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КАФЕДРА СТОМАТОЛОГИИ ДЕТСКОГО ВОЗРАСТА

А. Н. Антоненко, Ж. М. Бурак, Ю. В. Зенькевич

ДЕТСКАЯ ХИРУРГИЧЕСКАЯ СТОМАТОЛОГИЯ И ЧЕЛЮСТНО-ЛИЦЕВАЯ ХИРУРГИЯ

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Учебно-методическое пособие

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Посвящено вопросам детской челюстно-лицевой хирургии и хирургической стоматологии как части дисциплины стоматология детского возраста. Изложена этиология, патогенез, клиническая картина врожденных пороков развития, травм и опухолей челюстно-лицевой области, воспалительных заболеваний слюнных желез и лимфатических узлов, височно-нижнечелюстного сустава у детей. Рассмотрены современные представления о диагностике, лечении и профилактике данных заболеваний и их осложнений.

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Антоненко Анна Николаевна
Бурак Жанна Михайловна
Зенькевич Юрий Всеволодович

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LYMPHADENITIS OF THE MAXILLOFACIAL REGION IN CHILDREN. ABSCESSES, CELLULITIS AND ACTINOMYCOSIS OF THE MAXILLOFACIAL REGION IN CHILDREN. ETIOLOGY, CLINICAL MANIFESTATIONS, DIAGNOSIS, TREATMENT

Lymphadenopathy is a term meaning "disease of the lymph nodes". It could be due to infection, autoimmune disease or malignancy. Inflammation of a lymph node is called **lymphadenitis**. In practice, the distinction between lymphadenopathy and lymphadenitis is rarely made.

Lymphadenitis is rarely a primary disease. More often it is the *reaction* of the *regional lymph nodes* to the introduction and spread of infection from a distant focus. Lymphadenitis can be a symptom of periodontitis, osteomyelitis, stomatitis, angina, otitis, measles, chicken pox, etc. But, when the main focus is eliminated, lymphadenitis is considered as a separate disease.

Lymphadenitis is more common for **preschool children** than for adults. This is due to: the imperfection of cellular and humoral immunity, immature of lymph nodes, high incidence of injuries and inflammatory diseases of the skin and mucosa and high incidence of upper respiratory tract and the intensity of dental caries.

The most often inflammatory process affects **submandibular** lymph nodes. This is due to the fact, that they collect lymph from the face, oral cavity, teeth.

Classification of lymphadenitis:

1. ***By way of invasion:***

– Odontogenic;

– Non-odontogenic — as a result of respiratory and viral infection, specific infection (tuberculosis, syphilis, actinomycosis).

2. ***By the acuteness of process:***

– Acute (serous and purulent);

– Chronic (hyperplastic and purulent);

– Chronic specific (tuberculosis, actinomycosis, etc.).

ACUTE SEROUS ODONTOGENIC LYMPHADENITIS

The patient usually complains of painful "ball" during the palpation in the certain area. The child would note that it was a toothache before the enlargement of the lymph node.

General condition of a little patient is without significant changes: minimal rising of the body temperature, insignificant intoxication. During the *extraoral examination* soft tissue swelling of one of the anatomic areas is revealed. Lymph nodes therein are enlarged, elastic, freely movable, have a rounded shape and are painful during palpation. The skin above the lymph node is without changes. During the *oral cavity examination* the causative tooth with positive percussion can be revealed. In most cases the diagnosis of periapical inflammation is easily determined.

The treatment begins with sanitation of the primary site of infection. Doctor also prescribes physiotherapy (during first 2–3 days after the onset): phonophoresis of hydrocortisone, electrophoresis of dimexid with antibiotics, laser. Anti-inflammatory therapy, including antibiotics is necessary too.

ACUTE PURULENT ODONTOGENIC LYMPHADENITIS

Acute purulent lymphadenitis is the next stage of the serous lymphadenitis without treatment. It transforms to purulent form within 5–7 days after the onset of the disease.

Lymph node is enlarged and causes pulsative pain. Body temperature increases to 38–39, loss of appetite and behavior changes appear.

Clinical features. Doctor notes asymmetry of the face due to the swelling of the certain region. The skin over the affected node is swollen and hyperaemic. Lymph node is enlarged, painful, without clear borders (Fig. 1). Fluctuation is not always present because of the tight capsule of the lymph node. Opening of the mouth is not limited, the causative tooth can be easily found. Blood test reveals left shift of the leucocytes.

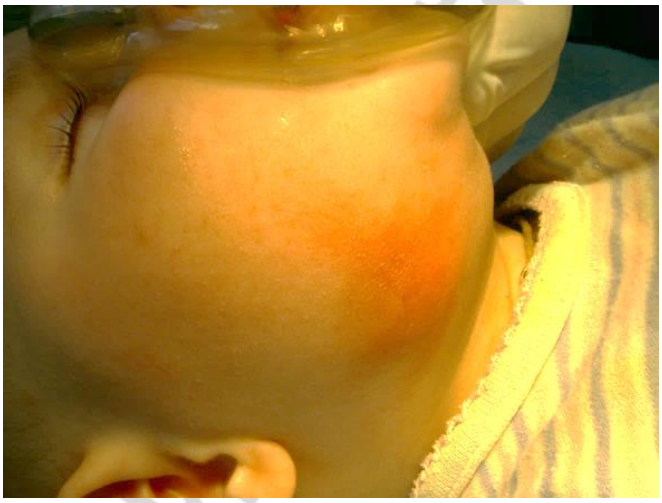


Figure 1. Acute purulent lymphadenitis of the right submandibular area (illustration is provided by A. K. Korsak)

Treatment. The main method of treatment of acute purulent lymphadenitis is the surgical one. It is performed in the hospital under the general anesthesia. Surgeon carries out primary surgical treatment of purulent foci, antiseptic washing and drainage of the wound. Complex medication treatment (antiinflammatory, antibacterial, desensitizing and physiotherapeutic) is also prescribed.

CHRONIC HYPERPLASTIC ODONTOGENIC LYMPHADENITIS

Chronic odontogenic lymphadenitis is rarely observed in children.

The patient complaints of the presence of a lump for a long time. It does not cause any discomfort to a child.

Clinical examination. The general condition of the patients is satisfactory, the body temperature is normal or subfebrile. The lymph node is enlarged

sometimes up to 3–4 cm in diameter (Fig. 2). It is round or oval with clear borders and mobile. The consistence is dense, the palpation is painless. The skin over the enlarged node has ordinary color, folds freely. During the oral cavity examination the causative tooth with pain in anamnesis is often revealed.

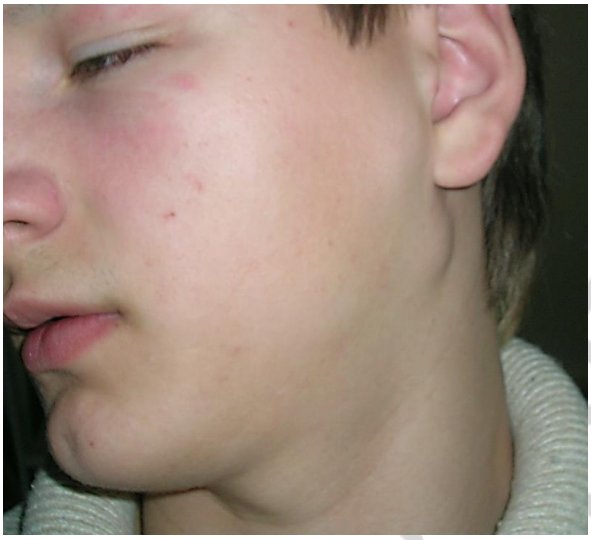


Figure. 2 Chronic hyperplastic lymphadenitis of the left submandibular region (illustration is provided by A. K. Korsak)

The treatment also begins with sanitation of the primary site of the infection. Complex medication treatment (anti-inflammatory, antibacterial, desensitizing and physiotherapeutic) is also prescribed.

CHRONIC ABSCESSED LYMPHADENITIS

The disease occurs within 3–4 weeks without treatment.

General condition of a child is satisfactory, the body temperature is normal. *Clinical course:* the skin over the enlarged node is hyperemic, cyanotic and thin. Affected lymph node is enlarged, painful and has clear borders. Fluctuation is present in the center of the lymph node during palpation. These are the symptoms of the lymph node abscess. Long-term abscesses can spontaneously be opened forming fistulas with purulent discharge.

The treatment of chronic abscessed lymphadenitis is the same one, but with adding the surgical method of the purulent focus.

ABSCESSES AND PHLEGMONS OF THE HEAD AND NECK

Abscess is a limited inflammation of the soft tissues with formation of the cavity with pus. It occurs as a result of the spread of the infection by lymphogenous, hematogenous pathways or along the length. There are subperiosteal abscesses (at periostitis, osteomyelitis) and abscesses of soft tissues of different localization. Abscesses can also be odontogenic and non-odontogenic.

Phlegmon is a diffuse purulent inflammation of the subcutaneous, intramuscular, intrafascial and other tissues without clear borders. There are several types of phlegmons: adenophlegmon — develops after suppuration of the lymph

nodes, osteophlegmon is a complication of osteomyelitis of the jaws and phlegmon, developing after the introduction of foreign bodies, due to trauma, etc.

The treatment of abscesses and phlegmon is emergency surgical and conservative. It is done in the hospital under the general anesthesia. The most important component of treatment is the primary surgical treatment of a purulent focus. It can be carried out by both intraoral and extraoral access.

Steps of primary surgical treatment:

1. Incision of the skin (mucosa), subcutaneous tissue and surface fascia.
2. Opening the purulent focus.
3. Taking exudate for microbiological research and determining sensitivity to antibiotics.
5. Washing the wound with solutions of antiseptics (3 % hydrogen peroxide, furagin 1 : 13 000, chlorhexidine 1 : 400 and 0.1 % iodinol).
6. Drainage of purulent focus (rubber strip, gauze with magnesium, sodium chloride and tubular, vacuum drains).

ACTINOMYCOSIS

Actinomycosis is a chronic specific infectious disease that develops as a result of the introduction of radiant fungi (actinomycetes) into the tissue. The disease is characterized by the formation in tissues *specific granulomas*, clinically manifested by limited or diffuse inflammatory infiltrates.

Etiology and pathogenesis. The causative agent of actinomycosis is a radiant fungus, or *actinomycete*. It is a representative of the normal microflora of the oral cavity. Actinomycetes are found in dental plaque, carious teeth, periodontal pockets, tonsils, etc.

In children actinomycosis often occurs in immunopathological conditions or as a result of decreasing the immunity after previous diseases. The ways of invasion of the actinomycosis infection are chronic odontogenic foci, oral mucosa, face and neck skin. From the place of invasion, actinomycetes are spread by contact, lymphogenous and hematogenous ways.

Clinical features. The clinical course of actinomycosis is predominantly chronic. Actinomycosis often develops in connective tissues, but in children the lymph nodes of the face and neck, the periosteum and bone of the lower jaw are mostly affected.

The subcutaneous form of actinomycosis is often localized in the buccal region near the odontogenic focus — the source of infection (Fig. 3). The disease begins with the appearance of a limited dense rounded infiltrate of subcutaneous fat with bluish skin above it — an actinomycotic granuloma. On palpation fluctuation is noted, however, when it is opened, pus is not released, only granulations occur. In the subsequent wound gradually scarring begins. However, new infiltrates appear next to it, in which the process spreads to adjacent areas of subcutaneous tissue — a symptom of quilt. Diagnosis is based on a typical clinical picture, biopsy, bacteriascopy.

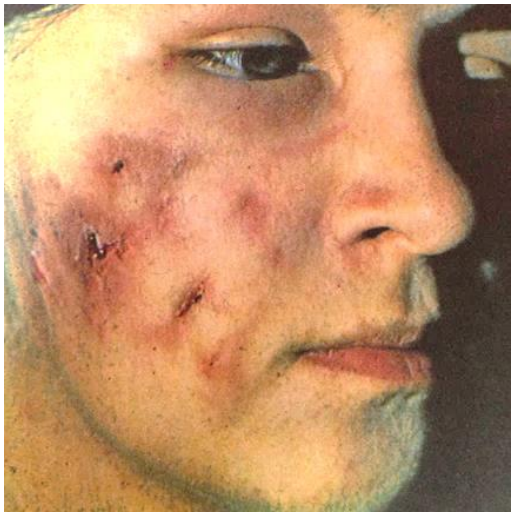


Figure. 3 Actinomycosis of the subcutaneous fat of the face

Treatment. Actinomycosis requires a long-term treatment (from 6 weeks to 12 months) with antibiotics: high dose of intravenous penicillin — 4 weeks; oral penicillin — 3 to 6 month. Other antimicrobial drugs — tetracycline, erythromycin and clindamycin — can be used in penicillin-allergic patients. In cases of abscesses, surgery is performed to drainage the abscesses.

INFLAMMATORY DISEASES OF SALIVARY GLANDS IN CHILDREN. ETIOLOGY, CLINICAL MANIFESTATIONS, DIAGNOSIS, TREATMENT

Saliva is produced by the three pairs of major salivary glands — parotid, submandibular and sublingual glands as well as many hundreds of small salivary glands — labial, buccal, lingual, palatal and the floor of the oral cavity which are localized in the mucosa. Any salivary gland consists of 3 parts: the parenchyma, interstitium and duct system. The majority of the diseases of salivary glands affect the *parotid or submandibular gland*, sublingual and minor salivary glands are less affected.

Classification of inflammatory diseases of the salivary glands. All sialadenites are divided:

1. According to etiology:

– bacterial;

– viral.

2. The nature of the inflammation:

– acute (epidemic and non-epidemic);

– chronic non-specific (parenchymal, interstitial, and sialodochitis);

– chronic specific (actinomycosis, syphilis, tuberculosis);

– calculous sialadenitis.

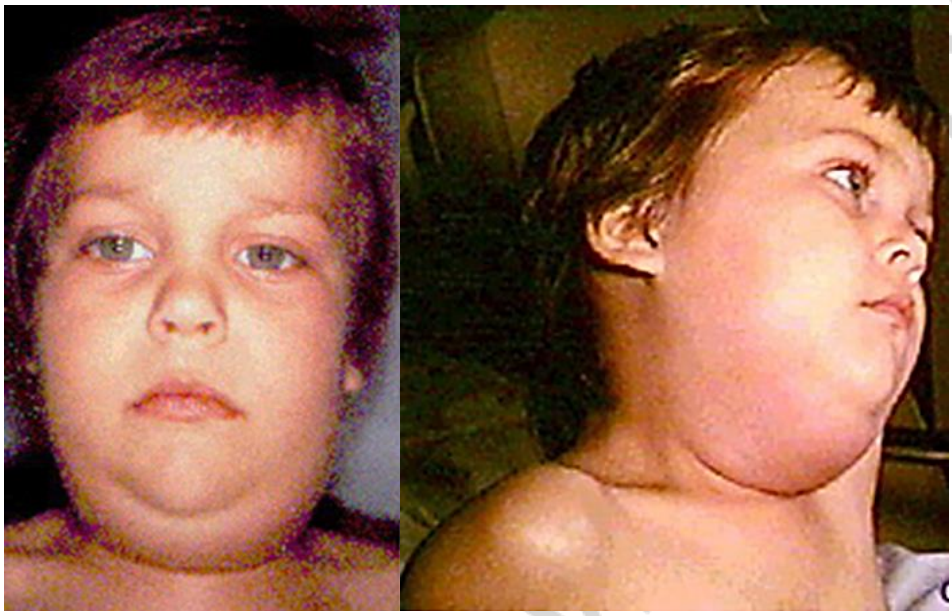
3. Stages:

– remission;

– exacerbation.

MUMPS (EPIDEMIC PAROTITIS)

Mumps is an acute infectious disease. It is a viral disease, caused by the Paramixovirus. Before the development and introduction of vaccination it was a common childhood disease all over the world. In 90 % of cases, *parotid salivary glands* are affected (Fig. 4). Infection spreads through airborne droplets. More often children from 7 to 10 years get mumps.



a ***b***

Figure 4 Appearance of a patient with mumps:
a — front view; *b* — side view

Clinical features. The patient usually complains of a headache, chills, soreness in the neck, joints and muscles. Body temperature increases to 38–39, loss of appetite, behavior changes appear. Parotid salivary glands most often are enlarged from the both sides. Glands are elastic, soft and painful during palpation. An enlarged and painful parotid gland knocks out the earlobe, sometimes narrows the external auditory canal. The salivation is decreased or stopped — dry mouth occurs. There is an increased level of amylase in biochemical blood test and diastase in urine.

*Common complication*s of mumps are orchitis, mastitis, changes in the pancreatic gland (pancreatitis).

The treatment of mumps is symptomatic. The patient is placed in bed for 7–10 days. Diet should stimulate secretion of saliva (sour drink, hard food, etc.). The patient must also maintain oral hygiene (mouth rinsing with antiseptics, irrigation). Warming dry compresses, UHF, gland massage and abundant drinking are prescribed. Healthy children should be isolated from the sick ones during 21 day from the onset of the disease.

Prevention of mumps includes active immunization with alive vaccine. The MMR (mumps, measles and rubella) vaccine is administrated to children in subcutaneous dose at the age of 12 month and then at 4–6 years old.

ACUTE NON-EPIDEMIC (BACTERIAL) SIALADENITIS

The causes of the development of acute inflammation of the salivary glands can be local and general.

Local causes are:

– injury of the gland;

– foreign body in the duct;

– oral reasons (stomatitis, gingivitis, dental plaque, complicated forms of caries, etc.).

Common causes include previous severe infectious diseases that can lead to the decreasing the immune forces of the body. Acute non-epidemic sialadenitis may occur after abdominal or genital surgery, when salivation is reflexively reduced, as well as in severe diseases of the cardiovascular system and oncological diseases.

Acute sialadenitis is clinically subdivided into ***serous*** and ***purulent*** forms.

Patients complain of spontaneous pain in the lateral area of the face (on the side of the gland lesion), an increase of the body temperature, etc.

Clinical features. Parotid gland is swollen and painful during palpation in the *serous* form. The mucous membrane in the area of parotid excretory duct is hyperaemic and swollen. Small amount of muddy, thick, viscous saliva appears after the massage of the parotid gland.

In *purulent* sialadenitis general condition of the child becomes worse. Locally there is purulent discharge from the parotid excretory duct in the mouth, hyperaemia of the skin of the parotid area. But if the disease progresses, purulent sialadenitis may transform into the phlegmon of the parotid region. Blood test reveals leukocytosis, increased number of neutrophils.

The treatment of acute sialadenitis is complex. First of all it is the treatment of the causative disease. Then the efforts of the doctor are directed to the normalization of salivation: washing the excretory ducts of the gland with antiseptic solutions or antibiotics, vacuum drainage, gentle massage of the gland. After normalization of salivation — diet, that stimulates salivation, and physiotherapy. Purulent sialadenitis requires the same treatment like any other purulent inflammatory process. The phlegmon of the parotid region is treated by surgery and medicamentous therapy.

CHRONIC PARENCHYMAL SIALADENITIS

The *etiology* of the disease is not clear. *Predisposing factors* are previous viral disease of the salivary gland (influenza, mumps, etc.), congenital pathology of the glands — polycystic gland (due to saliva retention), diabetes and another endocrine disease, decreased immunity. Parotid gland is usually affected.

Clinical picture of chronic parenchymal sialadenitis consists of exacerbation and remission.

Exacerbation occurs more often in cold seasons. The patient complains of a severe or moderate pain in the affected parotid gland. The body temperature

rises. The face of the child becomes asymmetrical. The affected parotid gland is dense and tuberous on palpation. Pus or muddy, viscous, bitter-salty saliva with lumps of mucus and pus is secreted during a massage through the stenon's main excretory duct. Duct foramen is wide and hyperemic (the symptom of "poppy seed" (Fig. 5)).

On contrasting sialograms and pantomosialograms (carried out in remission), the parenchyma of the gland is not clearly visible, a large number of small cavities (2–3 mm) are found. Ducts of II–V order are determined or intermittent. This X-ray picture is called the symptom of "grapes" (Fig. 6).

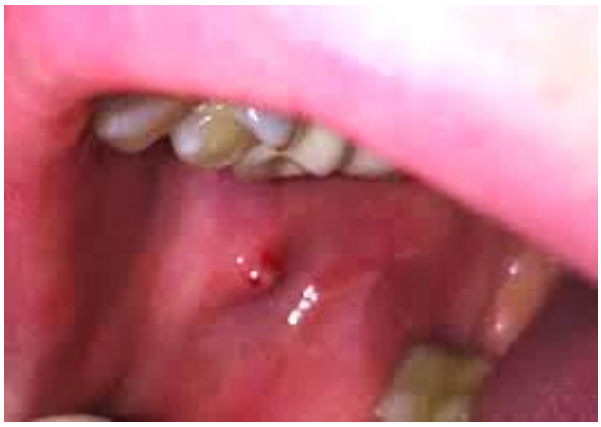


Figure 5. Symptom of "poppy seed" (illustration is provided by A. K. Korsak)

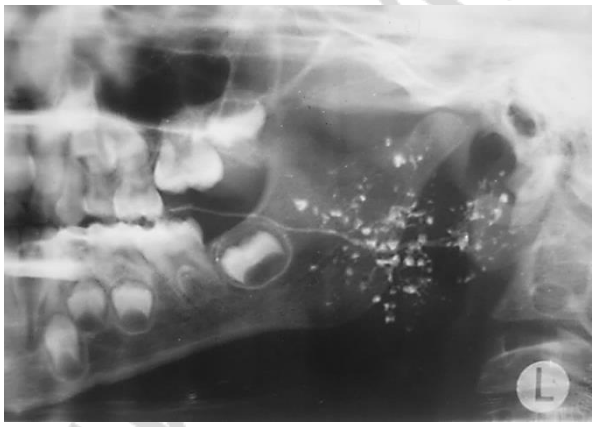


Figure 6. Symptom of "grapes" (illustration is provided by A. K. Korsak)

Chronic parenchymal sialadenitis in ***remission*** is characterized by an asymptomatic course. During examination, there are no changes in the region of the parotid glands, the mouth is opened well, from the parotid duct glands a sufficient amount of transparent saliva is secreted. However, in the morning saliva can be muddy and bitter-salty. This contrast sialography reveals the symptom of "grapes".

Treatment. Exacerbation requires washing the gland through the duct with antiseptic solutions, the massage of the gland. Furatsilin-novocaine mixture is used for normalization of trophic processes in the gland. 3 % aqueous solution of potassium iodide intraoral and by electrophoresis can be used. Physical methods of treatment: UHF, potassium iodide electrophoresis, helium-neon laser are also prescribed, but only after the normalization of salivation.

During *remission*, the main task is to prevent exacerbations:

1. Elimination of chronic foci of inflammation, sanitation of oral cavity, maintaining good oral hygiene.
2. Prescription of vitamin C, multivitamins to increase the body's resistance.
3. Gland blokage with furatsilin-novocaine mixture № 8–10.
4. Full-fledged diet, including proteins, vitamins, mineral salts and substances, that promote salivation.
5. Massage the gland in the morning within 10 minutes.

Parenchymatous parotitis should be differentiated from epidemic parotitis, lymphadenitis and tumor.

SALIVARY STONE DISEASE (SIALOLITHIASIS)

It is characterized by the formation of stones in the ducts of the salivary glands. The etiology of the disease is unknown. It is believed that the occurrence of salivary stone disease is associated with impaired mineral metabolism. Submandibular salivary glands are more often affected.

Clinical features. In the initial stage of salivary stone disease, the process is asymptomatic and the stone is detected accidentally. The first clinical sign is the retention of saliva. The patient complains of pain in the salivary gland and its enlargement during eating or thinking food (salivary colic).

The affected salivary gland is enlarged, soft and painful on palpation. If the stone is located in the gland, bimanual palpation along the mandibular duct will reveal induration — a stone. Mucosa around the duct entrance is hyperemic, if the stone is situated in the frontal or middle part of the duct (Fig. 7). Later symptoms of chronic sialadenitis join the disease. X-ray examination (especially X-ray of the mouth floor) is helpful for making the diagnosis (Fig. 8). But stones are not always clearly visible on X-ray. Defect of the filling is present on the contrast sialography.

Treatment. In exacerbation of sialadenitis and the localization of the stone in the excretory duct, the removal of salivary stone and subsequent treatment of the exacerbation of chronic sialadenitis is indicated. Stones in the anterior part of the submandibular duct can be removed by opening the duct, but if the stone is further back in the duct, or in the submandibular gland, then it is safer to remove it externally by a neck incision to avoid the damage to the lingual nerve. Extirpation of the submandibular gland is carried out if the stone is localized in the gland and in frequent exacerbations.

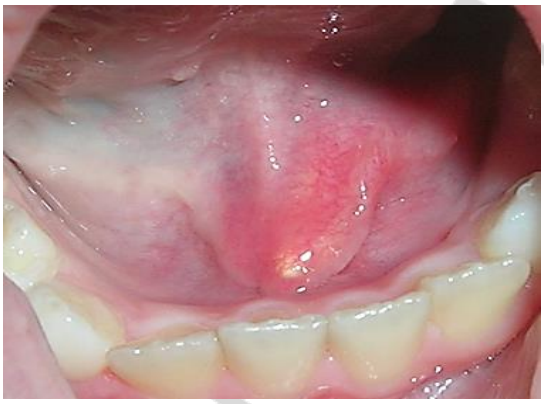


Figure 7. Sialolithiasis of the left submandibular gland, clinical view (illustration is provided by A. K. Korsak)

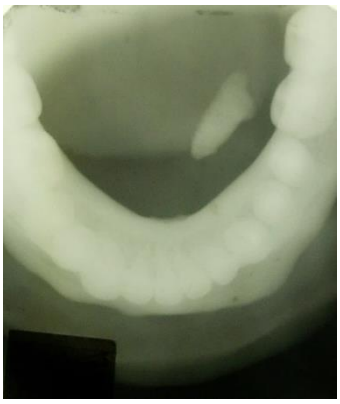


Figure 8. Sialolithiasis of the left submandibular gland, X-ray picture (illustration is provided by A. K. Korsak)

DISORDERS OF THE TEMPOROMANDIBULAR JOINT IN CHILDREN. ETIOLOGY, CLINICAL MANIFESTATIONS, DIAGNOSIS, TREATMENT

In different age periods, the TMJ has its own structural features, which often determine the clinical picture and the outcome of the disease.

In the **newborn**, the articular fossa is weakly expressed, flat, the articular tubercle of temporal bone is only marked. The head of condylar process is round, covered with thick layer of fibrous connective tissue. The articular disc (meniscus) is not formed yet and consists of connective tissue. In the neonatal period the lower jaw is located distally (infant retrogeny). Inflammation or injury rapidly destroys thin connective tissue of the joint, "nake" the bone and articular surfaces become fuse together.

Eruption of temporary teeth causes the first physiological bite increasing. The condylar head moves anteriorly. The anatomical elements of the joint become more pronounced (articular tubercle, fossa, condylar head and articular disc). Articular fossa deepens. The articular disc acquires a biconcave shape. Articular surfaces are covered with a thin connective cartilage, which is also quickly destroyed by trauma or inflammation and replaced by connective or bone tissue.

The second physiological bite increase occurs after ***eruption of the first permanent molars***. Mandible displaces medially and the head of the condylar process is situated in the center of the articular fossa. All anatomical joint elements are well expressed. Subsequently (after the eruption of the permanent canines), the occlusion is further increased and the mesial shift of the mandible progresses. The articular head is located at the base of the slope of the articular tubercle. By 12–14 years, all TMJ structures are completely formed.

Classification of the temporomandibular joint diseases:

1. Inflammatory diseases of the joint — arthritis (acute, chronic):
– Infectious;
– Non-infectious;
– Traumatic.
2. Arthrosis (osteoarthritis);
3. Bone ankylosis of TMJ;
4. Functional diseases of the TMJ:
– Junior dysfunction;
– Arthritis acute and chronic;
– Youth deforming arthrosis, etc.

ARTHRITIS OF TEMPOROMANDIBULAR JOINT

Infectious arthritis is divided into acute ***bacterial (septic)*** and ***reactive (aseptic)***.

Acute bacterial arthritis can be caused by a specific and non-specific infection. The infection can spread into the temporomandibular joint by hematogenous way from the primary focus (salmonellosis, gonorrhea, tonsillitis,

tuberculosis, brucellosis, mumps, rubella, etc.) or by contact way (over at distance) in osteomyelitis of the temporal bone, purulent inflammation of the middle ear, phlegmon of the parotid-masticatory region, etc. Direct infection of the temporomandibular joint can occur in case of joint puncturing or in case of trauma (out-and intra-articular fractures of the condyle).

Reactive and post-infectious arthritis is a group of inflammatory diseases of the joints in which microbial infection of the child's body is established, but neither the pathogen nor its antigens have been identified in the affected joint. Arthritis occurs simultaneously with an infection (reactive) or after 1–2 weeks (post-infectious). Dysentery, chlamydia, salmonellosis, syphilis, enteritis, viral hepatitis, etc. are the most often accompanied by such arthritis.

The clinical picture of infectious arthritis of the TMJ in children depends on etiology and mechanism of the disease. Arthritis in the first days is characterized by pain, restriction of the movement of the mandible, swelling and hyperaemia of the soft tissues, in case of injury — a violation of the bite, etc. Further, the clinical features depend on the main (causal) disease. *Treatment* of infectious arthritis of the TMJ in children is, as a rule, complex treatment of the underlying disease.

The clinical picture of reactive and post-infectious arthritis is also determined by the underlying disease, but aseptic arthritis flows more easily and without suppuration in the joint and therefore does not require surgical treatment.

The clinical picture of **non-infectious arthritis of the TMJ** depends on the underlying disease (rheumatism, systemic lupus erythematosus, hemophilia, etc.). There may be pain in the area of the joint, swelling, moderate skin hyperaemia, restriction of mobility of the lower jaw, pain in the masticatory muscles, fever, etc. The tasks of the dentist usually are: to identify the pathology of the TMJ, eliminate chronic joint microtrauma and sanitation of the oral cavity.

ARTHROSIS (FIBROUS ANKYLOSIS) OF TEMPOROMANDIBULAR JOINT

It is a chronic inflammatory disease of the joint, characterized by a combination of inflammatory, destructive and hyperplastic processes occurring in articular bones and cartilage (Fig. 9). Arthrosis occurs mainly in younger children after injury or inflammation (hematogenous or odontogenic osteomyelitis of the condyle, otitis media, fracture of the condylar process, etc.).

The area of the longitudinal growth of the mandible is located in the area of the condylar process, that's why the growth of this jaw slows down or stops completely, and an excessive bone formation occurs in the region of the neck of the condyle.

Clinical features. Arthrosis is characterized by limited mouth opening, delayed longitudinal growth of the lower jaw on the affected side with corresponding symptoms. Chin is shifted to the "sore" side, oblique or cross bite is in the oral cavity (Fig. 10).

On the X-ray joint gap is straight, the head is flat, the neck is thick and wide. The condylar process is shortened, osteosclerosis presents (Fig. 11).

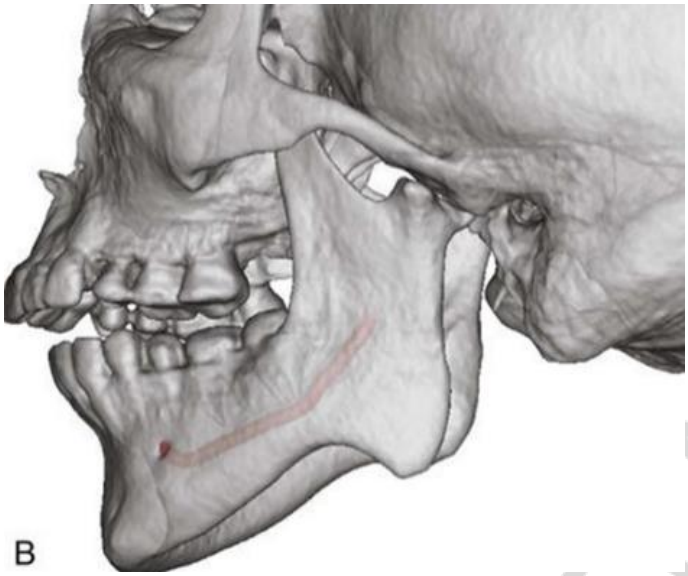
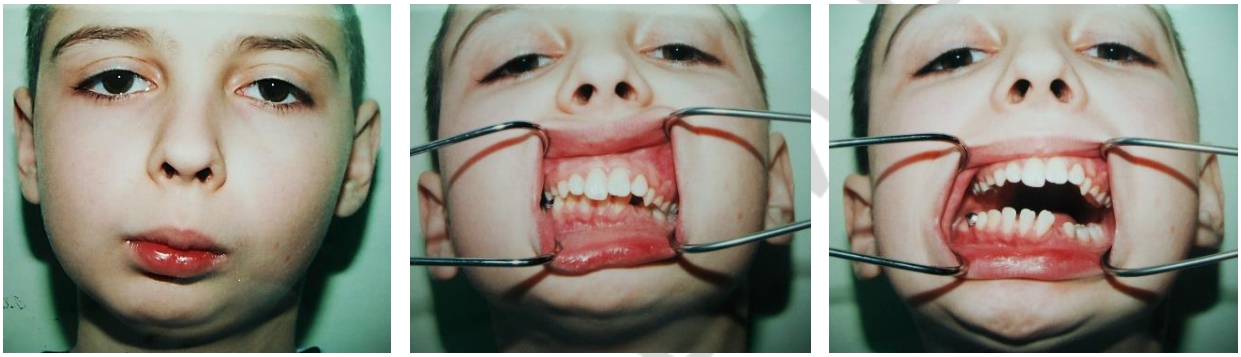


Figure 9. Osteoarthrosis of the temporomandibular joint



a ***b*** ***c***

Figure 10. Fibrous ankylosis of the right TMJ:
a — patient appearance; *b* — central occlusion; *c* — limited mouth opening (illustration is provided by A. K. Korsak)

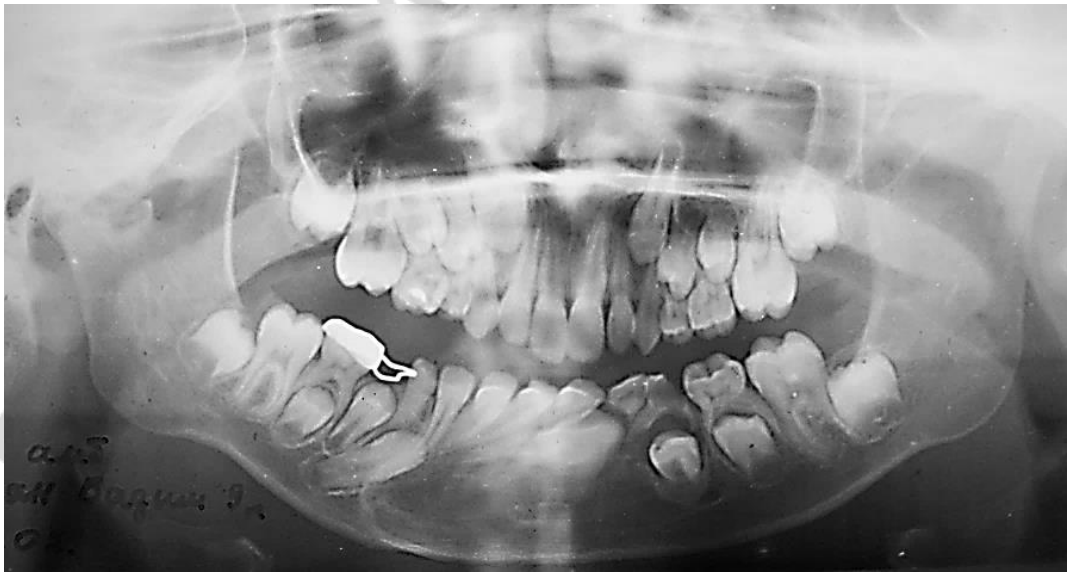


Figure 11. Orthopantomogram of the patient with the right TMJ fibrous ankylosis (illustration is provided by A. K. Korsak)

The treatment of children is complex and depends on the duration of the disease, its stabilization or progression, the age of the child, etc. Indications for surgery in arthrosis are: inability to eat in a natural way (vertical gap between the frontal teeth is less than 5 mm), violation of the functions of breathing and sleep, increasing reflected deformity of the upper jaw.

The purpose of the operation is creation of a false joint, normalization of the position of mandible and, possibly, osteoplasty with the elements of arthroplasty. After the surgery all children need active mechanical movements to form a false joint, as well as orthodontic treatment until the end of the growth of the facial skeleton.

BONE ANKYLOSIS OF THE TEMPOROMANDIBULAR JOINT

The *etiology and pathogenesis* of osteal ankylosis of the TMJ in children are the same as in osteoarthritis. Very often, arthrosis precedes bone ankylosis.

The clinical picture of ankylosis is similar to osteoarthrosis. However, in bone ankylosis, the patient cannot open the mouth at all, because mandible is immobile. X-ray reveals that joint elements are absent, bone growths occupy the joint space, articular cavities, etc.

Respiratory dysfunction occurs in the early stages of the disease, especially during sleep. The distal position of the mandible leads to dislocation of the tongue while sleeping with the development of dislocation asphyxia. Impaired respiratory function often requires an emergency mandibular surgery or a tracheotomy.

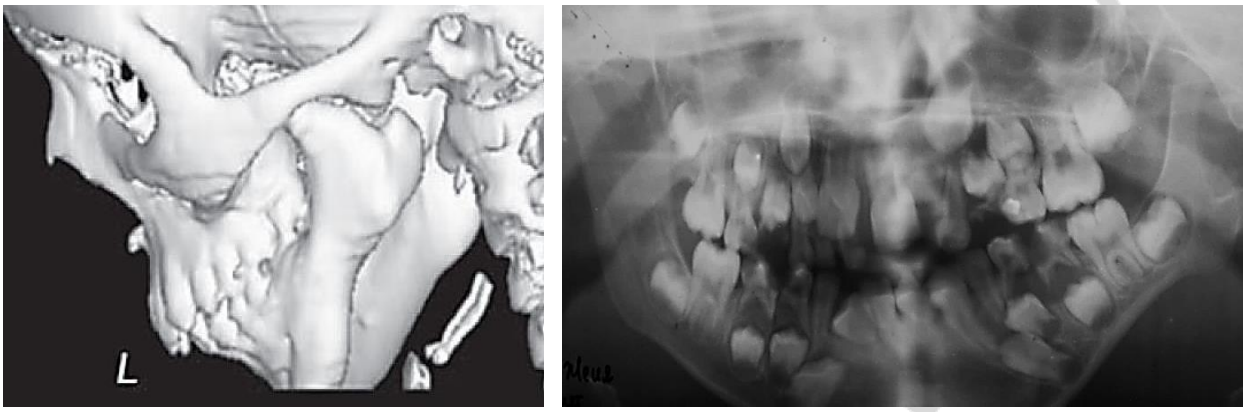
The treatment of bone ankylosis begins with a surgical intervention (regardless of the age of the child), the purpose of which is to create a false joint. In the future, orthodontic treatment is carried out to 15–16 years.

a ***b*** ***c***

Figure 12. Bilateral ankylosis of the temporomandibular joint (illustration is provided by A. K. Korsak):
a — “bird” face, front view; *b* — “bird” face, side view; *c* — CT

NEOARTHROSIS

Neoarthrosis, or pseudarthrosis (near or in the area of TMJ), is clinically characterized by asymmetry of the face, malocclusion, and limited or normal mouth opening. Radiographically, there are all signs of neoarthrosis: the presence of a gap between the bone fragments, the closure of the cortical bone plate, etc. (Fig. 13).



a ***b***

Figure 13. Neoarthrosis of temporomandibular joint on the right:
a — CT; *b* — orthopantomogram

The treatment of children with neoarthrosis of temporomandibular joint should begin immediately after diagnosis and includes orthodontic treatment until the end of the growth of facial bones. In the future, after 17 years, facial contour or osteoplasty of the lower jaw may be performed.

FUNCTIONAL DISEASES OF THE TMJ

Temporomandibular joint dysfunction is associated with the restructure of the skeleton and disproportion of its growth. Adolescences have intense bone growth, which is significantly ahead of the growth of condylar process and ligaments of the temporomandibular joint. In such a situation, an increased or normal load on the joint leads to overload and stretching of the ligamentous apparatus, chronic injury of the joint surfaces of the temporomandibular joint.

Youth dysfunction of TMJ. It is clinically characterized by the following symptoms: excessive opening of the mouth, which may be accompanied by the dislocation and subluxation of the lower jaw (habitual dislocation); crunching and clicking in the joints; possible pain; S-shaped movements of the lower jaw with maximum opening of the mouth due to asynchronous “work” of both TMJ, etc. Traumatic factor (hit to chin, dislocation of the lower jaw, etc.) is often present in anamnesis. Radiographically pathological changes are not detected in juvenile dysfunction of the TMJ.

The treatment of such dysfunction should begin with measures, that make rest to the joint: patient should not eat solid food, open mouth widely, avoid traumas of the lower jaw (sports), sometimes use a bandage, etc. Physiotherapy of the disease is carried out simultaneously: UHF, paraffin therapy, electrophoresis

with novocaine, potassium iodide, phonophoresis with hydrocortisone preparations, etc.

DISLOCATION OF THE TEMPOROMANDIBULAR JOINT

Dislocation is a complete persistent displacement of the articular ends of the bones. Traumatic dislocations of the TMJ can be divided into acute and chronic (habitual dislocation). Acute traumatic dislocations of the TMJ are divided into:

– unilateral and bilateral;

– anterior, posterior, lateral.

Anterior acute traumatic dislocations of the temporomandibular joint occur most often. Bilateral dislocation usually appears as a result of the hit to the mandible when shout, gapes, removing or treating a tooth, etc. Clinical features are: the mouth is open and the patient cannot close it on his own. In front of the auricles the retraction is determined, and the heads of the condyle processes are palpated under the zygomatic arch. Radiographically, the head of the condylar process is situated forward to articular tubercle, bone fractures are absent.

The treatment consists of reducing the dislocation of the TMJ. After anesthesia, the surgeon places his thumbs on the lower chewing teeth and presses downward and then posteriorly. It should be done very carefully as the patient can traumatize the surgeon's fingers. Then immobilization of the mandible is done for 7–10 days to prevent further stretching of the ligaments. On the first day after injury, cold is prescribed according to the scheme. Subsequently, physiotherapy (thermal procedures, etc.) are prescribed for the rapid relief of the inflammatory process.

TRAUMATIC INJURIES OF SOFT TISSUES AND TEETH IN CHILDREN. CLINICAL MANIFESTATIONS, DIAGNOSIS, EMERGENCY CARE AND TREATMENT

Trauma is an injury, caused by extrinsic agents, which violates the anatomical integrity and physiological functions of organs and tissues.

Facial soft tissue injuries are usual phenomenon in today's society. Motor vehicle accidents, accidental injuries, and falls comprise the majority of cases for complex facial wounds. The problem of injuries in maxillofacial area continues to be one of actual problems of maxillofacial surgery and dentistry. Currently, about 30 % of patients with maxillofacial trauma are treated in maxillofacial hospitals.

Maxillofacial soft tissue injuries in children in most cases is associated with trauma in everyday life, less often with street and traffic injuries. Mechanical non-gunshot trauma of soft tissues most often occurs in pediatric surgery. Thermal and chemical burns of the soft tissues of the face and oral mucosa, as well as frostbite, are much less common and generally occur in children up to 3 years.

Traumas of the maxillofacial area can be divided by the ***mechanism of occurrence*** (nature of damaging factors) in:

– mechanical (non-gunshot and gunshot);

– thermal (burns, frostbite, electrical accident);

– chemical;
– radiation;
– combined.

MECHANICAL SOFT TISSUE INJURIES

Mechanical soft tissue injuries can be *closed* and *open*. Closed damage don't include damage to the skin and mucous membrane throughout the thickness. It includes *bruises, abrasion, extravasation and hematoma.* Open soft tissue damage is called a *wound.* It is a soft tissue injury with violation of the integrity of the skin or mucosa and with possible damage of underlying tissue.

Contusion (bruise) is a mechanical tissue damage without disturbing the anatomical integrity of the skin or mucosa. Despite the fact that younger children (up to 5 years old) often fall, due to the elasticity of the skin of the face, the abundance of subcutaneous fat, presence the fatty lump of Bish, as well as the small height and weight, they often get bruises of the face.

Edema is clinically determined in case of contusion of a soft tissue injury in children. It is due to the abundance of subcutaneous fat, hydrophilicity of soft tissues, etc. There may also be hemorrhages, i.e. the skin has a cyanotic color. Palpation of the area of the injury is painful.

Cold (a bubble with ice) is recommended in contusion of a soft tissue. It is applied through a napkin to the damaged area for 30 minutes, with interruptions for 40–60 minutes. Cold allows to reduce edema of soft tissues, pain sensitivity and prevents hemorrhage into soft tissues. After 3–4 days of the injury, thermal procedures, UHF-therapy, etc. are prescribed.

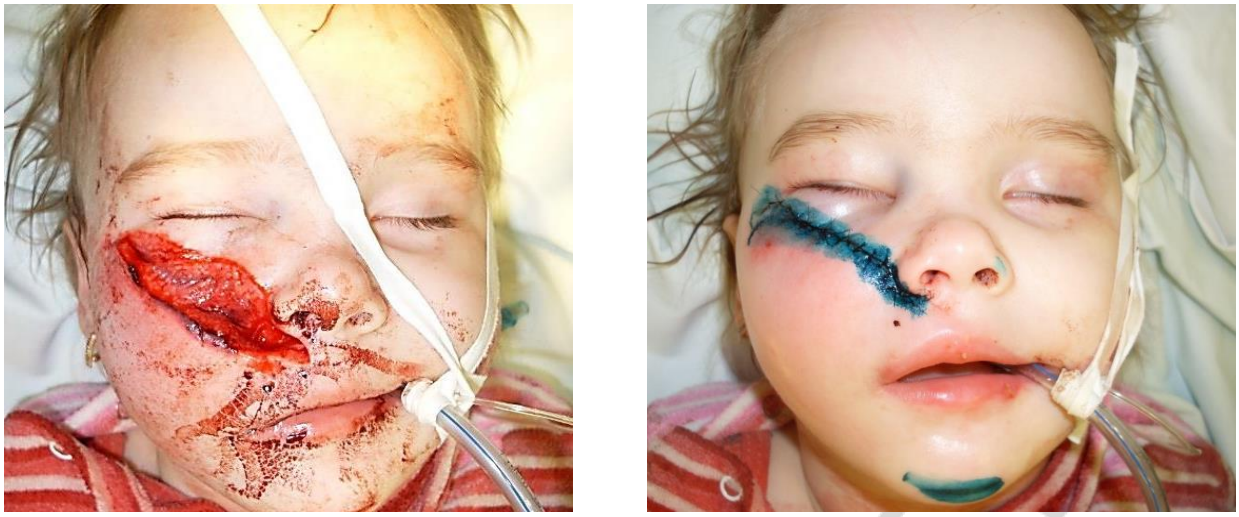
Contusions of the soft tissues of the maxillofacial area can be accompanied by formation of hematomas and / or interstitial hemorrhages.

Hematoma occurs due to the damage to blood vessels and separation of soft tissues with cavity formation. The most characteristic sign of hematoma of soft tissues is a symptom of fluctuation. The skin above the hematoma has a cyanotic color, the palpation of the tissues is painful. Hematomas of the soft tissues of the maxillofacial region can be independently reabsorbed forming an interstitial scar or suppurate.

The treatment of fresh hematomas of soft tissues of medium and large sizes includes: blood removing from the cavity with the syringe and application of a pressure bandage and cold (ice pack) on the damaged area. Physiotherapy procedures are prescribed after 3–4 days of the injury. In case of suppuration, hematoma should be opened and drained with further treatment, as in the treatment of any purulent-inflammatory process of soft tissues.

Hemorrhage (extravasation) is an impregnation (imbibition) of a soft tissue with blood (subcutaneous fat, muscles of the face, etc. Treatment of them practically does not differ from treatment of bruises of soft tissues.

Open soft tissue damage is called a ***wound***. It is a soft tissue injury with violation of the integrity of the skin or mucosa and with possible damage to the underlying tissue (Fig. 14).



a ***b***

Figure 14. Laceration of the upper lip, cheeks and molar area to the right:
a — preoperative view; *b* — postoperative view (illustration is provided by A. K. Korsak)

By the nature of tissue damage, wounds are divided into: punctured, chopped, cut, bruised, bitten, gunshot and others; wounds can also be penetrating and non-penetrating into the body cavities. In the maxillofacial area wounds can penetrate into the oral cavity, nasal cavity, maxillary sinus, etc. In addition, the wounds can be with or without damaging to the bones of the facial skeleton.

Features of wounds of the maxillofacial area in children

However, the wounds of the soft tissues of the face and oral cavity in children have their own anatomical, physiological and age peculiarities.

Soft tissues of the maxillofacial region have a good blood supply and innervation, which contributes to the high regeneration of tissues and their high resistance to development of the suppuration. Damage to soft tissues in children is accompanied by extensive swelling. Wounds of the maxillofacial area are characterized by excessive bleeding. It can be dangerous to a child's life, if large blood vessels are damaged. Wounds on children's faces always gape widely due to the facial muscle contractions, high elasticity of the skin and the presence of a thick layer of subcutaneous fat. Facial wounds in children are accompanied by severe pain and hypersalivation, which can lead to maceration of the facial skin, loss of fluid, etc. The wounds of the maxillofacial area are often heavily infected and contaminated by foreign bodies (earth, sand, etc.). It can cause their suppuration. Maxillofacial injuries frequently associated with damage to the upper respiratory tract, eyes, brain injury, and others.

Clinical picture of soft tissue injuries of the maxillofacial area in children doesn't often show a true size of the damage, and always seems to be more dangerous than actually is.

Features of surgical treatment of facial wounds

All wounds to the face and oral cavity can be tightly sutured in 48 hours, and under antibiotics cover — even in 72 hours after injury.

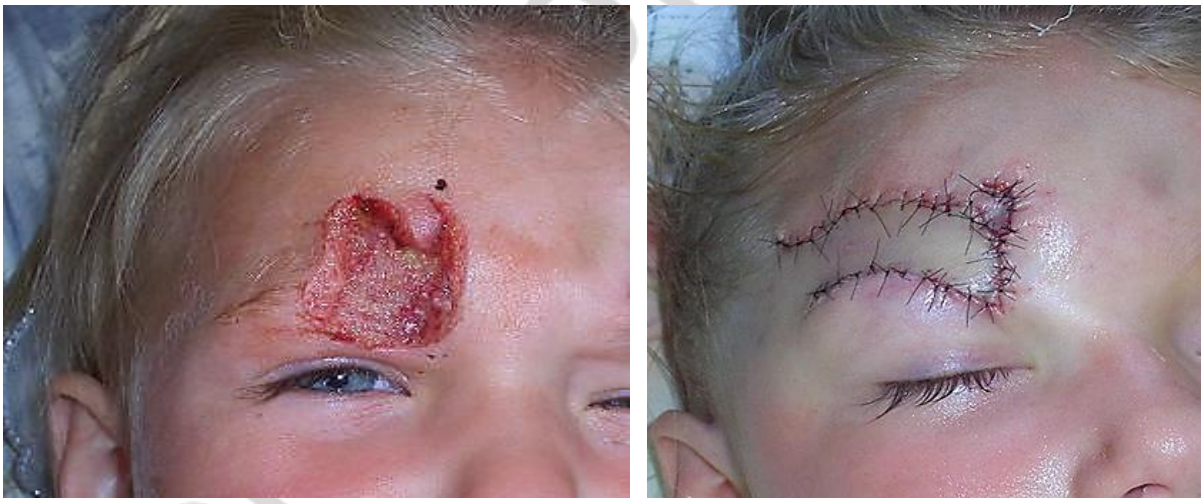
In primary surgical treatment of facial wounds, soft tissue should be carefully treated and only clear nonviable soft tissues are excised.

If maxillofacial wounds penetrate the oral cavity, nose, etc., first of all, they should be sutured from the side of the mucosa. It is done to prevent further infection of the tissues. Facial wounds should always be sutured in layers with suturing the mimic muscles and subcutaneous fat to obtain good cosmetic results. During primary surgical treatment wound margins on the face should be very carefully connected against the natural holes (red border of the lips, nose, etc.). Cutaneous wound margins should be well adapted and connected without tension. If the large branches of the facial nerve are damaged child should be consulted by neurosurgeon.

In simultaneous damage to the soft tissues of the face and fractures of the facial bones (or teeth), first primary surgical treatment of the bone fractures with fixation of the bone fragments is carried out. Secondly, primary surgical treatment of soft tissue wounds is performed.

Thin (6/0 or 5/0) monofilament suture with an atraumatic needle (nylon, polyester, ethylene, miralen, etc.) should be used to wounds of the facial skin. It allows to obtain a good cosmetic result. In many cases, it is possible to use cosmetic intracutaneous seam.

Soft tissue defect should be closed during initial surgical treatment of the wound. Tactics of the maxillofacial surgeon depends on the size of the soft tissue defect. Mobilization of wound margins is used to close small defects. Mattress or guide sutures can be used in medium-sized defects. Large defects can be closed by plasty of local tissues (Fig. 15).



a ***b***

Figure 15. Soft tissue defect plasty with local tissues:
a — preoperative view; *b* — postoperative view (illustration is provided by A. K. Korsak, V. I. Lapkowsky)

After removal of sutures, to get good cosmetic results the physiotherapy is prescribed: massage, paraffin therapy, electrophoresis or lidaza, hydrocortisone phonophoresis, laser therapy, magnetic therapy and others.

FACIAL BURNS

Facial burns account for approximately 2 % of all types of maxillofacial injury. They are relatively rare. Burns are mainly prevalent in young children. Depending on the causes of burn, they are divided into: thermal, chemical, radiation.

Thermal burns also include electric trauma.

Most face burns are caused by *thermal* factors. *Chemical* burns of the face are observed less frequently. Burns of the oral and pharyngeal mucosa are often caused by chemical factors, mainly acids and alkalis. *Radiation* injury of the face tissues in children in peacetime occur only after radial therapy of tumors of the maxillofacial region.

Thermal burns

Thermal burns are divided into 4 degrees according to the depth of tissue damage. The depth of damage is determined by the temperature of the damaging factor, the duration of its effect and the features of the structure of the skin in the area of the lesion.

Burns of the 1st degree are characterized by hyperemia of the skin, swelling of the tissues and pain. Only skin epidermis is affected. There is no appreciable scarring, only pigmentation of the affected skin areas is sometimes changed after them.

Burns of the second degree are characterized by deeper skin lesions of the papillary layer. The bubbles, filled with serous fluid are formed. The surface of the burn is usually epithelialized after 14–16 days.

Burns of the III A degree are characterized by necrosis of the tips or the entire papillary layer of the skin, but with the preservation of the sebaceous and sweat glands, as well as the hair follicles.

Burns of the III B degree are accompanied by necrosis of all layers of the skin. These burns heal by secondary tension, which leads to the formation of deforming scars.

Burns of the IV degree are accompanied by charring of the skin and necrosis of more deeply located tissues (muscles, bones, etc.). After burns of the III B–IV degrees, hypertrophic or keloid scars are formed.

Different depth of skin burns (from I to IV degree) forms uneven relief of the face on different areas of the face. The most protruding parts of the face are most deeply affected: nose, lips, zygomatic area, ears, chin.

The treatment of children with facial burns is performed in burn centers. And only children with isolated, superficial and small burns can be hospitalized in the department of maxillofacial surgery.

It begins with carrying out anti-shock measures, which include analgesics and applying an aseptic bandage. Antibacterial therapy is also carried out to prevent infection. After the initial surgical debridement, the ***open method*** of treatment is most acceptable, because the facial bandages quickly become soaked

with saliva, get dirty while eating and infected. However, a ***closed method*** of burn wounds treatment in children with the use of ointment dressings is also possible.

Chemical burns of the face and oral cavity

Chemical burns arise from the action of chemicals that can cause necrosis of tissues (acids, alkalis, etc.). Chemical burns (like thermal ones) are divided into ***4 degrees***, depending on the type and concentration of the chemical agent. However, the degree of chemical burns is very difficult to determine in the first hours and even days after trauma.

Strong acids and heavy metal salts lead to coagulation of proteins, that is *coagulative necrosis* of tissues. Alkalis cause *colliquative*, deep tissue necrosis.

Chemical burns of facial skin (lips, chin, etc.) and oral mucosa mainly occur in young children ***(up to 3 years)***, when domestic chemicals are entered into oral cavity of the child. Chemical burns of mucosa of the pharynx and esophagus occur in swallowing these chemicals.

Some chemicals due to the resorptive action can additionally cause *general poisoning* of the body. The pain syndrome appears later and is not so severe as in case of thermal burns. The *local clinical picture* depends on the type of chemical agent that caused the burn. So, alkalis dissolve the surface layers of the mucous membrane of the mouth.

The mucous membrane of the oral cavity becomes green in burns with hydrochloric acid, nitric acid changes color of mucosa to greyish-brown, and sulfuric acid — to black. Crystals of manganese-acid potassium stain mucous membrane of the mouth in black-brown color. There is a specific odor from the mouth, if a child takes concentrated acetic acid (30 %) or ammonia in the oral cavity. It is very important to know these symptoms of chemical burns to carry out rational medical care to a child. The oral mucosa in chemical burns is edematic and hyperemic, regardless of the type of the chemical substance. Areas of mucosal necrosis (in deep burns) are quickly covered with a thick fibrin film, under which epithelization of the wound occurs.

The treatment of chemical burns of the facial skin and mucosa of oral cavity begins with washing the skin and oral cavity with cold water. After clarifying the nature of the damaging factor, it is chemically neutralized. So, 1–2 % solution of baking soda is used in burns with acids. Weak (1–2 %) solutions of citric or acetic acids are used in case of alkalis burns. Further, the oral cavity is treated (rinsed) with a local anesthetic solution to reduce pain.

Further treatment of chemical burns in the hospital is the same as treatment of thermal one. Local anesthetics, weak antiseptics, keratoplastics, etc. are used for the treatment of the mucous membrane of the oral cavity.

FROSTBITE INJURY

Frostbite is a tissue damage caused by prolonged exposure to low temperature. Increased humidity, wind, and local and general circulatory disorders, that promote the development of frostbite. There are ***4 degrees*** of frostbite.

The degree (depth) of frostbite mainly depends on the time of low temperature action. Cold causes vasospasm, resulting in their thrombosis with a violation of tissue supply.

Frostbite of facial skin in children occurs relatively rare. The protruding parts of the face: nose, ears, cheeks, and chin are usually damaged.

The clinical picture of frostbite depends on the depth of tissue damage and the period of the disease. In the initial (pre-reactive) period the skin is pale, anesthesia or paresthesia, minor pain or tingling appear, tissues are cold, dense on touch. After the warming of frost-bitten tissues, a reactive period begins, when pain, swelling and cyanosis appear. In case of frostbite of the second degree, the epidermis is additionally exfoliated with the formation of blisters.

Frostbite of facial skin, especially in children, can also occur at temperatures above 0 °C, with high humidity and strong wind.

The treatment of frostbite begins with the elimination of low temperature. After that measures for quickly restoration of blood circulation in the affected tissues are taken. To do this, massage (rubbing) of the facial skin with a palm or soft cloth from the periphery to the center is performed. The facial skin is treated with alcohol and the blisters are opened in frostbite of 2 degree. After restoration of blood circulation, the frostbitten area is treated with alcohol, fish oil, synthomycin and other ointments. Subsequently physiotherapy procedures (UFO, UHF, etc.) are prescribed.

Prevention of skin frostbite in infants includes lubrication of their cheeks with a thin layer of fat before walking in the frosty days.

DENTAL INJURIES IN CHILDREN

Dental trauma is a violation of the anatomical integrity of the tooth or surrounding tissues, with a possible change in tooth position in the dentition.

Trauma of temporary teeth most often occurs at the age of 2–3 years, and permanent teeth injury — at 8–10 years. Boys have dental trauma 2 times more often than girls. The ***incisors*** are most often traumatized, mainly on the ***upper*** jaw.

Classification of dental injuries (N. M. Chuprinina, 1985)

1. Concussion of the tooth.

1.1. With the rupture of the neurovascular bundle (NVB).

1.2. Without breaking the NVB.

2. Luxation of the tooth.

2.1. A partial luxation.

2.1.1. With the rupture of the NVB.

2.1.2. Without breaking the NVB.

2.2. Avulsion.

2.3. Intrusive luxation.

3. Fracture of the tooth.

4. Trauma to the tooth germ.

5. Combined tooth trauma.

ICD-10-CM Diagnosis Codes (2017)

S00-T88 Injury, poisoning and certain other consequences of external causes
S00-S09 Injuries to the head

- S02 Fracture of the skull and facial bones
  - S02.5 Fracture of the tooth (traumatic)
    - S02.5XXA…… initial encounter for closed fracture
    - S02.5XXB…… initial encounter for open fracture
    - S02.5XXD…… subsequent encounter for fracture with routine healing
    - S02.5XXG…… subsequent encounter for fracture with delayed healing
    - S02.5XXK…… subsequent encounter for fracture with nonunion
      - S02/5XXS…… sequela
- S03 Dislocation and sprain of joints and ligaments of the head
  - S03.2 Dislocation of the tooth
    - S03.2XXA…… initial encounter
    - S03.2XXD…… subsequent encounter
    - S03. S03.2XXS…… sequela

Classification by Andreasen (1981)

1. Injuries to the dental hard tissue and pulp
   - crown infraction
   - crown fracture
     - uncomplicated crown fracture
       – enamel fracture
       – enamel dentin fracture without pulp exposure
     - complicated crown fracture
       – enamel dentin fracture with pulp exposure
2. Injuries to the dental hard tissue, pulp and to the alveolar process
   - crown root fracture
     - enamel dentin fracture
   - root fracture
     - dentin root fracture with pulp exposure
   - alveolar fracture in connection with tooth trauma
     - alveolar fracture in the maxilla
     - alveolar fracture in the mandible
3. Injuries to the periodontium
   - luxation
     - concussion (shock)
     - subluxation (loosening)
     - intrusion (central luxation)
     - extrusion (peripheral luxation)
     - lateral luxation
     - total luxation (exarticulation)

Concussion according to N. M. Chuprinina (it corresponds to concussion and subluxation according to Andreasen) is the easiest kind of dental trauma. In this case the periodontium is primarily damaged in the form of the rupture of a part of the fibers; damage to small blood vessels and nerves occurs. Teeth with *unformed roots* have the rupture of the NVB much less frequently than teeth with formed roots.

Clinical features of acute traumatic periodontitis occur in case of tooth concussion: toothache on biting, painful percussion, moderate tooth mobility. Moderate widening of the periodontal space can be also detected on the roentgenogram.

*Treatmen*t of tooth contusion begins with giving rest to the damaged tooth within 3–4 weeks. First of all, it can be achieved by the exception of solid food from the diet. In a permanent bite, you can temporarily disengage the bite using a kappa or uncoupling plate. Besides kappa or uncoupling plate can be used to temporary separation of the bite.

The dentist should also determine the pulp vitality of the damaged tooth. EDI should be carried out in 2–3 days after injury and then 1–2 times a week for 4 weeks. If signs of tooth pulp death are revealed, the endodontic treatment of permanent tooth should be done.

Dental dislocation is a damage to the ligamentous apparatus of the tooth, which leads to the displacement of the tooth from the socket and dentition. It often occurs as a result of hit on the tooth crown. Frontal upper teeth are usually affected. Dental dislocation can be divided in*: partial luxation (subluxation), avulsion and intrusion*.

In ***subluxation*** according to N. M. Chuprinina (corresponds to lateral luxation according to Andreasen), the tooth partially loses its connection with the tooth socket due to the rupture of periodontal fibers. Subluxed tooth becomes mobile, painful on percussion and dislocated. X-ray examination reveals widening of the periodontal space and “shortening” of the tooth root, if it is displaced orally or vestibularly.

Treatment. In a temporary bite, all the dislocated milk teeth must be extracted. Permanent subluxed teeth should be always preserved. Treatment begins with reposition and immobilization (fixation) of the dislocated teeth under local anesthesia.

Immobilization or fixation of teeth in children can be carried out in the following ways (Fig. 16):

1. Splint-brace. It is indicated in permanent occlusion with stable adjacent teeth. But this method is traumatic for periodontal tissues and labor-consuming for doctor.

2. Acrylic overlay splint and splint-kappa. It is usually made of plastic. Acrylic overlay splint and splint-kappa are used to fix teeth in a temporary, mixed and constant bite with insufficient number of adjacent teeth.

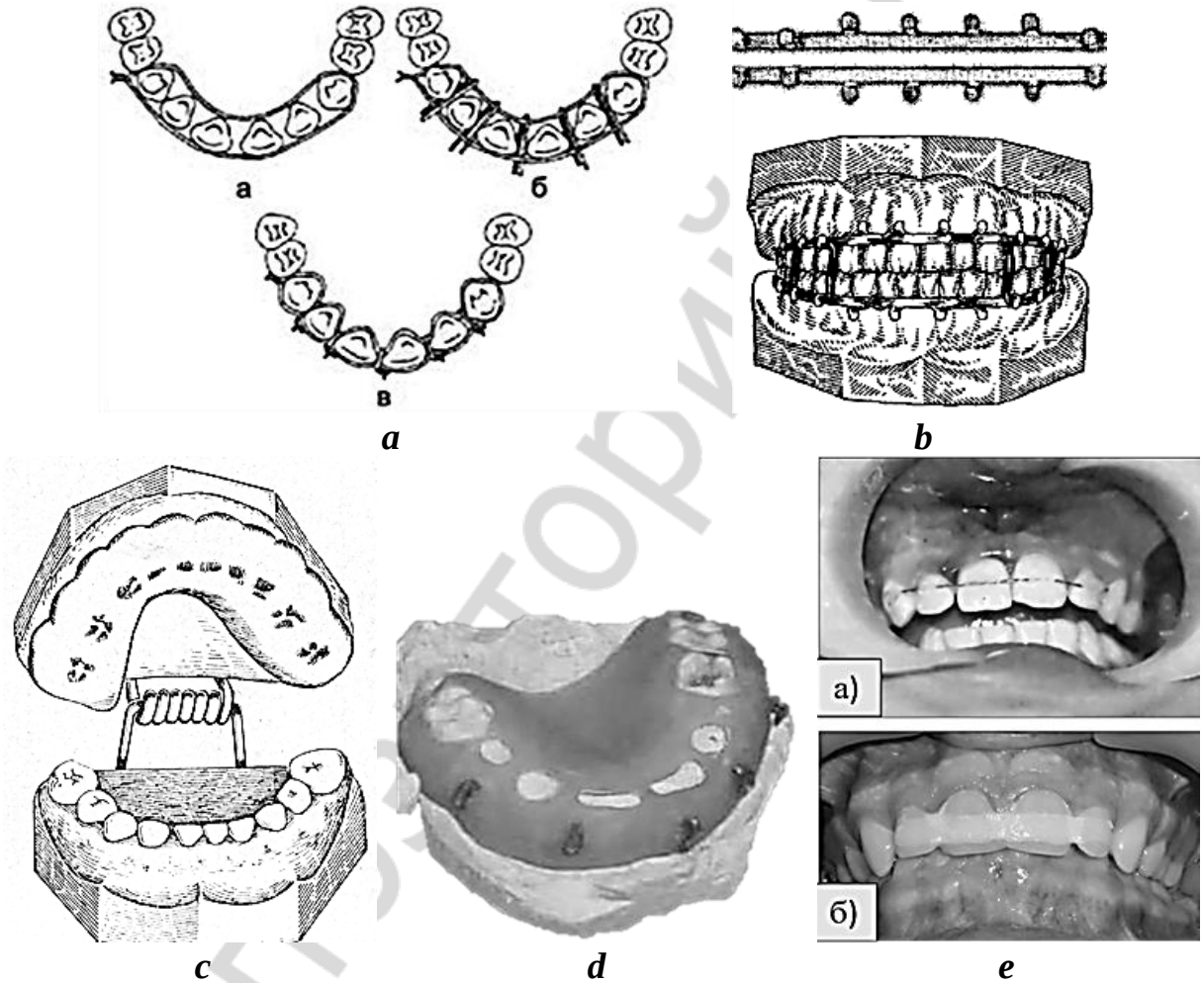
3. Composite materials are widely used for splinting nowadays. As a result wire arcs, or other splinting structures are fixed to teeth with composites.

Immobilization of the dislocated teeth is usually carried out during one month (4 weeks). The main task of a dentist after splinting the dislocated teeth is to determine the pulp vitality of the injured tooth. If the tooth pulp died, the endodontic treatment of the tooth should be done.

Avulsion according to N. M. Chuprinina (corresponds with total luxation according to Andreasen) means that the tooth loses its connection with the socket, falls out or is retained only by a circular ligament.

Intraoral examination reveals, that tooth is absent, it's socket bleed or filled with a fresh blood clot. Avulsed teeth often are brought in a "pocket" to the dentist.

The treatment of a complete dislocation begins with the assessment of indications and contraindications to ***tooth replantation***. It means returning the tooth to its own socket. It should be done as fast as possible.



a ***b*** ***c*** ***d*** ***e***

Figure 16. Methods of teeth fixation in children:
a — dental ligature binding; *b* — splint-brace; *c* — splint-kappa; *d* — acrylic overlay splint; *e* — composite splints

Contraindications to tooth replacement are the following:

1. Temporary teeth.
2. Permanent teeth with root fractures.
3. Destruction of the tooth socket.
4. Development of purulent inflammation in the tooth socket.

The operation of tooth replantation can be divided into the following stages:

1. Preparing the tooth for replantation.
2. Preparing the tooth socket for replantation.
3. Tooth replantation and its fixation in the socket.
4. Postoperative treatment and follow-up in dynamics.

In the first stage, the avulsed tooth is washed in physiological saline with antibiotics. During these manipulations, the tooth should be picked up only by its crown and not the root. This prevents damage to periodontal tissue on the root.

Preparation of the tooth socket for replantation includes removing the blood clot from the socket under local anesthesia and rinsing it with physiological saline with antibiotics. Then it is necessary to cause fresh bleeding, which promotes the regeneration of periodontium.

Endodontic treatment should be carried out in some days after replantation to minimize extra-socket time of the tooth and decrease possibility of root resorption or ankylosis in future.

In the third stage, the tooth is actually replanted. After that avulsed tooth should be fixed by any of the above methods for 4 weeks.

After 1–1.5 months after the tooth replacement surgery, the following ***types of tooth engraftment*** are possible:

1. Through the periodontium (syndesmosis).
2. By the type of synostosis (bone type) or root of the tooth grow together with the bone of the socket.
3. By mixed (periodontal-bone) type.

In a few years after tooth replantation resorption of the root of the implanted tooth may occur. The results of tooth replantation are most favorable, if no more than 1.5 hours have passed from the trauma.

Intrusion is the partial or total immersion of the tooth crown into the alveolus, and the root of the tooth into the spongy substance of the jaw as a result of trauma.

On admission patients complain that the tooth became shorter than other teeth or is not visible at all after the trauma. Clinical examination reveals the intruded tooth is situated above (for upper jaw) or below (for laver jaw) the occlusal plane. The tooth is stable, percussion is painful. Sometimes the crown of an injured tooth is not visible at all, because it is driven in the socket. On the X-ray, an embedded tooth in the bone tissue is determined. Periodontal space is not visible.

Intruded primary teeth are most often extracted. It is possible to preserve only temporary teeth with unformed roots and shallow intrusion. Permanent tooth should be most often preserved. There are two main tactics for the treatment of intrusion of the permanent tooth:

1. The waiting method: tooth reposition is not performed hoping for its possible re-eruption into the dentition.
2. Simultaneous reposition of the intruded tooth with its fixation in the correct position and further treatment like in subluxation.

Each of the above methods of treatment has its advantages and disadvantages. BSMU adhere to the wait-and-see tactics in the treatment of intruded teeth. We consider that one-stage reposition of the intruded tooth with forceps leads to partial or complete destruction of the tooth socket and death of the pulp of the intruded tooth.

FACE BONES FRACTURES. CLINICAL MANIFESTATIONS, DIAGNOSIS, TREATMENT, EMERGENCY, PREVENTION

Fractures of the facial bones in children are one of the most difficult problems of maxillofacial surgery and dentistry. Fractures of the lower jaw are the most frequent and take from 70 to 90 % among all facial fractures. They are followed by fractures of the upper jaw and zygomatic bone. The dentist should be able to provide emergency care to the child with maxillofacial injury, determine treatment methods (orthopedic or surgical), as well as to prevent the possible complications occurrence.

MANDIBULAR FRACTURES

Features of the mandibular fractures in children are the following:

– subperiosteal fractures;

– fractures of the “green branches” type;

– epiphysiolysis;

– reflected fractures occurring in condylar processes upon impact to the chin;

– localization.

Mandibular fractures are commonly localized in the condylar processes or in the projection of permanent canines germs or at midline. Mandibular fractures at other areas (the angle, the branch and the body of the mandible) are much rare.

Mandibular fracture in children has the following *clinical manifestations*:

– it is accompanied by soft tissue injuries of the maxillofacial area (abrasions, contusions, bruises, wounds);

– pain, “step”-symptom and “crepitation”-symptom are detected by palpation;

– malocclusion;

– wounds of the mucous membrane of the alveolar bone;

– teeth mobility;

– bleeding from the mouth;

– pathological mobility of the lower jaw;

– violation of chewing function;

– violation of the integrity of the lower jaw bone tissue, determined by X-ray.

The *diagnosis* is made on the basis of the clinical picture and radiographic examination in 2 projections

The treatment of mandibular fractures in children depends on location, type and severity of the injury.

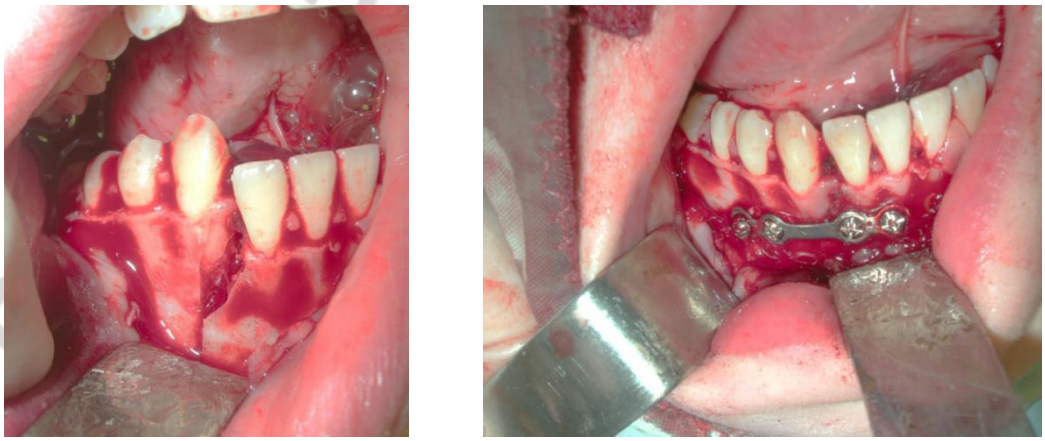
The treatment of *mandibular condylar process fractures* by “green branch” type is manufacturing and using a thick bandage, limiting lower jaw movements, sparing diet and anti-inflammatory therapy.

In case of fracture of unilateral *condylar process fracture* with bone fragments displacement the doctor should make individual plastic splint-kappa (removable splint) with inclined plane (Weber tire). Splint holds the lower jaw in the right proportion with the upper jaw and provides correct occlusion. In addition, for 2–2.5 weeks the child should wear a sling bandage. For the prevention of secondary deforming osteoarthritis these patients are assigned potassium iodide electrophoresis, ultrasound or phonophoresis of hydrocortisone, antibiotics, etc.

For children up to 10 years old with *complete fractures of both condylar processes* it is necessary to make individual plastic splint-kappa with hooking loops and jawbone. The fixation is carried out for 3–4 weeks. After removing the tires, mechanotherapy, therapeutic gymnastics physiotherapy, etc. should be carried out, aimed at preventing the development of secondary deforming osteoarthrosis. Wire splinting can be used after 11 years, because the children under the age of 11 do not have enough stable permanent teeth to use this treatment method and anatomical neck of the primary teeth is poorly defined.

The treatment of the child with a *mandibular body fracture* is manual reposition of fragments and fixing them with Tigerstedt splint (children older 11 years) or individual splint-kappa with a sling bandage (children up to 11 years old). Before fixing the fragments it is necessary to “decide the fate” of the teeth that are in the fracture line. The teeth with complicated caries and marginal periodontitis; dislocated and broken teeth; as well as teeth and follicles blocking bone fragments reposition should be extracted. All sound teeth remaining in the fracture fissure should be checked for pulp viability and, if necessary, their endodontic treatment should be done.

Surgical methods for the treatment of mandibular fractures may be used if reposition and fixation of fragments by orthopedic methods are impossible. From operational methods the surgeon can use bone suture, osteosynthesis with a Kirschner needle, miniplates (Fig. 17), etc.



a ***b***

Figure 17. Osteosynthesis of the mandible using miniplates:
a — preoperative view; *b* — postoperative view (illustration is provided by A. K. Korsak)

In children up to 2 years, as well as in persons with an insufficient number of teeth or teeth absence, immobilization according to Black or Kilgren may be used. A reliable fixation of the removable splint is carried out by applying a blanket wire stitch around the mandibular body and the splint on both sides of the fracture.

Antibiotic therapy (broad-spectrum antibiotics, tropic to the bone tissue) is used to prevent inflammatory complications of mandibular fracture. Active immunotherapy with staphylococcal toxoid is carried out for the same purpose. Physiotherapy is prescribed to accelerate healing and tonic effect. Good results are obtained when applying thymalin.

MAXILLARY FRACTURES

Upper jaw fractures are among the most severe face injuries. All of them are infected and open.

Classification of fractures of the upper jaw of Le Fort (Fig. 18):

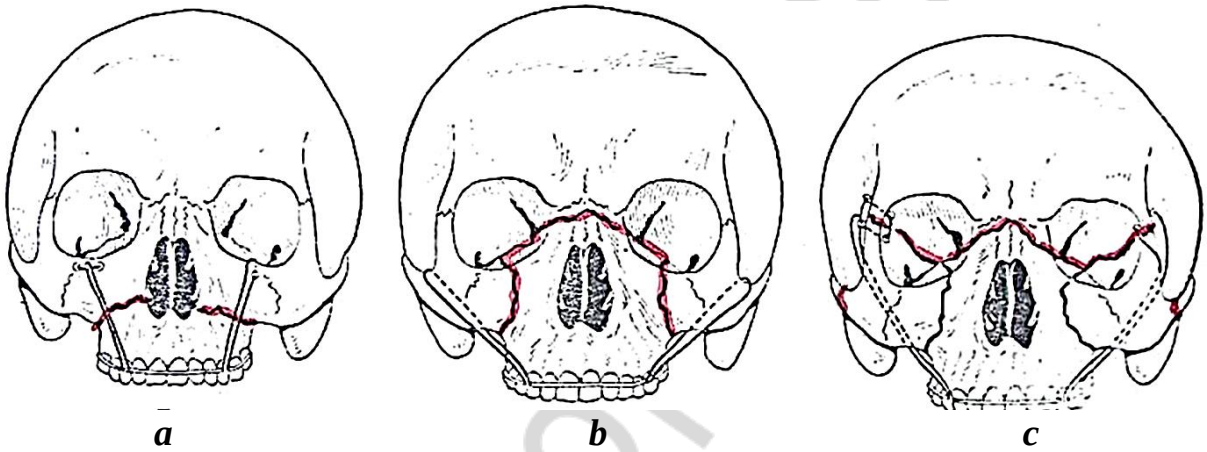


Figure 18. Le Fort classification of maxilla fractures:
a — Le Fort I; *b* — Le Fort II; *c* — Le Fort III

Le Fort I fracture (horizontal), otherwise known as a floating palate, may result from a force of injury directed low on the maxillary alveolar rim, or upper dental row, in a downward direction. The key component of these fractures, in addition to pterygoid plate involvement, is involvement of the lateral bony margin of the nasal opening. They also involve the medial and lateral buttresses, or walls, of the maxillary sinus, traveling through the face just above the alveolar ridge of the upper dental row. At the midline, the inferior nasal septum is involved.

Le Fort II fracture (pyramidal) may result from a blow to the lower or mid maxilla. The key component of these fractures beyond the pterygoid plate fractures is involvement of inferior orbital rim. When viewed from the front, the fracture is classically shaped like a pyramid. It extends from the nasal bridge at or below the nasofrontal suture through the superior medial wall of the maxilla, inferolaterally through the lacrimal bones which contain the tear ducts, and inferior orbital floor through or near the infraorbital foramen.

Le Fort III fracture (transverse), otherwise known as craniofacial dissociation, may follow impact to the nasal bridge or upper maxilla. The salient

feature of these fractures, beyond pterygoid plate involvement, is that they invariably involve the zygomatic arch, or cheek bone. These fractures begin at the nasofrontal and frontomaxillary sutures and extend posteriorly along the medial wall of the orbit, through the nasolacrimal groove and ethmoid air cells. The sphenoid is thickened posteriorly, limiting fracture extension into the optic canal. Instead, the fracture continues along the orbital floor and infraorbital fissure, continuing through the lateral orbital wall to the zygomaticofrontal junction and zygomatic arch. Within the nose, the fracture extends through the base of the perpendicular plate of the ethmoid air cells, the vomer, which are both part of the nasal septum. As with the other fractures, it also involves the junction of the pterygoids with the maxillary sinuses. CSF rhinorrhea, or leakage of the nutrient laden fluid that bathes the brain, is more commonly seen with these injuries due to ethmoid air cell disruption, as the air cells are located immediately beneath the skull base.

The following *local symptoms* are typical for the upper jaw fracture:

– the soft tissue injuries (bruises, wounds);

– severe swelling of the eyelids of both eyes;

– hemorrhage in the tissue around the eye and the conjunctiva (“glasses”-symptom score);

– bleeding from the nose, mouth and ears; liquorrhea can occur (“double spots” — symptom should be checked);

– lengthening and flattening of the middle part of the face;

– anesthesia or paresthesia of the upper lip, nose wing and suborbital area;

– diplopia (double vision);

– pain, crepitus, and “step sign” on palpation in the nose, on the edge of the infraorbital and upper-outer edge of the orbit, in the course of the zygomatic arch and the cheeks, alveolar ridge;

– malocclusion;

– upper teeth mobility;

– “cracked pot”-symptom on percussion of the maxillary teeth.

Correct *diagnosis* is possible only in the result of a complex examination of the patient with the involvement of related specialists (neuropathologist, ENT specialist, ophthalmologist, pediatrician). Facial bones radiography (radiography of the midface in direct projection, CT, etc.) is necessary to clarify the fracture localization.

The treatment for upper jaw fractures consists of:

1) surgical treatment of wounds of soft and bone tissues;

2) reposition and fixation of the jaw fragments;

3) drug therapy;

4) organization of special patient care;

5) prevention of fracture complications and traumatic brain injury.

Surgical treatment of wounds should be simultaneous, radical and fulfill, with good anesthesia. At first bone wound is treated in the depth, then around the periphery and after that the wounds of soft tissues are treated. The fixation of

fracture fragments can be carried out using orthopedic, surgical and combined treatment methods. Unilateral fragments of the alveolar process are usually easily set manually and fixed with a splint-kappa (removable splint) in children under 11 years of age and smooth splint in children after 11 years. Dislocated sound permanent teeth should be replanted. Endodontic treatment (if it is necessary) should be done only after consolidation of the jaw fragments and strengthening of the teeth in the alveolar sockets.

In children purely *orthopedic methods* for managing upper jaw fractures are practically not used, since these devices are very cumbersome and it is psychologically difficult for children to adapt to them.

Surgical methods are: bone suture, fixation of fragments with the help of Kirchner's needles, the method of the fronto-maxillary osteosynthesis, mini-plates, etc. In children these methods of fixation of the upper jaw fragments also have limited indications for use, since fractures within the dentition during the period of the primary and mixed occlusion can damage the permanent teeth follicles. In addition, the negative sides include damage to growth zones during surgery.

The treatment of children with upper jaw fractures can be carried out by *combined methods* (Federspiel, Adams technique, etc.), which combine orthopedic and surgical methods of treatment.

Federshpil method. A splint (removable commonly) is fixed on the upper jaw teeth. Then the wire is fixed on both sides (right and left) to the splint at the level of the molars and to the head plaster cap. The wire is carried out (with the help of an injection needle) through the soft tissues of the cheeks. Federshpil method cannot be used if the patient has a cranial injury and it is impossible to wear the head cap.

In such cases Adams method can be used. A splint is connected by wire through soft tissues to the intact bones of the facial skeleton (zygomatic processes of the frontal bone or to the zygomatic arch) on both sides. After 2.5–3 weeks after osteosynthesis, the wire ligatures on each side are crossed and removed.

Drug therapy is the same as for mandibular fractures treatment.

ZYGOMATIC BONE FRACTURES

Zygomatic bone fracture means zygomatic bone separation from the zygomatic process of the upper jaw, the frontal and temporal bones. The body of the zygomatic bone shifts down and inwards and, as a rule, damage occurs to the maxillary sinus too. In this case, bleeding from the corresponding nostril is determined. As a rule, the fracture of the zygomatic bone causes pinching of the infraorbital nerve.

Zygomatic arch fracture means disruption of the continuity of the temporal process of the zygomatic bone and the zygomatic process of the temporal bone.

Clinical signs of the zygomatic bone fracture are the following:

– soft tissue injuries of the zygomatic area (swelling, wounds, bleeding);

– severe swelling of the eyelids, and bleeding in the tissue around one eye;

– nasal bleeding from one nostril;

– limited mouth opening;

– anesthesia or paresthesia of soft tissues in the zone of innervation infraorbital nerve on the side of injury;

– diplopia;

– pain and "step-sign" on palpation on the edge of the infraorbital, upper-outer edge of the orbit, in the course of the zygomatic arch and on the cheekbones, the alveolar ridge;

– X-ray determined a violation of the integrity of the bone tissue in the joints of the zygomatic bone to other bones of the facial and cranial parts of skill (Fig. 19).

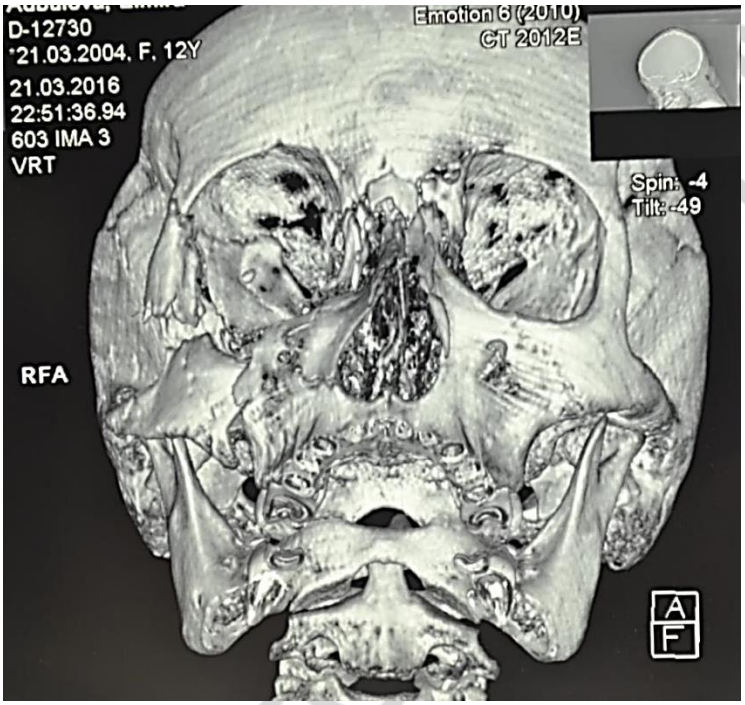


Figure 19. Fracture of the right zygomatic bone with dislocation, CT

The treatment is an operative reduction of fragments of the zygomatic bone or arch. Under general or local anesthesia, a skin incision is made up to a length of 5 mm at the intersection of the trago-nasal line and perpendicular through lateral edge of the orbit. A thin clip forms a tunnel through the soft tissue to the bone and through it the tip of the Limberg hook is inserted under the bone fragment. Under the action of a hook (outwards and upwards), the fragment is set off. The signs of proper reduction are disappearance of the "step" along the lower orbital margin, free mouth opening, restoring the face symmetry. Then the wound is sutured. In some cases, when the zygomatic bone is not fixed independently in the correct position after reduction, fragment is fixed with the bone suture or miniplates.

Drug therapy is the same as for mandibular fractures treatment.

GUNSHOT FRACTURES

Gunshot face bone fractures in children are rare. In case of gunshot mandibular fractures many bone fragments are formed (due to the massive cortical plate) that can be embedded in surrounding soft tissues.

Features of the gunshot fractures of the facial bones are the following:

– the fractures are always open;

– foreign bodies presence in the wound (bullets, shot, and other metal fragments, etc.), which requires X-ray examination;

– no typical localization;

– displacement of bone fragments in the direction of impact force of the projectile and wounding at least, depending on the thrust of the masticatory muscles;

– the fractures are comminuted often, sometimes with defects (shooting) bone;

– heavy contamination and infection, which is often the cause of abscesses and phlegmon of the maxillofacial region;

– they are much more common than traumatic, accompanied by life-threatening conditions: bleeding, asphyxia and traumatic shock.

Patients with gunshot wounds to the face is always in need of surgical treatment of the wound, which is best done in the early stages — up to 24 hours after the injury.

Wound treatment should be gentle. All the largest bone fragments that have retained contact with soft tissues should be carefully laid and covered with soft tissues.

Unviable bone fragments should be removed because they contribute to the wound suppuration and prevent the bone fractures consolidation. To clarify the bone fragment viability (vitality) it is necessary to bite its edge and, in case of bleeding, the fragment is vital and should be retained. Surgical treatment of bone wounds is necessary combined with the bone fragments reduction and fixation.

Gunshot wounds of the upper jaw almost always leads to the maxillary sinus trauma. In these cases, the sinus is filled with blood, fragments of bone, teeth; sometimes foreign bodies fall into it. Therefore, when such injuries maxillary sinus revision should be carried out.

In case of injuries to the upper jaw with damage to the nasal conchae, nasal septum, ethmoid bone, surgical treatment should be carried out together with the ENT specialist.

Complications of facial bones fractures

Complications of the injuries to the maxillofacial area according to the time of their occurrence are divided into:

– *immediate*: asphyxia, bleeding, traumatic shock;

– *nearest* (early): suppurative processes of the wounds, abscesses and cellulitis of soft tissues; traumatic osteomyelitis, traumatic sinusitis, secondary bleeding due to the melting of the thrombus, sepsis, etc.;

– *long-term* (late): scar deformation of soft tissues, soft tissue and bone defects, adentia and death of germs of permanent teeth, deformation of the jaws, wrong coalesced fracture of the bone, malocclusion, false joint, stunting jaws, TMJ disorders.

In addition, in children long-term complications (occur) which do not happen in adults. These include:

– the death of permanent teeth follicles;

– teeth anomalies and hypoplasia;

– teeth retention;

– stunted growth of the facial bones (especially the lower jaw).

Traumatic shock is the emotional or psychological state after trauma that may produce abnormal behavior. The most common types are hypovolemic shock from blood loss and neurogenic shock caused by a disruption of the integrity of the spinal cord.

Prevention and treatment of the traumatic shock include:

– local and general anesthesia;

– stop bleeding;

– blood loss compensation and hemodynamics normalization;

– maintenance of external respiration and the fight against asphyxia and hypoxia;

– temporary immobilization of bone fragments or transport;

– satisfaction of hunger and thirst;

– patient warming (in cold weather);

– timely (urgent) surgery.

Temporary immobilization methods in case of facial bones fractures are the following:

– standard transport bandage ;

– circular (chin-parietal) bandaging;

– submental soft sling bandage;

– net or tubular bandage according to the type of circular bandaging;

– lock jaw sticky plaster (from temple to temple across the chin);

– ligature binding the teeth of one jaw;

– ligature binding rostral teeth (at Ivy et al.), if the victim has no symptoms of traumatic brain injury (vomiting, nausea), and there is no danger of asphyxia.

The *main causes* of infectious and ***inflammatory complications*** (suppurative processes of the wounds, abscesses and cellulitis of soft tissues; traumatic osteomyelitis, traumatic sinusitis, etc.) are the following:

– teeth with complicated caries in fracture line;

– late submission of medical care for patients (hospitalization);

– late and inadequate fixation of bone fragments;

– infection of wounds from oral cavity and skin;

– violation of the treatment regimen.

Traumatic osteomyelitis *prevention* includes:

– after the repositioning of bone fragments, it needs timely, reliable and sufficiently long-term immobilization of the jaw;

– the decision to “fate” of the “caused” tooth, located in the fracture line;

– a thorough oral care (home oral hygiene);

– anti-inflammatory (antibacterial) therapy.

Children with facial bone fractures need follow-up care to prevent or reduce and manage ***long-term complications*** (Fig. 20). Children with chronic post-traumatic osteomyelitis need repeated inpatient treatment courses. Children with upper jaw fractures should be monitored by a neurologist, ENT doctor and ophthalmologist. Orthodontic treatment is carried out in case of violation of the formation and growth of the jaws.

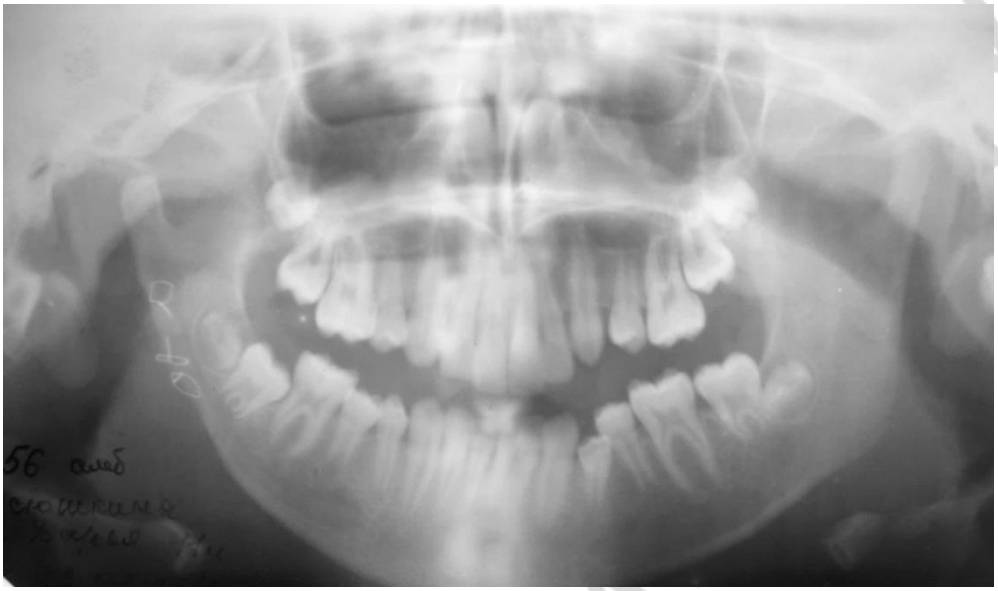


Figure 20. Underdevelopment of the mandible on the right as a long-term complication of fracture in the condyle (illustration is provided by A. K. Korsak)

Children with uncomplicated course, without damage to the permanent teeth follicles and growth zones are supervised during the 1st year after trauma and are removed from the follow-up after the normalization of the clinical and X-ray picture.

ANOMALIES OF LIPS AND TONGUE FRENULUM IN CHILDREN. SMALL VESTIBULE OF ORAL CAVITY. ANOMALIES OF NUMBER AND ERUPTION OF TEETH. CLINICAL MANIFESTATIONS, DIAGNOSIS AND TREATMENT

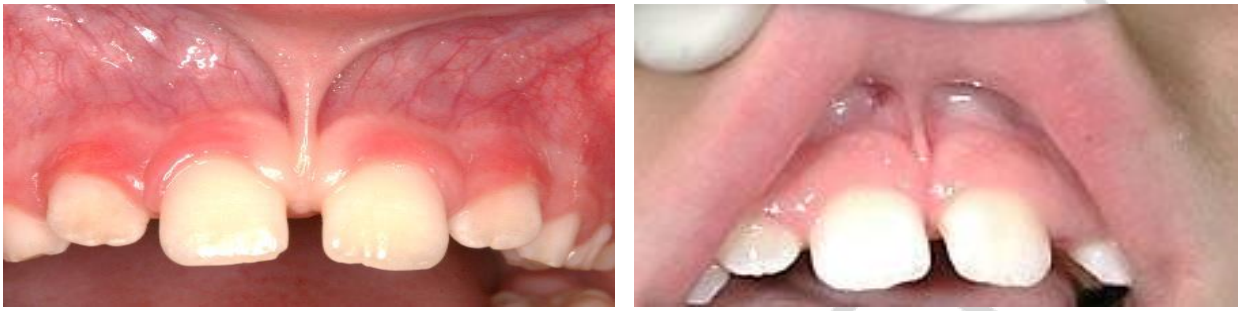
Anomalies of lips and tongue frenula are congenital malformations of maxillofacial area. It can cause chronic injury, periodontitis, violations of sucking, swallowing, speech in children. This pathology is very common in childhood. Plasty of short lip or tongue frenulum is the most common planned operation in pediatric maxillofacial surgery.

SHORT FRENULUM OF UPPER LIP

Frenulum of the upper lip is a plica of mucous membrane that separates oral vestibule on midline. Complaints of child with short frenulum often are aesthetic drawback — diastema between central incisors in upper jaw and marginal periodontitis. There are 2 types of short frenula of upper lip by clinical features.

The first type: frenulum is thick and powerful, triangular in shape and short. It connects with incisive papilla or interdental papilla between central incisors. Diastema is wide, more than 3 mm.

The second type: upper lip frenulum is short, thin, sometimes transparent, as duplication of mucous. It attaches to alveolar process between central incisors. Frenulum is low attached. Diastema is not wide, 2–3 mm.



a ***b***

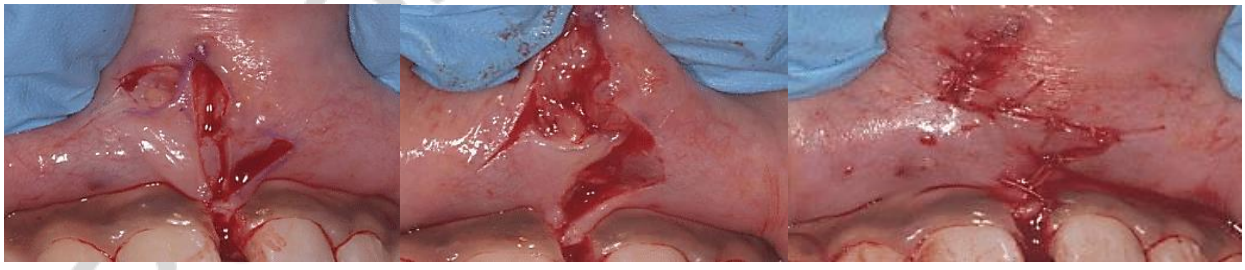
Figure 21. Two types of short frenula of upper lip:
a — 1st type; *b* — 2nd type

Also, attention should be paid to the width and shape of the diastema, as well as other anomalies from the central incisors. Children need X-ray examination for the diagnosis and treatment plan.

The type of short frenulum of the upper lip and diastema (according the age of the child) determines the method of surgical intervention and subsequent orthodontic treatment.

The indication for the operation of the plastiy of the short upper lip frenulum is the presence of a diastema and inflammatory changes in the area of the central incisors. Operative intervention is carried out in 7–8 years after the eruption of permanent central incisors. There are several ways to plastic the frenula of the upper lip.

Z-frenuloplasty (Limberg) is used to the first type of short upper lip frenula. After anesthesia surgeon makes two triangular flaps (Z-shape) with angles of at least 60°. Then flaps are repositioned to closure. After that the wound is sutured (Fig. 22).



a ***b*** ***c***

Figure 22. Operation of Z-plasty of upper lip frenula:
a — triangular flaps; *b* — reposition; *c* — sutured wound

The second type of upper lip frenulum require operation of **V-plasty** of frenula. After anesthesia surgeon makes V-shaped incision around the frenula.

Then flap is moved higher by 1–1.5 cm or excise. After that the wound is sutured (Fig. 23).

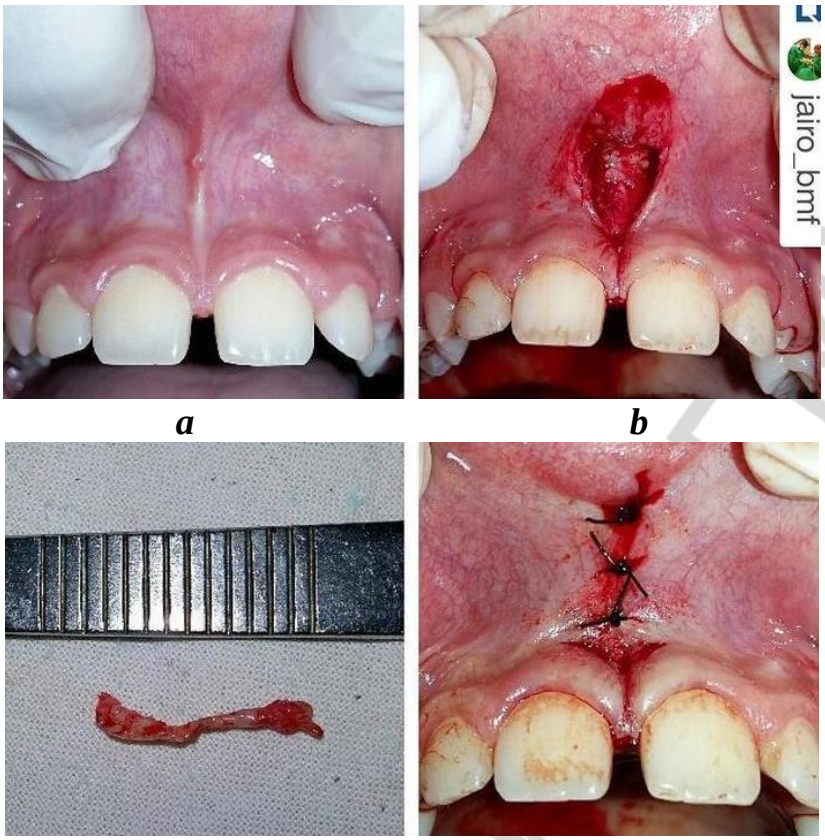


Figure 23. V-plastic operation of upper lip frenula:
a — preoperative view; *b* — incision; *c* — removed tissue; *d* — sutured wound

All children after plastic of the short upper lip frenula need orthodontic treatment, which should be started 10–14 days after the operation.

SHORT FRENULUM OF LOWER LIP

Short frenulum of lower lip can be independent illness or combined with a small oral vestibule. Children often complain of gums bleeding increasing at brushing and denudation of necks of lower central incisors.

Clinical examination of child should begin with the horizontal stretching of the lower lip. Gingival papilla between central lower incisors becomes pale or separates from the neck of the teeth (Fig. 24). Most patients have inflammation of gingival papilla between 31, 41, its bleeding and recession. Some patients also have pathological mobility of 41 and 31 teeth and pockets. In these cases, the X-ray reveals destructive changes in bone tissue with resorption of the interdental septum. Depth of oral vestibule on both sides is normal.

The plastic surgery of the short frenulum of the lower lip is performed no earlier than 7–8 years with a diastema between the lower central incisors, as well as inflammatory or inflammatory-dystrophic changes in the marginal periodontal of this area. Conservative treatment at dental therapist to normalize oral hygiene and eliminating inflammation should be done before operation.

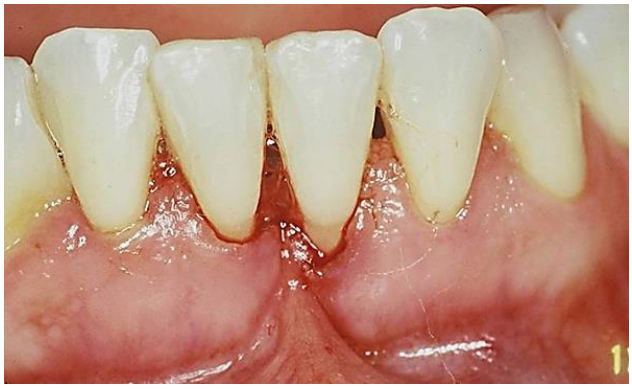
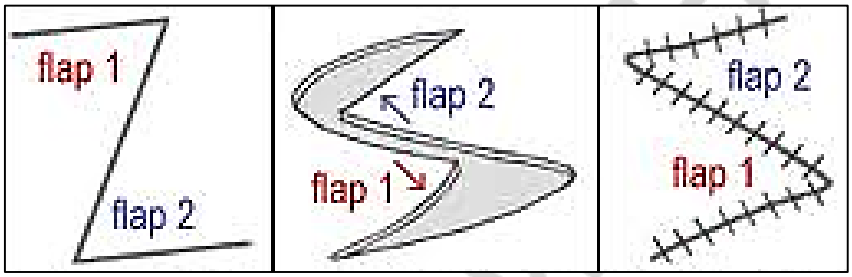


Figure 24. Short frenula of lower lip

Z-plasty of the short frenulum of the lower lip is most optimal technique. The aim of operation is elimination of tension by lengthening tissue. The steps of operation are the same one like in Z-plasty of short upper lip frenulum (Fig. 25).



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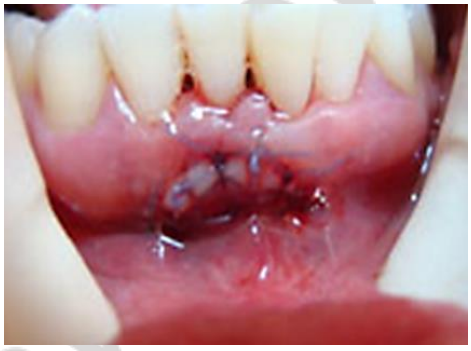


d

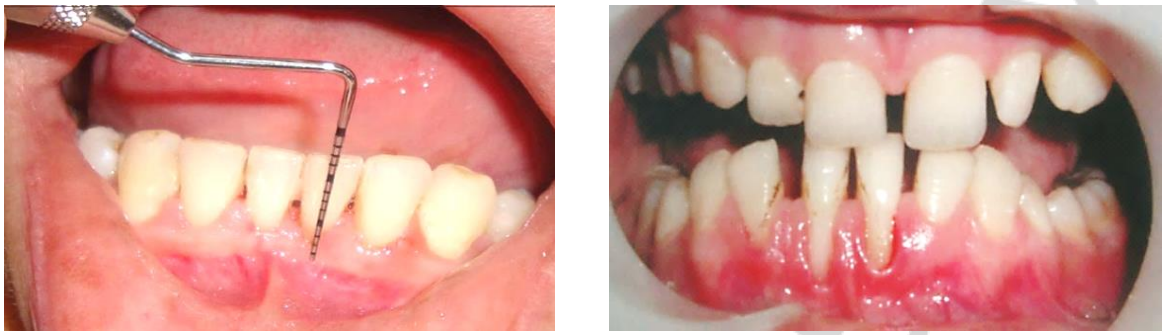
Figure 25. Operation of Z-plasty of lower lip frenulum:
a — triangular flaps; *b* — reposition; *c* — sutured wound (scheme), *d* — sutured wound (clinical view)

In the postoperative period, massage, physiotherapy and orthodontic treatment are prescribed.

SHALLOW VESTIBULE OF ORAL CAVITY

Shallow vestibule of mouth occurs at the age of 6–14 years in 7 % of cases. Depth of vestibule is distance from the base of gingival papillae of anterior teeth to unattached gingiva (Fig. 26). If this length is less than 5 mm, vestibule of mouth is shallow. It creates tension in the gums in the frontal region, which is a risk factor of periodontal disease due to chronic gingival injury during meals.

Children and their parents usually complain on gums bleeding increasing and denudation necks of lower front teeth. Clinical examination at developed disease revealed inflammatory and degenerative changes of marginal periodontium from 33 to 43 teeth (gum bleeding, recession (Fig. 26)), denudation of necks or roots, pathological movement, periodontal pockets lower front teeth). Shallow vestibule of mouth often (in 58.9 % of cases, BSMU, A. K. Korsak, 1995) combined with overcrowding of lower front teeth, deep bite, anomaly position of teeth and etc.



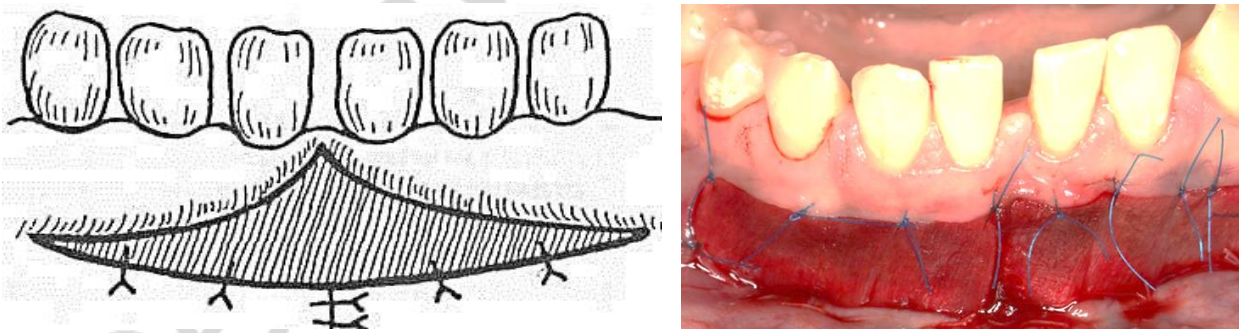
a ***b***

Figure 26. Clinical features of shallow vestibule of oral cavity:
a — depth measurement; *b* — gingival recession

Vestibuloplasty is indicated for children with a small vestibule of the oral cavity and the presence of local gingivitis, recession of the gum or marginal periodontitis. Operation is carried out after eruption of permanent frontal teeth and the formation of their roots.

There are “open” and “closed” types of plasty of oral vestibule.

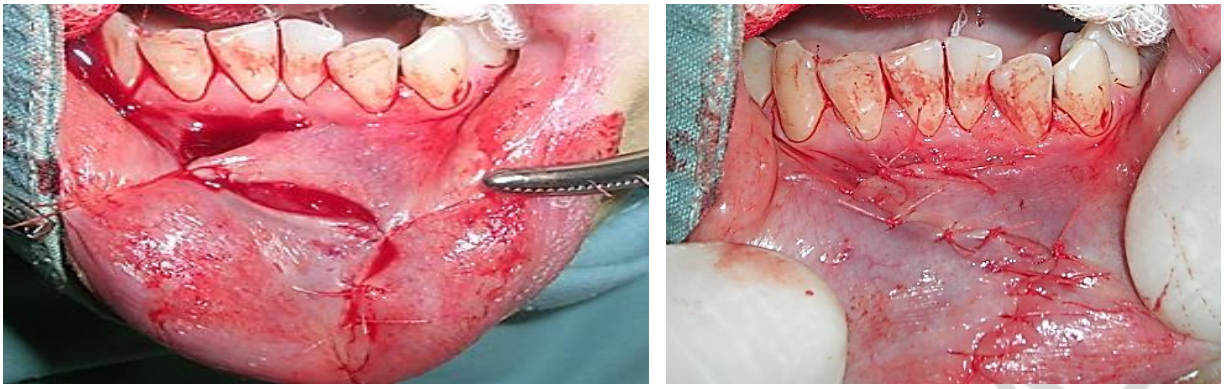
The first type — **“open” technique** by Kazanyan, Clark. Open wound at mucosa of lower lip or alveolar bone is formed after deepening of vestibule, which then heal by secondary tension (Fig. 27). Disadvantage of these techniques is high risk of recurrence as a result of rough scarring of soft tissues. Open technique is not used in children because of risk of growth area damage and developing of the bone priks in the area of incision.



a ***b***

Figure 27. “Open” types of plasty of oral vestibule:
a — operation scheme; *b* — postoperativeclinical view

The second type — **“closed” technique** (Z-plasty, Y-vestibuloplasty, and etc.). Surgeon closes wounds after deepening of vestibule with local tissues (Fig. 28). It is the most optimal method in children.



a ***b***

Figure 28. Z-plasty of shallow vestibule of mouth:
a — operative view; *b* — postoperative view

After the operation, a plate with a vestibular pelote is used to form oral vestibule and prevent the recurrence of the disease. Patients use it in 2–3 months after the operation.

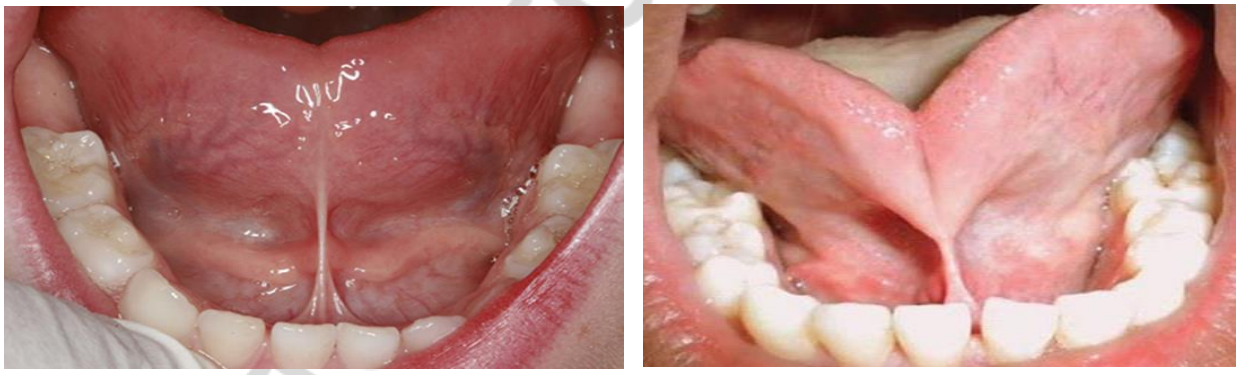
SHORT TONGUE FRENULUM

Short frenulum of the tongue is a congenital malformation, that restricts the tongue movements and leads to functional changes in the dentoalveolar system.

There are two types of short frenula of tongue by clinical features (A. K Korsak, 2000).

The first type: frenulum is short, thin, sometimes transparent, as duplication of mucosa (Fig. 29, *a*). Tongue mobility is limited and its tip is bifurcated.

The second type: frenulum is thick and powerful, and short (Fig. 29, *b*). Tongue mobility is also limited. Tongue tip is bifurcated, especially at function.



a ***b***

Figure 29. Types of short frenula of tongue:
a — 1st type; *b* — 2nd type

Infants with short frenulum of the tongue have difficulties in sucking. Insufficient mobility of the tongue at a later age can disrupt the swallowing and pronunciation of some sounds and also lead to bite anomalies (open, mesial prognathic bite).

Indications for ***surgical intervention in newborns*** are complaints of a violation of sucking and swallowing. If frenulum is short, thin and transparent

surgery can be done in polyclinic. Under topical anesthesia or without it doctor cuts short frenulum of tongue to normal borders of attachment by scissors (ankylotomy). But after this operation relapse can occur in 3–4 years old.

There are several ways of plasty of a short tongue frenulum, which depend on its kind. Operation is carried out under general anesthesia, and the tip of the tongue is fixed with a ligature.

Frenotomy — cutting the frenulum of the tongue from its lower surface to the full release of the tip of the tongue is produced at *first type* of short frenulum. Then, the tongue is pushed upwards and anteriorly, horizontal wound is transformed into a vertical one and sutured tightly.

a ***b*** ***c***

Figure 30. Frenotomy (ankylotomy) of tongue:
a — incision; *b* — wond transformation, *c* — sutured wound

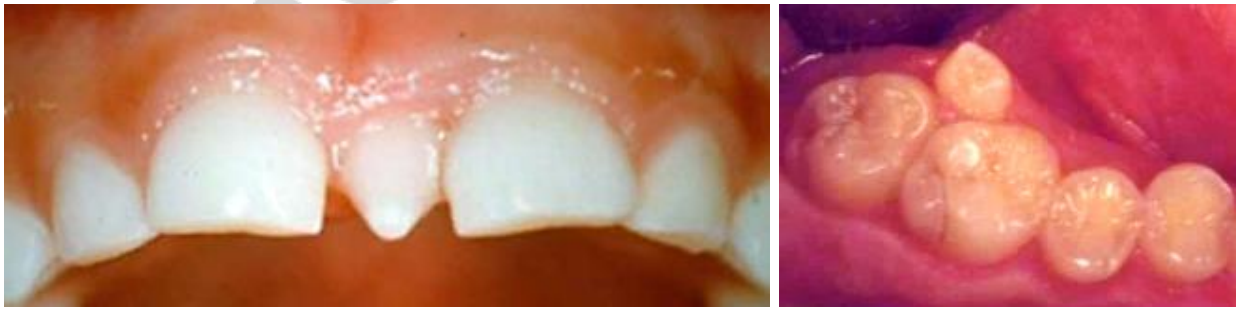
Second type of short frenula requires operation of Z-plasty. The median incision is carried out along the crest of the tongue frenulum.

Gymnastics for the tongue muscles and speech therapy are indicated after surgery to restore the mobility of the tongue and normalize the impaired functions to all children.

ANOMALIES OF NUMBER OF TEETH

Anomalies of the number of teeth include *hyperdontia (supernumerary teeth), hypodontia* and *anodontia*.

Supernumerary teeth (Fig. 31) are often localized (about 95 %) in area of upper incisors.



a ***b***

Figure 31. Supernumerary tooth:
a — mesiodens; *b* — paramolar

Presence of them in alveolar bone can cause retention of complete teeth or (and) their anomalies position in dentition (diastema, oral or vestibular position). The alveolar process is thickened, its palpation is painless, mucosa is pale pink. If a supernumerary tooth has erupted, it is easy to diagnose. Nevertheless, all patients with this pathology need an X-ray examination, because patient may have several supernumerary teeth.

The treatment of children with this pathology is complex — surgical and orthodontic. If supernumerary teeth cause anomaly position of complete permanent teeth or other pathology (pain, sinusitis, follicular cyst, osteomyelitis, etc.), these teeth should be extracted.

Anodontia is a congenital absence of all primary or permanent teeth. The cause often is ectodermal dysplasia, in which skin is dry, pale, wrinkled, hair and teeth germs are absent. **Hypodontia** *is* a congenital missing of one to six teeth. Surgical treatment of anodontia and hypodontia (dental implants, transplant of tooth germ) in children are not used.

ANOMALIES OF TEETH ERUPTION

Anomalies of teething include premature eruption and retention of teeth.

Premature eruption of permanent tooth requires saving such tooth, because root has not formed. Protective devices (plates, mouth guards, and others) are often prescribed, which patients use to the end of root formation.

Retained teeth — teeth, that are in alveolar process after optimal dates of eruption, which ended or ends root formation. About 3 % of children have it. Usually one or more permanent teeth are retained, but multiple retention of permanent teeth (cherubism) can occur too. Retention of upper incisors occurs more frequently (85.5 %) then a retention of the maxillary canines (13.0 %).

The causes of retention of individual teeth can be lack of space in the dentition, inflammatory processes in the area of the roots of the milk teeth, premature removal of milk teeth, supernumerary teeth, etc.

Clinical features of tooth retention are: alveolar process is thickened, painless palpation, mucosa is pale pink. Tooth is absent in dentition. Children also need X-ray examination.

Treatment of retained permanent teeth depends on the cause of anomaly, position of the tooth in the jaw and stage of root formation. If the retention of an individual tooth does not lead to the development of pathological conditions in the child, clinical and radiological monitoring is performed in dynamics and *treatment is not performed*.

Retained teeth are *extracted* at: abnormal anatomical shape, horizontal position, acute angle to the occlusal plane and absence of a space to it in the dentition.

Treatment with *tooth preservation* is often complex (surgical and orthodontic). The eruption of retained permanent tooth with unformed roots is stimulated by bite plates, irritating plate, massage and physiotherapy for 6–12 months. If roots have formed yet — surgeon expose crowns of retained teeth. Then orthodontic devices (braces) normalize the position of retained tooth.

CONGENITAL MALFORMATIONS OF THE MAXILLOFACIAL REGION IN CHILDREN (CYSTS, FISTULS, MALOCCLUSION). CLEFT LIP AND CLEFT PALATE. ETIOLOGY, CLINICAL MANIFESTATIONS, DIAGNOSIS, TREATMENT

Congenital malformations are structural abnormalities due to faulty development, present at birth, and are among the major causes of prenatal, perinatal and infant mortality and morbidity. They include gross and microscopic malformations, inborn errors of metabolism, mental retardation and cellular and molecular abnormalities. About 2–3 % of newborns have a single major malformation, and 0.7 % have multiple major defects.

The development of the maxillofacial area is coordinated by complex morphogenetic events and rapid proliferative expansion, and is thus highly susceptible to the harmful factors. During the first six to eight weeks of pregnancy, the shape of the embryo's head is formed (Fig. 32, 33). Five primitive tissue lobes grow (Fig. 33): Frontonasal Prominence, 2 Maxillar Prominences and 2 Mandibular Prominences.

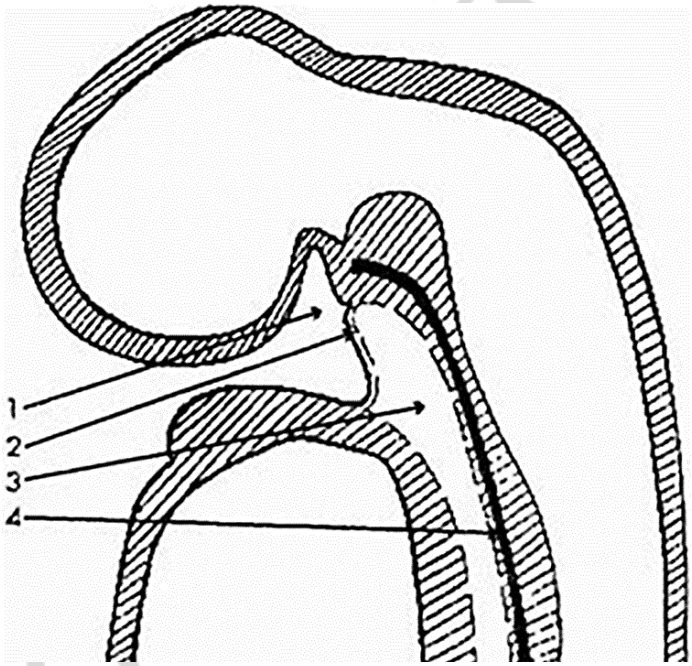


Figure 32. Longitudinal section through the head of the human embryo (scheme). Germ length 3 mm:
1 — oral fossa; *2* — pharyngeal membrane; *3* — foregut; *4* — chord

If these prominences fail to meet, a gap appears where the tissues should have joined (fused). This may happen in any single joining site, or simultaneously in several or all of them. The resulting birth defect reflects the locations and severity of individual fusion failures. Formation of the palate is the last step in joining the five embryonic prominences and involves the palatal shelves (back portions of the Maxillar Prominences), which grow towards each other until they fuse in the middle.

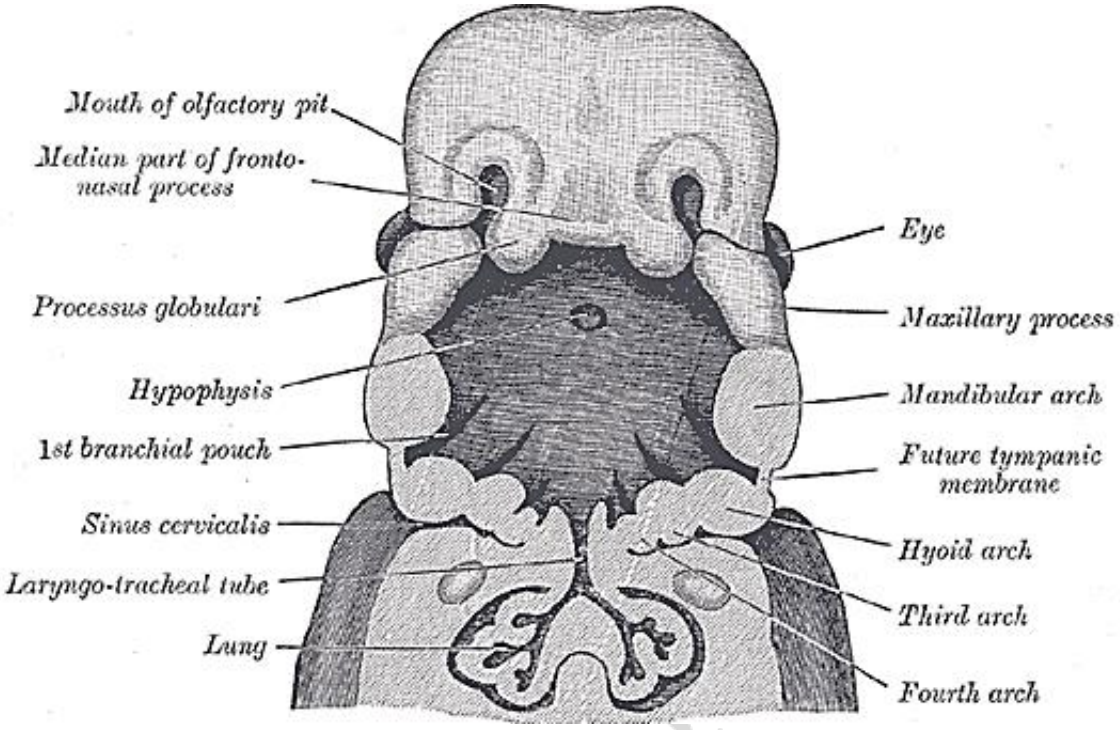


Figure 33. Front view of the head of the human embryo (scheme)

Causes of the congenital malformations in humans are the following:

1. Endogenous causes:

1.1. Changes in genetic structure (mutations).

1.2. Endocrine diseases.

1.3. "Over ripeness" of gametes.

1.4. Age of the parents.

2. Exogenous causes:

2.1. Physical factors (radiation, mechanical injury, hyperthermia).

2.2. Chemical factors (chemicals used in everyday life and industry (teratogenic poisons); hypoxia; drugs; malnutrition).

2.3. Biological factors (viruses, mycoplasma, protozoa infection).

Congenital malformations of the face and neck can be divided into:

1. Congenital anomalies of the system of the facial soft tissue and facial bone shaping (syndromes).

1.1. Fibrous dysplasia.

1.2. Syndrome I–II gill arches.

1.3. Maxillofacial dysostosis (Treacher Collins syndrome).

1.4. Craniofacial dysostosis (Crouzon syndrome).

1.5. Cranio-clavicular dysostosis

1.6. Robin syndrome and other syndromes.

1.7. Congenital cysts and fistulas, etc.

2. Congenital malformations of individual anatomical structures of the maxillofacial region.

2.1. Congenital cleft lip and cleft palate.

2.2. Congenital clefts of face.

2.3. Anomalies of lips and tongue frenulum

2.4. Small (shallow) vestibule of the oral cavity, etc.

3. Anomalies and defects of tooth development (adentia, retention, supernumerary teeth, etc.).

4. Dentoalveolar anomalies (malocclusion).

OBLIQUE FACIAL CLEFT

It is a severe congenital disorder resulting from nonunion (complete or incomplete) frontonasal and maxillary processes during embryonic development of the child. Cleft can be complete and incomplete, one- and two-sided. More frequent incomplete oblique facial cleft (Fig. 34).

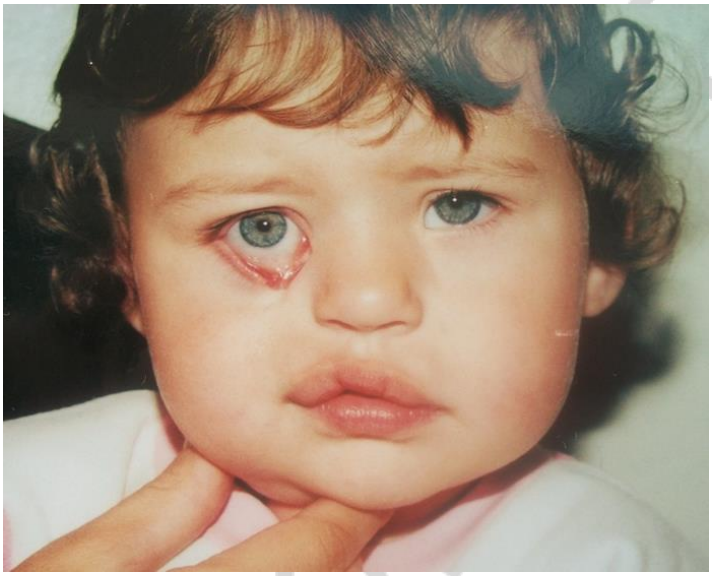


Figure 34. Oblique incomplete facial cleft in right (illustration is provided by A. K. Korsak)

Clinically cleft goes from the upper lip filtrum to the lower eyelid, where there is a coloboma. The treatment is surgical at different ages (plasty of the upper lip, lower eyelid, contour plastics).

PIERRE ROBIN SYNDROME (PRS)

It is a congenital facial abnormality including the following three main features:

1) cleft palate;

2) retrognathia (abnormal positioning of the jaw) of mandible;

3) glossoptosis (airway obstruction caused by backwards displacement of the tongue base). A genetic cause to PRS was recently identified.

It is generally diagnosed clinically shortly after birth. The infant usually has respiratory difficulty, especially when supine position and the main threat to life in these children is dislocation asphyxia. The cleft palate in PRS is often U-shaped and wider than in cleft palate that is not associated with this syndrome.

The treatment from birth is prevention of asphyxia. Further myogymnastics, plasty of the palate at preschool age, and orthodontic treatment are necessary.

VAN DER WOUD SYNDROME

It includes symmetrical congenital fistulas of the mucous glands of the lower lip in combination with congenital cleft lip and palate (Fig. 35). *The treatment* consists of the plasty of the upper lip and palate at the usual time. In addition, surgery is required excision of congenital fistulas of the lower lip at preschool age.

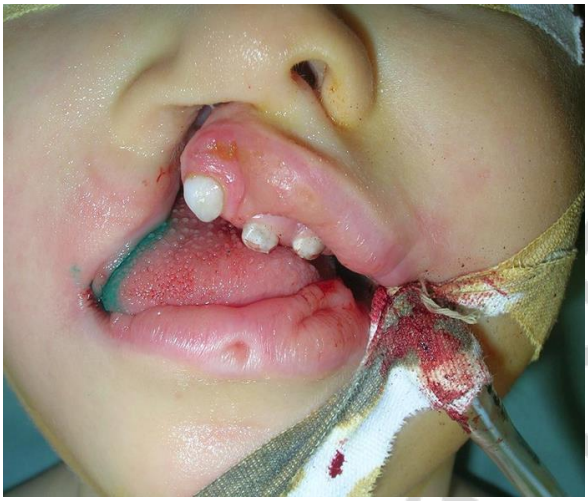
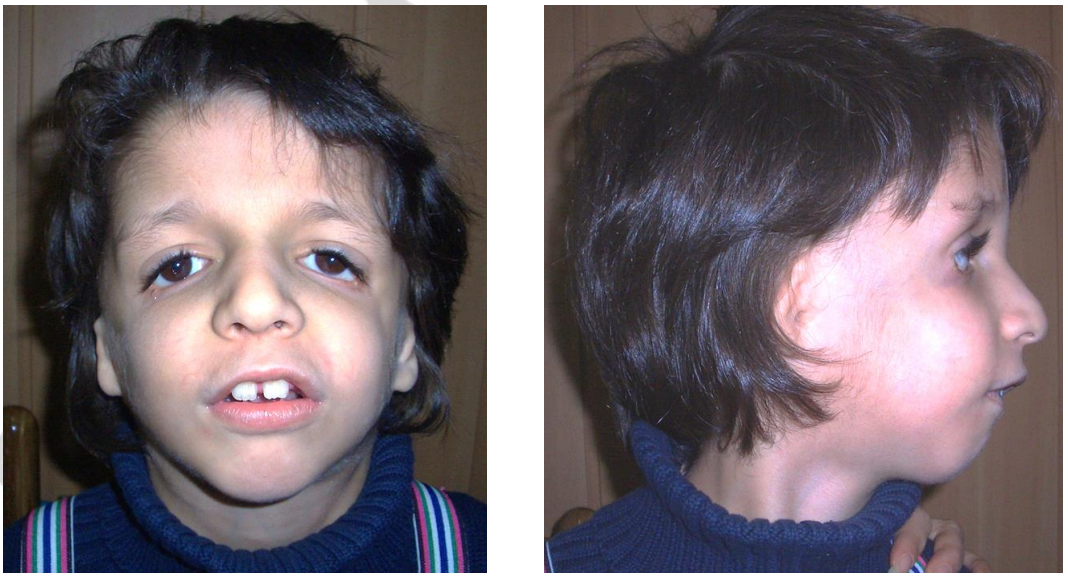


Figure 35. Van der Woud syndrome. Patient's appearance (illustration is provided by A. K. Korsak)

TREACHER COLLINS SYNDROME (TCS)

It is an autosomal dominant congenital disorder characterized by craniofacial deformities, involving ears, eyes and cheekbones and goes along with normal intelligence.

The typical features are the following: downward-slanting eyes, micrognathia (a small lower jaw), conductive hearing loss, underdeveloped zygoma, drooping part of the lateral lower eyelids, and malformed or absent ears (Fig. 36).



a ***b***

Figure 36. Treacher Collins syndrome. Patient's appearance:
a — front view; *b* — side view (illustration is provided by A. K. Korsak)

Hypoplasia (underdevelopment) of mandibular and zygomatic bones leads to a small and malformed jaw. Mandible hypoplasia can result in a malocclusion.

The external ear anomalies are small, rotated, or even absent auricles. Also symmetric, bilateral stenosis or atresia of the external auditory canals is described. In most cases, the ossicles and the middle ear cavity were dysmorphic. Inner ear malformations are rarely described. As a result of these abnormalities, a majority of the individuals with TCS have conductive hearing loss.

Most affected persons experience eye problems, varying from colobomata of the lower eyelids and aplasia of lid lashes to short, down-slanting palpebral fissures, and missing eyelashes. Vision loss can occur and is associated with strabismus, refractive errors and anisometropia.

Airway problems, which are often a result of mandibular hypoplasia, can occur.

The treatment is complex: surgical and orthodontic for aesthetic and functional indications.

CRANIOFACIAL (HEMIFACIAL) MICROSOMIA (1–2TH PHARYNGEAL ARCHES SYNDROME)

The pharyngeal (visceral, gill) arches are structures seen in the embryonic development of vertebrates that are recognizable precursors for many structures.

The 1st arch divides into a maxillary process and a mandibular process, giving rise to structures including the bones of the lower two-thirds of the face and the jaw. The maxillary process becomes the maxilla (or upper jaw), and palate while the mandibular process becomes the mandible or lower jaw. This arch also gives rise to the muscles of mastication. The second pharyngeal arch or hyoid arch, is the second of six pharyngeal arches that develops in fetal life during the fourth week of development and assists in forming the side and front of the neck.

Craniofacial (Hemifacial) Microsomia is a relatively common disorder in which the lower half of one side of the face is underdeveloped and does not grow normally having as a result facial asymmetry or/and malformative development of certain organs. The degree of malformation varies from mild degree that is just perceived up to very severe degree.

The organs that are commonly affected are the lower jaw (mandibular hypoplasia) and auricles. The upper jaw (maxillary hypoplasia) and the cheekbone are also affected but to a lesser extent.

In milder forms, the problem may be only the underdeveloped auricles or a small degree facial asymmetry. In the most severe forms, the lower jaw may be very malformative or not developed at all at the side of the face with this disorder (Fig. 37). The soft tissues are not developed properly giving to the face very asymmetrical appearance (Fig. 38).

The craniofacial (hemifacial) microsomia is not associated with mental retardation, except in rare cases in which there may coexist disorders of the central nervous system. The newborn usually has no feeding difficulty, and there is no problem with breastfeeding.

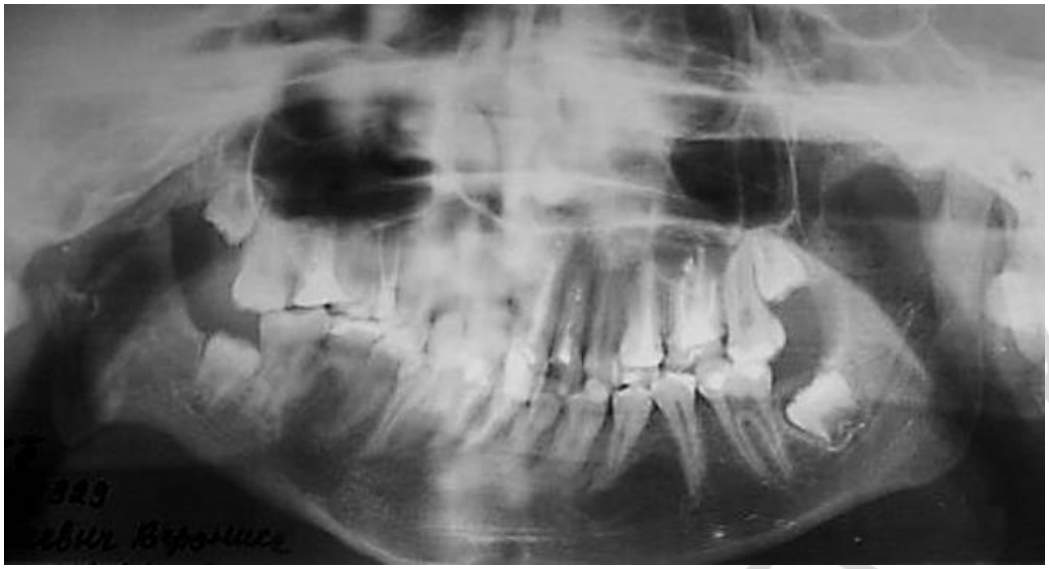
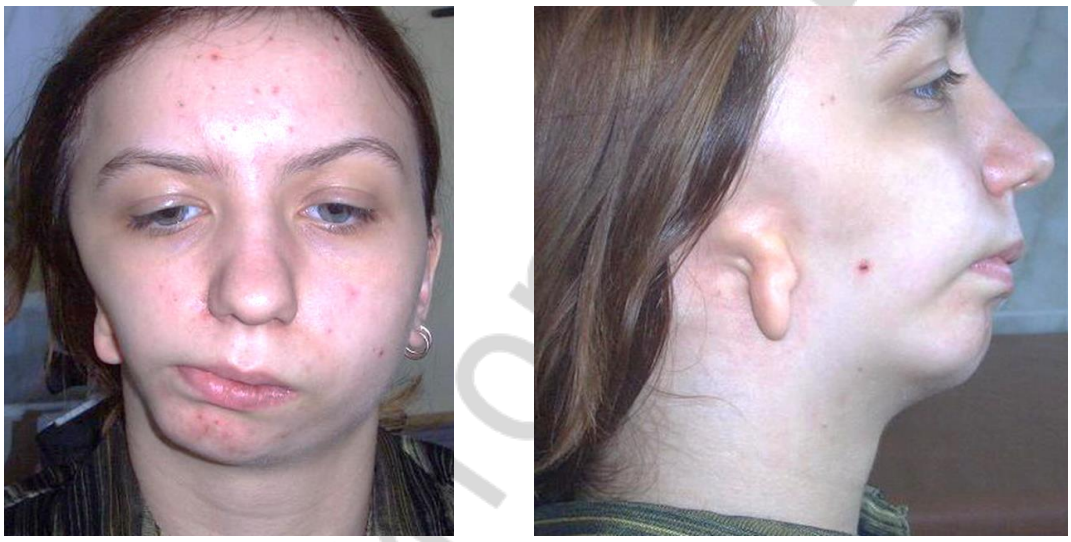


Figure 37. Craniofacial (Hemifacial) Microsomia. Mandible underdevelopment, X-ray picture (illustration is provided by A. K. Korsak)



a ***b***

Figure 38. Craniofacial (Hemifacial) Microsomia. Patient's appearance: *a* — front view; *b* — side view (illustration is provided by A. K. Korsak)

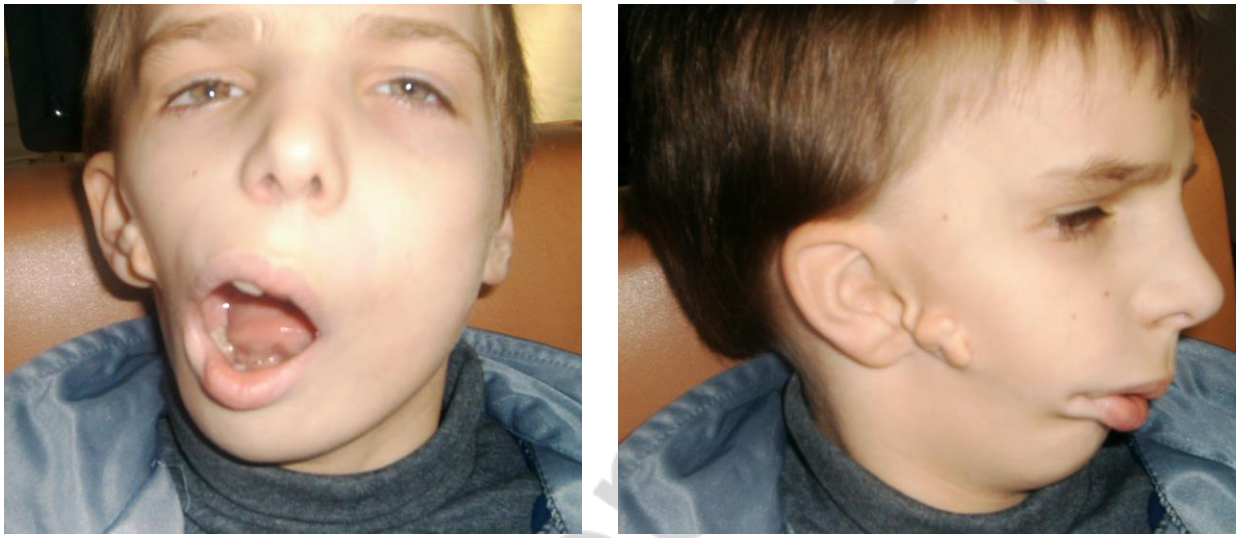
The treatment is long and complex aimed to restoring the size of the lower jaw, plastic of the auricle, functions normalization, etc.

If the pathology is unilateral and the unaffected side of the face is tested and found to be normal, there should be no attempt for surgical repair of the hearing mechanism on the affected side. This will render the subsequent reconstruction of the auricle extremely complex or do it impossible. It shouldn't be performed any surgical intervention in the early stages of child's life except the removal of skin morphomas in front of the auricle or mouth slit correction. Surgical rehabilitation starts commonly from the age of 6 years. Orthodontic care is necessary both before and after surgery. In severe cases in which a large part of the lower jaw is absent, it will be required major surgeries, which are performed in older age as well. Soft tissues correction should be done after face bones rehabilitation.

The external ear is restored last in order to ensure its accurate placement in the correct position.

GOLDENHAR SYNDROME (GS)

The main markers of Goldenhar syndrome are incomplete development of the ear, nose, soft palate, lip, and mandible on usually one side of the body. Additionally, some patients will have growing issues with internal organs, especially heart, kidneys, and lungs. Typically, the organ will either not be present on one side or will be underdeveloped. Note that while it is more usual for there to be problems on only one side, it has been known for defects to occur bilaterally (approximate incidence 10 % of confirmed GS cases).



a ***b***

Figure 39. Goldenhar syndrome. Patient's appearance:
a — front view; *b* — side view (illustration is provided by A. K. Korsak)

Other problems can include severe scoliosis (twisting of the vertebrae), limbal dermoids, and hearing loss (see hearing loss with craniofacial syndromes), and deafness or blindness in one or both ears/eyes.

The treatment is long and complex.

CROUZON SYNDROME (CRANIOFACIAL DYSOSTOSIS)

It is an autosomal dominant genetic disorder known as a branchial arch syndrome. This syndrome affects the first branchial (or pharyngeal) arch, which is the precursor of the maxilla and mandible.

As a result of the changes to the developing embryo, the symptoms are very pronounced features, especially in the face. Low-set ears are a typical characteristic, as in all of the disorders which are called branchial arch syndromes. The reason for this abnormality is that ears on a foetus are much lower than those on an adult. During normal development, the ears "travel" upward on the head; however, in Crouzon patients, this pattern of development is disrupted. Ear canal malformations are extremely common, generally resulting in some hearing loss.

The other notable characteristic of Crouzon syndrome is craniosynostosis, it usually presents as brachycephaly resulting in the appearance of a short and broad head. Exophthalmos (bulging eyes due to shallow eye sockets after early fusion of surrounding bones), hypertelorism (greater than normal distance between the eyes) and psittichorhina (beak-like nose) are also symptoms (Fig. 40). Additionally, external strabismus is a common occurrence. Lastly, hypoplastic maxilla (insufficient growth of the midface) results in relative mandibular prognathism (chin appears to protrude despite normal growth of mandible) and gives the effect of the patient having a concave face and the mesial bite.

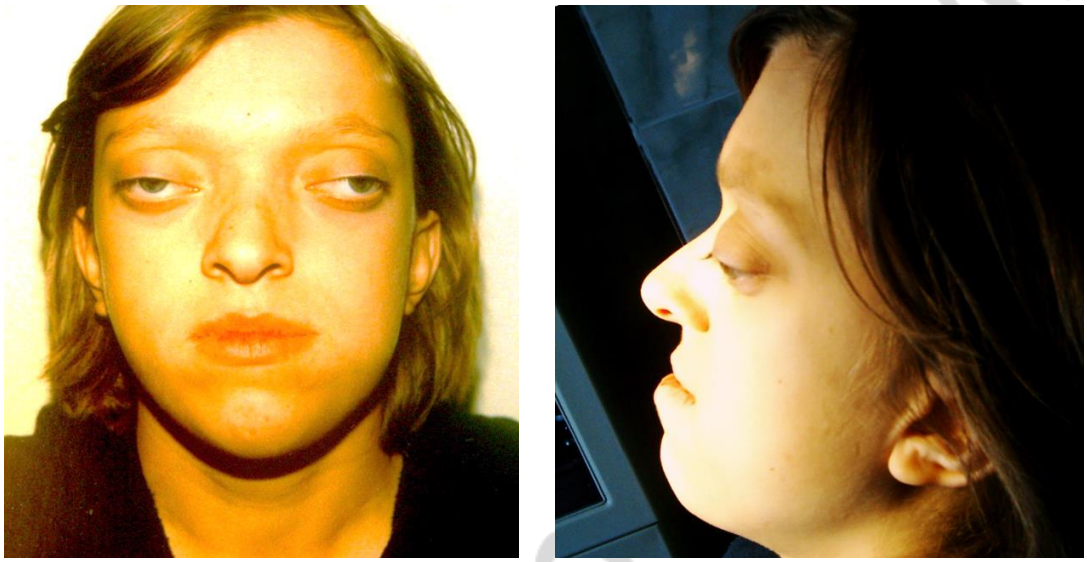


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Figure 40. Crouzon syndrome. Patient's appearance:
a — front view; *b* — side view (illustration is provided by A. K. Korsak)

The treatment is complex: surgical and orthodontic for aesthetic and functional indications.

CLEIDOCRANIAL DYSOSTOSIS (CLEIDOCRANIAL DYSPLASIA)

Cleidocranial dysostosis is a general skeletal condition so named from the collarbone (cleido-) and cranium deformities which people with it often have.

Common features are the following:

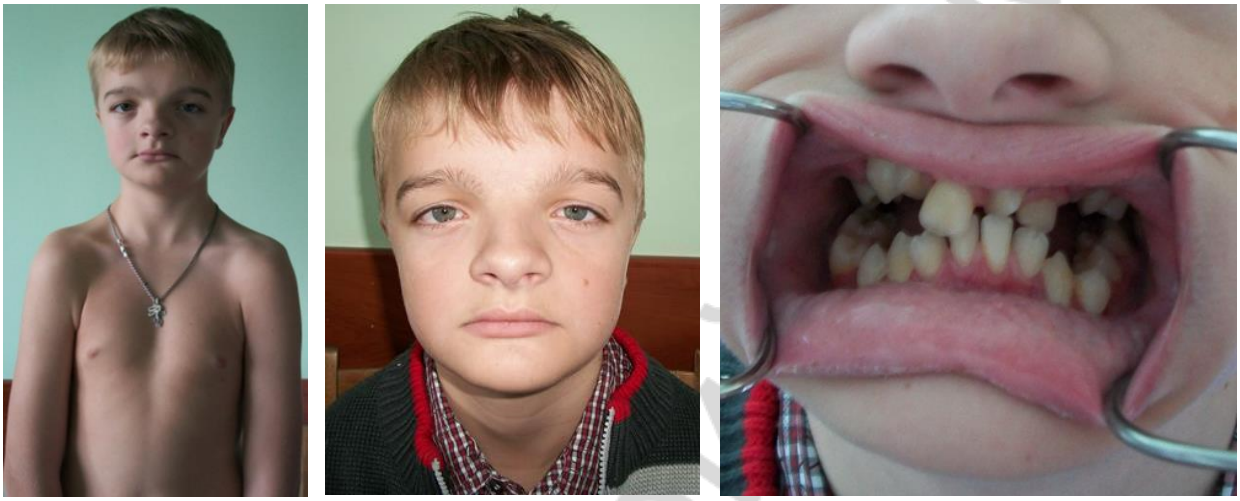
• Clavicles (collarbones) can be partly missing leaving only the medial part of the bone. In 10 % cases, they are completely missing (Fig. 41, *a*). If the collarbones are completely missing or reduced to small vestiges, this allows hypermobility of the shoulders including ability to touch the shoulders together in front of the chest. The defect is bilateral 80 % of cases. Partial collarbones may cause nerve damage symptoms and therefore have to be removed by surgery.

• The mandible is prognathic due to hypoplasia of maxilla (micrognathism) and other facial bones.

• The permanent teeth include supernumerary teeth. Unless these supernumeraries are removed they will crowd the adult teeth in what already may be an underdeveloped jaw. If so, the supernumeraries will probably need to be

removed to make space for the adult teeth. Up to 13 supernumarary teeth have been observed. Teeth may also be displaced (Fig. 41, *c*). Cementum formation may be deficient.

- Failure of eruption of permanent teeth.
- Open skull sutures, large fontanelles.
- Hypertelorism (Fig. 41, *b*).
- Delayed ossification of bones forming symphysis pubis, producing a widened symphysis.
- Short distal phalanges, sometimes causing short and wide fingers.
- Vertebral abnormalities, etc.



a ***b*** ***c***

Figure 41. Cleidocranial dysostosis. Patient's appearance:
a — partly missing clavicles; face, front view; *b* — bite pathology (illustration is provided by A. K. Korsak)

The treatment is complex: surgical and orthodontic for aesthetic and functional indications.

MEDIAN CLEFT OF NOSE

It is formed by the confluence of nasal disorders plates frontonasal tuber during embryonic development of the child. Clinical pathology manifests itself in the form of a split nose tip and a small groove that runs up the back of the nose due to differences alariacartilages. The tip of the nose is wide, flat, nasal septum is shortened. Sometimes hidden cleft extends above the bone of the nose, and even his forehead. Bridge of the nose for these children a broad, flattened, and under the skin bony crevice can be palpated. The orbits of these children are located widely (hypertelorism). The children have a typical wedge-shaped growth of hair on the midline of the forehead (Fig. 42). Median cleft of the nose can be combined with the teeth on the upper jaw abnormalities, cleft lip, congenital fistulas of the lips and other congenital disorders.

The treatment of median cleft nose is surgery, depending on the severity of the disease. In mild cases plasty of alaria cartilage and tip of the nose can be

carried. This operation can be carried out at an earlier age. In more severe cases, a complete (including bone) rhinoplasty, which is carried out, usually after 17 years. In addition, in patients with severe hypertelorism in adults, reconstructive surgery on the frontal bone of the upper jaw, cheek bones to normalize the shape of the face may be performed. These rare and complex operations are conducted in several large clinics of Maxillofacial Surgery.

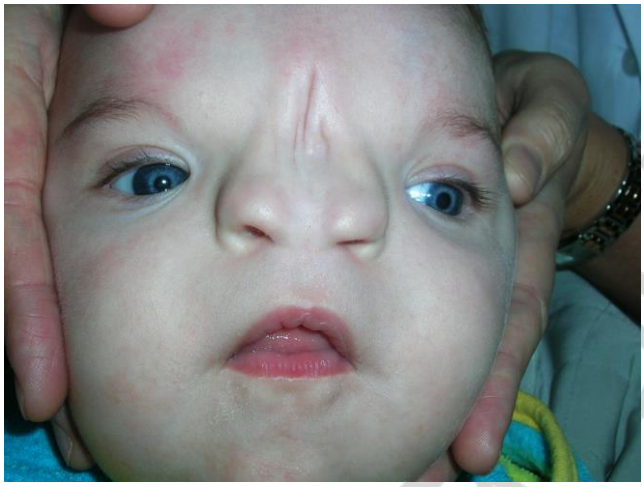


Figure 42. Median cleft of nose. Patient's appearance (illustration is provided by A. K. Korsak)

TRANSVERSE FACIAL CLEFT (MACROSTOMIA)

It is a congenital abnormality that manifested by unusually wide mouth. It is unusual for macrostomia to occur on its own and it is included as a symptom for many diseases including craniofacial microsomia. The pathology results from improper development and fusion of the mandibular and maxillary processes. This can lead to a variety of abnormalities involving skin, subcutaneous tissue, facial muscles, and the mucous membrane. The severity of each abnormality can vary from minor to severe. The origin of the disorder is not yet fully understood, it could have multiple causes.

The treatment is surgical correction in the preschool years.

CONGENITAL CLEFT LIP AND PALATE (OROFACIAL CLEFT)

It is a group of congenital conditions that includes cleft lip, cleft palate, and both together.

Congenital cleft lip and palate are divided into (Fig. 43, 44):

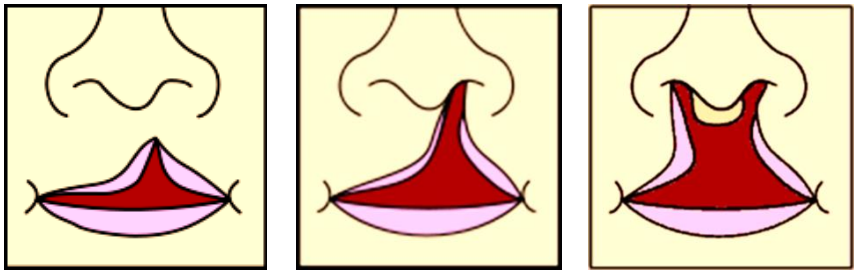
1) isolated congenital cleft lip (hidden, partial (incomplete), complete), uni- and bilateral;

2) isolated congenital cleft soft and hard palate (hidden, partial (incomplete), complete);

3) complete congenital cleft lip, alveolar process and palate (uni- and bilateral);

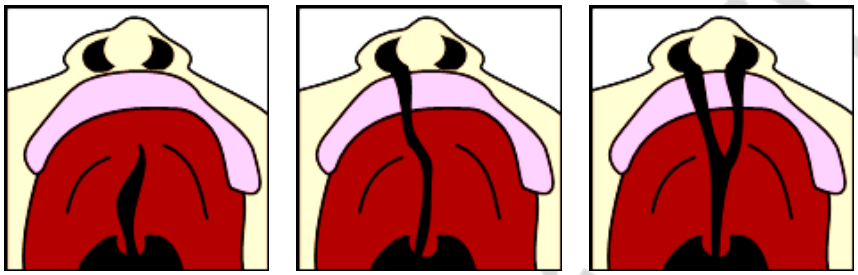
4) combined congenital cleft lip, alveolar process and palate.

Congenital cleft lip and palate is accompanied by anatomical and functional changes.



a ***b*** ***c***

Figure 43. Types of the cleft lip:
a — unilateral incomplete cleft lip; *b* — unilateral complete cleft lip; *c* — bilateral complete cleft lip



a ***b*** ***c***

Figure 44. Types of the cleft palate:
a — incomplete cleft palate; *b* — unilateral complete cleft lip and palate; *c* — bilateral complete cleft lip and palate

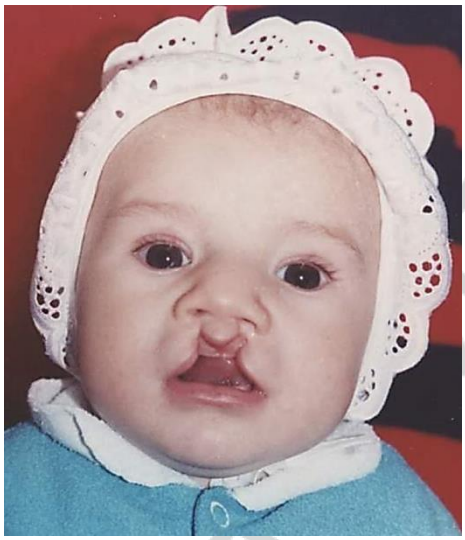


Figure 45. Bilateral cleft lip and palate. Patient's appearance (illustration is provided by A. K. Korsak)

Anatomical changes in congenital clefts of lip and palate are the following (Fig. 45):

- the splitting of the upper lip;
- the shortening of the upper lip;
- typical nose deformity (due to lack of soft tissues);
- the splitting and deformation of the alveolar bone;
- malformations of tooth development;
- cleft palate;
- shortening the soft palate;
- extension of the oropharyngeal ring.

Primary functional disorders (due to lack of tightness in the oral cavity and communication of the oral cavity and the nasal cavity) in congenital cleft lip and palate affect breathing, sucking, swallowing, speeches, hearing (it occurs due to middle ear inflammation after nasopharyngitis and eustachitis), chewing, etc. Dysfunction is most pronounced when complete cleft lip and palate. Secondary functional changes develops with the age of the child and manifest themselves in changes in the mental state due to the esthetic disorders and decreased immunity due to the

development of frequent concomitant inflammatory processes of the nasopharynx, trachea, bronchi, gastrointestinal tract, middle ear, etc.

The treatment should be complex and requires the participation of many specialties for child care.

Feeding the newborn with cleft lip and palate is difficult due to the functional disorders. The best choice is breast feeding but methods of feeding depend on the severity of anatomical disorders.

Newborns with isolated cleft lip can and should suck the mother's breast. When hidden and incomplete cleft lip the child sucks the breast, pressing the nipple to the normally developed alveolar process, compensating for the inadequacy of the lips by the tongue. With a complete cleft lip to create tightness it is necessary to reduce the edges of the cleft with mother’s fingers.

Children with hidden cleft soft palate can and should be fed in natural way. Children with isolated incomplete cleft palate mostly can also suck their mother’s breasts. To prevent food from entering the airways during feeding the child should be kept upright or semi vertical. The children with complete cleft lip and palate often cannot suck the mother’s breast without special devices (obturators). Its use improves feeding ability, and further benefits normalization of speech function. A baby can also be fed from a large elastic nipple or a nipple with a “petal” (a variant “nipple in a nipple”). In case of failure, the child can be fed from a spoon or pipette in semi vertical position.

The use of the gastrointestinal probe for feeding children with cleft lip and palate is inadmissible. This leads to the extinction of the function of sucking and swallowing.

Treatment at a speech therapist is absolutely necessary and it is divided into preoperative and postoperative course. The treatment should be started in children from an early age (1–2 years old and no later than 2.5 years) and finish only after normalization of the function of speech.

The purpose of the surgery is to restore the correct anatomical shape of the upper lip, palate and nose, which will contribute to the normalization of functions and esthetics. Surgical correction starts from *cheiloplasty* (upper lip plasty) at the age of 4–8 months. In case of bilateral cleft lip the cheiloplasty can be performed in one or in two stages with an interval of 2–2.5 months. The tasks of the operation are the following:

- to close the cleft lip;
- to extend the upper lip;
- to eliminate the nose deformation.

Modern methods of cheiloplasty allow to get good anatomical results. However, with age in 70–80 % of patients, various deformities of the lip and nose are detected. Corrective surgeries in the upper lip and the vestibule of the oral cavity give good results when they are carried out starting from 4–6-year-old child. Operations to correct the nose deformation for aesthetic reasons are carried out at the age of 12–14 years (sometimes later) when the maxillofacial area growth is finished.

In case of cleft palate its repair should be done in two steps: *veloplasty* (soft palate plasty) at the age of 4 months – 2.5 years and *uranoplasty* (hard palate plasty) at the age of up to 4–5 years. The tasks of the operation are the following:

- to close the cleft palate;
- to extend the soft palate;
- to narrow the oropharyngeal ring.

The earlier the uranoplasty is performed, the faster the functions are normalized, but the more the growth of the upper jaw is disturbed.

Orthodontic care for children with cleft lip and palate has the following tasks:

1. Creating conditions for optimal feeding during newborn and in infancy.
2. Normalization of the shape and size of the upper alveolar process and dental arch as before, and after surgery.
3. Elimination of congenital underdevelopment of the upper jaw, both before and after surgery.
4. Manufacturing orthodontic appliances that provide optimal conditions for wound healing and tissue formation in the postoperative period.
5. Controlling the growth of the upper jaw and ensuring the formation of the normal occlusion after surgery.
6. Elimination of tooth position anomalies.
7. Prevention of secondary deformities of the mandible and lower dentition.
8. Dental prosthetics according to functional and aesthetic indications.

Pediatrician, ENT doctor, anesthetist, neuropsychiatrist, pediatric dentist (high LCI is common in such patients), audiologist, medical geneticist, exercise therapy methodologist are the other specialists which are necessary for the complex care of the children with cleft lip and palate.

PREVENTION OF CONGENITAL PATHOLOGY OF THE MAXILLOFACIAL AREA

Congenital malformation prevention includes:

1) healthy lifestyle;

2) medical and genetic counseling;

3) prenatal diagnosis (ultrasound, amniocentesis, chorionic villus sampling, fetal karyotype determination, etc.).

Ultrasound diagnosis should be carried out in 10–13 weeks, from 16 to 22 per week in the third trimester of pregnancy by a doctor. As a result, ultrasound can detect up to 60–70 % of the structural defects of the fetus (cerebrospinal hernia, missing limbs, heart defects, cleft lip and palate, and others.). In addition, analysis of amniotic fluid or placental biopsy and some biochemical indices of maternal blood (60 % efficiency) can be carried out for the prenatal diagnosis of congenital malformations.

ODONTOGENIC JAW CYSTS IN CHILDREN. PATHOGENESIS, CLINICAL MANIFESTATIONS, RADIOLOGICAL SIGNS, DIAGNOSIS AND TREATMENT

Odontogenic cysts — benign tumor-like formations, that consist of mature cellular elements. They are usually found at the age of 7–12 years. About 90 % of children have radicular tooth-containing cysts. Cysts develop in lower jaw 3–4 times more often than in upper one.

A cyst is hollow formation, lined with connective tissue and filled with a yellow liquid containing cholesterol. Cyst membrane consists of 3 layers: the outer — dense connective tissue, medium — granulation tissue, and inner — stratified squamous epithelium (Fig. 46).

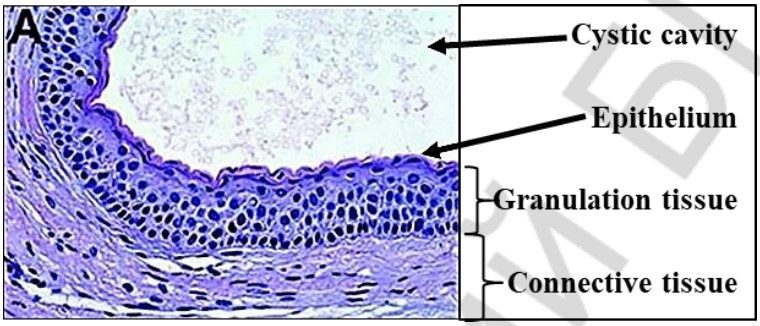


Figure 46. Structure of odontogenic cyst

Classification of cysts of the jaws according to it pathogenesis:

1) Inflammatory cysts:
– radicular (periapical);
– radicular tooth-containing;
– paradental cysts.

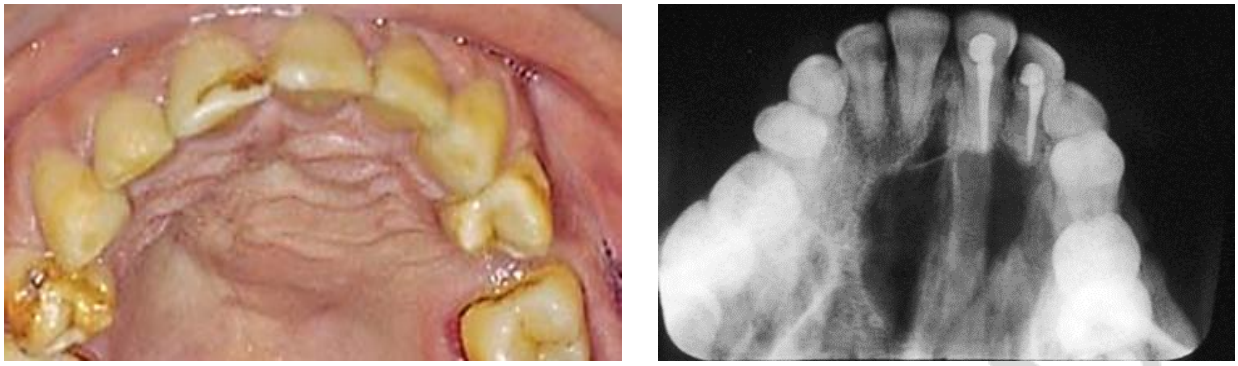
2) Developmental cysts:
– follicular (dentigerous);
– eruption cyst;
– primordial (keratocyst).

The widest spread inflammatory cysts in children are radicular tooth-containing and periapical cysts.

PERIAPICAL (RADICULAR) CYST

Periapical cyst usually occurs in older children from permanent maxillary incisors. The cause is chronic infection of tooth, which spreads to apex and into adjacent bone. It leads to apical periodontitis, granuloma formation and then cyst. These cysts are situated on apex of tooth and usually less than 1 cm.

Clinical features are the following. Radicular cyst can be asymptomatic and diagnosed incidentally during X-ray examination. Alveolar bone at area of causative teeth is thickened, mucosa is not changed in color or have expressed vascular pattern, palpation is painless. Causative tooth has a deep restoration or large carious lesion and painless percussion (Fig. 47).



a ***b***

Figure 47. Radicular cyst from 2.1 and 2.2 teeth:
a — Clinical view; *b* — X-ray picture

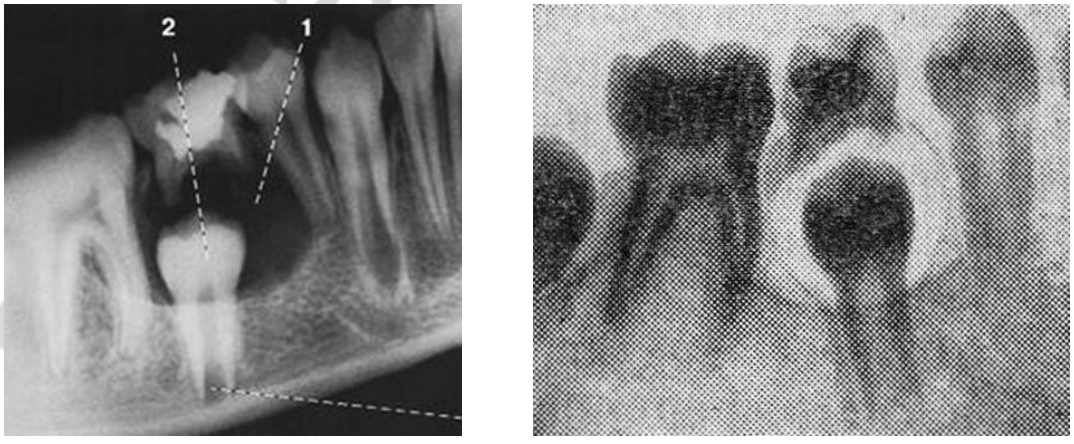
Periapical cysts are divided into *festering* and *non-festering*, according to clinical features. In case of festering radicular cyst, a clinical picture of acute periostitis or osteomyelitis develops.

The leading method of diagnosing radicular cysts is *radiography* (intraoral x-rays of the teeth, orthopantomography, x-ray of the jaws and paranasal sinuses). On the x-ray, the radicular cyst looks like round, unilocular formation with clear borders connected with root of causative tooth. A simple additional method of diagnosing the root cyst is a diagnostic puncture. Periapical cyst contains a yellowish liquid with cholesterol crystals.

Surgical treatment is cystectomy (see treatment of cysts), the root canals of the "causative" tooth are sealed before operation, and during surgery apex of the tooth is resected.

RADICULAR TOOTH-CONTAINING CYST

"Tooth-containing" cyst is radicular cyst from the milk tooth, which contains germ of permanent tooth. The pathogenesis is the same as radicular cyst at early stages. Further, as a result of growth of the cyst, its membrane covers and displaces germ of permanent tooth. Most often the cysts are developing from temporary lower molars (90 %).



a ***b***

Figure 48. X-ray of radicular "tooth-containing" cyst from temporary lower molars:
a — case 1; *b* — case 2

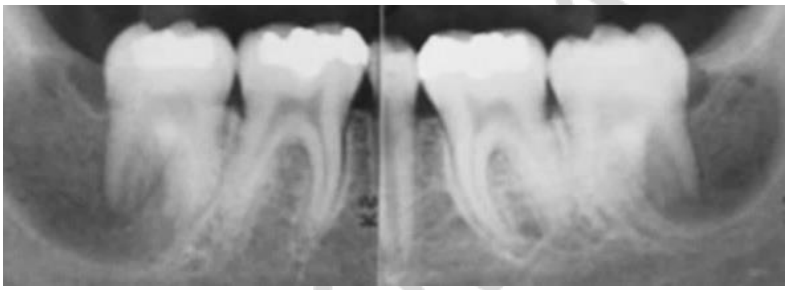
Clinical features are practically no different from root cysts, except that "causative" is a temporary tooth. Radiological findings are: oval foci of destruction of bone with clear borders from root of milk "causative" tooth, which contains germ of permanent one. Crown of permanent tooth is included in cystic cavity to neck and its root is outside the cyst.

The treatment is cystotomy (see treatment of cysts) with mandatory removal of the "causative" milk tooth during the operation.

PARADENTAL CYSTS

Paradental cyst often occurs in adults as a result of complicated eruption 38 and 48 teeth. Typical localization of the paradental cyst — in the region of the angle, the branch of the jaw. It is always connected with the wisdom teeth. The pathogenesis of it are the following. Behind lower wisdom teeth forms bone pocket with epithelial cells in it, that proliferate and turn in cyst. Morphologically the paradental cyst is the same as the root cyst.

On the x-ray paradental cyst looks like rounded focus of bone destruction with clear borders behind lower wisdom tooth, which part of crown is outside cyst (Fig. 49).



a ***b***

Figure 49. X-ray of paradental cysts from lower wisdom molars:
a — right side; *b* — left side

The treatment of the paradental cysts is extraction of wisdom teeth and cystectomy.

FOLLICULAR (DENTIGEROUS) CYST

Follicular cyst is slow growing benign and developmental odontogenic cyst. A hyperplastic follicle has a very similar appearance to a dentigerous cyst. If the lesion edge is more than 5 mm from the enamel, it is classified as a dentigerous cyst. Over 75 % of all cases of follicular cyst are located in the mandible. It is the second most common cysts after periapical cysts.

Pathogenesis. Dentigerous cyst arises from reduced enamel epithelium of the dental follicle of an unerupted tooth. It is formed by hydrostatic force exerted by the accumulation of fluid between reduced enamel epithelium and the tooth crown of unerupted teeth. Cyst encloses crown and is attached to neck at cementoenamel junction (Fig. 50). Follicular cyst exclusively occurs in permanent dentition. The source of development are often germs of: wisdom teeth, upper canines, premolars and supernumerary teeth.

X-ray examination revealed unilocular pericoronal rounded foci of bone destruction with clear borders centred on an unerupted tooth. The border of the cyst is continuous at the cemento-enamel junction of the unerupted tooth (Fig. 51). As cyst grows, it will displace the associated tooth apically. It may displace or resorb adjacent teeth as it enlarges.

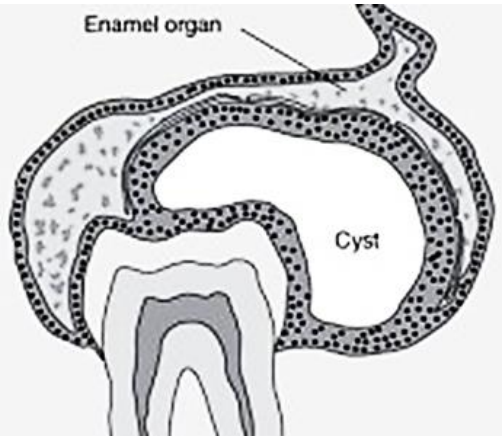


Figure 50. Scheme of dentigerous cyst

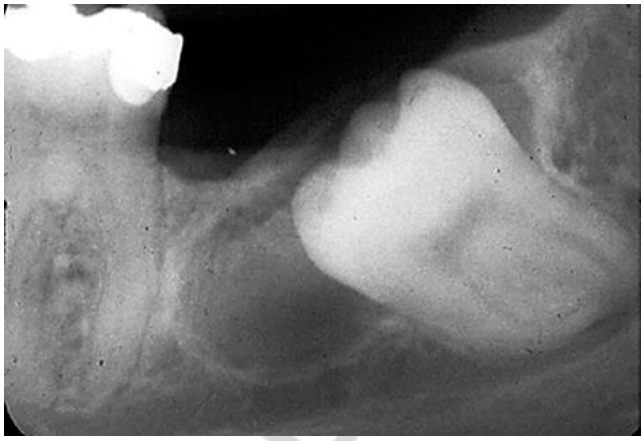


Figure 51. X-ray picture of dentigerous cyst

ODONTOGENIC KERATOCYST (PRIMORDIAL CYST)

Odontogenic keratocyst is benign cystic neoplasms involving jaw. It is locally aggressive and tend to recur after excision. 70 % of primordial cysts occur in the area of lower wisdom teeth and ramus region.

Pathogenesis. It arises from reduced enamel epithelium, dental lamina rests and malassez rests of supernumerary tooth germ. The thin fibrous cyst membrane is consist of squamous keratinizing epithelium. There are fluid and keratin mass in the cyst cavity.

Clinical presentation. This cyst is often discovered incidentally. But jaw swelling and pain can also present. On the X-ray there is focus of unilocular oval destruction of bone with clear smooth borders (Fig. 52). Resorption of the root is often present. Mandibular cysts grow along of the bone (anteroposterior). In maxilla, they expand into maxillary sinus.

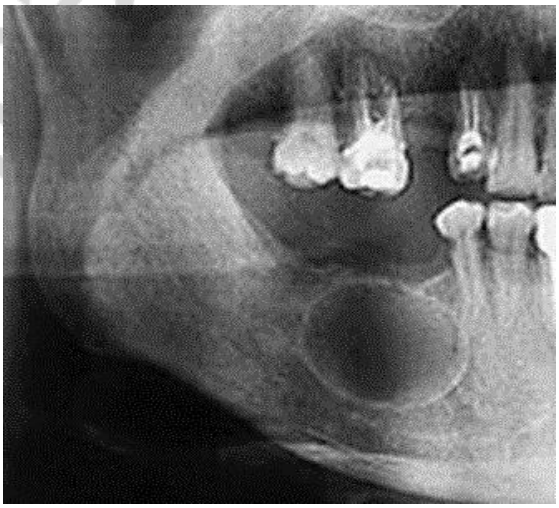


Figure 52. X-ray of odontogenic keratocyst of mandibule

ERUPTION CYST

The eruption cyst is the soft tissue analogue of the follicular cyst. It is developmental cyst. This cyst occurs as a result of fluid accumulation within the follicular space of an erupting tooth.

Clinical features. It appears as soft blue to dark red mass on the alveolar process in place of any erupting tooth, particularly molars and canines (Fig. 53). Radiographically it is negative as it is soft tissue cyst above the crown of unerupted tooth.

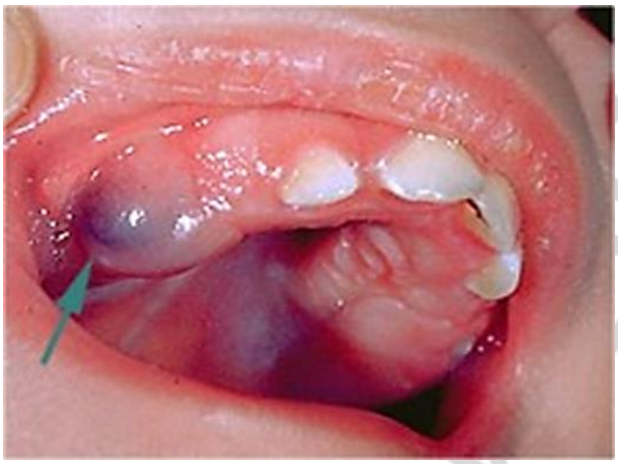


Figure 53. The eruption cyst of second upper right temporary molar

The treatment isn't needed, because the tooth erupts through the lesion, the cyst disappears spontaneously without complication.

SURGICAL TREATMENT OF CYSTS

There are two main surgical ways of treating the jaw cysts described by Parch in 1895. It is a **cystotomy** (so-called Parch I) and **cystectomy** (Parch II and Parch III).

Indications to "causative" teeth extraction:

– all temporary teeth;

– all destroyed (roots) permanent teeth;

– multirooted permanent teeth;

– permanent teeth, which roots are more than 50 % appear in cyst cavity.

Operation of tooth extraction should be performed only during the main operation.

Cystotomy

The aim of operation is a creation of a wide connection between the cyst cavity and oral cavity to stop growth of cysts and bone tissue regeneration, preservation of teeth germs. This operation is easier then cystectomy and widely used in pediatric practice.

Indications to operation of cystotomy are the following:

– radicular tooth-containing cyst;

– large cysts (except maxillary cysts, that grew into maxillary sinus);

– threat of a pathological fracture of the jaw;
– suppurative cysts (acute stage);
– serious condition of the patient.

Cystotomy technique steps (Fig. 54):

1. Anesthesia.
2. Incision (oval or trapeziform).
3. Removal of bone.
4. Removal of front cystic membrane.
5. Irrigation of cystic cavity by antiseptic fluids.
6. Suturing (mucoperiosteal flap is turned into cystic cavity and sutured to it.
7. Packing (tampon with iodoform).
8. Maintenance of cystic cavity: instruct the patient to clean and irrigate the cavity regularly with oral antiseptic.

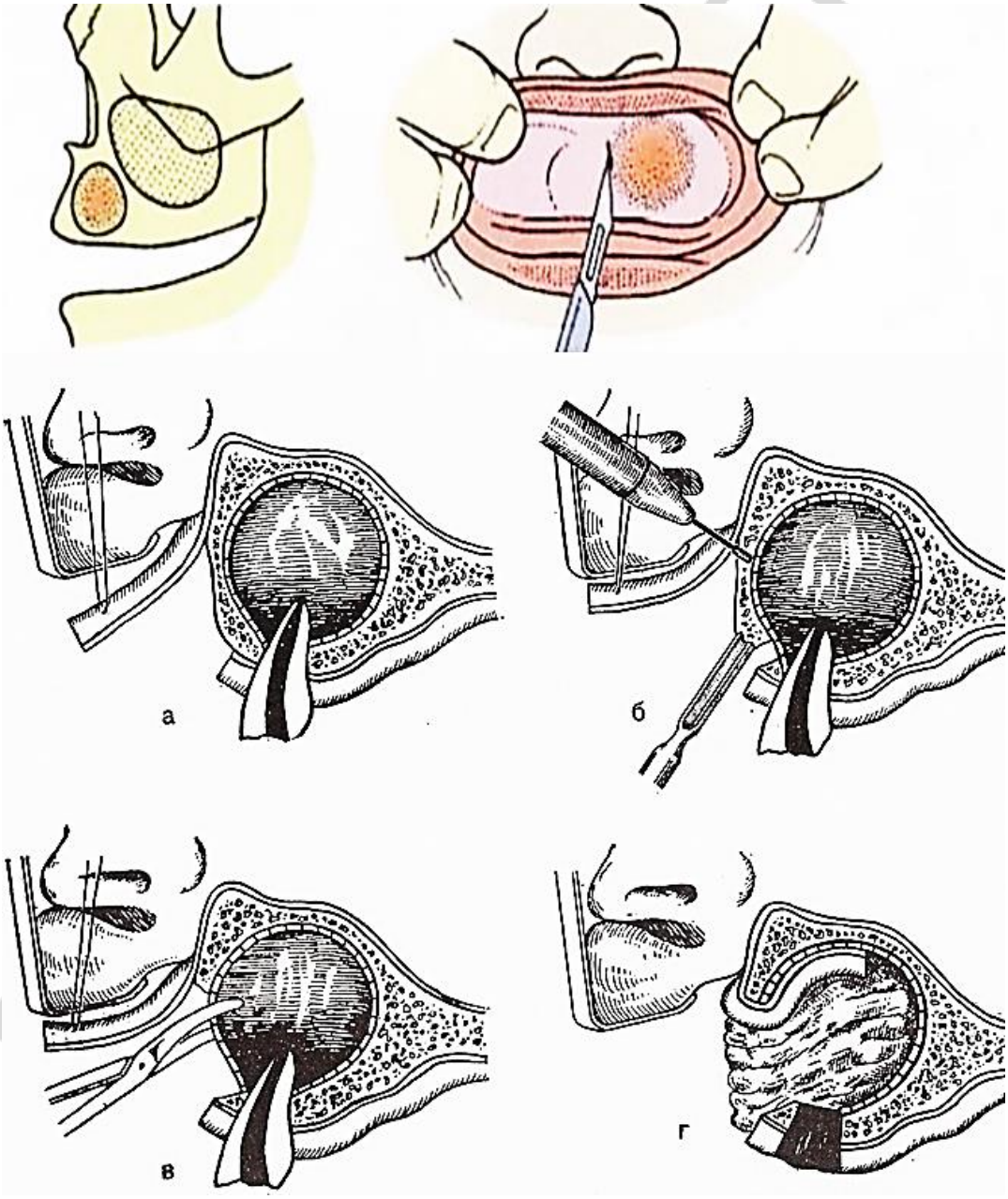


Figure 54. Stages and technique of cystotomy

Cystectomy

This operation is recommended in all other cases: radicular, follicular paradental cysts and odontogenic keratocyst. The aim of cystectomy is a *complete removal of cyst membrane* and suturing the wound.

Steps of cystectomy operation (Parch II):

1. Anesthesia.
2. Incision (oval or trapeziform), it size should be more then front wall of the cyst to 4–5 mm.
3. Removal of bone.
4. Enucleation and removal of cystic membrane together with its contents (by dental curette).
5. Irrigation of cystic cavity by antiseptic fluids.
6. *Resection* of opened roots tops *or tooth extraction*.
7. Suturing (mucoperiosteal flap is replaced and sutured in it proper location).
8. Management of wound: instruct the patient to clean and irrigate the suturing wound regularly with oral antiseptic.

Parch III: full removal of the cystic membrane and secondary healing under the cotton.

Prevention of odontogenic inflammatory cysts is timely and qualitative treatment of temporary and permanent teeth with complicated caries and pericoronitis.

BENIGN TUMORS AND TUMOR-LIKE FORMATION OF THE SOFT TISSUES IN THE MAXILLOFACIAL REGION IN CHILDREN. CLINICAL MANIFESTATIONS, DIAGNOSIS, TREATMENT

Tumor (neoplasms) is a new growth of tissue in which cell multiplication is uncontrolled and progressive.

Tumors are divided into *benign* and *malignant*. Benign means not dangerous to health or well-being; malignant means dangerous to health. The term “malignant” is also used to describe dangerous medical and psychological conditions other than cancer. Some malignant conditions are very treatable, whereas some benign conditions may be life-threatening.

Differences of malignant and benign tumors are the following.

1. Invasion of nearby tissues. Malignant tumors have poor boundaries. Unlike benign tumors which can *press on* nearby structures, malignant tumors can *penetrate into* nearby structures.
2. Ability to spread (metastasize). Unlike benign tumors, malignant tumor cells have the ability to break away from the tumor and travel either locally, or through the bloodstream or lymphatic system. Most deaths from cancer (roughly 90 percent) occur due to this ability of malignant tumors to spread.
3. Likelihood and location of recurrence. Benign tumors may recur after removal, in the region where they were first located. In contrast, malignant tumors

recur more often, and may recur locally (as with benign tumors), regionally (for example, in lymph nodes near the original tumor), or distantly (in organs or regions far from the original tumor).

4. Cells. There are many important differences between benign and malignant cells. The malignant cells differ in their stickiness (attachment to surrounding cells). They also differ in their ability to spread (they lack "adhesion molecules" which keep normal cells in place), the way in which the cells communicate with each other, in their growth, and ultimately in their immortality or lack of cell death over time.

Tumors of maxillofacial area in children have the following features:

– dizontogenetic nature ;
– capacity for spontaneous regression;
– faster growth compared with adults;
– connective-tissue tumors predominate;
– soft tissue tumors are more common in younger children;
– tumors of bones are more common in older children;
– complexity of interpreting histology.

Tumors of maxillofacial area in children are divided into:

– soft tissue tumors:
a) epithelial nature;
b) the connective tissue nature;
– salivary glands tumors:
a) epithelial nature;
b) the connective tissue nature;
– facial bones tumors:
a) osteogenic nature;
b) nonosteogenic nature;
c) odontogenic nature.

All children with tumors of maxillofacial area should be under the constant follow-up supervision from the day of tumor diagnosis to 15 years, after which the patient is transferred to an adult network or supervision is finished (if anatomical structure and functions are normal).

PAPILLOMA

Papilloma (plural papillomas or papillomata) (papillo- + -oma) is a benign epithelial tumor growing exophytically (outwardly projecting) in nipple-like and often finger-like fronds. In this context papilla refers to the projection created by the tumor, not a tumor on an already existing papilla (such as the nipple).

When used without context, it frequently refers to infections (squamous cell papilloma) caused by human papillomavirus (HPV), such as warts. Oral verruca vulgaris, or oral warts, are exophytic papillomatous lesions indistinguishable clinically from oral squamous cell papillomas. Like their skin counterpart, the common wart (verruca vulgaris), they are a viral disorder associated with

the human papillomavirus (HPV) and may be spread to the oral cavity in children through autoinoculation by finger or thumb sucking.

Although the histopathologic differences between squamous papilloma and verruca vulgaris are subtle, these lesions are distinguishable from one another. Histologically, the papilloma is seen as a proliferation of the spinous cell layer in a papillary pattern, often with hyperkeratosis, acanthosis, and basilar hyperplasia. Mitotic figures may be prominent. The supporting fibrous connective tissue stroma often contains prominent numbers of small blood vessels as well as an inflammatory cell infiltrate. HPV may, however, be seen in squamous cell papillomas.

The presence of papillomatosis often with convergence of rete ridges centrally, hyperkeratosis (either hyperparakeratosis or hyperorthokeratosis, or both) a coarse keratohyalin granular cell layer and vacuolated cells with pyknotic nuclei (koilocytes) may be used to differentiate verruca vulgaris from a squamous papilloma. Hence, from the above discussion both squamous papilloma and verrucous vulgaris should probably be considered benign, virus-induced epithelial hyperplasias and the identification of HPV does not appear to offer any diagnostic advantage because the histopathologic features alone allow for differentiation of one from the other.

The treatment of either the oral squamous papilloma or verruca vulgaris is best accomplished by complete surgical excision of the lesion, including the base, cryosurgery, electrocoagulation and using antiviral remedies.

NEVUS

Nevus (or nevi if multiple) is a nonspecific medical term for a visible, circumscribed, chronic lesion of the skin or mucosa. The term originates from nævus, which is Latin for “birthmark”, however, a nevus can be either congenital (present at birth) or acquired.

Congenital nevi are present at birth and result from a proliferation of benign melanocytes in the dermis, epidermis, or both. Occasionally, nevi that are not present at birth but are histologically identical to congenital nevi may develop during the first 2 years of life. This is referred to as congenital nevus tardive.

The treatment is complete removing the tumor with some millimeters of heavy skin near the nevus area.

It is necessary to remember that to clarify the diagnosis of nevus incisional biopsy is contraindicated (NO INCISIAL BIOPSY, ONLY COMPLETE REMOVING!!!!) due to risk of malignancy.

Signs of nevus malignancy are the following:

– the color changes;
– it gets smaller or bigger;
– it changes in shape, texture, or height;
– the skin on the surface becomes dry or scaly;
– it becomes hard or feels lumpy;
– it starts to itch;
– it bleeds or oozes.

TRICHILEMMAL CYST

A trichilemmal cyst, also known as a wen, pilar cyst or isthmus-catagen cyst, is a common cyst that forms from a hair follicle. They are most often found on the scalp. The cysts are smooth, mobile and filled with keratin, a protein component found in hair, nails, skin, and horns. They are, however, clinically and histologically distinct from Trichilemmal Horns, which are much more rare and not limited to the scalp. Trichilemmal cysts may run in families and they may or may not be inflamed and tender, often depending on whether they have ruptured. Rarely, these cysts may grow more extensively and form rapidly multiplying trichilemmal tumors, also called proliferating trichilemmal cysts, which are benign but may grow aggressively at the cyst site. Trichilemmal cysts can become cancerous very rarely.

Trichilemmal cysts are derived from the outer root sheath of the hair follicle. Their origin is unknown, but it has been suggested that they are produced by budding from the external root sheath as a genetically determined structural aberration. They arise preferentially in areas of high hair follicle concentrations, therefore, 90 % of cases occur on the scalp. They are solitary in 30 % of cases and multiple in 70 % of cases.

Histologically, they are lined with stratified squamous epithelium that lacks a granular cell layer and are filled with compact “wet” keratin. Areas consistent with proliferation can be found in some cysts. In rare cases, this leads to formation of a tumor, known as a proliferating trichilemmal cyst. The tumor is clinically benign, although it may display nuclear atypia, dyskeratotic cells, and mitotic figures. These features can be misleading, and a diagnosis of squamous cell carcinoma may be mistakenly rendered.

Surgical excision is required to treat a trichilemmal cyst.

THYROGLOSSAL DUCT CYST

It is an uncommon developmental cyst. The thyroid gland rudiment develops around the fourth embryonic week, between the derivatives of the first and second branchial arches at the base of the tongue which is recognized later as the foramen cecum. A hollow stalk known as the thyroglossal duct, extends from this foramen cecum through the neck to the thyroid gland. By about the tenth week this duct breaks up and disappears, but cysts may form from residues of this duct at any point along its course.

It can occur anywhere in the midline along the course of the embryonic thyroglossal duct, which extends from the foramen cecum of the tongue into the deep fascia near the thyroid isthmus. Common sites of occurrence are floor of the mouth, area around the hyoid bone and thyroid cartilage region.

Clinical picture: classically swellings are seen in the midline, slight lateral positional variations may be seen. On palpation the swellings are soft, tender and movable. Pathognomonic is the movement of the cyst during swallowing and

protrusion of the tongue. At times they may cause dysphagia, dysphoria or dyspnea. The size may vary from 1 to 5 cm in diameter.

Pathology: the epithelial lining is variable, cysts present above the level of the hyoid bone are lined with stratified squamous epithelium while those present below the hyoid bone are lined with ciliated respiratory type or columnar epithelium. However, a single cyst may show different epithelium from one area to another. In the fibrous wall, lymphoid tissue, thyroid tissue or mucous glands may be seen.

The treatment. Complete radical surgical excision of the cyst along with its tract is essential to prevent recurrence. Owing to its proximity to the hyoid bone, a central part of the hyoid bone, approximately 1 to 2 cm may require to be removed during surgery.

BRANCHIAL CLEFT CYST

Branchial arch remnants can give rise to cervical cysts, which are more common than intraoral branchial cysts. It has been postulated that these cysts develop from:

– epithelial remnants of the branchial clefts and pouches;

– residual cervical sinus epithelium;

– salivary gland inclusions in parotid lymph nodes which undergo cystic changes;

– cystic changes within cervical lymph nodes of epithelial inclusions, hence the term benign lymphoepithelial cyst.

The most common location of them is at the angle of the mandible anterior to the sternocleidomastoid muscle or the parotid region, less commonly they are seen in the floor of the mouth and the ventral surface of the tongue. Other sites of location are the soft palate, anterior palatine pillar and buccal vestibule.

Clinical features: the lesions vary in size. The neck lesions may reach a size of 10 cm. They are seen as a soft, fluctuant mass at or above cervical sites. Some may develop a fistulous tract and drain externally. The *intraoral lesions* range in size from 1 to 10 mm. They are submucosal and freely mobile, often they may be confused as mucoceles, lipomas or irritation fibromas, at times, they may drain intraorally via a fistulous tract.

Pathology: the *cervical cysts* are lined with ciliated or nonciliated, pseudostratified columnar epithelium that may contain goblet cells, beneath the epithelium lymphoid tissue may be seen as typical germinal centers or a diffuse, dense infiltrate of lymphocytes. Lumen will contain mucus and desquamated parakeratolic cells. *Intraoral cysts* are usually lined with stratified squamous epithelium and lumen will contain a watery fluid. Lymphoid tissue envelops the cystic lining.

The treatment: aspiration or drainage of these cysts is followed by recurrence. *They are best treated by complete surgical excision by a cervical or intraoral approach.*

DERMOID AND EPIDERMOID CYSTS

Dermoid and epidermoid cysts are a form of cystic teratoma, which is lined with epithelium and in addition reveals the presence of skin appendages, e.g. hair, sebaceous glands or teeth. These are nonodontogenic developmental cysts that are thought to arise from epithelial rests persisting in the midline after fusion of the mandible and hyoid branchial arches, it is comprised of a combination of ectoderm, mesoderm and endodermal elements. The difference between dermoid and epidermoid is that epidermal cyst does not contain any skin appendages.

In 5 percent of the cases, one of the tissue elements can become malignant.

Lateral dermoids are rarely seen, median dermoids are seen in the midline in the floor of the mouth above the geniohyoid muscle or inferior to the geniohyoid muscle, which may be present, superior or inferior to the mylohyoid muscle.

Clinical features. Swellings may be seen as midline swellings or rarely as lateral swellings, in the floor of the mouth and neck. Those above the geniohyoid muscle, elevate the tongue, causing difficulty with mastication and speech. Those present inferior to the geniohyoid muscle, cause a submental swelling, that has been aptly described as a double chin. They are small in infancy but enlarge over the years to several centimeters in diameter. On palpation, a doughlike feel is appreciated.

The treatment: surgical excision is the best choice of treatment. An intraoral approach is advisable for cysts present in the floor of the mouth. For very large cysts present inferior to the mylohyoid musle, an extraoral approach via a horizontal submandibular incision in the midline may be required for better access.

SEBACEOUS CYST

It contains keratin, not sebum, and originates from sebaceous glands.

Surgical excision of a sebaceous cyst is a simple procedure to remove the sac and its contents completely.

HEMANGIOMA

A hemangioma is a benign vascular tumor (derived from blood vessel cell types). The word “hemangioma” comes from the Greek haema- (αίμα), “blood”; angeio (αγγείο), “vessel”; -oma (-ωμα), “tumor”.

The most common form is infantile hemangioma, known colloquially as a “strawberry mark”. An infantile hemangioma is one of the most common benign tumors of infancy and occurs in approximately 5–10 % of infants. Infantile hemangiomas are composed of an increased number of unique endothelial cells that line blood vessels. They occur more frequently in female (3 : 1), premature and low birth weight infants. Infantile hemangiomas usually appear within the first weeks of life and grow most rapidly during the first three to six months of life. For most hemangiomas, 80 % of infantile hemangioma size is generally reached by 3 months of age. Usually, growth and proliferation is complete and involution commences by twelve months of age, however, involution occurs slowly over many years with a majority of infantile hemangioma regression occurring by five

years of age. Although infantile hemangiomas spontaneously regress over time, some may leave residual redundant fibrofatty tissue, scar, residual telangiectasia, or pigmentary changes, but regression of these tumors on the face we saw only in 2–3 % of cases.

Hemangiomas occur as single lesions mostly. Clinical manifestaions are red/blue lesions that occur in skin, lips, tongue and buccal mucosa. The lesion blanches when compressed. Intraosseous lesions also occur and seen on X-ray as multilocular radiolucency

Lips, tongue and buccal mucosa are the most common locations of the hemangiomas manifest. Damaged lips cause a cosmetics deformation, but also contributes to speech dysfunction and swallowing the mass effect. Involving tongue often determines dysarthria or dysphagia, but can compromise also breathing.

Hemangiomas can be classified as:

– Capillary;
– Cavernous;
– "Ramose";
– Combine;
– Mixed:
  - Telangiectasia;
  - "Wine stain" (macular lesions).

Hemangiomas can lead to:

– bleeding, especially if the hemangioma is traumatized;
– dysphagia, dyspnea (in the digestive or respiratory locations);
– upper airway obstruction;
– secondary infection;
– skin changes (consistency, appearance, or texture of the skin);
– visual disturbances (strabismus, amblyopia);
– ulcer (in 5–10 % of cases);
– psycho-emotional disorders where the hemangioma is large and visible.

Kasabach–Merritt syndrome (KMS) is also known as hemangioma with thrombocytopenia or hemangioma thrombocytopenia syndrome. It is named after two paediatricians (Haig Haigouni Kasabach and Katharine Krom Merritt) who first described the disease. The syndrome is characterized by large or rapidly enlarging vascular tumor, thrombocytopenia, microangiophatic hemolytic anemia, and coagulophathy as a result of platelet and red blood cells trapping and activation of the clotting system within vasculature of hemangioma. The disease is rare (usually infants suffer), can be life-threatening, has a mortality rate of about 30 %.

The following diagnostic methods may be used to confirm KMS diagnosis: blood counts, clotting studies, ultrasound, computerized axial tomography scan, magnetic resonance imaging (MRI), angiography, tumor biopsy. The patients show severe thrombocytopenia, low fibrinogen levels, high fibrin degradation products, and microangiopathic hemolysis.

Treatment of KMS should be complex and include thrombocytopenia and coagulopathy management and vascular tumor control. There is no consensus treatment guideline now.

The treatment of hemangiomas may be *surgical* and *conservative*.

The kinds of surgical treatment of hemangiomas are the following:

– surgical removal of the hemangioma;

– surgical removal of the damaged organ or damaged area;

– tying off the main artery that supplies blood to the hemangiomas;

– sclerosing theraphy — injection of a sclerosing (clotting) medication which causes clotting of the channels;

– laser treatment;

– embolization of the blood vessels (injection of material into the blood vessels to block the blood inflow);

– electrodestruction;

– cryosurgery.

Conservative treatment of the hemangiomas includes:

1. Steroid medications:

– Intralesional steroids — small amount of steroids may be injected directly into hemangiomas — often one or two treatments at 3 to 4 week intervals. This form of treatment is typically used for small hemangiomas (1–3 cm) on the face. The main potential side effects are skin atrophy (thinning) or systemic absorption.

– Topical cortisone - Clobetasol, a strong steroid ointment, applied 2x/day can be used to treat relatively flat hemangiomas, particularly if they are not becoming thick during the growth phase. The main potential side effects are skin atrophy (thinning) or systemic absorption.

– Oral corticosteroids (usually prednisolone) have been used for more than 40 years to treat hemangiomas. They are quite effective at stopping hemangioma growth and actually shrink hemangiomas in approximately 30 % of cases. Oral steroids have many potential side effects. They are typically given for several months and toward the end of treatment the dosage is gradually reduced.

2. Using beta-blockers:

– An oral (systemic) medication called Propranolol: this is classically a heart medication that has been safely used to treat symptomatic hemangiomas. The use of propranolol — a medication normally used for treating high blood pressure — for hemangiomas was first reported in 2008. Propranolol has been reported to stop hemangiomas from growing and causing actual shrinkage of hemangiomas. Studies are underway to determine the safety and efficacy of this treatment option. Potential side effects include low blood sugar, slowing of the heart rate, and lowering of blood pressure. Patients with asthma should not be treated with this medication.

– Topical timolol — a beta-blocker which is licensed as an eye drop for treating glaucoma. It has been shown to be effective in treating hemangiomas that are relatively flat, either because they have not yet thickened or because they have never grown to be thick. Potential known side effects include skin irritation; it

should be avoided in ulcerated hemangiomas or in larger areas of skin as there may be systemic absorption.

3. Topical imiquimod 5 % — has been published for superficial hemangioma treatment, however there is frequently a significant amount of inflammation and crusting that may make treatment difficult. The potential for absorption and immune effects are not known.

LYMPHANGIOMA

Lymphangiomas are malformations of the lymphatic system and classified on a histologic basis into the following types:

– capillary lymphangiomas;
– cavernous lymphangiomas;
– cystic hygroma;
– mixed types (hemlymphangioma, etc.).

The capillary lymphangioma is typically composed of a proliferation of thin-walled, endothelium-lined channels primarily devoid of erythrocytes. The cavernous lymphangioma is characterized by the presence of dilated sinusoidal endothelium-lined vascular channels devoid of erythrocytes. The cystic hygroma is a macroscopic form of the cavernous lymphangioma, with large sinusoidal spaces lined with a single layer of endothelial cells that form multilocular cystic masses of varying sizes.

Lymphangiomas of the oral soft tissues occur most commonly on the tongue, lips, and buccal mucosa. They are often elevated and nodular in appearance and may have the same color as the surrounding mucosa. Treatment is generally not indicated for small oral mucosal lymphangiomas.

Although *cystic hygromas* can occur virtually anywhere in the body and may be found in sites of the oral cavity, such as the tongue, floor of the mouth, they most frequently appear as a mass in the neck, occasionally extending into the mediastinum. Most commonly presenting as an asymptomatic soft tissue mass, they are usually slow growing; however, they may undergo sudden enlargement in the presence of trauma, inflammation, internal hemorrhage, or respiratory tract infection. Large cystic hygromas may encroach on the airway and esophagus, leading to difficulty in swallowing and even causing airway obstruction, dislocation or stenotic asphyxia. Typically they appear in the first few months of life, some may be diagnosed at birth or before the age of 2 years.

The treatment. Early excision is recommended, even in the newborn, because although they are not malignant, they tend to infiltrate into the local tissues in and amongst muscle fibers and nerves making them difficult and hazardous to remove at a later date. There is no satisfactory nonsurgical treatment — a wide variety of sclerosing agents have been tried, but unsuccessfully. Surgical extirpation is difficult and tedious. Every effort must be made to remove the cyst wall as completely as possible to avoid a recurrence.

Cystic Hygroma can also be treated with OK432 (Picibanil) under the ultrasound control in emergency aid department due to risk of sepsis.

LIPOMAS AND OTHER LIPOMATOUS LESIONS

Lipomas are the most common soft-tissue tumor and contain tissue histologically identical to adipose fat. The incidence of lipomas is up to 2.1 per 100 individuals. Lipomas are radiolucent on radiographs and computed tomographic (CT) images and are isointense relative to subcutaneous fat on MR images obtained with all pulse sequences. The classic lipoma is composed entirely of fat, without areas of nodularity or thickened septations. Of note, a substantial percentage of benign lipomas demonstrate nonadipose features. In a study by Kransdorf et al., 31 % (11 of 35) of lipomas showed nonadipose content, which the authors attributed to fat necrosis and associated calcification, fibrosis, inflammation, and myxoid change. Lipoma variants, such as angiolipoma and myolipoma, are another group of tumors that are predominantly fat containing but demonstrate nonadipose features that may be difficult to dismiss as a benign lipoma.

The important differential diagnosis for a benign lipoma includes a well-differentiated liposarcoma, which may also demonstrate a large fat component. It is important to remember that other subtypes of liposarcoma (dedifferentiated, myxoid, and pleomorphic) may contain minimal or no visible fat. Features found to favor a diagnosis of well-differentiated liposarcoma include lesion size greater than 10 cm, presence of thick (> 2 mm) septae (diffuse or focal), presence of globular and/or nodular nonadipose areas or masses, and lesion composition of less than 75 % fat. Well-differentiated liposarcomas must also be distinguished from benign inter- and intramuscular lipomas. Intramuscular lipomas vary greatly in size, can have well-defined or infiltrative margins, and can appear to have septae owing to intermingled muscle fibers. However, the muscle fibers should be isointense to normal muscle on both T1- and T2-weighted MR images and, when viewed in the longitudinal plane, should maintain their native architecture.

In the past, a distinction was made between atypical lipomatous tumors and well-differentiated liposarcomas.

The treatment is the complete surgical removing of this tumor.

FIBROMA

Fibromas (or fibroid tumors or fibroids) are benign tumors that are composed of fibrous or connective tissue. They can grow in all organs, arising from mesenchyme tissue. The term “fibroblastic” or “fibromatous” is used to describe tumors of the fibrous connective tissue. When the term fibroma is used without modifier, it is usually considered benign, with the term fibrosarcoma reserved for malignant tumors.

The ***hard fibroma*** (fibroma durum) consists of many fibres and few cells, e.g. in skin it is called dermatofibroma (fibroma simplex or nodulus cutaneous), might be a special form is the keloid, which derives from hyperplastic growth of scar.

The ***soft fibroma*** (fibroma molle) or fibroma with a shaft (acrochordon, skin tag, fibroma pendulans) consists of many loosely connected cells and less fibroid tissue. It mostly appears in the neck, armpits or groins.

The ***fibroma cavernosum*** or angiofibroma, consists of many often dilated vessels, it is a vasoactive tumor occurring almost exclusively in adolescent males.

The ***cystic fibroma*** (fibroma cysticum) has central softening or dilated lymphatic vessel.

MYOMA

Myoma (myoblastoma or Abrikosov's tumor) is a benign dystontogenetic tumor from immature muscle tissue (myoblasts). It is often found in newborns and infants and is localized in the tongue, alveolar process, etc. The cherry-colored tumor has a round shape, dense, painless on palpation, in some cases on a thin stalk.

The treatment is surgical, i.e. previous excision of the tumor within healthy tissue, regardless of the age of the child. The tumor may recur.

NEUROFIBROMATOSIS

The term refers to several genetically inherited conditions that are clinically and genetically different and carry a high possibility of tumor formation. This disorder is divided into neurofibromatosis type 1, neurofibromatosis type 2 and schwannomatosis. The neurofibromatoses are considered as members of the neurocutaneous syndromes (phakomatoses).

Neurofibromatosis type I, in which the nerve tissue grows tumors (neurofibromas) that may be benign and may cause serious damage by compressing nerves and other tissues.

Neurofibromatosis type II, in which bilateral acoustic neuromas (tumors of the vestibulocochlear nerve or cranial nerve 8 (CN VIII) also known as schwannoma) develop, often leading to hearing loss.

Schwannomatosis, in which painful schwannomas develop on spinal and peripheral nerves.

The diagnosis of neurofibromatosis is done via the following means: radiograph, MRI or computer tomography (CT) scan, electroencephalography, slit-lamp examination (with regard to optic pathway gliomas), genetic testing, and histology.

Treatment. Surgical removal of tumors is an option, however the risks involved should be assessed first. With regard to optic pathway gliomas the preferred treatment is chemotherapy. However, radiotherapy isn't recommended in children who present with this disorder. It is recommended that children diagnosed with neurofibromatosis type I at an early age have an examination each year, which allows any potential growths or changes related to the disorder to be monitored.

BENIGN OSTEOGENIC AND NON OSTEOGENIC TUMORS AND TUMOR LIKE FORMATIONS OF THE FACIAL BONES IN CHILDREN. CLINICAL MANIFESTATIONS, DIAGNOSIS, TREATMENT

Tumors of the facial bones are divided into osteogenic and non osteogenic, benign and malignant (Table 1).

Table 1

Classification of facial bone tumors

Tissue of origin	Benign tumors	Malignant tumors
Bone forming	Osteoma Osteoid Osteoma Osteoblastoma	Osteosarcoma

Tissue of origin	Benign tumors	Malignant tumors
Cartilage forming	Chondroma Osteochondroma Chondroblastoma	Chondrosarcoma
Fibrous tissue	Fibroma	Fibrosarcoma
Giant-cell tumor	Benign Osteoclastoma	Malignant osteoclastoma
Marrow tumors		Ewing's Sarcoma Myeloma
Vascular tumors	Haemangioma	Haemangiosarcoma
Other connective tissue	Fibrous histocytoma Lipoma	Malignant fibrous histocytoma Liposarcoma
Other tumors	Neurofibroma	Adamantoma

The most common tumors of the facial bones are considered below.

OSTEOMA

Osteomas are benign tumors composed of bone. The bones of the face are subject to a variety of bony overgrowths that are difficult to classify. Osteomas vary greatly in size. They may be attached by a pedicle; or may grow from a wide base. The pedenculated type occurs more frequently in the mandible. The skull is the most common site for the development of an osteoma, with the calvarium and frontal sinus areas being affected most frequently.

X-ray examination provides information concerning the shape and size and its relationship and attachment to the bone. If the tumor consists chiefly of dense, laminated bone with a few Haversian canals, the radiographic density is much greater than that of a tumor containing spongy bone and abundant marrow space.

Osteomas of the mandible are rare. The tumors with associated supernumary teeth and sometimes with compound odontomas and hypercementosis may develop in patients with Gardner syndrome. Impacted supernumerary and permanent teeth are seen with multiple polyposis of large intestine and epidermoid cysts of skin. Malignant change can occur in these polyps and the syndrome is transmitted as an autosomal dominant trait.

There are two forms of osteoma: “spongy” and compact. Osteomas also can be divide into central and peripheral.

FIBROUS DYSPLASIA (FD)

It is a non-neoplastic tumour-like congenital process, manifested as a localised defect in osteoblastic differentiation and maturation, with replacement of normal bone with large fibrous stroma and islands of immature woven bone. FD has a varied radiographic appearance. If they are asymptomatic, they do not require treatment.

Clinical picture. Craniofacial involvement may occur both as true craniofacial fibrous dysplasia, considered a form of monoostotic fibrous dysplasia (despite multiple cranial bones being affected) which accounting for 10–25 % of monoostotic cases or as part of polyostotic fibrous dysplasia. The craniofacial bones are affected in up to 50 % of polyostotic cases. Occasionally it is seen in the setting of McCune-Albright syndrome. Clinical manifestations are usually cosmetic or due to mass effect on cranial structures: cranial asymmetry, facial deformity, nasal stuffiness, proptosis, and visual impairment (unilateral blindness).

Similar to fibrous dysplasia elsewhere, affected bones demonstrate a variety of *radiographic features* ranging from lucency to sclerosis. *Plain radiograph* may reveal blistering/bubbling cystic skull vault lesions, commonly cross sutures, sclerotic skull base, widened diploic space with displacement of outer table, inner table spared (this is in contrast to Paget disease, in which case the inner table is involved) and obliteration of paranasal sinuses.

CT appearance. Affected bones are usually expanded with an intact cortex and lose the normal cortico-medullary differentiation, being replaced classically by a homogeneous ground glass appearance, although mixed lucencies and sclerosis are also common. The margin between abnormal and normal bone is often difficult to identify, the two regions blending with each other, however on occasion a relatively sharp demarcation may be present. Sometimes there the mixed regions of sclerosis and lucency are reminiscent of Paget disease, and are thus referred to as “pagetoid”. When the maxilla or mandible are involved, resorption of the roots of teeth is uncommon.

MRI appearance is variable depending of the degree of lucencies versus sclerosis:

– T1: heterogeneous signal, usually intermediate;

– T2: heterogeneous signal, usually low, but may have regions of higher signal;

– T1 C+ (Gd): heterogeneous contrast enhancement.

Differential diagnosis should be done with:

– cemento-ossifying fibroma (histology may be similar, however trabeculae are rimmed by osteoblasts; usually more sharply defined);

– intraosseous meningioma (may appear very similar; usually abuts intracranial compartment);

– Paget disease (predilection for skull vault; usually spares facial skeleton);

– sclerotic metastases (usually little expansion; usually has different demographics).

The treatment is reserved for cases where function is being threatened (particularly the airway or vision) and is surgical, although much controversy exists over the best approach (e.g. early vs late intervention; minimal vs radical resection). As the affected region is often large and involves complex facial anatomy, complete resection is usually not possible. Likewise reconstruction can be challenging. Historically radiotherapy was used to attempt to control growth. It was not very efficient but encouraged sarcomatous degeneration, and therefore it is contraindicated now.

CHERUBISM

It has historically been considered a variant of fibrous dysplasia, but in reality is likely a distinct entity. Cherubism is inherited as an autosomal dominant disorder of variable penetrance, with onset in early childhood (typically in the 3–4 years of age). Interestingly penetrance is dependent on gender: 100 % in males, 50–70 % in females.

A "cherub" is a toddler or baby angel, often portrayed in art to have chubby cheeks and an upward gaze. In fact, such a divine being is more accurately called a putto, but in modern English usage the terms have become blurred, and patients with cherubism are implied to have cherub-like faces.

Histological features are indistinguishable from a giant cell granuloma.

Clinical picture is due to the following characteristic cosmetic changes in the face:

– bilateral usually symmetric jaw fullness with a slight upward turning of eyes;

– bilateral expansile multiloculated cystic masses with symmetric involvement of mandible and maxilla.

Additionally, submandibular lymph node enlargement may also be present. The teeth in the affected regions may be lost, and tooth eruption delayed.

Radiographic features consist of lucent expanded regions within the maxilla and mandible, with soap-bubble appearance. As the lesion ages, it often becomes sclerotic and may reduce in size.

Despite the pronounced changes, the disease stabilises and often regresses without the need for *treatment*. In other cases orthodontic & surgical treatment is used.

MCCUNE-ALBRIGHT SYNDROME

McCune-Albright syndrome is a genetic disorder characterised by the association of:

– endocrinopathy: precocious puberty;

– polyostotic fibrous dysplasia: more severe than in sporadic cases;

– cutaneous pigmentation: coast of Maine "cafe au lait" spots.

Clinical picture is variable and includes:

– leg pain, limp, pathological fracture: ~ 75 %;

– abnormal vaginal bleeding: ~ 25 %;

– coast of Maine cafe-au-lait spots (predominantly on back of trunk: ~ 40 % (range 30–50 %); often ipsilateral to bone lesions; irregular borders);

– hypercortisolism (Cushing syndrome): ~ 7 %.

The treatment is dictated by both the affected tissues and the extent to which they are affected. Surgical intervention may be necessary for some skeletal abnormalities. Conservative treatment (special drugs) is aimed to reducing bone pain, decreasing tumor growth and managing endocrine abnormalities.

OSTEOBLASTOMA

It is a rare benign tumor that accounts for less than 1 % of all bone tumors and most commonly involves the spine and sacrum of young individuals. Less than 10 % of osteoblastoma are localized to skull and nearly half of these cases affecting the mandible, especially the posterior segments. The first well-documented case of osteoblastoma of the jaw bones is attributed to Borello and Sedano in 1967.

This central bone tumor usually occurs in young adults, with a mean age of 20 years; with a male to female ratio of 2 : 1. The lesion is characterized clinically by pain, which is traditionally said to be unresponsive to pain and swelling at the tumor site, the duration being just a few weeks to a year or a more.

There are two main clinicopathological entities of osteoblastoma:

– *benign* form, which grows slowly over many years and has a well-defined sclerotic margin, is fairly well vascularized with a mild inflammatory response;

– *aggressive* form. The aggressive form of osteoblastomas exhibits locally aggressive behavior with a propensity to recur and has atypical histopathological features, often making differentiation from low-grade osteosarcoma difficult.

Osteoblastomas can also be classified as cortical, medullary, and periosteal types depending on which component of the bone is involved. Those involving the jaws are either medullary or periosteal with the cortical variant commonly seen in the extragnathic sites.

According to the clinic-radiological picture osteoblastomas can be divided into cystic, cellular and lytic forms.

Radiographically the lesion may appear as radiolucent that can be either ill or well defined containing variable amounts of mineralization. Although, conventional radiography play an important role in diagnosis; however, the final diagnosis can only be confirmed after histopathological examination.

The treatment is surgical. In CELLULAR & CYSTIC forms we used the excochleation of the tumor, in the LYTIC form the resection of the part of jaw is used.

FIBROMA

It is a primary nonosteogenic tumor of bone, consisting of connective tissue elements. In childhood there are two kinds of fibromas: *desmoplastic fibroma* and *ossificans fibroma* (fibroosteoma).

The treatment consists of wide local excision to prevent otherwise frequent recurrences.

INTRAOSSEOUS HEMANGIOMA

Intraosseous hemangioma is a hemangioma located into the bone. This tumor is also known as vascular hamartoma. Hemangioma is classified as benign, but rarely may be locally aggressive.

Clinical picture. These tumors are growing slowly and are generally asymptomatic unless they exert mass effect on sensitive structures. Occasionally they may present as a swelling or a palpable mass, especially in the skull. When large and strategically located they may present with a pathological fracture.

X-ray of the jaw bone structure becomes the coarse character. Sometimes round shape pockets of destruction, fringed with sclerotic rim are visible. There is somewhat racemose lesions around the central vacuum chamber sometimes. Divergence of the roots of teeth and their resorption are possible, sometimes the teeth are as if "hanging in the air". On the upper jaw radiological picture is accompanied by a darkening of the maxillary sinus, in childhood bone hemangioma may cause a delay in the formation of the permanent teeth roots. Sometimes resorption of interdental septa, resembling bone resorption in periodontitis, is radiologically determined.

Diagnosis of hemangiomas with a combination of soft tissue lesion is not difficult usually, the separate injury of bone is difficult. In this case, the available method of diagnosis is a diagnostic puncture of the tumor. The emergence of a large number of clean syringe blood indicates hemangioma.

The treatment is reserved for symptomatic lesions, and a number of options exist: radiation therapy; embolization to reduce intraoperative blood loss; surgical resection, especially if complicated by spinal cord compression; vertebroplasty; intralesional ethanol injection

ODONTOGENIC TUMORS OF FACIAL BONES IN CHILDREN. CLINICAL MANIFESTATIONS, DIAGNOSIS, TREATMENT

Odontogenic tumor is a neoplasm of the cells or tissues that initiated odontogenic processes. Examples include:

– Adenomatoid odontogenic tumor;
– Ameloblastoma, a type of odontogenic tumor involving ameloblasts;
– Calcifying epithelial odontogenic tumor;
– Keratocystic odontogenic tumor;
– Odontogenic myxoma;
– Odontoma.

The most common odontogenic tumors in children are ameloblastoma, odontoma and cementoma.

AMELOBLASTOMA

Ameloblastoma (from the early English word amel, meaning enamel + the Greek word blastos, meaning germ) is a rare, benign tumor of odontogenic epithelium (ameloblasts, or outside portion, of the teeth during development) much more commonly appearing in the lower jaw than the upper jaw. It was recognized in 1827 by Cusack. This type of odontogenic neoplasm was designated as an adamantinoma in 1885 by the French physician Louis-Charles Malassez. It was finally renamed to the modern name ameloblastoma in 1930 by Ivey and Churchill.

While these tumors are rarely malignant or metastatic, and progress slowly, the resulting lesions can cause severe abnormalities of the face and jaw. Additionally, because abnormal cell growth easily infiltrates and destroys surrounding bony tissues, wide surgical excision is required to treat this disorder. If an aggressive tumor is left untreated, it can obstruct the nasal and oral airways making it impossible to breathe without oropharyngeal intervention.

Clinical picture. Ameloblastomas are often associated with the presence of unerupted teeth. Symptoms include painless swelling, facial deformity if severe enough, pain if the swelling impinges on other structures, loose teeth, ulcers, and periodontal (gum) disease. Lesions will occur in the mandible and maxilla, although 75 % occur in the ascending ramus area and will result in extensive and grotesque deformitites of the mandible and maxilla. In the maxilla it can extend into the maxillary sinus and floor of the nose. The lesion has a tendency to expand the bony cortices because slow growth rate of the lesion allows time for periosteum to develop thin shell of bone ahead of the expanding lesion. This shell of bone cracks when palpated and this phenomenon is referred to as “Egg Shell Cracking” or crepitus, an important diagnostic feature.

Ameloblastoma is tentatively *diagnosed* through radiographic examination and must be confirmed by histological examination (e.g., biopsy). *Radiographically*, it appears as a lucency in the bone of varying size and features — sometimes it is a single, well-demarcated lesion whereas it often demonstrates as a multiloculated “soap bubble” appearance. Resorption of roots of involved teeth can be seen in some cases, but is not unique to ameloblastoma. The disease is most often found in the posterior body and angle of the mandible, but can occur anywhere in either the maxilla or mandible.

There are 3 forms of ameloblastomas according to clinical and radiological manifestations: unicystic, solid, multicystic.

Multicystic ameloblastomas account for 80–90 % of cases which are classically manifested on CT as expansile “soap-bubble” lesions, with well-demarcated borders and no matrix calcification. Occasionally erosion of the adjacent tooth roots can be seen. When larger it may also erode through cortex into adjacent soft tissues.

On MRI ameloblastomas in general demonstrate a mixed solid and cystic pattern, with a thick irregular wall, often with solid papillary structures projecting into the lesion. These components tend to enhance vividly which is very helpful to distinguish them from other lucent lesions of the mandible.

Histopathology will show cells that have the tendency to move the nucleus away from the basement membrane. This process is referred to as "Reverse Polarization". The follicular type will have outer arrangement of columnar or palisaded ameloblast like cells and inner zone of triangular shaped cells resembling stellate reticulum in bell stage. The central cells sometimes degenerate to form central microcysts. The plexiform type has epithelium that proliferates in a "Fish Net Pattern". The plexiform ameloblastoma shows epithelium proliferating in a "cord like fashion", hence the name "plexiform". There are layers of cells in between the proliferating epithelium with a well-formed desmosomal junctions, simulating spindle cell layers.

The six basic different histopathological variants of ameloblastoma are described: desmoplastic (about 9 %), granular cell (extremely rare and in older patients), basal cell (extremely rare and in older patients), plexiform (about 30 %), follicular (about 30 %), and acanthomatous (extremely rare and in older patients).

Differential diagnosis should be done with:

– dentigerous cyst: the relationship between ameloblastomas and dentigerous cysts is a controversial one, 20 % of ameloblastomas thought to arise from pre-existing dentigerous cysts;

– odontogenic keratocyst (OKC): usually unilocular with thin poorly enhancing walls;

– odontogenic myxoma: can be almost indistinguishable;

– aneurysmal bone cyst (ABC);

– fibrous dysplasia.

Treatment. Ameoloblastomas tend to be treated by surgical en-bloc resection. Local curettage is associated with a high rate of local recurrence (45–90 %).

Simple unilocular lesions are less common but have a better prognosis and can be treated only by curettage:

– simple (no nodule) variant will not be diagnosable on radiography, as it will be indistinguishable from other more common cysts;

– luminal variant, has a single nodule projecting into the cyst;

– mural variant has multiple nodules (often only microscopic) in the wall of the cyst. The latter has an elevated risk of recurrence.

Malignant behaviour is seen and the appropriate treatment is necessary in two forms: ameloblastic carcinoma (it demonstrate frankly malignant histology) and malignant ameloblastoma (it metastases despite well differentiated "benign" histology).

ODONTOMA (ODONTOME)

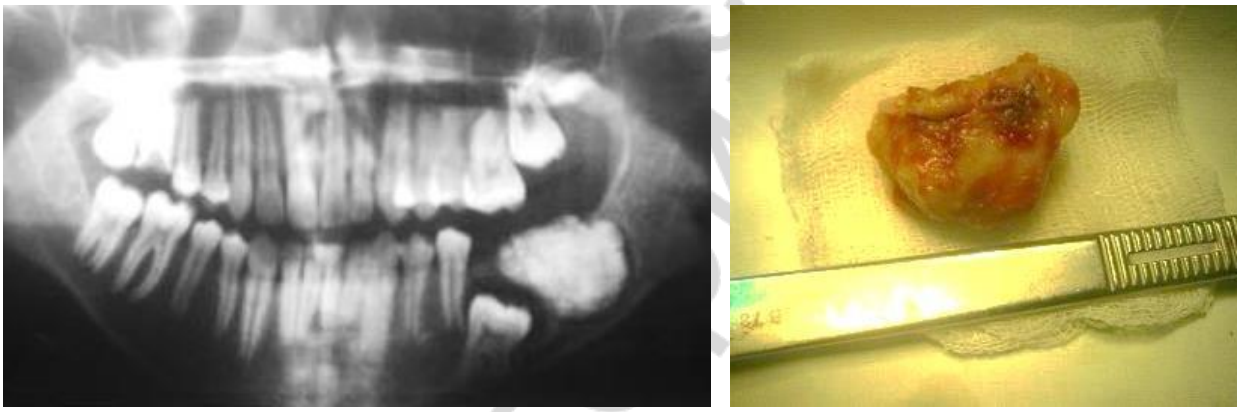
It is a benign tumour of odontogenic origin. Specifically, it is a dental hamartoma, meaning that it is composed of normal dental tissue that has grown in an irregular way.

The average age of people found with an odontoma is 14. The condition is frequently associated with one or more unerupted teeth. Though most cases are found impacted within the jaw; there are instances where odontomas have erupted into the oral cavity.

There are two main types of odontoma: compound and complex.

A *compound odontoma* still has the three separate dental tissues (enamel, dentin and cementum), but may present a lobulated appearance where there is no definitive demarcation of separate tissues between the individual "toothlets" (or denticles). It usually appears in the anterior maxilla.

The *complex odontoma* is unrecognizable as dental tissues, usually presenting as a radioopaque area with varying densities. It usually appears in the posterior maxilla or in the mandible (Fig. 55).



a ***b***

Figure 55. Odontoma (illustration is provided by A. K. Korsak):
a — X-ray picture; *b* — postoperative view

In addition to the above forms, the *dilated odontoma* is an infrequent developmental alteration that appears in any area of the dental arches and can affect deciduous, permanent and supernumerary teeth. It is the most extreme form of *Dens invaginatus* (developmental anomaly resulting from invagination of a portion of crown forming within the enamel organ during odontogenesis).

CEMENTOMA

Cementoma detected radiographically in the form of round or oval, homogeneous dense tissue associated with a tooth root. Tooth roots are undeveloped, unformed. Periodontal fissure is absent. The presence of focal calcifications is necessary. *The treatment* is surgical. The tumor is removed together with the capsule and tooth which cementum was the source of tumor growth.

BENIGN TUMORS OF SALIVARY GLANDS IN CHILDREN. CLINICAL MANIFESTATIONS, DIAGNOSIS, TREATMENT

Tumors of the salivary glands are very diverse and occur more frequently in older children. Large salivary glands are commonly affected. Histologically salivary gland tumors are divided into epithelial and non-epithelial, benign (mature) and malignant (immature). Epithelial tumors make up the majority of neoplasms of the salivary glands.

Classification of salivary glands tumors according to V. V. Panikarovsky:

1. Benign epithelial: adenomas, adenolymphomas, "mixed" tumors.
2. Epithelial malignant: cancers, carcinomas, mucoepidermoid tumors.
3. Non-epithelial benign tumors: angiomas, neuromas, lipomas, fibromas, etc.
4. Salivary glands cysts.

PLEOMORPHIC ADENOMA

It is a benign neoplastic tumor of the salivary glands. It is the most common type of salivary gland tumor and the most common tumor of the parotid gland. It derives its name from the architectural pleomorphic (variable appearance) seen by light microscopy. It is also known as "mixed tumor, salivary gland type", which describes its pleomorphic appearance as opposed to its dual origin from epithelial and myoepithelial elements.

The tumor is usually solitary and presents as a slow growing, painless, firm single nodular mass. Isolated nodules are generally outgrowths of the main nodule rather than a multinodular presentation. It is usually mobile unless found in the palate and can cause atrophy of the mandibular ramus when located in the parotid gland. When found in the parotid tail, it may present as an eversion of the ear lobe. Though it is classified as a benign tumor, pleomorphic adenomas have the capacity to grow to large proportions and may undergo malignant transformation, to form carcinoma ex pleiomorphic adenoma, a risk that increases with time. Although it is "benign" the tumor is aneuploid, it can recur after resection, it invades normal adjacent tissue and distant metastases have been reported after long (+10 years) time intervals.

Histologically, it is highly variable in appearance, even within individual tumors.

Classically it is biphasic and is characterized by an admixture of polygonal epithelial and spindleshaped myoepithelial elements in a variable background stroma that may be mucoid, myxoid, cartilaginous or hyaline. Epithelial elements may be arranged in duct-like structures, sheets, clumps and/or interlacing strands and consist of polygonal, spindle or stellate-shaped cells (hence pleiomorphism). Areas of squamous metaplasia and epithelial pearls may be present. The tumor is not enveloped, but it is surrounded by a fibrous pseudocapsule of varying thickness. The tumor extends through normal glandular parenchyma in the form of finger-like pseudopodia, but this is not a sign of malignant transformation.

The diagnosis of salivary gland tumors utilizes both histopathological sampling and radiographic studies. Histopathological sampling procedures include fine needle aspiration and core needle biopsy (bigger needle comparing to fine needle aspiration biopsy). Both of these procedures can be done in an outpatient setting. Diagnostic imaging techniques for salivary gland tumors include ultrasound, CT and MRI. Fine needle aspiration biopsy can determine whether the tumor is malignant in nature with sensitivity around 90 % and distinguish primary salivary tumor from metastatic disease. Core needle biopsy is more invasive but is more accurate compared to fine needle aspiration biopsy with diagnostic accuracy greater than 97 %. Furthermore, core needle biopsy allows more accurate histological typing of the tumor. In terms of imaging studies, ultrasound can determine and characterize superficial parotid tumors. Certain types of salivary gland tumors have certain sonographic characteristics on ultrasound. Ultrasound is also frequently used to guide fine needle aspiration biopsy or core needle biopsy. CT allows direct, bilateral visualization of the salivary gland tumor and provides information about overall dimension and tissue invasion. CT is excellent for demonstrating bony invasion. MRI provides superior soft tissue delineation such as perineural invasion when compared to CT only.

The treatment for salivary gland tumor is surgical resection. Benign tumors of the parotid gland are treated with superficial or total parotidectomy due to high incidence of recurrence. The facial nerve should be preserved whenever possible. The benign tumors of the submandibular gland are treated by simple excision with preservation of mandibular branch of the trigeminal nerve, the hypoglossal nerve, and the lingual nerve. Benign tumors of minor salivary glands are treated similarly.

ADENOMA

Adenoma of the salivary glands is extremely rare. The parotid salivary glands are mainly affected. The tumor grows slowly, has elastic consistency and relatively smooth surface. It is displaceable on palpation, delimited from surrounding tissues by a fibrous capsule. The child does not complain of pain. The skin over has the normal tumor color and normal saliva is secreted from the salivary gland duct. The diagnosis is established on the basis of morphological research (cytological — before and histological — after surgery). It is necessary to differentiate with a “mixed tumor” (pleomorphic adenoma), vascular tumors of the salivary glands (based on clinical and morphological data), from malignant tumors (based on their rapid growth, pain, infiltrating growth, early metastasis, recurrence, morphological research data). *The treatment* is surgical. The tumor is removed with a capsule. Relapses are extremely rare.

RANULA

It is a type of mucocele found on the floor of the mouth. Ranula manifested as a swelling consisting of collected mucin from a ruptured salivary gland duct, which is usually caused by local trauma. The latin *rana* means frog, and a ranula is so named because its appearance is sometimes compared to a frog’s underbelly.

The gland that most likely causes a ranula is the sublingual gland. Nonetheless, the submandibular gland and minor salivary glands may be involved.

An oral ranula is a fluctuant swelling with a bluish translucent color that somewhat resembles the underbelly of a frog Rana. If it is deeper it does not have this bluish appearance. If it is large (2 or more cm), it may hide the salivary gland and affect the tongue location. Most frequently it stems from the sublingual salivary gland, but also from the submandibular gland. Though normally above the mylohyoid muscle, if a ranula is found deeper in the floor of the mouth, it can appear to have a normal color. A ranula below the mylohyoid muscle is referred to as a "plunging or cervical ranula", and produces swelling of the neck with or without swelling in the floor of the mouth. Ranulas measure several centimeters in diameter and are usually larger than mucoceles. As a result, when ranulas are present the tongue may be elevated. As with mucoceles, ranulas may be subject to recurrent swelling with occasional rupturing of its contents. When pressed, they may not blanch.

Ranulas are usually asymptomatic, although they may change gradually in size, shrinking and swelling, making most ranulas hard to detect. The overlying skin is usually intact. The mass is not fixed and is also not tender. The mass is not connected with the thyroid gland or lymph nodes. The mass may not be well defined. If it gets large enough it may interfere with swallowing, and cervical ranulas may even interfere with breathing. Some pain may be connected with very large ranulas.

Microscopically, ranulas are cystic saliva filled distensions of salivary gland ducts on the floor of the mouth alongside the tongue, and are lined with epithelium. A salivary mucocele, in contrast is not lined with epithelium.

The treatment of ranulas could involve either a procedure known as "marsupialization" or more often excision of both the gland and lesion. Ranulas are likely to reoccur if the sublingual gland or other gland causing them is not also removed with the lesion. There is little morbidity or mortality connected with treatment.

RETENTION CYSTS

Retention cysts of the mucous membrane of the lips, cheeks, frenulum of the tongue are rather common in children. Most often, these cysts result from the closure of the excretory ducts of the small salivary glands of the oral mucosa (trauma, inflammation) and delayed secretion, or as a result of congenital malformations. *Clinically*, the cyst appears as a bluish, elastic formation of a rounded shape, with a diameter of approximately 1 cm, with clearly defined boundaries. These cysts often open up on their own, and then reappear.

The treatment of retention cysts of the oral mucosa is surgical — cystectomy with the gland in order to avoid relapse.

MALIGNANT TUMORS OF THE MAXILLOFACIAL REGION AND NECK IN CHILDREN. CLINICAL MANIFESTATIONS, DIAGNOSIS, TREATMENT

Malignant tumors of the maxillofacial region in children are much less common than benign ones. Tumors of a connective tissue nature (sarcoma) and tumors of the lymph nodes (lymphogranulomatosis, etc.) predominate in childhood. Malignant tumors in children grow quickly and almost always metastasize, which requires early diagnosis, timely and radical treatment.

Early diagnosis of malignant tumors in children is difficult due to poor symptomatology at early stages of development (discomfort, parasthesia, etc.). Pain occurs when the tumor spreads significantly. The first symptoms of a malignant tumor in a child are often similar with inflammatory diseases signs: swelling, fever, tooth mobility, etc. As a result, the dentist can take a tumor for the inflammatory process and prescribes therapy (tooth extraction, tissue dissection, physiotherapy), which aggravates the malignancy course.

The basis for the early diagnosis of tumors is oncological alertness of pediatric dentists, the main point of which is that any increase in the volume of soft tissues and facial bones should be considered to exclude a tumor.

MELANOMA

It is gradually increasing single pigmented, nodulate tumor, surrounded by erythematous halo (Fig. 56). Gradually, tumor becomes a mushroom shape and about the hearth the child elements appear. In most cases, the primary melanoma does not reach large sizes, as early metastases occur and the disease progresses rapidly. Metastases occur initially in the regional lymph nodes (lymphogenous), and holds hematogenous metastasis to the lungs, liver, brain, bone, and others. Radioisotope study is used for the diagnosis of melanoma.

The treatment is only surgical (*no incisive biopsy*!!!).

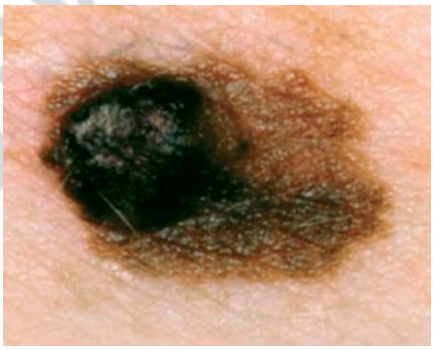


Figure 58. Melanoma

LYMPHOGRANULOMATOSIS. (HODGKIN'S DISEASE)

It refers to malignant lymphoma, and about 15 % of all malignancies in children. Defeat cervical lymph nodes is observed in 80 % of cases. In some cases, the children in addition to cervical nodes, affected lymph nodes of the parotid

region. Lymph nodes are mobile, painless, of soft elastic consistency. A characteristic is to maintain the mobility of the lymph nodes even with a significant increase in their size. In the future, multiple nodes are merged into a single conglomerate. The process may be local and generalized. At the same time the defeat of the cervical lymph nodes combined with the defeat of the other groups of lymph nodes, spleen, and others. It is typical bilateral involvement of lymph nodes and not the cohesion of the skin and underlying tissues.

The stage of Hodgkin lymphoma describes the size and position of the cancer and whether it has spread over. Staging is very important because this guides how much treatment you will need. The number of cycles of chemotherapy you receive is decided by the stage. The staging system for Hodgkin lymphoma is as follows:

– Stage 1. Only one group of lymph nodes is affected and the lymphoma is only on one side of the diaphragm (the sheet of muscle under the lungs that controls breathing).

– Stage 2. Two or more groups of lymph nodes are affected, but they are only on one side of the diaphragm.

– Stage 3. The lymphoma is in lymph nodes both above and below the diaphragm.

– Stage 4. The lymphoma has spread outside the lymph nodes to other organs such as the liver, lungs or bone marrow.

As well as giving each stage a number, doctors also use a letter code — either A, B or E:

A — child has no symptoms.

B — child has one or more of the following symptoms: a fever, night sweats or significant weight loss.

E — the lymphoma has grown from the lymph gland to extranodal tissue (tissue in places outside the lymph nodes).

A number and letter is used in the staging of every child with Hodgkin lymphoma.

MUCOEPIDERMOID CARCINOMA

Mucoepidermoid carcinoma is the most common type of minor salivary gland malignancy. *Clinically* in the initial stages it is usually not much different from the "mixed tumor". However, limited mobility, lack of clear boundaries, fixation of the skin over a tumor, etc. are signs of malignancy.

The appearance of pain, pain during palpation and facial nerve involvement indicate the presence of a malignant tumor of the parotid salivary gland. This tumor is not encapsulated and is characterized *histologically* by squamous cells, mucus-secreting cells, and intermediate cells.

Surgery is the recommended *treatment* for localised resectable disease. When the tumour is incompletely resected (positive margins) post-operative radiotherapy gives local control comparable to a complete resection (clear margins).

RHABDOMYOSARCOMA

Rhabdomyosarcoma is the most common malignant tumor of soft tissue in children (about 50 %). It often occurs in those organs where normally is no mature skeletal muscle (in the wall of bladder, nasopharynx, vagina). In children aged 2–6 years, it is often localized in the neck and head (nasopharynx), in teenagers — in the genital and retroperitoneal areas. Rhabdomyosarcoma is extremely rare in adults.

OSTEOSARCOMA

It is a cancerous tumor in a bone. Specifically, it is an aggressive malignant neoplasm that arises from primitive transformed cells of mesenchymal origin (and thus a sarcoma) and that exhibits osteoblastic differentiation and produces malignant osteoid. Osteosarcoma is the most common histological form of primary bone cancer. It is most prevalent in children and young adults.

Clinical picture. Many patients first complain of pain that may be worse at night, may be intermittent and of varying intensity and may have been occurring for some time. Teenagers who are active in sports often complain of pain in the bones. If the tumor is large, it can present as overt localised swelling. Sometimes a sudden fracture is the first symptom, because affected bone is not as strong as normal bone and may fracture abnormally with minor trauma. In cases of more deep-seated tumors that are not as close to the skin, such as those originating in the pelvis, localised swelling may not be apparent.

A complete radical, surgical, *en bloc* resection of the tumor, is *the treatment* of choice in osteosarcoma. Current standard treatment is to use neoadjuvant chemotherapy (chemotherapy given before surgery) followed by surgical resection.

EWING'S SARCOMA

It is a type of cancer that forms in bone or soft tissue, most common in adolescents about 13 years old. *Symptoms* are very similar to osteomyelitis and may include swelling and pain at the site of the tumor, fever, and a bone fracture.

The general condition changes and the patient suffers from increasing weakness and fever. ESR and the number of leukocytes increases, secondary anemia is sometimes found. A wave-like course is a feature of the disease: after some time the pain subsides, the swelling becomes less pronounced, the mobility of the teeth decreases slightly. But after some time the disease becomes acute with a new force. Ewing's sarcoma is sometimes characterized by a history of injury. Ewing's sarcoma metastasizes to regional and other lymph nodes, which is different from true osteosarcoma.

X-ray picture with Ewing's sarcoma is not typical. At the onset of the disease, one or more lesions of bone tissue appear with fuzzy contours in the spongy substance, and then in the cortical layer. Periosteal bone formation is absent.

Diagnosis is based on biopsy of the tumor.

The treatment includes chemotherapy, radiation therapy, surgery, and stem cell transplant. Targeted therapy and immunotherapy are being studied.

TREATMENT OF PRIMARY MALIGNANT TUMORS

The main method of treatment of primary malignant tumors of the maxillofacial area in children is a combination of surgery with radiation and chemotherapy. When prescribing radiation therapy to a child, it should be remembered that the most pronounced radiation damage occurs in growing and developing systems, organs and tissues.

All children who have received a course of treatment for a malignant neoplasm should be under the constant follow-up supervision of an oncologist and a pediatric dentist from the day of tumor diagnosis to 15 years, after which the patient is transferred to an adult network.

Objectives of follow-up: to control the possibility of tumor recurrence, to restore the functions and anatomical integrity of organs and tissues, and to ensure adequate growth and development of the maxillofacial area.

All children with tissues and organs defects after treatment should be received prostheses in the hospital, 2–3 weeks after surgery. Further monitoring of the child is carried out by the orthodontist at the place of residence.

Children operated due to malignant tumors are observed with an interval of 1, 3, 6 months and then 2 times a year. During examinations, the general and local condition of the child (weakness, temperature, pain, lymph nodes, skin and mucous membrane color, teeth, etc.) is assessed; a complete blood count is performed. Periodic control radiological examination is carried out.

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