palate are some of the most common malformations in humans, and they account for about 10-20% of all malformations.

Ambroise Paré (1575) speaks of "bec de lievre" - "rabbit lip" referring to cleft lip cases. Ternovski introduces the term "non-union". Limberg A. (1937) writes about "cleft upper lip", and Dorofeev (1937) about "congenital cleft upper lip".

Incidence: The first report on the incidence of clefts is found in the works of St. Petersburg physician Frabelius (1864), who provides statistics on the number of children with clefts in an orphanage. From 180 000 children, 188 were found to have clefts (1:1525).

The incidence of cleft lips and/or cleft palates not caused by syndromes was reported to be in the range of 1:700 (Bender, P. L., 2000). 1:750 (Young, G. Deskin, R., 2008) to 1:850 (Georgiev, E., 1974), in newborns. Cleft lips (with or without cleft palate) are more common in males (males: females = 2:1), and isolated cleft palates are more common in females (males:females = 1:2), with more than 70% of children with cleft lips having cleft palate as well (Frolova, L. E., 1974; Diewert, V. M. Wang, K. Y., 1993).

About 21-37% of children born with cleft lip and/or cleft palate have other abnormalities, such as cardiovascular (24-51%), musculoskeletal, facial and urogenital malformations (Stoll, C. et al., 2000). There are over 100 syndromes associated with cleft lip and/or palate.

Syndromes associated with cleft lip and/or palate (Lees, M., 2001)

- Velocardiofacial syndrome
- Pierre Robin syndrome
- Goldenhar syndrome
- Ectrodactyly-ectodermal dysplasia-clefting syndrome
- Gorlin syndrome
- Oto-palato-digital syndrome
- Oral-facial-digital syndrome
- Smith-Lemli-Opitz syndrome
- Stickler syndrome
- Treacher Collins syndrome
- Van der Woude syndrome
- de Lange syndrome
- Kabuki syndrome
- Fetal alcohol syndrome
- Fetal phenytoin syndrome
- Fetal valproate syndrome
**Etiology:** Numerous studies conducted so far have not completely determined the causes for the development of cleft lip and cleft palate. Various exogenous and endogenous factors have adverse effects, impairing the normal development of the fetus in its earliest stages.

Historically, the attitude towards people born with clefts has been contradictory. In the ancient Egypt, the priests considered people born with clefts as gods, surrounded them with reverence, and after their death, such people were embalmed. In ancient Rome and in Greece, children born with clefts were thrown off cliffs, and during the Middle Ages, their mothers were also killed. A birth of a child with a cleft was considered a punishment for committing a sin.

Long-lasting clinical observations show that children with clefts are completely normal and are no different from other people. There are many famous people born with clefts (doctors, philosophers, etc.). It is, therefore, very important nowadays to know the adverse factors and to ensure prevention and/or treat such children adequately.

Presently, it is assumed that most of the cases of cleft lip and/or cleft palate are caused by a combination of genetic and environmental factors which alter the complex process of morphogenesis (Amaratunga, N.A., 1989; Young, G., Deskin, R., 2008). The environmental factors that increase the risk of clefts include: maternal disease (diabetes), smoking and alcohol intake during pregnancy, poor nutrition during pregnancy (avitaminosis A and B2), certain medications (ethanol, thalidomide, aminopterin, corticosteroid therapy or increased corticosteroid secretion in early pregnancy), infections (rubella, toxoplasmosis), ionizing radiation (X-rays and radiotherapy) during pregnancy, mental trauma (emotional disorders increase the release of ACTH by the anterior pituitary gland which results in subsequent increase in the secretion of cortisone by the adrenal glands).

Many children with clefts are born in families who do not have a family history of cleft lip or cleft palate and by mothers who had a normal pregnancy. If both parents do not have clefts, but have a child with a cleft, then the probability for them to have another child with a cleft is 3 to 5%. If both parents have clefts, but have a child without a cleft, then the risk for them to have a child with a cleft is 5%. If more than one parent and/or children have a cleft, then the risk of their future children to be born with clefts is high (Children's craniofacial association, USA, 2009).

**Embryogenesis:** The maxillofacial area is formed by the fusion of 5 processes (frontonasal, two maxillary and two mandibular) during the human embryonic development. At the end of the third week of the embryonic development, the above mentioned processes define the primary oral fossa, or stomadeum. At the end of the first month of intrauterine life, when the length of the fetus is about 10.5 mm, the mandibular processes fuse together. Meanwhile, the frontonasal process quickly develops forward and splits into three additional processes - medial and lateral nasal processes. From the caudal end of the medial nasal process, the two globular processes are formed.
By the 40th day of intrauterine life, the globular processes fuse together with the lateral nasal processes in area of the nostril, and by the third month they fuse together to form the medial part of the lip and the intermaxillary bone.

The ala of nose and lateral surfaces of nose are formed from the lateral nasal processes. The nasal root, the nasal tip, the nasal septum, the medial part of the upper lip with the philtrum, the vomer and the intermaxillary bone are formed from the medial nasal process. The maxillary processes form the lateral parts of the upper lip, the upper part of cheeks and the maxillary bones with the nasal turbinates and the palatal processes.

The primary palate (the triangular area of the hard palate, situated in front of the incisive foramen next to a point laterally of the lateral incisor, which includes a portion of the alveolar ridge with 4 incisors) is formed during the first 4-7 weeks, and the secondary palate (the part posterior to the incisive foramen and the whole soft palate) is formed during the first 6-9 weeks of embryonic development. The palate changes its position from vertical to horizontal and the tongue moves down and forward (Young, G., Deskin, R., 2008).

The primary palate is formed by the fusion of the maxillary and the medial nasal process, and during the second month, the development of the final palate that separates the oral and the nasal cavities begins. The hard and soft palate and the palatopharyngeal arches are formed from the palatal plates, emerging from the inner surface of the maxillary processes. In the beginning, these plates are situated vertically and their sides fit to the tongue. With the growth of the mandible and the displacement of the tongue, the plates turn from vertical to the horizontal position. They gradually fuse together and with the nasal septum.

The parts forming the lip and the alveolar ridge fuse early, and the parts forming the hard and the soft palate fuse later. This explains the formation of clefts of the upper lip and the alveolar ridge and the formation of isolated clefts of the palate.

Classification: Numerous classifications of clefts can be found in the literature, involving upper lip clefts (cheiloschisis) and palate clefts (palatoschisis), as well as clefts involving the lips and the alveolar ridge (cheilognathoschisis) and clefts involving the lips, the alveolar ridge and the palate (cheilognathopalatoschisis) - Veau, V., 1938; Kernahan, A., Stark, R., 1958; Vasiliev, G. A., 1959; Kavrakirov, V., 1961; Kernahan, D. A., 1971; Frolova, L. E., 1974.

Classification of upper lip clefts according to Frolova, L. E. (1974):

- Incomplete congenital cleft of the upper lip
  - unilateral (right-sided or left-sided)
  - bilateral (symmetrical or asymmetrical)
- Complete congenital cleft of the upper lip
  - unilateral (right-sided or left-sided)
  - bilateral (symmetrical or asymmetrical)
- Atypical congenital cleft of the upper lip (facial coloboma)
Classification of clefts of the palate according to Dubov, M. D. (1960):

- Crossing
  - unilateral
  - bilateral

- Non-crossing
  - complete
  - incomplete
  - hidden (submucosal)

Classification of clefts of the upper lip and the palate (WHO, 1971):

A. Cleft lip (unilateral – left-sided, right-sided; bilateral)
   - marginal (within the red portion of the lip)
   - incomplete (within the body of the lip)
   - complete, non-penetrating (extends to the nasal vestibule)
   - Complete, penetrating (passes through the nasal vestibule)

B. Clefts of the lip and the alveolar ridge.
   - marginal (within the red portion of the lip)
   - incomplete (within the body of the alveolar ridge)
   - complete, non-penetrating (extends to the incisive foramen)
   - complete, penetrating (passes through the incisive foramen)

C. Clefts of the lip and the palate

D. Clefts of the lip, the alveolar ridge and the palate
   - non-crossing
   - crossing

E. Clefts of the palate (medial only)
   - uvula (within the uvula)
   - soft palate (within the soft palate)
   - incomplete (within the soft palate and only partially in the hard palate)
   - complete, penetrating (extends to the incisive foramen)
   - complete, non-penetrating (passes through the incisive foramen)

Clinical presentation: Clinical symptoms of cleft lip and/or palate are related to cosmetic, functional (dental, speech, swallowing, hearing, face growth) and emotional changes (Young, G., Deskin, R., 2008).

- Cosmetic (anatomical) changes.

   In children who have only a cleft lip without an alveolar ridge cleft or a palate cleft, problems are of cosmetic nature only. Depending on the cleft type, characteristic anatomical and cosmetic changes are observed which determine both the esthetics of the face and the choice of a treatment method. In the case of incomplete clefts of the lips (harelip), the width of the lip is different, but bottom of the nose always remains closed.
In case of a unilateral complete cleft of the lip, the floor of the nose communicates with the oral cavity; the maxilla on the side of the cleft is hypoplastic; the columella is shifted to the normal side, the ala of the nose on the side of the cleft is displaced sideways, backwards and downwards; the muscles of the lip are included in the ala of the nose and in the columella.

In case of a cleft of the lip and alveolar ridge, often the intermediate bone on the side of the cleft is displaced in outward and cranial direction, and the hard plate is normal. There are significant anatomical and esthetic changes in clefts involving the lip, the alveolar ridge and the palate. The large maxillary process is turned outward, the small process is located dorsally and it stretches the nostril. Cartilage and soft tissue edges of the cleft have a different degree of hypoplasia. The most severe anatomical and cosmetic changes are those in bilateral crossing cleft involving the lip, alveolar ridge and the palate. In this type of clefts, the intermediate jaw and the probibium are in front of the vomer and the nose, they continue forward and cause a significant disfigurement of the face. The probibium is mobile and the lateral jaw processes may be turned inwards and touch the vomer.
In individual children, clefts of the lips, alveolar ridge and the palate, and of the lateral part of the face, the so called "facial coloboma", which are extremely hard to treat, are very rare.

- **Functional changes**
  - Difficulties with eating

  Children born with a cleft upper lip cannot latch on the nipple of the mother's breast and often give up sucking.

  Children with cleft palates also have difficulties sucking due to the cleft (gap) in the roof of the mouth and the inability to create vacuum. The food goes into the nose and the children give up eating. Such children need a special feeding position and feeding bottles with modified or special teats which allow proper feeding. Such children are not able to suck and many mothers feed their children by placing the breast milk in a bottle with a palatal teat.

  - Ear infections and hearing loss

  A common complication in cleft palate is a decrease in hearing. Various acquired impairments of the eardrum (dark pigmentation, straining, scars, perforations) and obstruction of the Eustachian tube are observed. Often the tonsils are enlarged, but inflammation occurs rarely. Some children with cleft palate are at an increased risk of developing frequent ear infections. Malfunctions result in the collection of fluid in the middle ear. The combination of
presence of fluid and recurrent ear infections can result in scars on the eardrum membrane and in hearing loss.

- **Speech and linguistic problems**

  Speech problems are a serious complication in cleft palate. As a result of the impaired palate integrity, air passes not only through the mouth, but also through the nose, which leads to voice abnormalities (nasal speech – rhinolalia). The speech is faint and the vocal sounds cannot be clearly pronounced. Speech disorders, or dyslalia, occur. There are also changes in facial gestures when talking, such as contractions of the nostrils and lifting the upper lip (to prevent air from exiting through the nose). Such children are depressed and avoid contact and conversations.

  Children with cleft palate have nasal speech because air is released through the cleft of the roof of the mouth and goes out of the nose. They may also have difficulty keeping enough air pressure to produce a clear sound. Due to the opening of the roof of the mouth, the muscle function may be reduced, which may result in slow speech or abnormal speech.

  After palate reconstruction, most children achieve speech that is close to normal. A number of studies have shown that speech problems are found in about 5% of the children in the normal population. In children with clefts, the level of such problems is significantly higher - 33% of the 5-year-olds, and 14% of the 12-year-olds (Fox, V. A., Dodd, B., Howard, D., 2002). Some children may need additional speech therapy or additional surgery when they are older in order to improve their residual nasal speech.

- **Dental problems**

  Children with cleft of the palate or the alveolar ridge may have dental problems - supernumerary teeth (20%), dystopic teeth (30%), missing teeth (50%), malocclusion (100%) or abnormal shape of primary or permanent teeth (Young, G., Deskin, R., 2008). In addition, the maxilla may not grow at the same rate as the mandible, requiring further corrections later in life (treatment by orthodontist).

- **Respiratory problems**

  Children with cleft lip or palate often suffer from inflammation of the upper respiratory tract, bronchi and lungs due to the inability of the air to be cleaned and warmed before going directly into the lungs. In children with clefts, breathing through the nose is impaired; it becomes deep and rapid (40-60 per minute (normal rate 15-20)).

  **Care for children born with clefts.** Care should begin as early as the prenatal period, when the presence of a cleft is discovered by ultrasonography. At that moment, the preparation of the parents should also start, and it should be focused on preparing them psychologically and on showing them the right way to raise and treat the child.

  At the first meeting with the parents, it is necessary to explain to them carefully and respectfully that this is a congenital disorder and it is not rare. It is necessary to explain to the parents that they are not to blame and that the modern plastic surgery is able to eliminate this defect. They should know that children with clefts are in no way inferior to their peers and
that a number of celebrities have had clefts (for example, the famous German plastic surgeon Prof. Moeller).

It must be stressed that children with congenital clefts are a pediatric problem first, and then a surgical problem. Children born with clefts need multidisciplinary care, including pediatrician, plastic surgeon, dentist, orthodontist, otorhinolaryngologist, psychologist, orthopedic surgeon and speech and voice therapist.

Special attention should be paid to the way the child is fed, first by the medical staff and then by the mother. The child should be taught to obtain nutrition by a spoon or a baby bottle, but never with a feeding tube. In case of a cleft palate, baby bottles with special plugs are used so that the food does not go into the nose, which can make the child refuse eating. This is a difficult and important task requiring much patience and time. According to Wassmund, the mother has to spend at least 4-5 hours a day feeding her child.

The dental practitioner should have enough information on the problem and should provide the necessary guidelines for child care. Next, he/she should refer the children to treatment at specialized wards, by knowing the requirements related to child’s age and general condition, and the timing for the surgery.

**Proper time for surgical treatment** Closing a cleft requires surgical procedure. The proper time for the treatment of cleft lip and cleft palate is different, and it depends on their functions and the effects they have on the growth of the jaw bones. Various surgical techniques and time points for the surgery have been proposed, but the principle goal of surgery is to achieve the following by careful muscular reconstruction (Delaire, J., 1978):

- cosmetic reconstruction of the normal appearance of the baby at the right time
- functional reconstruction of the lip or palate in order to allow normal feeding and to create a functionally adequate palate allowing for the development of normal speech
- creating conditions for optimal growth and development of the face in order to prevent deformities

These principles apply to both primary and secondary cleft corrections because good function is a prerequisite for good facial esthetics.

Over the years, the understanding of the appropriate time for surgical treatment has changed. So, according to Veau, V. (1938), the appropriate age for **cleft lip** treatment is no less than two months, whereas a large group of authors suggest that the weight of the child should be over 6 kg and the age at least 4 months (Burian, F., 1966; Stelman, R., 1978, Roland, C., 1982). A third group of authors recommend that the child should be operated immediately after birth (in the first two days) or after the age of 30 days (Frolova, L. E., 1974, Georgiev, E., 1975). The early surgery has a number of advantages: the lowest risk of infections is after birth and the risk increases significantly after the 3rd month; prevents psychological traumas, as the child leaves the maternity hospital healthy; allows for forming of the facial expression complex and helps for the proper and harmonious forming and development of the craniofacial system. According to Frolova, after the birth the child should rest, and the pediatrician should determine whether there are any contraindications for the
surgery (other congenital abnormalities). If it is not possible to perform the surgery in the first 2-3 days, the surgery should be postponed until day 11 to 30 (need to clarify the condition; period for physiological jaundice to resolve – day 3 to 10; physiological weight loss - day 2 to 10; increase in bleeding – day 2 to 11).

Surgery in the first 2 to 3 days should only be performed if:

- child’s weight is between 2,500-3,300 g
- respiration rate is up to 60/min and the heart rate is 120-140/min
- WBC are 21 G/L and the decrease on day 9-10 to 10-12 G/L
- Hb - 140% in the neonate and decrease to 110-100% on day 9-10

When the child is crying, the pulse accelerates, and it slows down by 20-30/min during sleep.

Today, few surgeons practice lip reconstruction in the neonatal period because the results are not better than with later surgery and there is no psychological benefit for the parents (Slade, P., Emerson, D.J. M., Freedlander, E., 1999). Most surgeons perform reconstruction the lip and often the alveolar area, and the frontal part of the hard palate between the 3rd and 6th months after birth (Precious, D. S., et al., 2001; Hodgkinson, P., et al., 2005; Children's craniofacial association, USA, 2009).

When discussing the time for surgical reconstruction of a cleft lip, it is necessary to consider what its type is - unilateral or bilateral (partial or complete).

In the case of a unilateral complete cleft, early reconstruction will prevent the rapid growth of the small fragment, which results in retroversion of the entire dentoalveolar segment and underdevelopment of the premaxilla - a problem that is difficult to correct at a later time. It is better to wait until the 6th month when the upper central incisors are well developed and even have erupted. This time is physiologically logical (Precious, D. S., 2000).

In the case of a bilateral complete cleft, where the premaxilla is elongated and displaced forward, it is preferable to close both sides of the cleft lip at the same time, in the first 4 months. Three months later, the dentoalveolar elements of the premaxilla and the lateral segments are usually arranged adequately, so it is possible to perform gingivoperiosteoplasty, a procedure that contributes to the rapid transversal development of the maxilla (Precious, D.S., et al., 2001).

The opinions of surgeons regarding the time for surgery of cleft palate are also contradictory. The early authors (Brofi, Lein) performed surgery in the first days after birth. Later authors like Veau, V. (1938), Ivy (1939), Rosenthal (1940) performed surgery when the child was over 3-4-5 years old, while Bernadsky, Yu. (1974) proposed surgery to be performed after the child becomes 6-7 years of age. Authors, such as Limberg, A., Langenbeck suggest surgery after 10-13 years of age.

A number of radiographic and cephalometric studies carried out in children with cleft palate of different ages showed that maxilla is underdeveloped in all directions (Graber, Krogmann). Early surgery worsen the deformities, so they proposed surgery to be carried out
after 4 years of age. At this age, the jaw has reached 5/6 of its size and deformities caused by the surgery performed after that age are minor.

Speech therapists prefer surgery to be performed at a younger age (after the first year), that is, at the beginning of speech development. However, this slows the development of the maxilla and in the case of a traumatic surgery, results in its deformation.

Orthodontists prefer later surgery (4-6 years) in order not to impair the growth of the jaws. However, this results in poor speech results.

After the 1980s, a number of surgeons performed surgery at a younger age, i.e., after the eruption of the primary second molars (2 to 2 1/2 years) - Frolova, L. M., Stelmah, R., Yovchev, V. Speech development is not impaired and the growth of jaw bones is less likely to become complicated because of the advanced mineralization of the maxilla and the presence of stable bite.

Nowadays, plastic surgery of cleft palate is performed at the age of 8 months, using one-step technique for the soft and hard palate (Hodgkinson, P., et al. 2005), or between 6 and 12 months, using two-step operation - the first step is performed at the age of 6 months, when the soft palate and the cleft of the lip are closed, and the second surgery of the hard palate is performed at 1 year of age (Precious, D. S. et al., 2001). According to data provided by the Children's craniofacial association, USA, 2009, cleft lips should be reconstructed at the age of 3 to 6 months and cleft palates between 9 to 12 months, but before the age of 2. Late closure of the palate worsens speech (Sell, D., 1992).

In alveolar cleft, osteoplasty should be performed at the age of 5 ½ to 6 years, which is a prerequisite for normal anatomical and functional development of the alveolar ridge and the dental arch (Precious, D. S., et al., 2001).

Treatment

The dawn of the cheiloplasty is associated with the name of Celsius (25 BC). The first written source, according to Boo Chai (1966), dates back to 390, when the lip of the governor of the 6th Chinese province Wey Young-Chi was reconstructed by an unknown Chinese surgeon.

The Papal Decree of 1215, prohibiting surgery and blood-letting, retarded the development of surgery until the 16th century.

The modern development of the cheiloplasty is associated with the name of Veau, V. (1938).

- **Fundamental principles of the treatment of cleft lip (cheiloplasty)**
  - The treatment methods for cleft upper lip can be divided into three groups:
    1) refreshment of the edges of the cleft and suturing the cleft (Yperman, J., 1300; Mirault, 1845)
    2) covering the defect by immobilizing the adjacent soft and bone tissues (Diffenbach, 1845)
    3) covering the defect by modelling flaps from the adjacent soft tissues (Veau, V., 1938; Limberg, A., 1939; Frolova, L. E., 1974; Millard, D. R., 1958)
Nowadays, the methods of Group 3 are used exclusively, and their goal is:
- reconstructing the form of the red portion with a correct outline of the Cupid's bow
- creating a lip that is long and mobile in transverse direction, and corresponds to the healthy side
- forming the ala of the nose and the nostril, which are severely deformed in cases of crossing clefts and in some of the non-crossing clefts

In our country, Kavrakirov, V. (1961), using his extensive experience in the field of cheiloplasty, developed his own method, which consists of the following steps:
- planning the surgery and outlining the main incisions
- making incisions in tissues
- dissection and mobilization of the tissues of the lip
- suturing.

- **Fundamental principles of the treatment of cleft palate (uranoplasty)**
  
  The first attempt for surgical reconstruction of a cleft palate was made by the dentist Lemonnier and the operation was described by Robert (1776).

  The fundamental principles of surgical reconstruction of cleft palate are the following:
  - closing the fissure
  - retrotransposition
  - constriction of mesopharynx

  **Closing the fissure** was developed by Langenbeck (1859), and it includes refreshing the edges of the defect, dissecting and relocating mucoperiosteal flaps to the midline and suturing them. Langenhagen keeps the nutrient base of the flaps at the front and at the back, and in order to reduce tension, he cuts the muscles of the soft palate in the area of the hamulus (performing myotomy). In 1889, Billroth performed resection of the medial plate of the ala of the sphenoid bone at the level of the hamulus, thus relocating the muscles of the soft palate. Performing a myotomy, according to Billroth disturbs the function of the muscles.
Retrotransposition was developed by Ernst and Hale (1915) and further developed by Lvov, P. (1925). Ernst and Hale move the mucoperiosteal flaps backwards, but in two steps (after cutting the palatal neurovascular bundles and restoring the nutrition of the flaps, they move them backwards). Lvov moves the mucoperiosteal flaps backwards after removing the posterior part of the greater palatine foramen. Thus, in one step, he extends the soft palate using retrotransposition.

Constriction of mesopharynx was developed by Ernst and Hale (1925). Unsatisfactory speech results after uranoplasty have shown that the cause is the widened oral part of the mesopharynx. This gave Ernst and Hale the idea to perform constriction of mesopharynx by extending the lateral incisions of the hard palate to the lingual surface of the alveolar ridge of the mandible. The incision goes backwards, downwards and inwards along plica pterygomandibularis, cutting the mucosa only. This incision reveals the pharyngeal vestibule and moves the lateral wall of the pharynx inwards, along with the muscles of the soft palate.
Bibliography


79. Colotne SH. The mucogingival surgical procedures which were employed in re-establishing the integrity of the gingival (III). The free mucosal graft. Quintessence Int. 1977;8(7):53–61.

319
103. Dimova–Gabrovska M.: Disclusion in excursive mandibular movements by CMD-patients –computerized analysis at laterotrusion, PRAEMEDICUS Since 1925, Medical University Sofia, 2016, 33,1, 33-37
321


315. Sarachev, E. Lymphatic system disorders in maxillofacial area and cervical region, Plodvid, 1999: 78.
324. Shashikiran, N. D., Reddy, V.V., Naga, N.B. Knowledge and attitude of 2000 parents( urban and rural-1000 each) with regard to avulsed permanent incisors and their emergency management,in and around Davangere, J. Indian Soc.

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- www.cancerwall.com/pleomorphic-adenoma/
- www.headandneckcancerguide.org/
- www.atlasgeneticsoncology.org/Tumors/SalivGlandOverviewID5328
- www/bharathreddymoola/tmj-dislocation
- www/ennbeee/tmj-ankylosis
- www/prashanthlakshman3/parotidectomy
- www/shaimaazakaria9/salivary-gland-pathology-46004405
- www.dipika005/tmj-imaging
- www.drotornyipious/tmj-anatomy
- www.drotornyipious/tmj-anatomy
- www.exodontia.info/Jaw_Dislocation.html
- www.indiandentalacademy/tmj-iiorthodontic-courses-by-indian-dental-academy
- www.physio-pedia.com/TMJ_Anatomy
- www.physio-pedia.com/TMJ_Anatomy
- www.slideshare.net/kcpanopio/canalicular-adenoma
- www.toothandtips.com/what-is-tmj-symptoms-relief/
- www/pathologyoutlines.com/topic/salivaryglandsoncocytoma.html
- www/pathologyoutlines.com/topic/salivaryglandssebaceousadenoma.
- www.ghorayeb.com/ParotidAcinicCellCarcinoma