

**Courses of lectures of inflammatory diseases, localized
in the maxilla-facial region**

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Introduction

In the present study guide for maxilla-facial surgery and dental surgery, briefly, are described some facial and neck diseases namely etiology, pathogenesis, diagnostics, of the clinical progression and treatment of the given diseased peculiarities. Collection of lecture material about inflammatory processes localized in the maxilla-facial region will help students and physician residents of dental departments in the given speciality study. The present manual is composed in accordance with syllabus approved for students and physician-residents of dental department, of the State University of Medicine and Pharmacy “Nicolae Testemitanu” of Republic Of Moldova. The manual contains the lecture material for students of 3rd year of stomatological department.

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I Chapter

Topic № 1

Inflammatory processes in maxillofacial region

Ethiology and pathogenesis of odontogenic inflammatory diseases

Inflammatory diseases of maxillofacial area inherently are infectious inflammatory processes, (that is) i.e. are caused by microbes the majority of which under ordinary conditions which perennate on skin integument and oral mucosa. During the skin integument disintegration and mucous coat, affect of acentric paradontium and also the hard tooth tissues degradation with its opening of pulp cavity, these microbes invade in subjacent tissues. Depending on localization of site of entry for microbes can be distinguished odontogenic, stomatogenic, tonsillogenic, rinogenic and dermatogenic infectious inflammatory processes.

Ethiology

Activator of odontogenic inflammatory diseases - are microorganisms, which usually are included in the composition of permanent oral cavity microflora: staphylococcus, streptococcus, enterococcus, diplococci, gram-positive and gram – negative cocci. According to the data of different authors, the microflora of odontogenic pockets` infection most commonly is presented by staphylococcus pure culture (Staphylococcus albus, St. aureus, St. epidermidis). The nascence probability of infectious inflammatory process depends largely on the state of microorganism immunologic reactivity.

Pathogenesis.

The term “odontogenic inflammatory diseases” – is collective. It includes a variety of enough edgy form of the disease in clinical concern (parodontitis, periostitis, osteomyelitis, apostasis and phlegmon, lymphadenitis, odontogenic genyantritis). The influence of admitted in the organism serum protein, which possesses antigenic properties, take place the elaboration of antistubstance, just this underlie in sensibilization of the organism. Against this background the local insertion of challenge size of antigen is accompanied by penetration of the last in the bloodstream, where forms the antigen-antistubstance complex.

Heterothallic leukocytes phagocytizing immune complexes in the same time disturb cell membrane, which deals to lysosomal enzymes - inflammatory mediator. This is accompanied by activation of 3rd (third) thrombocytic factor and can be the reason of inopexia adducting to disorder microcirculation and tissue necrosis. Rise of lancinating odontogenic inflammatory diseases in maxillofacial area often forego for influence on patient body different general factors: cooling, overwarming, athletic and emotional overexertion, immoderate ultraviolet irradiation, intercurrent diseases. The generalization of odontogenic infectious inflammatory processes from the area of its first location can be by extension, lymphogenous or haematogenous. The purulence, appeared in parodontium melt bony tissue and penetrate through roughness compact stratum under the periosteum, and after melt of periosteum - in interfascial compartments attached to maxilla. Through the osteon canals and alimention canals antigens achieve richly vascular periosteum in interfascial pacefollower. The presence of dense network capillary contributes to penetration of antigens through its membrane in vasculature. Here they ally with circulating in the blood antistubstances and form complexes, the presence of which is determined appearance of immunopathological reaction. Factors determinated the appearance of caries and the volume of its affection.

Virulence infectious beginnings depend from the properties and quantity of microbes.

General immunological responsiveness of the body - is the capability in time and in a proper manner use the safety adaptive reactions directed to deletion, growth inhibition and limitation of pathogen generalization zone largely takes measure of course of odontogenic infectious inflammatory processes and caries evidence. Local tissues immunity of maxillofacial area depends from the tissues capability of this area produce and accumulate factors of nonspecific and specific defense. For example, oral liquid contains enough high concentration of lysozyme (albumin with the properties of mucolytic enzyme) and secretory immunoglobulin A, which participate in the maintenance of microbial flora in mouth cavity.

Disturbed blood circulation (microcirculation).

Parodontitis

Acutus and chronic parodontitis.

Classification.

Source and character of infection	Disease	Form of disease	
Odontogenic and stomatogenic	Parodontitis	Acute: Serosal Suppurative	Chronic: Fibrotic Granulating Granulomatous Chronic recrudescence

Clinical picture

Acute parodontitis

1. Serosal stadium.

During developed serosal parodontitis appears not acute, dull aching pain in affected tooth, which increase in the night. Pressing on the tooth during occlusion becomes tenderer. Pains do not irradiate the patient exact point out the affected tooth. Then appear the sensation as if growing and prolongation of the tooth. Soft-tissue swelling of the face usually does not occur. Submandibular lymph nodes enlarge, becomes a bit painful. Inflammatory changes of the mucosa are absent, but sometimes the gingival is hydropic. Not large tooth mobility is marked and pain reaction during the vertical percussion (tapping). Such clinical picture is typically for serosal stage of inflammation which usually is not short-term.

2. Suppurative stadium

With the transference to the other stadium the pain intensity grows. The pain becomes acute, systaltic, more often than not irradiate in temple, ear, eye or cervix. The pain increases from the physical exertion, warm and body horizontal attitude. The affected tooth is mercurial. A touch to it causes the acute pain. Patient cannot occlude tooth and therefore often keeps his mouth semi open. The gingival of this tooth is hyperemic and hydropic. Periostenum of alveolar bone on the circumscribed portion relevant view of the apex of the tooth root is infiltrated. Palpation of the transitory fold and gingival along the all root becomes more painful. Some patients have soft tissue oedema apparent in different degree. Submandibular and submental lymph nodes are increased and tendered.

Because of pains the ingestion is hampered, the sleep is disturbed, appears the discomfort, general weakness and brokenness-down. Physiognomy is full suffering and anxious. Somatic temperature is 37.3-37.5 °C, some patient have higher. The number of leucocytes increases (9 – 11*10⁹/l). In the white blood cells is presented neutrocytosis due to increase of microxyphil (70- 72%) and stab (8 – 10%) leucocytes. Erythrocyte sedimentation rate (ESR) is increased till 12 – 15 mm/h (millimeters per hour).

During the acute serosal suppurative parodontitis on the X-ray picture the periodontal fissure isn't changed and the destruction of the alveolar bony tissue is not apparent. But in some cases on the third (3rd) - fifth (5th) day of disease can be observed the turbidity of compact alveolar plate in the region of its fundus.

Differential diagnostics. Acute parodontitis should be differentiated from the diffuse suppurative endodontitis, radicular cyst, acute odontogenic maxillary sinusitis, periostitis and maxilla osteomyelitis.

During the endodontitis the pain is periodical, but during the suppurative parodontitis – is permanent. During the endodontitis do not appear changes in the gingiva. The tooth reaction to the cold is sharply positive during the endodontitis and during the parodontitis is absent.

During the radicular cyst the for the most part bulging of the alveolar crest is bounded, sometimes the bone on this place is absent, tooth are quite often dislocated. During the cyst happens considerable bone resorption, which is detected by the help of X-ray investigation and during the acute parodontitis such changes do not determine.

Self-existing tooth pains (odontalgia) which appear during the acute odontogenic maxillary sinusitis, and also eccentric pains along the devarication of the maxillary nerve, sickliness during the tooth percussion, swelling of the check and inferior eyelid embarrass the differential diagnostics of the acute parodontitis. Distinguished characters of the acute odontogenic maxillary sinusitis are stuffiness of the one part of the nose, the presence of serosal-suppurative or suppurative elimination from the nose, intensive headaches, the abnormality of the transparence of the maxillary sinus on the X-ray picture.

Treatment.

During the treatment of the acute parodontitis in the first place it is necessary to create a free outflow of exudation from the periapical region. This in mainly cases causes the remitting of the inflammatory process, prevents its generalization on the surrounding tissues, reduces the pain; normalize the general state of the patient. If the affected tooth is no greatly destroyed by the curiosity and in the future can be treated and filled, then the drainage of the inflammatory locus is performed through the root canal.

In cases when the drainage of the apostem in periodont through the root canal isn't succeed because of impassability or the tooth is greatly destroyed and its preservation isn't effectually, resort to the surgical treatment (surgical intervention) – extraction of a tooth; the outflow of the exudation happens through an alveolus. If the suppurative parodontitis is accompanied with the limited serosal periostitis it is depicted the discission of the infiltrative locus of the mucosa and periostenium along the transitory fold of affected tooth. This de-stress inflammatory tissues and creates conditions for outflow of the serosal exudation – surgical treatment.

For cause lead the medicamentous, antibacterial therapy by sulfanilamides. To the debilitated patient with pronounced appearance of intoxication could be applied the treatment by the antibiotics.

Topic № 2

Periostitis

Definition. Classification.

Periostitis: Inflammation of the periosteum, a dense membrane composed of fibrous connective tissue that closely wraps (invests) all bone, except the bone of articulating surfaces in joints which are covered by synovial membranes.

Periostitis includes the 3 symptoms listed below:

- Pain in the front of lower leg
- Exacerbation of symptoms with repetitive activity
- Symptom improvement with rest

Acute periostitis

The maxilla periostitis most off all (74-75% of cases) develops due to recrudescence of the chronic inflammatory process in periodont. In consequence of bone resorption during the chronic parodontitis the infection from the periodont`s tissue easy penetrate to periostenium.

Conservative treatment of the tooth, also, can be complicated by the periostenium inflammation. The disease also develops after the tooth extraction surgery, when it is performed traumatic with the affection of the tooth tissue and gingiva, in the cases of incomplete tooth extraction concerning acute or aggravated chronic parodontitis.

Pathanatomy

Acute odontogenic periostitis of maxilla is infectious – inflammatory process originated as complication of tooth disease and tissues of periodont. Most often it proceeds in the form of circumscribed inflammation of the periostenium of the alveolar ridge through some teeth.

In periost and adjacent soft tissue take place significant changes in vessels (plethora, stasis and hemorrhage). Appears expressed leucocytic infiltration in cellular tissue. Accumulation of seropurulent exudate can provoke the abruption of the periostenium from the bone. In the center of infiltrate appears necrosis with the suppurative fusion and fusion of infectious inflammatory process outwards.

Clinical picture

Clinical aspects depend from the patient body responsiveness, type of inflammatory reaction, infection virulence and the localization of inflammatory process. In this period the well-being deteriorate, arise asthenia, distortion, the body temperature arise. Appears the headache, disappear appetite, sleep disturbance.

Pain senses in the tooth region irradiate in temporal fossa, ear, eye, neck (cervix). In the future the pain intensity fall. In the periostenium appears edema of perignathic soft tissues. Arising bullation changes face configuration. Regional lymph glands increase and become more painful. Difficult and painful opening of the mouth, appearing as a result of inflammatory contracture masticatory and wing muscles occurs not so often, during the inflammatory process in the region of big root teeth of mandibular bone.

Changes in the mouth cavity: appears hyperemia and edema of mucosa transitory fold and adjacent locus of the check through some teeth. The mouth mucosa breaks out by the accretion. The transitory fold is fluttering. A big bulging - subperiosteal abscess appears when the process comes in the suppurative form and the exudation accumulates under the periostenium of alveolar bone along the transitory fold. When we examine the tooth, serving as a source of infection, often is established that its cavity and root canals are filled by ichorization. In some cases, there is a deep pathologic dental pocket. In this period the pain reaction, during the tooth percussion, is

not pronounced and sometimes is absent. During the X-ray study of the alveolar bone and maxilla body changes specific to the acute periostitis do not emerge.

Usually the body temperature during the periostitis is within 37,3-37,8 °C. During the blood examine in the period of disease development is registered augmentation of leucocytes quantity (10-12×10⁹/l, sometimes more). Frequently appears neutrocytosis at the cost of enlargement of the quantity of microxyphil (till 70-75%) and stab leukocytes (8-20%). At the same time the percentage composition of lymphocytes (till 10-20%) and eosinocytes diminish. In a few days from the beginning of disease Blood Sedimentation Rate (BSR) is equal to 15-20 mm/h. In acute stage of the disease in some patient urine appears albumin (till 0,33g/l). Sometime we can observe a bit quantity of leucocytes (10-20 per high power field).

Upper lip and wing of nose swell very much during the vestibular localization of periostitis of the maxilla in the region of central and lateral incisor.

If the source of infection is eye tooth then the swelling dominates in a part of buccal and infraorbital region. The swelling dominates in the buccal, infraorbital and jugal region during the localization of the process in the region of small root teeth. The inflation dominates in the jugal, buccal and upper region of parotic- masticatory region during the periostenium inflammation respectively to the second and third big root teeth.

Acute purulent periostitis of upper maxilla with the localization on the palatal surface more often appears as a result of infection spread. The periostitis from the palatal part is characterized by peculiar course. At the very beginning appear intense aching then systolic pains in the region of hard palate. Inflammatory infiltrate lifts the mucosa. In many cases it occupies a significant part of respective half of hard palate. Mucosa over the focus of inflammation and in it surrounding turn red.

Underlip and chin swell during the periostitis of the submaxilla (mandibular bone) from the vestibular side in the region of central and lateral incisors. Sometimes the source of infection is big root teeth which form collateral swelling. It occupies lower and middle section of buccal and region parotic- masticatory regions.

Development of the mandibular bone periostitis as a result of big root teeth pathology begins more often from the inner surface of alveolar bone as this side is thinner then external surface. In the region of alveolar bone and sublingual region appears hyperemia and swelling of mucosa during the periostitis of the tongue surface of the alveolar bone. Sublingual roller on the respective side increased and bulge between tongue and mandibular.

Differential diagnosis.

Acute purulent periostitis of maxilla should be differentiated from the acute parodontitis, osteomyelitis, apostem and phlegmon and lymphadenitis. Sometimes periostitis is mistaken for inflation of sublingual and submandibular gland and their ducts.

Treatment.

Main remedial measures during the acute purulent periostitis consist in surgical prosection of inflammatory nidus and creation of free outflow of formed exudate. More often it is applied local anesthesia – conduction and infiltration anesthesia. In the capacity of anesthetic it is used 1-2% of trimecaine solution or 1% of lidocaine.

Insertion of anesthetize solution in the periostenium is painful, that why during the infiltration anesthesia, an anesthetic is inserted in the sound tissue on the border with infiltrate.

Discission during the periostitis make as section of mucosa and periostenium through the transitory fold in length 2-2,5cm to all tissue depth till the bone. For free outflow of exudate and prevention of part agglutination in the wound is inserted on 1-2 days thin drainage, which is fabricated from the surgical gloves or polyethylene pellicle.

During the discission of the periostenium apostasis, placed on the hard palate, dissect moderate portion of soft tissues from the abscess side (triangular or oval shape). This is made with the aim to avoid wound sides agglutination.

Consequently with the discission of the periostenium apostasis, it is executed the tooth extraction, which served as source of infection, in the case if its future preservation isn't possible

(the tooth with greatly broken crownwork, which does not perform functional value and respond to treatment). In other cases the tooth must be saved. After inflammatory process interruption, the tooth is treated and filled. Some patients after tooth treatment have to surgical intervention – apex of root resection or reimplantation.

Not always the surgical intervention for discission of subperiosteniun apostasis and tooth extraction can be performed consequently. Surgical intervention is performed in some days, after acute inflammatory processes disappear or diminish, if general state of the patient is bad and also in the cases when tooth extraction can afford some technical difficulties.

After surgical intervention for speedy resorption of inflammatory infiltrate, it is placed on mouth rinsing by warm (40-42°) solution of potassium permanganate (1:3000).

Chronic periostitis.

The chronic form of disease develops seldom. Predominantly appears at children in the age of 9-13 years and young peoples. Not frequently chronic ostitis proceeds without pronounced symptomatology (are absent local clinical aspects and fever response). The reason of chronic periostitis and chronic ostitis more often is odontogenic infection. Such patients have soft pains in the maxilla, little bullation of perignathic tissue, sometimes can be raised body temperature. In alveolar bone region (sometimes maxilla body) appears limited, dense, a bit painful infiltrate. Mucosa unite (seal) with infiltrate and appears insignificant hyperemia. Patients do not observe developing disease that`s why do not address to doctor. The treatment of the chronic periostitis has its peculiarities. The treatment begins from elimination of the cause which proves pathological process.

Topic № 3

Osteomyelitis

Odontogenic maxilla osteomyelitis.

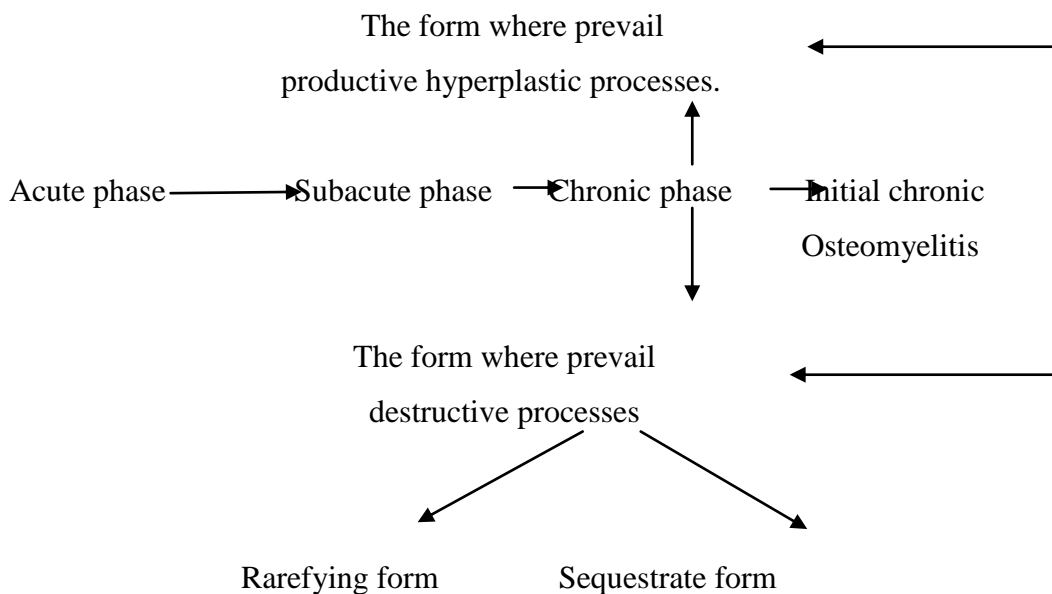
Terminology and classification. Under the term osteomyelitis it is understood not only the inflammatory process in bone marrow, but also in all structural parts of the bone and in surrounding its soft tissue.

Maxilla osteomyelitis represents infectious purulo-necrotic process, which develops in the bone and in its surrounding tissues under the influence of aggressive factors of physical, chemical or biological nature against the preliminary sensibilization and neurohumoral dislocations.

Maxilla osteomyelitis can be: odontogenic (stomatogenic), traumatic, toxic, hematogenic and specific. There are distinguished three phases (periods) of disease course: acute, subacute and chronic.

Depending on the spread process the osteomyelitis can be circumscribed and diffuse. During the circumscribed osteomyelitis the pathological process is localized in the region of two-three tooth parodontium. Diffuse osteomyelitis is characterized by the character of total affection of a half or the whole maxilla.

Classification of the odontogenic osteomyelitis.



Pathological anatomy

During the odontogenic maxilla osteomyelitis the process comprise all components of the bone: bone marrow, the ground substance of the bone, periostenium. Infectious – inflammatory process spreads on perignathic soft tissue in which abscesses and phlegmons are formed. Acute phase of the odontogenic osteomyelitis it is characterized by the diffuse suppurative inflammation of all bone elements without pronounced process demarcation. It is occurred by the edema, plethora and leucocytic infiltration of bone marrow.

In the chronic phase of odontogenic osteomyelitis clever becomes apparent the regions of osteonecrosis, round which the surrounding healthy bone dispersion took place.

In period of 1-2 moths the formation of sequester usually runs down (full separation of regions with osteonecrosis from the unaffected bone). In some cases these are singular or multiple small sequesters (miliary), in other cases - a big regions of the maxilla full-thickness.

Maxilla defect, which appeared in case of sequester formation, is filled by newly formed bony and granulation tissue.

Clinical picture of odontogenic osteomyelitis.

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

In the acute phase of disease, in the beginning patients complain of the pain in the region of one tooth, which is the source of infection. However soon to these join signs of periodont and other nearby teeth inflammation. The pain increases, becomes tearing, irradiating on devarication of trifacial nerve in eye-socket, temporal region and ear.

One of typical complaints during the mandible osteomyelitis is disturbance of the interfacial sensitivity of the lower lip red border (Vincent syndrome), mouth threshold mucosa, the chin of the relevant part (numbness, makes one fill creepy). In case of purulo-inflammatory process in the soft tissues development, the pain moves to outside of the maxilla. Appear complaints typing to perignathic phlegmon (swelling, maxilla contractions, and pain during the deglutition, mastication). Practically permanent it is observed headaches, general weakness, rise of body temperature, appetite and sleep disturbance.

Patients are pale. The pulse is rapid, in some cases arrhythmic. In the region of affected maxilla appears infiltration and edema of soft tissues. It is fetid smell from the mouth. "Casual" tooth at the beginning is immovable, but in the future become impaired. Become impaired and nearly tooth: their percussion is painful. Gum and transitory fold in the tooth region, involved in the purulo-inflammatory process, are edematous and hyperemic. Their palpation is sharply painful. In certain cases appear subgums abscesses. During the penetration of the purulence in cells surfaces appear abscesses and phlegmons of perignathic tissues. In these cases appear the infiltration of the tissues of the dense consistence and hyperemia of skin integument. Near with infiltrate appears pronounced collateral edema of soft tissues. Reflection of the inflammatory reaction of the osteomyelitis also can be lymphadenitis. Infiltration of the soft tissue not frequently spreads on the masseters, which leads to the maxilla contracture (contraction). The most permanent and early osteomyelitis symptoms of the mandible are thickness of its border, disturbance of the interfacial sensitivity of the lower lip red border and the chin skin, changes of tooth electroexcitability. The disturbance of the sensitivity of soft tissues during mandibular osteomyelitis becomes the name of Vincent in honor of the author who described it.

Systemic body reaction is manifested by fever, heart and breath acceleration, rigor, general in evenings, changes in the blood and urine. Body temperature rises till 39-40 C.

It is assumed that the acute phase of maxilla osteomyelitis is attended by neutrocytosis (12-15·10⁹/l) with appearance of new form of neutrophilic leukocytes.

In hard cases we observed neutrocytosis (17·10⁹/l).

BSR(Blood Sedimentation Rate) as a rule is raised till 40-60mm/h.

Acute phase of the upper maxilla osteomyelitis is characterized more easy course, shortening of disease duration, absence of large destructive bone tissue. The upper maxilla osteomyelitis seldom is complicated by the heavy phlegmons. Such variety of osteomyelitis clinical course of upper maxilla is down to the fact of its anatomy-topographical peculiarities – good vascularization, presence of big quantity of apertures in cortical substance, which assists in quick evacuating of the purulent exudate under the periostenum or under the mucosa. To the upper maxilla do not adjoin massive musculature stratum, significant cellule spaces, therefore various phlegmons, purulent leakages during the osteomyelitis of this bone appear seldom.

During the localization of affected bone tissue in the region of upper maxilla hump purulent can spread in wing-shaped palatal fossa, and then through infraorbital fissure on the eye socket pacerollower. In such cases in the beginning appear eyelids edema and infraorbital region, then their infiltration. The purulence can penetrate in infratemporal fossa and maxilla wing-shaped space, causing the heavy clinical course of disease. During the odontogenic osteomyelitis of upper maxilla in some cases in inflammatory process is involved upper maxillar cavity.

Diagnostics

Diagnostics of the acute phase of the odontogenic osteomyelitis as often as not is difficult.

The acute phase of the odontogenic osteomyelitis should differentiate from following diseases: 1) acute parodontitis, 2) acute purulent periostitis, 3) isolating inflammatory process of face soft tissues (abscesses, phlegmons), 4) matured cysts in maxilla facial region (odontogenic, dermatoid, epidormoid).

The absence of affection features of the maxilla periosteum and surrounded soft tissues distinguish acute purulent parodontitis from the osteomyelitis. Focus of inflammation during the parodontitis is limited by the one alveolar socket. Gum and mucosa of transitory fold can be edematous, painful during the palpation. Percussion and pressure on the affected tooth causes pain; the tooth becomes flexible.

Acute purulent periostitis is accompanied by the disturbance of the general state of the patient, subfebrile temperature, moderate changes from blood side. In the process are involved periosteum and soft tissues, which as a rule, leads to the collateral edema and periosteal apposition formation. The patients with acute odontogenic osteomyelitis is more distinct expressed general organism's reactivity also the changes in blood than at patients with periostitis. The maxilla is bulged; take place the mobility of some teeth, neurological disorder of the soft tissues and disturbance of teeth electroirritation. Maxilla facial cysts with the growth causes deformation of soft tissues or maxilla. Suppuration with the typical feature of acute purulent process appears secondary.

Odontogenic osteomyelitis: classification, pathogenic etiology, clinical picture, diagnosis, treatment.

Subacute phase.

Osteomyelitis subacute phase is short-term, but oftener it is lasted 1.5-2 weeks. Subacute phase of the maxilla osteomyelitis is characterized by the stabilization of the inflammatory process. Operative wounds are cleaned from the necrotic tissues, granulate, and decreased flow of pus and edema. Teeth located in the region of the inflammatory nidus, becomes more flexible. Typical symptom of the osteomyelitis acute phase is improvement of the general state of the patient: disappear weakness, become normal sleep and appetite, descend body temperature, significantly diminished leucocytosis and ESR (erythrocyte sedimentation rate).

Chronic osteomyelitis phase.

Chronic phase of the maxilla osteomyelitis is the very prolonged. Pains in the maxilla region calm down, diminished the infiltration of soft tissues. In the section location and other skin parts of the mouth mucosa appear fistulas, from which is effused pus. The tearing away of the sequestrum is accompanied by the appearance of the granulation and fistulous passages. In the region of the osteomyelitis nidus the maxilla is thickened, teeth are usually flexible.

In the cases of the extensive destruction of the lower maxilla body can occur pathological fracture. In the result of exudate elimination delay through fistulas and formation of purulent swell can appear intensification of the inflammatory process. Soft tissues are infiltrated again, appear the signs of purulent-resorption fever.

Diagnostics

Important place in the diagnostics of the chronic maxilla osteomyelitis belong to X-ray examination. To 10-14 twenty-four hours of disease (subacute phase) it is founded focal or diffusive clarification, osteoporosis. Then become apparent one or some focuses of destruction of irregular shape. The major diagnostic value during the osteomyelitis has sequestration, which is characteristic for chronic phase of disease. Sequester boundary is bitterly distinguished on the background of more transparent surrounding bones' elements. During the osteomyelitis of the lower maxilla the sequestration is distinguished only in the end on the 3 (third) – 4 (fourth) week. Sequesters can be different by form – rounded, oval, multangular with irregular edges, central, peripheral, complete.

Chronic odontogenic maxilla osteomyelitis is differentiated from specific maxilla affection (actinomycosis, tuberculosis, syphilis), benign and malignant tumors.

Actinomycosis

During this disease it is generated a lot of fistulas with the crumbly purulence. The bone is swollen during the actinomycosis. It has the look of dense fusiform tumor, inside which become apparent cystic spaces with sings of serosal-purulent exudate.

For **tuberculous** bone affection it is characteristic gentle flow (months, years), sharp morbidity and lymphadenitis. In the pathological process are involved other bones, appears indrow cicatrice.

Syphilis

Maxilla syphilis appears as a result of gummous affection of the bone or periostenium. For the syphilitic bone affection it is characteristic appearance of nidus of specific mollities (necrotic form) and ossifying periostitis (hyperplastic form). During the suspicion for syphilis it is used serological sedimental reactions.

Chronic maxilla osteomyelitis should be differentiated from benign tumors and tumor-like diseases (maturate odontogenic cyst, osteoclastoma, osteoid osteoma, eosinophilic granuloma and e.t.c.), and also malignant tumors.

Treatment

The extraction of the “causative” tooth in the incipience of the acute odontogenic osteomyelitis is basic and obligatory type of therapy of this disease. Alongside with antibacterial, desensitise and desintoxicated medication are used anticoagulants, fibrinolytic and sedative medications. Mercurial intact teeth with are neighboring with “causative”, should be ligated or splinted. For organism desensibilization and reduction of vessel wall penetrability it is used 10% calcium chloride brine, antihistaminic (diphenylhydramine, suprastin).

In subacute phase of the maxilla osteomyelitis it is continued antibacterial therapy.

In the chronic phase of maxilla osteomyelitis in the aggravation cases of inflammatory process it is continued antibacterial, anti-inflammatory treatment. It is executed sequestrectomy. Sequesters on the upper maxilla usually are extracted at 3-4 weeks from the beginning of disease, on the lower later – at 5-8 weeks.

Medicinal treatment: Deintoxication - NaCl (Ringer physiological saline solution) 0,09%, appointed 30-60 ml on the g/kg (for example weight 80 kg, consequently 80 kg multiply on 30 ml will be about 2,4 l liquid intravenously), desintoxication is usually appointed in the acute phase of osteomyelitis to remove the intocsication. Solutions for the desintocsications – solution Hemodez, Refortan, Glucose 50% 500-1000 ml intravenous by drop infusion (after level of sugar in blood examination).

Antibiotic therapy: it is appointed in all phases of osteomyelitis. Usually it is appointed osteotropic antibiotic (which penetrate selectively in the bone tissue -Solution Lincomicini 30% -2 ml intramuscular 2-3 time per day, 5-10 days or solution Kanamicini, is very well to appoint together with sulfanilamides. Desensibilization therapy (antiallergic): solution Suprastini, Dimidroli 1%-1ml, Fencaroli. Analgesic medications (especially in the acute phase and great pains: solution Analigini 50%-2ml, Naclofen, Ketonal, Tab. Dexalгинi. Vitaminotherapy and immunotherapy.

Topic № 4

Diseases of teeth eruption

From the abnormality of teeth eruption, more often occurs hampered eruption, irregular tooth location, appearing in the process of eruption (dystopia), incomplete tooth eruption through maxilla bone tissue or mucosa (half retention), delay of full formed tooth eruption through solid bone substance (retention).

Hampered lower wisdom tooth eruption - (Pericoronitis)

The eruption of the lower wisdom teeth happens mainly in the age of 18-25 years, sometimes later. More often lower wisdom tooth is two-root with big and great pronounced crown, often with curved edges. Definite significance during the hampered eruption of the lower third molar have:

- 1) Absence of predecessor of milk-tooth that is why the bone structure under the teeth is made denser;
- 2) Thick and dense compact bone layer along the external and internal maxilla surface together with external and slanting line;
- 3) Lack of place in the branch;
- 4) Thick mucous membrane, including fibres of check muscle and upper constrictor of the throat, which creates the soft-tissue barrier for tooth eruption.

Sheinberg V.M. and Jitnischky G. D. (1970) developed the classification of lower wisdom tooth position depending on its axis position regard to distal portion of lower maxilla alveolar part and its branch:

- 1) Vertical (wisdom tooth axis is parallel to second molar axis);
- 2) Medial-slanting (wisdom tooth axis is inclined to second molar axis);
- 3) Distal-slanting (wisdom tooth axis is inclined to lower maxilla branch);
- 4) Horizontal (wisdom tooth axis is perpendicular to second molar axis);
- 5) Lingual (tooth axis is declined in lingual part);
- 6) Buccal (tooth axis is declined in buccal part);
- 7) Combined (various combinations of pointed above positions).

After the eruption of the upper part of the crown and exposition of the one or two medial humps of lower wisdom tooth the mucosa membrane covers it in the form of hood. Under it accumulates plentiful microflora. J. Moore (1985) and also J. Sowrai (1985) consider that during the hampered tooth eruption of lower wisdom tooth it is necessarily to distinguish acute and chronic pericoronitis.

Acute pericoronitis.

Acute Pericoronitis represents the preliminary process of gingiva tissue inflammation and acentric periodont during the hampered lower wisdom tooth eruption.

Ethiology.

Pericoronitis appears as a result of activation of usual microflora of mouth cavity, in which prevail anaerobic and facultatively – anaerobic types of bacteria.

Pathogenesis.

During the lower tooth eruption the mucosa membrane under the medial humps mummified; remaining part of the crown remain covered by the mucosa membrane – hood. In the pericoronal spatium (between tooth crown and internal hood surface) fall food debris, cells of desquamated epithelium, slime. Trauma of the hood mucosa membrane during the mastication causes to formation on its surface anabrosis and ulcerations and local immunity lowering. As a result appear inflammatory phenomenon in tissues, which surround the crown of the noncompletely erupted wisdom tooth.

Clinical picture.

Acute pericoronitis can be catarrhal and purulent. The catarrhal form develops in the beginning of disease. Patients complain to the pain during the mastication in the region of the erupted wisdom tooth. General state of patients is satisfactory, body temperature is normal. In the submandibular region is defined painful and increased lymph gland. Mouth opening is free. The wisdom teeth are covered with hyperemic or edematous hood of mucosa membrane. Usually there are seen only one or both medial humps of the tooth. In some cases all tooth crown is under the hood and only level up it can see tooth. Eliminations from the hood is not observed, its palpation is painful. This form of pericoronitis proceeds productive and in case of timely treatment the process cut short rapidly.

Purulent pericoronitis

Purulent pericoronitis is characterized by permanent pain behind of second molar and increasing during the mastication. Pain “gives back” in the ear, temporal region. Pain appears during the deglutition (odontogenic angina). The general state of the patient suffers, the body temperature increases till 37.2-37.5 °C. Mouth opening becomes restricted and painful (inflamed contracture of I degree). During the progression of inflammatory phenomenon, the mouth opening is more restricted (contracture of II degree). Submandibular lymph glands are increased, their palpation is painful. The mucosa membrane of the wisdom tooth is hyperemic, edematous as in the region of infiltrate and elevated upwards hood, as in the region aliform-mandibular rugosity and lower fornix vestibule of mouth on the level of lower molars. Inflammatory processes invade on the mucosa membrane of the palatoglossal arch, check and soft palate. During the pressure on the hood from under it effuse purulent contents, appears sharp pain.

Diagnosis is made on the base of clinical picture and results of X-ray examination. On the base of X-ray picture of the mandibular in the basal view is possible to argue about the position of the lower wisdom tooth, state of its periodont and surrounding bone, attitude to the branch and canal of lower maxilla.

Chronic pericoronitis.

In the lower wisdom tooth the processes develops in the course of frequent traumatizing of the hood and repeated aggravations of inflammatory process. Purulence delay and granulation development leads to the bone resorption. Depending on tooth position covered it fully or partially by mucosa membrane and disease duration chronic pericoronitis can be attended by:

- 1) Destruction of the alveolar bone part vertically or horizontally;
- 2) Development of marginal granulating or granulomatous parodontitis.

Clinical picture.

Clinical picture of the chronic pericoronitis is various. In the same cases patients complain on the hamper mastication on the affected part, morbidity of the hood under the wisdom tooth, in other cases only on the discomfort in the mouth cavity, fetor from the parts of wisdom tooth.

In the submandibular distal region during the examination occur increased and painful lymph glands. Mucous membrane under the partially erupted wisdom tooth is more often hyperemic and edematous. From underneath of the hood in one case effuse scanty serosal secretion, in other cases purulent exudate. Exteriorly the mucosa hood can be seen expression; its edge is often cicatricial changed. During the excavation intubation occur hemorrhage. Palpation of tissues is painful along the external and internal surface of the mandibular alveolar part. Chronic pericoronitis can aggravate.

Diagnostics.

The disease is diagnosed on the base of clinical picture and X-ray data, during which are registered destructive changes to anterior and behind tooth, and also downwards during its horizontal position.

Differential diagnostics is made with chronic pulpitis and parodontitis, in certain cases – with neuralgia of third branch of trifacial nerve.

Behind molar periostitis.

Clinical picture.

The disease develops in consequence of disturbance of exudate outflow during the pericoronitis, and also expansion of purulent infection from the marginal periodont and from under hood on the periostenium behind molar caveola and pacefollower of behind molar space, where abscess is forming. The pain becomes more intensive. General state suffers, appears weakness, body temperature increases till 38-38.5 °C. Inflammatory contracture is sharp expressed (II-III degree), mastication of the food becomes impossible and sleep suffers.

The patient is pale; it is registered evident tissue edema in the after portion of submandibular and lower part of buccal region. Submandibular lymph glands are increased and painful. Examination of the mouth cavity is managed to perform only after forcible release of maxilla. Palpation of the hood and surrounding tissues is sharp painful. In behind molar region it is enlisted infiltrate, which exceed on external, rarer, internal surface of alveolar maxilla part.

Diagnosis.

It is necessary to take into consideration clinical picture with expressed appearance, X-ray pictures date and laboratory indicants of blood (moderate leucocytosis, shift of the leukogram in the left and ESR increase till 25-30 mm/s).

Differential diagnostics. The disease should be differentiated from the abscess of wing-shaped- mandibular and peritonsillar spaces, sublingual region.

Treatment.

Pericoronitis and behind molar periostitis is treated predominantly in clinic conditions. Measures complex depends from the evidence of inflammatory phenomenon, general and local picture and also from the X-ray picture data.

In the first place, it is necessary to liquidate acute inflammatory appearances. During the catarrhal form of pericoronitis can be effective only the treatable therapy under the hood by the antiseptic liquids from the syringe with the dull needle. During the purulent pericoronitis it is made pericoronarotomy - hood discission, which covers the tooth crown and create the conditions of the exudate outflow.

Discission during the behind molar periostitis is made through the mucous membrane from the subiculum of wing-shaped – mandibular rugosity down to the fornix of buccal cavity. During the inflammatory infiltration of the tissue of buccal cavity fornix the discission is prolonged along the alveolar part of the maxilla on the molar level. The wound is drained by the rubber strip. It is necessary daily bandaging.

During the pericoronitis and behind molar periostitis the good curative effect is given by the single or double blocking with anesthetic by type of conduction and infiltrative anesthesia. It is justified the intake of the sulfanilamidis anti-inflammatory and antihistaminic means. During acute retromolar periostitis there are putted on the antibacterial means during 6-7 days. During the chronic pericoronitis, after tooth removal should be carefully scraped out pathological granular enlargements and to stitch the wound completely. In the case if there is a big bone defect should be introduced in the bone cavity granules of the hydroxylapatite, Colapol and stitch the wound completely.

Prognosis is advantageous generally.

Pericoronitis and behind molar periostitis can be the reason of purulent lymphadenitis of submandibular region, actinomycosis, ulcerative stomatitis. More often repeated aggravation of the processes, as often as not, leads to the death of compact layer of the lower maxilla branch and development of the second cortical osteomyelitis.

Eruption cysts.

The development of the eruption cyst is tighten with the remains of embrional epithelium or encapsulated epithelium because of inflammation. Such cyst is named - follicular.

Clinical picture. The cyst is difficult to distinguish from the acute pericoronitis, but more often symptoms argue about chronic pericoronitis. Diagnostics is based on the X-ray picture: it is shown up the place of bone destruction with sharp bounds adjacent to lower third molar.

Parodontal cyst can be situated:

- 1) Behind of the tooth crown;
- 2) Near the distal surface of the distal root;
- 3) Near the medial root surface and tooth crown;
- 4) In the root region and periapical region;
- 5) In two present cyst's cavity.

Differential diagnostics is made with cysts and odontogenic tumors of the maxilla.

Treatment. When cysts are little sizes, it is eliminated together with the lower wisdom tooth.

Handicap eruption of other teeth

Inflammatory events more often are seen during the premolar eruption, canines on the both maxilla and third upper root tooth. Depending on wisdom tooth placement, pressure on the neighboring teeth appear pains, swelling of mucous membrane, covering the alveolar bone from one and other part, sometimes phenomena of acute parodontitis.

Diagnostics. Clinical diagnosis is proved out by the X-ray data.

Differential diagnostics is made with the odontogenic inflammatory diseases, cyst's purulence.

Treatment. Depending on clinical picture is placed on the same treatment as during the pericoronitis and periostitis, abscesses, phlegmons and maxilla osteomyelitis, accordingly to the therapy principles of such diseases. During the remitting of inflammatory processes the question about tooth removal is discussed, which cannot erupt and is misplaced, half impacted or impacted.

Prognosis during the handicap tooth eruption is advantageous.

Preventive measures of the disease of teeth eruption consists in control of right maxilla development and teeth eruption, mouth cavity sanitation and timely orthodontic treatment, observance of mouth cavity hygiene rules.

After inflammatory processes remitting it is necessary to deal with a problem of tooth destiny. If it is situated right and in the alveolar part is enough place for it, then the reason of the hampered eruption is solid mucous membrane covered its crown. Mucous membrane is exsected by scalpel or curved scissors.

Topic № 5

The role of the head and cervix interfacial and intramuscular spaces during the spread of infection in maxillofacial soft tissues.

Trucks of infection spread.

Quick spread of the purulent exudate is caused by such anatomical peculiarities of maxillofacial region as presence of big quantity of fatty tissue, which forms a wide range of interlocking cellular spaces, abundant net of blood-vessels, well-marked the system of lymph vessels and nodes.

To the front part of the face are related regions of chin, mouth, nose and eye-socket. The lateral part of the face consist cheek and parotid-masticatory regions. In the stomatology also is accepted in addition to mark out: infraorbital and malar regions and also temporal region.

The front part of the neck includes supraglottic and sublingual regions. To suprahyoid region are related tissues of submandibular and submental triangles participating in the formation of the mouth floor. In sublingual region are distinguished carotic and scapular-tracheal triangles. Outside to them is linked the region of placement of sternal-clavicular-mastoid muscle.

Fascia and cellular face spaces

Interfacial infraorbital cellulose is placed between orbicular muscle of eye and square muscle of the lower lip, melt to the lateral side of external nose. To it permeate branches of facial artery and vein and malar branch of facial nerve. More deeply is situated the cellulose of canine fossa. In deeply cellulose infraorbital region permeate infraorbital vessels-nerve fascicle, descending from infraorbital canal. Cheek fat body, its fascial compartment can serve as infection conductor in cheek region from the parotid-masticatory, temporal and other regions.

In after part of the region, in the fascia parotid notch, during the lamination, forms bed of parotid gland. In its fascial bed are placed auriculotemporal and facial nerves, outside carotid artery with its branches, retromandibular vein, superficial and deep parotid lymph nodes. Implication in the inflammatory process of the facial nerve` branches leads to its affection, and destruction of the wall of the external carotid artery can lead to profuse bleeding.

Between folium of the temporal fascia and maxillary arch is encompassed interaponevrotic cellular space of temporal region. The content of the space: middle temporal artery and vein, zygomaticotemporal branch of zygomatic nerve. Interaponevrotic space directly goes on cellular space of zygomatic region, which is encompassed between zygomatic bone and superficial folium of temporal fascia.

Pterygo-temporal space is restricted from the outside by the lower part of temporal muscle, but from inside lateral pterygoid muscle. In friable cellulose of this space go on venous pterygoid plexus, uppermaxilla artery and its branches, lowermaxilla nerve. Between lateral and medial pterygoid muscles there is infrapterygoid space. In the cellulose of the pterygopalatine fossa is placed uppermaxilla artery, uppermaxilla nerve, pterygopalatine ganglion maxillary nerve.

Phlegmons of the infratemporal and pterygopalatine fossa appears as a result contagion from the part of third molars of upper and lower maxilla.

Eye-socket fatty tissue of the adjacents to its bony walls. In the fatty tissue of the eye socket there are muscles of the eye ball, veins and nerves. Infection penetrates in the eye socket

through lower orbital cleft, from the canine fossa through infraorbital canal, and during the thrombophlebitis by face's veins.

Maxillary sinus - is the biggest from the paranasal sinuses. Its volume consists 10-12 cm³, varying from 3 till 20 cm³.

In the sinus are distinguished upper, inner, front-lateral, back-lateral and lower walls. Inner sinus wall delimits its from the nose cavity. Its least thickness is on the middle of lower edge, and the largest in the region of anteroinferior angle, what should be taken into consideration during the sinus punctuation and formation of inosculation with the nose cavity.

The space contains bid vessels and nerves (inner carotid artery, inner jugular vein, glossopharyngeal additional and sublingual nerve, upper neck sympathetic ganglion).

Fascia and cellular spaces of the neck front part

Among the neck lower bound in front superficial neck fascia also laminates and attaches to the presternum by two foliums, between which is encompassed episternal interaponevrotic space. This space is filled by the fatty tissue, in which are situated horizontal parts of frontal jugular veins.

Medial neck cellular space, surrounded the neck organs, can be by the way of odontogenic infection spread in the frontal and after mediastinal.

Fascial axil of the main vessel-nerve fascicle contains own compartments of general carotid artery and inner jugular vein with surrounding it numerous lymph ganglions.

Prespinal cellular space is situated between the spondyls bodies and prespinal neck fascia.

Blood and lymph system of the neck and face

Blood vessels of the face and neck are characterized by the abundance of the intrasystem and intersystem inosculations, which caused the infection spread. Hematogenic infection spread can lead to venous thrombosis and sepsis.

The main role plays lymph system in the appearance and spread of inflammatory diseases in maxillofacial region. In the result of penetration of microflora in the lymph vessels and regional lymph glands from the focus of acute or chronic inflammation develops catarrhal, and then purulent lymphadenitis.

The disease which is localized superficially not frequently constitutes a danger considering the inclination to spread depthward along the cellulose, blood and lymph vessels. Localization and possible way of spread are shown on the picture 1.

Anatomical ways of contamination of brain tunics are:

- 1) Cheek cellulose surface – canine fossa cellulose – infraorbital canal – eye socket fatty tissue – upper infraorbital cleft – skull cavity;
- 2) Fatty body of the cheek – eye socket fatty tissue;
- 3) Infraorbital cellulose – angular vein – orbital veins – cavernous sinus;
- 4) Cheek fatty body – infratemporal fossa cellulose and pterygoid plexus – oval and round pore – skull cavity.

In the mediastinal infection can penetrate by two main ways: along the duct of neck organs and along the superficial folium of neck fascia, consequently captivate fascial axils

submandibular gland, the main neck fascicle vessels, sternal-clavicular-mastoid muscle, episternal and supraclavicular interaponevrotic space.

Depending on the anatomico-topographical localization odontogenic abscesses and phlegmons can be divided into 4 groups conditionally:

1. Tissue abscesses and phlegmons, which adjoin to the upper maxilla:
 - a) superficial - suborbital, buccal, zygomatic regions;
 - b) deep – subtemporal and pterygopalatal fosa;
 - c) secondary – temporal region, eye socket region.
2. Tissue abscesses and phlegmons, which adjoin to the lower mandibular:
 - a) superficial – submandibular, submental triangles;
 - b) deep – pterygomandibular, peripharyngeal, sublingual spaces;
 - c) secondary – retromandibular region, frontal regions and cervix (neck) spaces.
3. Tongue abscesses and phlegmons.
4. Widespread phlegmons of face and cervix.

Canine Fossa Abscess

Anatomic Location. The canine fossa, which is where this type of abscess develops, is a small space between the levator labii superioris and the levator anguli oris muscles.

Buccal Space Abscess

Anatomic Location. The space in which this abscess develops is between the buccinator and masseter muscles. Superiorly, it communicates with the pterygopalatine space; inferiorly with the pterygomandibular space. The spread of pus in the buccal space depends on the position of the apices of the responsible teeth relative to the attachment of the buccinators muscle.

Infratemporal Abscess

Anatomic Location. The space in which this abscess develops is the superior extension of the pterygomandibular space. Laterally, this space is bounded by the ramus of the mandible and the temporalis muscle, while medially, it is bounded by the medial and lateral pterygoid muscles, and is continuous with the temporal fossa. Important anatomic structures, such as the mandibular nerve, mylohyoid nerve, lingual nerve, buccal nerve, chorda tympani nerve, and the maxillary artery, are found in this space. Part of the pterygoid venous plexus is also found inside this space.

Temporal Abscess

Anatomic Location. The temporal space is the superior continuation of the infratemporal space. This space is divided into superficial and deep temporal spaces. The superficial temporal space is bounded laterally by the temporal fascia and medially by the temporalis muscle, while the deep temporal space is found between the medial surface of the temporalis muscle and the temporal bone.

Mental Abscess

Anatomic Location. The accumulation of pus in this space is located at the anterior region of the mandible, near the bone, and, more specifically, underneath the mentalis muscle, with spread of the infection towards the symphysis menti.

Submental Abscess

Anatomic Location. The submental space in which this abscess develops is bounded superiorly by the mylohyoid muscle, laterally and on both sides by the anterior belly of the digastrics

muscle, inferiorly by the superficial layer of the deep cervical fascia that is above the hyoid bone, and finally, by the platysma muscle and overlying skin. This space contains the anterior jugular vein and the submental lymph nodes.

Sublingual Abscess

There are two sublingual spaces above the mylohyoid muscle, to the right and left of the midline. These spaces are divided by dense fascia. Abscesses formed in these spaces are known as sublingual abscesses.

Anatomic Location. The sublingual space is bounded superiorly by the mucosa of the floor of the mouth, inferiorly by the mylohyoid muscle, anteriorly and laterally by the inner surface of the body of the mandible, medially by the lingual septum, and posteriorly by the hyoid bone. This space contains the submandibular duct (Wharton's duct), the sublingual gland, the sublingual and lingual nerve, terminal branches of the lingual artery, and part of the submandibular gland

Submandibular Abscess

Anatomic Location. The submandibular space is bounded laterally by the inferior border of the body of the mandible, medially by the anterior belly of the digastric muscle, posteriorly by the stylohyoid ligament and the posterior belly of the digastric muscle, superiorly by the mylohyoid and hyoglossus muscles, and inferiorly by the superficial layer of the deep cervical fascia. This space contains the submandibular salivary gland and the submandibular lymph nodes.

Submasseteric Abscess

Anatomic Location. The space in which this abscess develops is cleft-shaped and is located between the masseter muscle and the lateral surface of the ramus of the mandible. Posteriorly it is bounded by the parotid gland, and anteriorly it is bounded by the mucosa of the retromolar area.

Pterygomandibular Abscess

Anatomic Location. This space is bounded laterally by the medial surface of the ramus of the mandible, medially by the medial pterygoid muscle, superiorly by the lateral pterygoid muscle, anteriorly by the pterygomandibular raphe, and posteriorly by the parotid gland. The pterygomandibular space contains the mandibular neurovascular bundle, lingual nerve, and part of the buccal fat pad. It communicates with the pterygopalatal, infratemporal, submandibular, and lateral pharyngeal spaces.

Lateral Pharyngeal Abscess

Anatomic Location. The lateral pharyngeal space is conical shaped, with the base facing the skull while the apex faces the carotid sheath. It is bounded by the lateral wall of the pharynx, the medial pterygoid muscle, the styloid process and the associated attached muscles and ligaments, and the parotid gland. The lateral pharyngeal space contains the internal carotid artery, the internal jugular vein with the respective lymph nodes, the glossopharyngeal nerve, hypoglossal nerve, vagus nerve, and accessory nerve. It communicates directly with the submandibular space, as well as with the brain by way of foramina of the skull.

Retropharyngeal Abscess

Anatomic Location. The retropharyngeal space is located posterior to the soft tissue of the posterior wall of the pharynx and is bounded anteriorly by the superior pharyngeal constrictor muscle and the associated fascia, posteriorly by the prevertebral fascia, superiorly by the base of the skull, and inferiorly by the posterior mediastinum.

Parotid Space Abscess

Anatomic Location. The space in which this abscess develops is located in the area of the ramus of the mandible and, more specifically, between the layers of the fascia investing the parotid gland. It communicates with the lateral pharyngeal and the submandibular spaces. It contains the parotid gland and its duct, the external carotid artery, the superficial temporal and facial artery, the retromandibular vein, the auriculotemporal nerve, and the facial nerve. In order to understand how odontogenic infections are treated, the dentist must be familiar with the terminology concerning infection and the pathophysiology of inflammation, which are described below.

Inflammation is the localized reaction of vascular in the development of an exudate rich in proteins and cells. This reaction is protective and aims at limiting or eliminating the irritant with various procedures while the mechanism of tissue repair is triggered. Depending on the duration and severity, inflammation is distinguished as acute, subacute or chronic.

Acute Inflammation. This is characterized by rapid progression and is associated with typical signs and symptoms. If it does not regress completely, it may become subacute or chronic.

Subacute Inflammation. This is considered a transition phase between acute and chronic inflammation.

Chronic Inflammation. This procedure presents a prolonged time frame with slight clinical symptoms and is characterized mainly by the development of connective tissue.

The natural progression of inflammation is distinguished into various phases. Initially vascular reactions with exudate are observed (serous phase), and then the cellular factors are triggered (exudative or cellular phase). The inflammation finally resolves and the destroyed tissues are repaired. On the other hand, chronic inflammation is characterized by factors of reparation and healing. Therefore, while acute inflammation is exudative, chronic inflammation is productive (exudative and reparative). Understanding the differences between these types of inflammation is important for therapeutic treatment

Serous Phase. This is a procedure that lasts approximately 36 h, and is characterized by local inflammatory edema, hyperemia or redness with elevated temperature, and pain. Serous exudate is observed at this stage, which contains proteins and rarely polymorphonuclear leukocytes.

Cellular Phase. This is the progression of the serous phase. It is characterized by massive accumulation of polymorphonuclear leukocytes, especially neutrophil granulocytes, leading to pus formation. If pus forms in a newly developed cavity, it is called an abscess. If it develops in a cavity that already exists, e.g., the maxillary sinus, it is called an empyema .

Fundamental Principles of Treatment of Infection

In order to treat an acute dentoalveolar infection as well as a fascial space abscess correctly, the following are considered absolutely necessary:

Take a detailed medical history from the patient. Drainage of pus, when its presence in tissues is established. This is achieved by way of the root canal, with an intraoral incision, with an extraoral incision, and through the alveolus of the extraction. Without evacuation of pus, that is with administration of antibiotics alone, the infection will not resolve.

Drilling of the responsible tooth during the initial phase of inflammation, to drain exudate through the root canal, together with heat therapy. In this way, spread of inflammation is avoided and the patient is relieved of the pain. Drainage may also be performed with trephination of the buccal bone, when the root canal is inaccessible.

Antisepsis of the area with an antiseptic solution before the incision.

Anesthesia of the area where incision and drainage of the abscess are to be performed, with the block technique together with peripheral infiltration anesthesia at some distance from the inflamed area, in order to avoid the risk of existing microbes spreading into deep tissues.

Planning of the incision so that:

- – Injury of ducts (Wharton, Stensen) and large vessels and nerves is avoided/
- – Sufficient drainage is allowed. The incision is performed superficially, at the lowest point of the accumulation, to avoid pain and facilitate evacuation of pus under gravity.
- – The incision is not performed in areas that are noticeable, for esthetic reasons; if possible, it is performed intraorally.

Incision and drainage of the abscess should be performed at the appropriate time. This is when the pus has accumulated in the soft tissues and fluctuates during palpation, that is when pressed between the thumb and middle finger, there is a wave-like movement of the fluid inside the abscess. If the incision is premature, there is usually a small amount of bleeding, no pain relief for the patient and the edema does not subside.

The exact localization of pus in the soft tissues (if there is no fluctuation present) and the incision for drainage must be performed after interpretation of certain data; for example, ascertaining the softest point of swelling during palpation, redness of the skin or mucosa, and the most painful point to pressure.

This area indicates where the superficial incision with a scalpel is to be made. If there is no indication of accumulation of pus to begin with, hot intraoral rinses with chamomile are recommended to speed up development of the abscess and to ensure that the abscess is mature.

Avoid the application of hot compresses extraorally, because this entails an increased risk of evacuation of pus towards the skin (spontaneous drainage).

Drainage of the abscess is initially performed with a hemostat, which, inserted into the cavity of the abscess with closed beaks, is used to gently explore the cavity with open beaks and is withdrawn again with open beaks. At the same time as the blunt dissection is being performed, the soft tissues of the region are gently massaged, to facilitate evacuation of pus.

Placement of a rubber drain inside the cavity and stabilization with a suture on one lip of the incision, aiming to keep the incision open for continuous drainage of newly accumulated pus.

Removal of the responsible tooth as soon as possible, to ensure immediate drainage of the inflammatory material, and elimination of the site of infection. Extraction is avoided if the tooth can be preserved, or if there is an increased risk of serious complications in cases where removal of the tooth is extremely difficult.

Administration of antibiotics, when swelling is generally diffuse and spreading, and especially if there is fever present, and infection spreads to the fascial spaces, regardless of whether there is an indication of the presence of pus.

Antibiotic therapy is usually empiric, given the fact that it takes time to obtain the results from a culture sample. Because the microorganisms isolated most often in odontogenic infections are streptococci (aerobic and anaerobic), penicillin remains the antibiotic of choice for treatment.

Canine Fossa Abscess

Anatomic Location. The canine fossa, which is where this type of abscess develops, is a small space between the levator labii superioris and the levator anguli oris muscles.

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

Clinical Presentation. This is characterized by edema, localized in the infraorbital region, which spreads towards the medial canthus of the eye, lower eyelid, and side of the nose as far as the corner of the mouth. There is also obliteration of the nasolabial fold, and somewhat of the mucolabial fold.

The edema at the infraorbital region is painful during palpation, and later on the skin becomes taut and shiny due to suppuration, while its color is reddish.

Treatment. The incision for drainage is performed intraorally at the mucobuccal fold (parallel to the alveolar bone), in the canine region. A hemostat is then inserted, which is placed at the depth of the purulent accumulation until it comes into contact with bone, while the index finger of the nondominant hand palpates the infraorbital margin. Finally, a rubber drain is placed, which is stabilized with a suture on the mucosa.

Palatal abscess

Clinical Presentation. As far as the palatal abscess is concerned, it manifests as a circumscribed swelling, respective to the responsible tooth. The mucosa appears reddish, while sensitivity is observed during palpation and fluctuation.

Treatment. Incision and drainage of palatal abscesses require special attention to ensure avoiding injury to the greater palatine artery, vein, and nerve. Therefore, the incision must not be made perpendicular to the course of the aforementioned vessels and nerve, but near the border of the gingivae or towards the midline and parallel to the dental arch. Drainage of the abscess is achieved with a curved hemostat. After drainage, the patient is relieved of pain, and resolution of the abscess, in other words the healing stage, begins.

Zygomatic (malar) region

Boundaries of anatomical region, space: upper – anteroinferior section of temporal region and lower edge of the eye socket, lower-anterosuperior section of buccal region, frontal – bulge-maxilla suture, dorsal – bulge-temporal suture.

The main sources and ways of 14,15,16,24,25,26 teeth. Secondary affection as a result of infection extension from the buccal and subtemporal regions.

Local signs of inflammatory - infectious processes: pronounced inflation of zygomatic region tissues, Hyperemia of the skin of zygomatic region, mastication difficulty because of moderate jaws contracture and pain increase because of mouth opening.

Surgical approach for drain of inflammatory-infectious locus: from the side of skin integument the section in the zygomatic region performs taking into consideration position of face nerves branches.

Temporal Abscess

Etiology. Infection of the temporal space is caused by the spread of infection from the infratemporal space, with which it communicates.

Clinical Presentation. It is characterized by painful edema of the temporal fascia, trismus (the temporalis and medial pterygoid muscles are involved), and pain during palpation of the edema.

Treatment. The incision for drainage is performed horizontally, at the margin of the scalp hair and approximately 3 cm above the zygomatic arch. It then continues carefully between the two layers of the temporal fascia as far as the temporalis muscle. A curved hemostat is used to drain the abscess.

Infratemporal Abscess

Etiology. Infections of the infratemporal space may be caused by infected root canals of posterior teeth of the maxilla and mandible, by way of the pterygomandibular space, and may also be the result of a posterior superior alveolar nerve block and an inferior alveolar nerve block.

Clinical Presentation. Trismus and pain during opening of the mouth with lateral deviation towards the affected side, edema at the region anterior to the ear which extends above the zygomatic arch, as well as edema of the eyelids are observed.

Treatment. The incision for drainage of the abscess is made intraorally, at the depth of the mucobuccal fold, and, more specifically, laterally (buccally) to the maxillary third molar and medially to the coronoid process, in a superoposterior direction. A hemostat is inserted into the suppurated space, in a superior direction. Drainage of the abscess may be performed extraorally in certain cases. The incision is performed on the skin in a superior direction, and extends approximately 3 cm. The starting point of the incision is the angle created by the junction of the frontal and temporal processes of the zygomatic bone.

Drainage of the abscess is achieved with a curved hemostat, which is inserted through the skin into the purulent accumulation

Pterygomandibular Abscess

Etiology. An abscess of this space is caused mainly by infection of mandibular third molars or the result of an inferior alveolar nerve block, if the penetration site of the needle is infected (pericoronitis).

Clinical Presentation. Severe trismus and slight extraoral edema beneath the angle of the mandible are observed. Intraorally, edema of the soft palate of the affected side is present, as is displacement of the uvula and lateral pharyngeal wall, while there is difficulty in swallowing.

Treatment. The incision for drainage is performed on the mucosa of the oral cavity and, more specifically, along the mesial temporal. The incision must be 1.5 cm long and 3–4 mm deep. A curved hemostat is then inserted, which proceeds posteriorly and laterally until it comes into contact with the medial surface of the ramus. The abscess is drained, permitting the evacuation of pus along the shaft of the instrument.

Eye-socket region

Boundaries of anatomical region, space: eye socket region correspond to the eye socket parietes.

The main sources and ways of 34,35,36,44,45,46 teeth. Secondary affection as a result of infection extension from the upper maxillary cavity, subtemporal region, pterygo-maxillary space, suborbital region, pterygo-palatal fosa.

Local signs of inflammatory - infectious processes: Pronounced eyelid edema, exophthalmos, Hyperemia of eyelid skin. Visibility deterioration because of eyelid edema, deflection of eyeball, compression of eye nerve.

Surgical approach for drain of inflammatory-infectious locus: From the side of skin integument section along the lower-external or upper-external eye socket edge. From the side of upper-maxilla cavity the exertion of the dorsal section of eye socket floor.

Localization of abscess, phlegmon.	Boundaries of anatomical region, space	The main sources and ways of.		Local signs of inflammatory- infectious processes.				Surgical approach for drain of inflammatory-infectious locus
				(bulge by means of edema, tissue infiltration)	reddening	Pain (atrest)	Functional damage and its reasons of appearance	
Suborbital region.	Upper – lower edge of eye socket, lower-alveolar bone of the upper maxilla, inner-edge of piriform fenestra, external – maxilla check bone suture.	5 4 3 2	2 3 4 5	Pronounced tissue swelling of suborbital region, edema of upper and lower eyelid.	Hyperemia of the skin of the suborbital region.	Moderate	Vision disorders because of eyelid closure.	Under the deep localization of the processes (in the region of canine fossa) intraoral section goes by the upper fornix threshold. Under the superficial localization of the processes, the section from the part of skin integument is parallel to lower edge of the eye socket or along the nasolabial sulcus.
		teeth. Secondary affection as a result of infection extension from check region and lateral part of the nose.						
Zygomatic (malar) region.	Upper – antereinferior section of temporal region and lower edge of the eye socket, lower-anterosuperior section of buccal region, frontal – bulge-maxilla suture, dorsal –bulge-temporal suture.	6 5 4	4 5 6	Pronounced inflation of zygomatic region tissues.	Hyperemia of the skin of zygomatic region.	Moderate	Mastication difficulty because of moderate jaws contracture and pain increase because of mouth opening.	From the side of skin integument the section in the zygomatic region performs taking into consideration position of face nerves branches.
		teeth. Secondary affection as a result of infection extension from the buccal and subtemporal regions.						
Eye socket	Eye socket region correspond to the eye socket parietes.	5 4 3 3 4 5		Pronounced eyelid edema, exophthalmos.	Hyperemia of eyelid skin.	Severe headache	Visibility deterioration because of eyelid edema, deflection of eyeball, compression of eye nerve.	From the side of skin integument section along the lower-external or upper-external eye socket edge. From the side of upper-maxilla cavity the exertion of the dorsal section of eye socket floor.
		teeth. Secondary affection as a result of infection extension from the upper maxillary cavity, subtemporal region, aliform-maxilla space, suborbital region, aliform-palatal caveola.						
Buccal region	Upper-lower edge of the zygomatic bone,	8 7 6 5 4	4 5 6 7 8	Pronounced swelling of	Hyperemia of the	Mod	Restriction during mouth	From the side of skin integuments

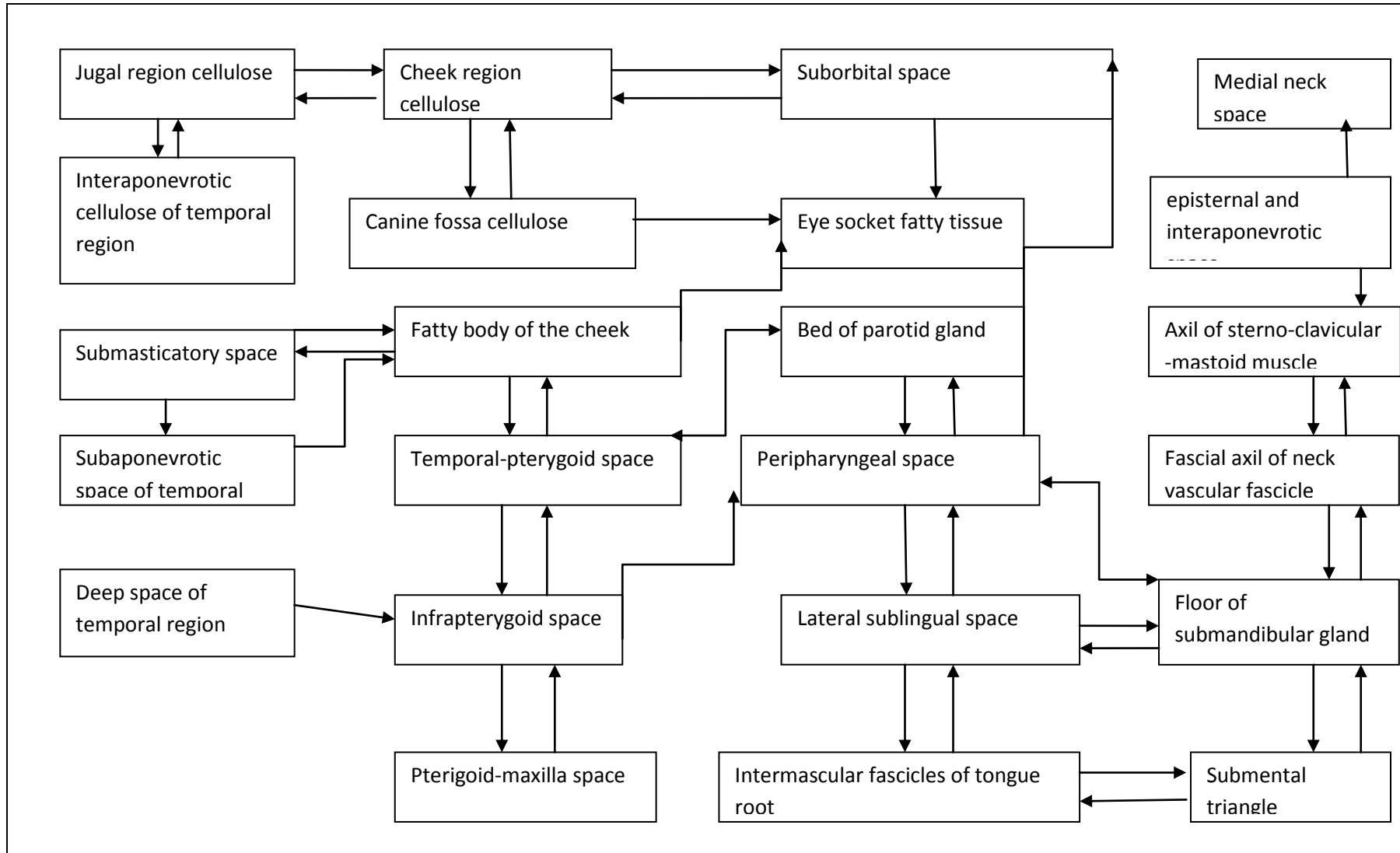
	lower edge on the mandibular, frontal line which connect zygomatic-maxilla suture with the mouth angle, dorsal-frontal edge of mastication muscle.	87 6 5 4 4 5 6 7 8 teeth. Secondary affection as a result of infection extension from the suborbital, parotid – mastication and subtemporal regions.	the cheek (under the superficial localization of the processes).	mucosa membrane of the cheek.	erate	opening because of pain.	section in the buccal region parallel to way of face nerves branches or along to nosolabial fold. From the side on mouth cavity sections are made parallel to way of excretory duct parotid-salivary gland (above or below it)
Infratemporal fossa.	Upper –subtemporal crista of main bone, frontal-knot of upper maxilla and zygomatic bone, dorsal-belemnold with the beginning from its muscles, inner-external palate of aliform crest of the main bone, external – surface of the branch of the mandibular.	8 7 7 8 teeth. Infection during the anesthesia. Secondary affection as a result of infection extension from buccal, parotid-mastication regions, from the aliform-maxilla space.	Infiltrate in the dorsal side of the vestibule of mouth fornix.	Hyperemia of the mucosa membrane of vestibule of mouth fornix.	Severe. Can be irradiation of the pain in the eye.	Moderate restriction during mouth opening and amplitude of lateral movement of lower maxilla (in the health side).	From the side of mouth cavity section is performed through transitory fold of vestibule of mouth fornix under big root teeth. From the side of skin integuments the section is performed along the frontal edge of the temporal muscle, in the subzygomatic region, in the submaxilla region (with the ablation of the medial aliform muscle from the lower maxilla).
Temporal region.	Upper and dorsal temporal line, lower – infratemporal crest of main bone , frontal – zygomatic and frontal bones, inner temporal field, formed by the temporal parietal and the main bones, external jugal bridge.	Secondary affection as a result of infection extension from the subtemporal caveola of parotid- masticatory region. Infectious – inflammatory skin affection, the contamination of the temporal region wound.	During the superficial localization of the processes is observed pronounced swelling of the tissues of the temporal region. In the case of is deep process localization it is observed moderate swelling.	Hyperemia of the skin of the temporal region. Hyperemia is not typically.	Mod erate Severe	Difficulty of mastication because of pain. Hard limiting of mouth opening.	From the side of skin teguments the section is made parallel to upper edge of jugal bridge and radial sections parallel to fibres way of temporal muscle. From the side of skin teguments the section is made along the frontal edge of temporal muscle, arcual section along the line of muscle fixture to the calvarial bones.
Parotid-masticatory	Upper – lower edge of zygomatic bone and		During the superficial	Hyperemia of the skin	Mod	Difficulty during the	From the side of sin teguments the

region.	jugal bridge, lower - lower body edge of lower maxilla, frontal – frontal edge of masticatory muscle, dorsal – dorsal edge mandibular branch.	8 8 teeth. Secondary affection as a result of infection extension from the buccal region, subtemporal caveola, parotid saliva gland and also by the lymphogenic way.	localization of the processes it is observed pronounced tissue swelling of the parotid-masticatory region. During the deep processes localization it is observed moderate swelling.	of the parotid-masticatory region. Hyperemia is not typically.	erate Severe	mastication (because of the pain and moderate contraction of masticatory muscle). Hard limiting of mouth opening.	section is made parallel to way of the facial nerve and parallel to lower edge of mandibular. From the side of skin teguments the section is bordering the angle of mandibular with the ablation of masticatory muscle from the lower maxilla.
Retromandibular region	Upper – lower side of external auditory meatus, lower-lower pole of parotid gland, frontal – dorsal edge of the branch of the mandibular and medial aliform muscle, dorsal – mastoid bone of temporal bone and breast-clavicular mastoid muscle, inner – belemnold of temporal bone with attached to it muscles, external – parotid-masticatory fascia.	Secondary affection as a result of infection extension from the parotid-masticatory and submandibular regions of aliform-maxilla space.	Pronounced tissues swelling of dorsal region.	Moderate skin hyperemia of retromandibular region.	Severe	Mastication and ingestion are difficult because of pain.	From the side of skin teguments the section is parallel to dorsal edge of mandibular branch.
Pterygo-maxillary space	External –inner surface of the mandibular branch, inner, dorsal and lower external surface medial aliform muscle, upper-lateral aliform muscle and interaliform fascia, frontal buccal - pharyngeal suture.	8 8 teeth. Infection durin the anesthesia.	Bulge in the region aliform-maxillary fold.	Hyperemia mucosa membrane in the region of aliform-maxillary fold.	Severe.	Hard limiting of mouth opening, violation of deglutition because of the pain.	From the side of mouth cavity the section of mucosa membrane is made along the aliform-maxillary fold. From the side of skin teguments the section is made edging the angle of lower maxilla with the ablation of medial aliform muscle from the lower maxilla.
		Any tooth of the mandibular, more often, 87654 45678 Inflammatory- infectious affection and wounds of the mouth cavity mucosa.					
Sublingual space	Upper – mucosa membrane of mouth floor, lower – maxillary-sublingual muscle, external – the surface of the mandibular body, inner – sublingual-lingual and submental-lingual muscle.	Any tooth of the mandibular, more often, 87654 45678 Inflammatory- infectious affection and wounds of the mouth cavity mucosa.	Pronounced bulge of the sublingual region tissues.	Hyperenia of the mouth floor mucosa.	Moderate	Violation of mastication, deglutition and speech because of pain.	From the side of the mouth cavity the section of the mouth floor mucosa is made parallel to inner surface of mandibular. From the side of skin teguments the section is made in

							submandibular region with the interception of the parts of maxillary-sublingual muscle fibres.
Alveololingual groove	Upper-mouth floor mucosa, lower – maxillary-sublingual muscle (its dorsal part), external – inner surface of the mandibular body (on molar level), inner – lateral surface of the tongue root, dorsal – floor of frontal faucial pillars.	8 7 6 6 7 8 teeth. Infectious – inflammatory affections and injuries of mouth cavity mucosa.	Pronounced bulge of the tissues of posterolateral section of the mouth cavity floor.	Mucosa membrane hyperemia of the of the posterolateral section of the mouth cavity floor.	Moderate	Hard violation of deglutition and moderate jaw's contraction because of pain.	From the side of mouth cavity the section of the mouth cavity floor mucosa is made parallel to inner surface of mandibular on the level of big root teeth.
Peripharyngeal space	External – medial aliform muscle, parotid salivary gland, inner – lateral side of the throat and muscle which elevate and strain soft palate, frontal – aliform – maxillary suture, dorsal – lateral fascial spurs passing to pre-spinal fascia to throat side, sublingual muscle, upper – skull base.	Palatine tonsils extension. Injury of throat wall. Secondary affection as a result of infection extension from aliform-maxillary space, submandibular and parotid-mastication regions.	Tissue bulge in the region of lateral throat wall.	Hyperemia of the mucosa of the lateral throat wall.	Moderate	Hard violation of deglutition because of severe pains, moderate jaws contracture, rough breathing.	From the side of skin teguments the section is edging the angle of mandibular.
Submental region	Anterosuperior – lower edge of mandibular mental region, dorsal – maxillary-lingual muscle, external – frontal venters of right and left twoventors muscles, lower – sublingual bone.	3 2 1 1 2 3 teeth. Secondary affection as a result of infection extension from the submaxillary and sublingual regions.	Pronounced tissue bulge submental region.	Hyperemia of the skin of the submental region	Moderate	Mastication and deglutition are difficult because of pain.	From the side of skin teguments the section in the mental region is made parallel the edge of mandibular or along the middle line.
Submandibular region	Upper-inner – maxillary sublingual muscle, external – inner body surface of the mandibular, frontal – lower – the frontal belly of the twoventors muscle, dorsal-lower – dorsal belly of twoventors muscle.	8 7 6 5 4 4 5 6 7 8 teeth. Secondary affection as a result of infection extension from the sublingual and submental regions, aliform-maxillary space and also by lymphogenic way.	Pronounced tissue bulge of submental region.	Pronounced hyperemia under the external localization of the processes (above the cutaneous muscle)	Moderate	Mastication, deglutition and speech are difficult because of pain.	From the side of skin teguments the section in the submental region is along the edge of mandibular, drawing off at 1.5-2 cm to down to it.
Mouth cavity floor	I. Top floor: upper – mucosa membrane of the mouth cavity floor, lower – maxillary-sublingual	Nidus of infections in region of	Hard edema of the sublingual region	Hyperemia of the mucosa membrane of the	Moderate	Hard violation mastication, deglutition, speech	From the side of skin teguments the section in the submandibular region is made

	<p>muscle, anteroexternal – inner surface of mandibular, dorsal – base of tongue.</p> <p>II. Ground floor: upper – maxillary-sublingual muscle, anteroexternal – inner surface of mandibular, dorsal – muscles, which attach to the styloid processes and dorsal ventor of the two ventors muscle, lower – skin of the right and left submandibular and mental regions.</p>	<p>5 4 3 2 1 1 2 3 4 5</p> <p>teeth and inflammatory affections of the mucosa membrane of the mouth cavity floor. Secondary affection as a result of the inflammation extension from the surrounding spaces extension and by the lymphogenic way.</p> <p>Nidus of infection in the region</p>	tissues.	mouth cavity floor.		<p>because of severe pains. It is possible breathing problems up to asphixia.</p> <p>Hard violation mastication, deglutition, speech because of severe pains. It is possible breathing problems.</p>	<p>right and left or collar-like section with the particular intersection of maxillary-sublingual fibres.</p> <p>From the side of skin teguments the section in the submandibular region is made right and left or cross section parallel to upper cervical ruga.</p>
		<p>8 7 6 6 7 8</p> <p>teeth. Secondary affection as a result of the inflammation extension from the surrounding spaces extension and by the lymphogenic way.</p>	Pronounced tissue bulge of submental and submandibular regions from both parts.	Hyperemia of skin of submental and submandibular regions.	Moderate		
Base of tongue root.	Upper – own tongue muscles, lower – maxillary-sublingual muscle, external – mental-lingual muscle of right and left side.	Infection is performed during tongue injury. Secondary affection as a result infection extension from the lingual tonsil, from the sublingual, submental, submandibular regions.	Pronounced tissue swelling of sublingual region, tongue expansion.	Hyperemia of the mouth cavity mucosa.	Severe	Hard violation of mastication, deglutition because of the pain, it is possible breathing problems.	From the side of skin teguments the section in the submental region is made along the middle line or along cross line.

Localization of purulent processes of the face and neck and their ways of spread.



Topic № 6

Complications of the inflammatory face and neck diseases

Mediastinitis

More often purulent mediastinitis appears in the result of mechanic and chemical damage of esophagus with the following mediastinal cellulose infection. However infection can penetrate into the mediastinal through interfascial neck cellulose during deep phlegmons of odontogenic origin. Odontogenic mediastinitis develops in the result of spread of purulent exudates through mediastinal during the phlegmon of peripharyngeal space and mouth cavity floor, through the way of neck neurovascular fascicle, and also through periesophageal and pretracheal cellulose.

Odontogenic mediastinitis could appear at lightning speed, proceed simultaneously with phlegmons of mouth cavity floor and neck, therefore its diagnostics is not easy.

Clinical picture. The general state of a patient bitter aggravates. Body temperature rise till 39-40°C, oftentimes is observed rigor. Pulse riches 110-140 bits in the minute, becomes arrhythmic, of poor volume and tension. Appears bitter dyspnea, shallow breast, the number of respiratory movement reaches 45-50 in a minute. One of the main symptoms during acute purulent mediastinitis is the pain behind the breast bone or in the depth of breast. During the tilt of a head the pain increase (Gerke symptom). Also pains can increase during the deep breath, the attempt to ingest food. The characteristic is permanent hacking deteriorates patient state. During the entrance is observed tissue retraction in the region of sterna notch (Ravich-Sherbo symptom). The position of the patient during the mediastinitis is enforced. One is with the inverted head or is lying sidelong with arcuated to the alvus foot; the chin is pressed to the breast. In the blood is observed expressed leucocytosis, a bitter shift of leucogram in left, a significant increase of ESR.

Important diagnostic method during the mediastinitis is X-ray examination. X-ray examination is made in three projections (frontal, lateral and slanting) usually. In the X-ray picture are taking into consideration mediastinum shadow, transudes in the pleural space.

Surgical intervention during the mediastinitis should be made immediately after diagnosis establishment and correct preparation.

Access to the mediastinum is made through the discission along the frontal edge of sternocleidomastoid muscle, beginning from the level of upper edge of thyroid cartilage and on 2-3 cm lower sternoclavicular abarticulation. After transection of the middle neck fascia, omo-sublingual muscle, to subject cellulose separate the dorsal edge of lateral part of thyroid gland. Neurovascular fascicle is lead to the outside. Along the lateral and frontal surface of weasand penetrate into the cellulose of frontal mediastinum, but along the weasand walls - in the cellulose of a dorsal mediastinum. Mediastinum is drained with the help of polyethylen or polyvinylchloride pipes, through which is bathes the purulent cavity by antibiotics and antiseptics solutions.

In the case of «contact», prognosis of an odontogenic mediastinitis always is very complicated, the disease is highly dangerous. It proceeds especial hard and unfavorable in the prognostic concern putrefactive-necrotic and putrefactive mediastinitis. During the post mortem examination at majority of died were detected purulent pleurisy, purulent pericarditis, deep dystrophic changes in liver and nephros.

Thrombophlebitis of facial veins. Thrombosis of cavernous sinus.

More often thrombosis of face and cavernous sinus are complications of the face furuncles and carbuncles, acute sinusit, phlegmons of temporal and pterygoid-palatal fossa.

In the thrombophlebitis pathogenesis of facial veins and cavernous sinus is important the presence of a dense net of lymph and venous facial vessels with the multiple anastomosis, connection of facial veins, nose cavity and pterygoid-palatal fossa with the eyesocket veins of cavernous sinus, lowering of the organism reactivity after catarrhal and virus diseases, microbial allergy and autoallergy during the inflammatory processes in maxilla-facial region, mechanic damage of skin pustule.

The main anastomosis, connecting the deep facial veins, pterygoid plexus with eyesocket veins, veins of hard brain-tunic, with the cavernous sinus, is lower orbital vein. In the anastomosis of facial veins with the cavities of hard brain-tunic seals are absent almost. The direction of veins blood flow could change during the inflammatory processes. In usual conditions a part of blood leaded from eyesocket along the angle vein into facial. During the inflammatory process, in the region of upper lip, blood through angle vein refluxes in eyesocket veins.

It is known that thrombophlebitis of facial veins appears after stripping of skin pustules by patients or accidental pustule traumatizing more often. Mechanic trauma in the pustule region is accompanied by the damage of endothelium of small blood vessels, among them veins, which promote the thrombophlebitis development.

Thrombophlebitis of facial veins.

The disease is characterized by the appearance along the angle and facial vein of painful “bands” of infiltrate tissue, skin hyperemia with the cyanotic color, and development of edema long after infiltrate. Subdermal veins are enlarged, radiate. It is obvious expressed intoxication, high body temperature, rigor, general sickness, leucocytosis with left formula shift, high ESR. Take place significant hemostasis change: shortens the time of dark blood clotting, rise the content of blood fibrinogen, appears fibrinogen B fraction, rise the factor XIII activity, inhibits fibrinolysis.

During the abscess of thrombosed veins and infiltrates it is performed a surgical treatment with the active pustules drain. For prevention of thrombosis of cavernous sinus, S.D. Sidorov (1979) recommends, to ligate angle or facial vein depending on process localization.

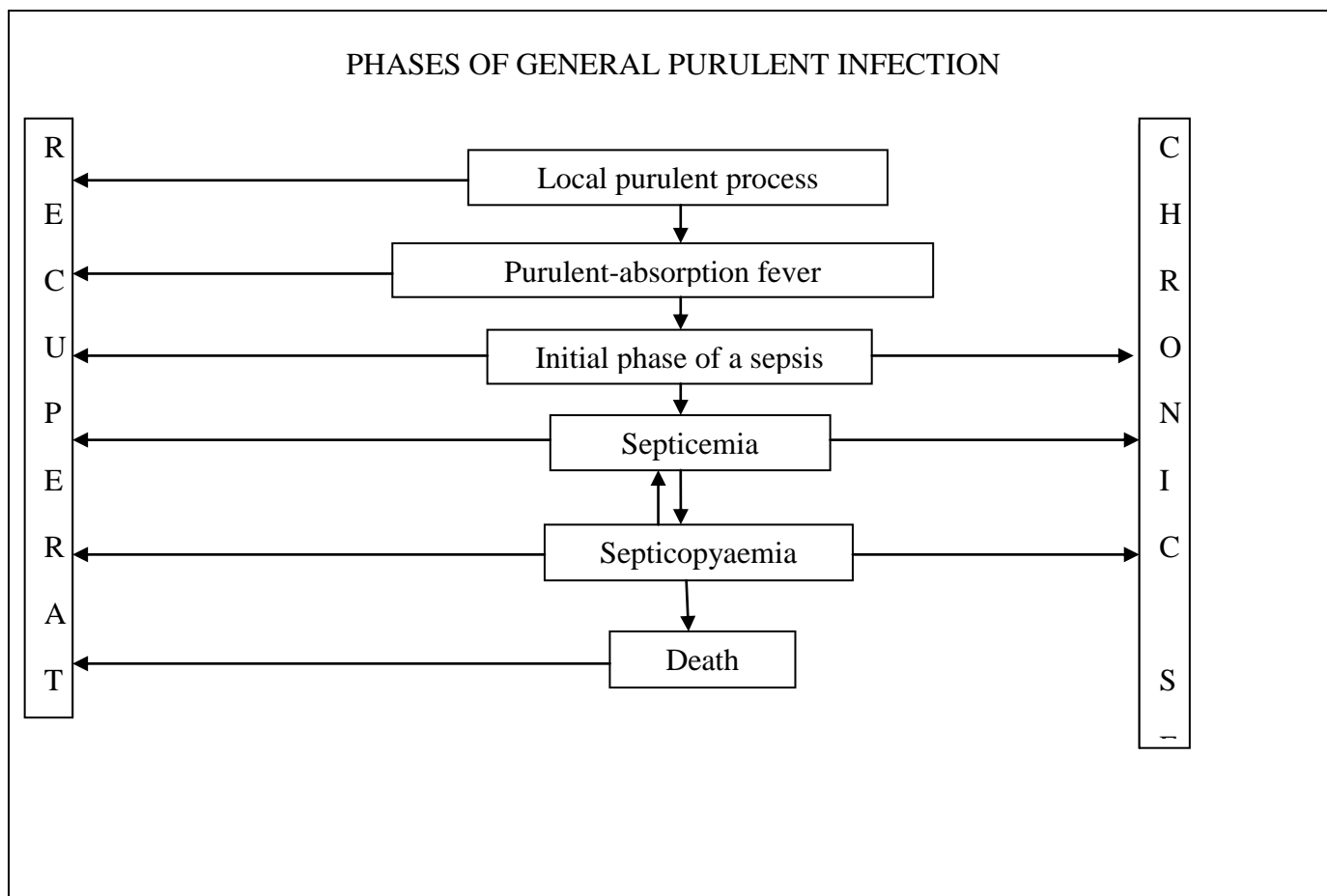
More difficult face thrombophlebitis complication is **cavernous sinus thrombosis**, which is concerned to intracranial complications.

Clinical picture. Appears a strong headache, sharply painfulness in eyes region, general weakness, and rigor. Body temperature achieves 38-40° C. To the local manifestation belong edema and hyperemia of eyelid and front skin, infiltration of arcula soft tissue, exophtalm chemosis conjunctivitis, phthalmoplegia, mydriasis, and hyperemia of eye floor. In a circumferential blood the number of leucocytes achieves 15-20·10⁹/l, ESR increase till 40-60 mm/h.

In the early stage of cavernous sinus thrombosis, M.P.Oskolkova and T.K. Supiev (1974) supervised expressed cyanosis of lip mucosa membrane, front skin, nose, ears.

Sepsis.

In most cases sepsis complicates acute inflammatory diseases of maxilla-facial region, to which is characteristic a range of general symptoms (high body temperature, as often as not rigor, shift of the leukocyte formula in left), appeared during the sepsis. Diagnostic of this dangerous complication is difficult, much less, that sepsis does not have specific features.



Purulent-absorption fever – a general syndrome, bound up with local suppurate process.

In the case when after elimination of purulent focus (phlegmon opening and available leaks, assurance of a good drainage), leading of etiotropic and patogenetic medicated therapy the occurrence of purulent-absorption fever do not disappear, and from the blood sift out pathogenic microbial flora, it is diagnosed the *initial phase of the sepsis*.

If the general state of a patient remains grave (body temperature is higher than 38°C, convulse rigor, insomnia), pyaemic focuses are not determined, and from the blood is sift out a pathogenic flora, is diagnosed *septicemia* (toxic phase).

Septicopyaemia-is characteristic to fever, organism`s intoxication, but in different organs and tissues appear metastatic pustules and abscesses.

Intracranial complications.

Edema of the brain-tunic, with the occurrence of liquor hypertension, appears under the influence of inflammatory process in the maxillary antrum. The main features of disease are decrement in visual acuity, affection of abduct, eye motive and trifacial nerve.

Purulent meningitis develops during the purulent fusion of cavernous sinus walls usually.

For purulent meningitis is characteristic acute beginning of disease, body temperature rising till 39-40° C, strong headache, nausea and vomit. The consciousness is dejected, it is observed soporose state.

II Chapter

Topic № 1

Lymphadenitis and phlegmonous adenitis

Lymphadenitis – is an inflammatory process in the lymph gland, phlegmonous adenitis - is a purulent inflammation of the lymph gland, reason of which is lymphadenitis.

Anatomical data.

Note that the tonsillar, submaxillary, and submental nodes drain portions of the mouth and throat as well as the more superficial tissues of the face.

Knowledge of the lymphatic system is important to a sound clinical habit: whenever a malignant or inflammatory lesion is observed, look for involvement of the regional lymph nodes that drain it; whenever a node is enlarged or tender, look for a source in the area that it drains.

THE NECK

Survey. Inspect the neck, noting its symmetry and any masses or scars. Look for enlargement of the parotid or submaxillary glands, and note any visible nodes.

Lymph nodes. Palpate the lymph nodes. Using the pads of your index and middle fingers, move the skin over the underlying tissues in each area rather than moving your fingers over the skin. The patient should be relaxed, with neck flexed slightly forward and, if needed, slightly toward the side of the examination. You can usually examine both sides at once. For the submental node, however, it is helpful to feel with one hand while bracing the top of the head with the other.

Note their size, shape, delimitation (discrete or matted together), mobility, consistency, and tenderness. Small, mobile, discrete, non-tender nodes are frequently found in normal person.

Enlarged or tender nodes, if unexplained, call for reexamination of the regions they drain, and careful assessment of lymph nodes elsewhere so that you can distinguish between regional and generalized lymphadenopathy.

Lymph glands in the maxilla-facial region of the frontal and lateral neck part are divided into several groups, depending on their position.

Lymph glands of the mental region are localized in the space between the frontalis muscle and sublingual bone. Their number usually fluctuates from 1 to 4. Frontal mental gland is situated near the apex of mental region. Dorsal are situated a little anterior from the sublingual bone.

Submental lymph glands receive the lymph from tissues, which surrounding frontal lower teeth, from the frontal part of the alveolar bone and mandibular body, tongue root and frontal part of sublingual region, lower lip and adjacent cheek parts.

Submandibular lymph glands are situated in the submandibular triangle and lie outside salivary gland capsule in the form of chain along the mandibular edge.

In submandibular glands fall tissue lymph vessels, surrounding set of mandibular teeth – from the canine level to third molar, from the corresponding to these teeth parts of alveolar bone and mandibular body.

Sublingual lymph glands are in the tight connection with tongue lymph vessels.

Facial or buccal lymph glands - are not big, not always detected formations, situated along the facial vein.

Parotid lymph glands with dimensions till 0.5 cm, usually by the number 3-5, are situated as superficially, under parotid mastication fascia, as deeply in the substance itself of salivary gland. To these glands matches lymph vessels from the lateral face`s parts, from the buccal mucosa membrane, nose, eyelids, the brow skin, earflap, parotid gland.

Ethiology and pathogenesis.

The reason of maxilla-facial region lymphadenitis can be an odontogenic infection: during acute periodontitis and aggravation of this chronic process, suppuration of the root cyst, odontogenic process in periostenum, maxilla bone, perignathic soft tissues. All lymphadenitis appeared in the result of an odontogenic infection, are named odontogenic. Maxilla-facial region lymphadenitis can develop as a result of infection expansion during infectious diseases and damage of mouth mucosa membrane (stomatogenic), from tonsils (tonsillogenic), from the tissue of external, drum and inner ear (otogenic).

More rarely the damage of lymph glands of the maxilla-facial region can be connected with the diseases and damage of skin integuments of the face and head.

The reason of the lymphadenitis is pyogenic non specific infection, specifically coccal flora, where the mainstream significance has staphylococcus. All lymphadenitis caused by this infection, are defined as non specific process. In other cases, rarely, lymphadenitis can be caused by the lambent myco , tuberculosis mycobacteria (specific lymphadenitis), or by virus (infectious mononucleosis).

Classification of the lymphangitis

- According to etiology: odontogenic and nonodontogenic, rinogenic, tonsillogenic, infectious and traumatic, dermatogenic, tumoral, stomatogenic, specific and nonspecific.
- According to pathogenesis: lymphangitis, lymphadenitis, adeno-abscesses, adeno-flegmons.
- According to the clinical picture: acute (serosal, purulent) and chronic (purulent, proliferate)
- Deep and superficial.

Lymphangitis – inflammation of the lymph vessels.

- Lymphangitis develops in the result of penetration of microflora, toxins in lymph tracts. Peculiarities of the lymph vessels – length and caliber of the lymph vessels are more reduced in comparison with other area of the body that explains the absence of the clinical features of lymphangitis in inflammation processes of the lymph system from this level.

Clinical picture.

Maxilla-facial lymphadenitis develops as reaction of lymph nodes on any inflammatory process. Lymphadenitis can be the one of the disease symptom (acute or aggravation of the

chronic periodontitis, periostitis or osteomyelitis of the maxilla and other), and also as independent disease.

Distinguish two types of lymphadenitis: acute and chronic lymphadenitis. Acute lymphadenitis can proceed in serosal and purulent forms. Chronic lymphadenitis proceeds in the hyperplastic form, it can transfer in the purulent form.

Acute serosal lymphadenitis.

Acute serosal lymphadenitis of the maxilla-facial and neck region (lymphadenitis acuta) – is the primary stage of the inflammatory changes of lymph nodes. Appear the morbidity of one or several lymph nodes. The distinct can be palpated in the nature of roundish or oval form, which have soft – elastic consistence. General state during the acute serosal lymphadenitis suffers insufficiently, but at the individual patients are observed temperature reaction, not exceeding 38 C, deterioration of general state.

Acute serosal lymphadenitis can melt in purulent lymphadenitis. Appear considerate increase and indurations of one or some lymph nodes. They are painless. Appearance of the morbidity shows the aggravation of inflammation in the main focus. During the chronic non specific lymphadenitis lymph nodes, as a rule, remains flexible do not solder with surrounding tissues and skin.

Acute purulent lymphadenitis (lymphadenitis acuta purulenta).

Acute purulent inflammation of lymph nodes of the facial and neck region appears rarer than chronic (blood changes, leucocytosis, ESR increase).

Purulent fusion of the lymph nodes allows to establish future growthing of morbidity, increasing of swell in their circle, in mainly cases slowly - in some days, and sometimes 1-2 weeks. Consequently can be observed progressive infiltration of the adjacent to lymph node tissues, causing its movement limitation, sealing of some teeth in one packet, and then mollities and fluctuation.

During the acute lymphadenitis general incompatibles - is indisposition, brokenness – can be absent or can be expressed insignificant. Body temperature is 37.6-37.8 C. in the blood is observed similar changes, as during the odontogenic abscesses of maxilla-facial region.

Phlegmonous adenitis (adenophlegmone).

Sometimes occurs the capsule fusion of the lymph node and the purulence penetrate in the surrounding it cellulose. In the result develops phlegmonous inflammation - phlegmonous adenitis, accompanied by the surrounding tissues edema.

Phlegmonous adenitis in the result of odontogenic infection is localized predominantly in the submandibular and submental region, rarer - on the lateral part of the neck.

At majority patients the body temperature does not exceed 38-38.5 C, during the phlegmonous adenitis. Happens rigor. In the blood moderate leucocytosis and neutrophilia, ESR is increased.

Chronic lymphadenitis. (Lymphadenitis chronica).

Chronic lymphadenitis is the outcome of the acute process in lymph node. In some cases there is chronic lymphadenitis with the unexpressed acute stage.

Clinic of the chronic lymphadenitis is characterized by node increasing, by roundish or oval form, precise and even contours, nonsolder with surrounding tissues. During the palpation, the lymph node has solid elastic consistence, painless. The general state of the patients do not suffer, the body temperature is in norm.

Such picture of the chronic inflammatory process is accepted to designate as chronic hyperplastic lymphadenitis.

In separate cases, in the result of lymph node chronic inflammation takes place the moderate granulation tissue enlargement, which replace by itself lymphoid tissue, spreads outward the node and grows to skin thereby make it thinner. During the eruption of the thinned region appears the fistulous tract. In the region of such process can develops the suppuration, but proliferative reaction is more expressed. About the purulent nature shows only discharge of purulent from the fistulous tract, often poor, or its accumulation under the scab on the skin surface.

The chronic hyperplastic lymphadenitis should be differentiated with odontogenic subdermal and dermal granuloma, subdermal form of the actinomycosis, scrofuloderma and main with the tumor metastasis.

Treatment.

During the acute lymphadenitis first of all it is necessary by the way of corresponding intervention in the region of primary inflammation focus (teeth extraction during periostitis, manipulation and sanitation of the extracted teeth cavity during the alveolitis and other), prevent the future entry of microorganisms in the lymph nodes.

During the serosal lymphadenitis the treatment could be conservative. Physiotherapeutic procedures are justified, Bandage with Dimecsid solution (in dilution with sol.Furacilini 1/3), vitamins, analgesics and antisensitizer are put on.

In the case of ineffectiveness of the leaded therapy the stimulating arrangements are justified (autohemotherapy, vitaminotherapy). It is necessary to scoop from the purulent focus remains of the halfbroken lymph node. In the first days after opening of the purulent focus it is effectually to introduce in the wound drainage, bandage with the Levomykoli ointment (which promote the supuration outflow).

During the phlegmonous adenitis the purulent cavity is opened according to the general rules of phlegmons opening.

Topic № 2

Furuncles and carbuncles

Furuncle is acute suppurative inflammation of hair follicle and surrounding its tissues. In the following the inflammation goes into oil gland and surrounding connective tissue.

Multiple affections by furuncles are named furunculosis.

Ethiology: It is caused oftener by aureococcus, rarer by white staphylococcus, sometimes by other microorganisms (for example: streptococcus).

Contributory cause to the development is skin diseases and microtraumas. The significant role in the furuncle and furunculosis appearance plays the weakening of organisms phylactic power in consequence of exhaustive chronic diseases: avitaminosis, diabetes, unfavorable meteorological conditions, cooling, hyperthermia, nervous and endocrine system disorder, soiling of the skin by the coarse dust grains (lime, cement, coal), in particular in combination with microtrauma during the shave, stripping acne.

Usually face furuncles are localized in the lip and chin region, rarer on the front and cheeks skin.

Histological changes in the initial stage are characterized by the pustule formation in the follicle entrance. Pustule contains neutrophilous leucocytes, the significant quantity of staphylococcus and fibrin. In the following staphylococcus descending along the hair follicle till the papillary bodies, cause the inflammation that leads to the formation of the inflammatory infiltrate with the following necrosis of the surrounding tissues. Around the necrosis zone connective tissue fuses and pus is formed. Suppurative exudate accumulates under the epidermis, around the follicle entrance, and then together with the necrotic bolt and dead hair comes outside. The defect fills by granulation and cicatrix is formed.

Clinical picture

- I. First stage (infiltrative) appears blurred limited redness and swelling, accompanied by an insignificant sickliness. During 1-2 days in the region of the hair follicle entrance is formed limited nodule, subjected tissues infiltrate, the skin hyperemia in the infiltrate region increase, appears sharp sickliness. Furuncle face character is expressed edema border tissues with the node (it becomes clear by the friability of the hypoderm).
- II. Second stage (abscess) development of the furuncle is characterized by the suppuration and necrosis. Through 3-4 days from the disease beginning occurs purulent tissue fusion, which clinical is apparent by the fluctuation. After spontaneous or artificial opening of the furuncle exudates small quantity of pus with the blood admixture. The epidermis defect place is seen necrotic furuncle bolt, which together with the pus spontaneously slough or is moved away by the pincers. After this on the furuncle place appears small bleed ulcer, which is filled by granulations. Infiltration and edema of the furuncle border tissues decrease. During the timely and intensive therapy the tissue infiltration gradually decrease, ensue the abortive process flow without abscess.
- III. The third stage is characterized by the wound healing with the formation of small gently indrawn cicatrix. In the case of an extensive accumulation of necrotic bolt appears so cold abscess furuncle.

Face furuncles often are accompanied by the regional lymphadenitis, but suppuration of the lymph nodes appears seldom.

Such patients have headache, chilling, general indisposition, which serve as manifestation of the organism's intoxication.

Especially difficult clinical course is characteristic for upper lip furuncles, in the nasolabial triangle. Because of relatively not frequent appearance of threatening aggravations (thrombophlebitis of facial nerves and cavernous sinus, senses, meningoencephalitis) such furuncles are named "malignant". The malignant flow of the furuncle usually ensues after the attempt to its stripping in the initial stage of disease.

Furuncle can be single, or sometimes simultaneously or consecutive one after another appears a lot of inflammation focuses on the different skin region - so cold furunculosis. The appearance of many furuncles on the restricted body region is called local furunculosis.

The prognosis during the uncomplicated furuncle is favorable. Furuncles and furunculosis should be differentiated with anthrax and some infectious granuloma (tuberculosis, actinomycosis, syphilis).

Face furuncle can be aggravated by the carbuncle formation during what at the same time is affected several hair follicles.

Carbuncle is named acute purulent-necrotic inflammation of several hair pouches and oil glands with the formation of the general infiltrate and vast skin necrosis and subdermal cellulose.

Etiology and pathogenesis of the carbuncle is the same as at the furuncle. Carbuncle oftener appears at the elderly, weak patients.

Carbuncle development, as furuncle, begins from the pustule formation. However, during the carbuncle quickly appears dense sharply painful infiltrate, spread not only all over skin layers but also over the subdermal fatty cellulose. The skin above the infiltrate has blue-purple coloration.

For carbuncle is characteristic appearance of several purulent-necrotic "heads". Afterwards, in the central part of the carbuncle happen softening and rejection of necrotizing tissue.

Face carbuncle, often than furuncle, are aggravated by thrombophlebitis of facial veins, cavernous sinus and septicopyemia.

Facial vein and cavernous sinus thrombosis more often are complications of face furuncles and carbuncles. In the facial veins and cavernous sinus thrombophlebitis pathogenesis the significance have the presence of dense net of lymph and venous face vessels with multiple anastomosis, facial vein connection, nose cavity and pterigoid-palatal fossa with eyesocket veins, cavernous sinus, lowering of the body reactivity after catarrhal and viral disease, microbial allergy and autoallergy during the inflammatory processes of the maxilla-facial region, the mechanical damage of skin pustules. The main anastomosis connecting deep face veins, pterigoid plexus with eyesocket veins, veins of hard brain tunic, with cavernous sinus, is lower orbital vein. In the anastomosis of face vein with the cavities of hard brain tunic the seal almost is absent. The blood flow direction in veins can change during the inflammatory processes. In common conditions a part of blood is turned off eyesocket across the angle vein in the facial. During the inflammatory process in the upper lip region blood across the angle vein reflows in the eyesocket veins.

Complications of furuncles and carbuncles

Thrombophlebitis of facial nerves is characterized by appearance on the course of angle or facial vein of painful "bands" of infiltrative tissue. During the disease occur significant changes of hemostasis: reduces the time of venous blood clotting, increase the content of blood

fibrinogen, appears the fraction of B fibrinogen, increase the activity of XIII factor, inhibits fibrinolysis.

More difficult complication of face thrombophlebitis is **cavernous sinus thrombosis**, which belongs to intracranial complications. Such patients usually appear intense headache, sharp sickliness in the eye region, general asthenia, rigor. Body temperature acquires 38-40° C. To local appearance belong edema and skin eyelid and front hyperemia, infiltration of orbit soft tissue, exophthalm, mydriasis and eye floor hyperemia. Mentioned symptoms can progress and on the part opposite to inflammatory focus. In the circumferential blood the quantity of leucocytes achieve $15-20 \cdot 10^9/l$, erythrocyte Sedimentation rate (ESR) increases till 40-60 mm/h.

Treatment:

Uncomplicated furuncles in the first stage are treated ambulatory as a rule. First of all should be eliminate any external irritator – shaving, a small tissue trauma in the furuncle region, overlapping on the focus and surrounding tissues of the semi-spirituos bandage at one hour (to do not cause skin irritation) during 3-5 days.

Furuncles of the II stage and carbuncles are treated only in the day and night clinic.

General therapy provides the fight with the infection (suppression of vital activator activity) and intoxication.

Local treatment in the disease acute phase is performed for creation of pus outflow and exudate from the suppurative focus

Antibacterial therapy:

Pluripotential antibiotics:- *Ceftriaxone* 1 gr - 2 times per 24-hour

Antibiotics of tight activation spectrum:

- Erythromycin 0,5-4 times per day.

Disintoxication therapy – the liquid is introduced intravenously by dropping way 30-60 ml on 1 kg:

- *Ringer solution*, - *Refortan solution*, - *physical solution*.

Densensitise means: Analgyn, dymedrol, suprastin.

With the prophylaxis aim of cavernous sinus thrombosis is indicated anticoagulants of the direct action:

- *Heparin* 2500-5000 UN i/v by dropping with interval 4-6 hours, then i/m.

With the aim to reduce factor XIII activity and increase of fibrinolysis the nicotinuric acid is indicated 0,05 gr 1-2 times per 24-hours.

Surgical treatment during the face furuncles usually is performed in the case of abscess, after discession the cavity is drained, the bandage with Levomicoli ointment is overlapped.

During the carbuncles is made the wide opening of the inflammatory infiltrate.

To the means of nonspecific stimulating therapy are related hemotransfusions, *Pyrogenal*, *Imunnofan* 0, 005%-1ml i/v.

Active specific immunotherapy is made by the staphylococcic antifungin, bacteriophage, staphylococcic anatoxin. Passive specific immunotherapy is performed hyperimmune antistaphylococcic plasma and gammaglobulin, which render specific antistahylococcic activity.

Physiotherapy

- Ultrasound therapy trough 1-2 days after erythema remitting.

- For resolution of rudimentary infiltrates is indicated paraffinotherapy, electrophoresis with Sol.Lidazi.

Topic № 3

Specific inflammation diseases in the maxilla-facial region

In the maxilla-facial region special group are inflammatory processes, caused by the specific activators, such as: lambent fungus, pallidum treponema, microbacteria of tuberculosis.

Actinomycosis

Actinomycosis, or lambent fungus disease, is chronic infectious disease, appeared in the result of implementation in the organism of actinomycetes (lambent fungus).

In the mouth cavity actinomycetes live permanent. They are in the teeth accretion, carious teeth cavity, pathological subgingival spaces, on tonsils. Actinomycetes are the main dental tartar stroma.

The considerable importance in the actinomycosis development have general reasons, which lower immunity, and also local moments, such as: inflammation, trauma, breach of the normal symbiosis of actinomycetes and other microflora.

Clinical picture.

The clinical picture of the disease depends from the individual peculiarities of the organism.

Actinomycosis of the maxilla-facial region differs by the significant variety. Often the primary period of this disease course is unnoticed, do not accompanied by pains and flows without temperature increase. With reference to this, the patient, in the primary periods of the disease, does not hurry to the doctor, and refers approach to the hospital only when the disease process is aggravated, in other words with the firm inflammatory inflation or during the breaking specific granuloma - abscess formation.

Usually clinical flow of the actinomycosis is chronic, during the breaking of specific granuloma the disease takes the acute course of disease.

On the primary stages of disease is observed inflammation infiltrate without sharp borders and characteristic to the actinomycosis tissue density.

During the localization of the actinomycosis process in the skin and in its neighboring tissues (subdermal cellulose) rather quickly, in the course of 2-3 weeks, happens the limitation of the focus, their mollities and self-existing opening of degrading granuloma.

It should be distinguished following clinical forms of actinomycosis of maxilla-facial and neck region.

1.Dermal; 2.Subdermal; 3.Submucosal, 4.Mucosal, 5.Odontogenic actinomycosis granuloma; 6.Subdermal-muscle (deep); 7.Actinomycosis of maxilla periost; 8.Actinomycosis of lymph nodes; 9.Actinomycosis of maxilla bone; 10.Actinomycosis of mouth cavity organs – tongue, tonsils, salivary glands, supramaxillary cavity.

Dermal form of the actinomycosis occurs relative seldom. Disease appears in the result of odontogenic infection penetration, as a damage of skin integuments. Patients complain on the insignificant pains and induration on the small extends of cheek skin, submandibular region, neck or during the interview point out on the gradual increase and induration of the focus.

Dental actinomycosis courses without temperature increasing. During the examination it is observed inflammatory infiltration of the skin, emerges one or several focuses, which grow outside.

Subdermal form of actinomycosis is characterized by the development of the pathological process in the subdermal cellulose, as a rule, directly nearby odontogenic focus, which is the portal of entry of specific infection. Patients complain on pains and inflation in the buccal or other regions; submandibular, parotid-masticatory, retromandibular regions, neck.

During the examination the inflation is diffuse, during the palpation in the subdermal cellulose is defined roundish infiltrate, in the beginning solid and painless, but forward, in proportion to fusion granulomas are in the center, soft and painless. In the period of decay of specific granuloma skin solders together with subject tissues, becomes hotpink to red.

Submucosal form of the actinomycosis occur relative seldom. The submucosal form of the actinomycosis courses without temperature rise or its insignificant rise until the subfebrile numbers, with moderate painful sensation in the damage focus centers. Pains increase during the movement – mouth opening, deglutition, speech. In the process dynamics increase the sensation of special discomfort “debride”.

During the palpation it is observed solid infiltrate of roundish form. During the process infiltrate is limited, becomes more solid, the mucosa membrane under it solder, gets cloudy, often acquire pale color. Submucosal actinomycosis focuses in the region of aliform-maxillary fold, peritonsillar region is characterized by significant solid tissues, which together with hypertrophic amygdale remind the picture of malignant tumor.

Focus opening allows mentioning the presence of clearly restricted region, filled by saniopurulent exudate and granulations.

Actinomycosis of the mouth cavity mucosa membrane occurs seldom. During the affection of the mucosa membrane of the mouth cavity lambent fungus penetrate through damaged and inflamed mouth mucosa membrane.

Traumatic factors can be debride, such as: herb`s awns, grasses, fish bones. Favorite place of affection is mucosa membrane of the lower lip and cheek, sublingual region, lower and lateral side of tongue. The mucosa membrane in the place of affection has red, sometimes cyanotic color.

Odontogenic actinomycetic granuloma. The formation in the tissues of the periodont of the primary actinomycetic granuloma happens more often as not. During the localization of the actinomycetic odontogenic granuloma in the derma and in the subdermal cellulose is observed a band, along the transitory fold, coming from tooth to the focus in soft tissues.

Subdermal – muscle (deep) form of the actinomycosis develops in the subdermal, intermuscular, interfascial cellulose.

It is localized in the submandibular, buccal or parotid-masticatory regions, and also affects tissues of temporal, suborbital, zygomatic, subtemporal fossa, aliform-maxillary space and lateral part of the neck.

During the acute phase it is observed cyanosis of the skin integuments over the infiltrate; appeared in separate infiltrate parts focuses of mollities remind not big forming abscesses.

Actinomycosis of lymph nodes. At all times between doctors indurates the opinion that actinomycosis do not affect lymph nodes.

Actinomycosis of lymph nodes appears as a result of odontogenic penetration of infection. During the actinomycosis of lymph nodes the process is localized in buccal and supramaxillary lymph nodes.

Clinical picture is various. Patients complain on the limited, slightly sickly, solid inflation corresponding to one of the lymph nodes group. From the anamnesis is apparent that affection of the lymph nodes develops slowly and droopingly, appeared less sickly lymph node increase slowly and around it grows tissues infiltration.

Actinomycosis of maxilla periost. It is affected predominantly the periostenum from the vestibular part of the mandibular, more often it is marginal process on the level of first lower molar. Clinical it is observed solid infiltrate, more often along the transitory fold, its flatness, mucosa membrane over it is red, sometimes bluish color.

Actinomycosis of the maxilla bone occurs seldom. In the early periods patients complain on the insignificant pains in the region of the affected bone part. During the immediate neighboring intraosteal focus with the mandibular canal is damaged sensibility in the region of facial nerve arborization. Hereafter pains become more intensive, can accept the character of neurologic pains, often it is observed the edema and infiltration of soft tissues or periosteal thickness of the bone, develops the inflammatory contracture of the mastication muscles.

According to X-ray picture, the primary destructive actinomycosis of the maxilla is characterized by the presence of one or several symphysic cavities of roundish form, but always clearly circumscribed.

Actinomycosis of the mouth cavity organs – actinomycosis of the tongue, tonsils, salivary glands, maxillary cavity. – occur comparatively rare and represent significant difficulties for diagnostics.

Actinomycosis of the maxillary cavity occurs rarely. Infection penetrates by rinogenic and rarer by odontogenic way.

Clinical it is observed hinder nasal breathing, sometimes purulent discharge from the nose. The frontal side of the maxilla is thickened; the mucosa membrane of the transitory fold is thickened by the periostenum.

According to X-ray examination actinomycosis of the maxillary cavity is characterized by its homogeneous blackening with the well-marked cavity walls.

Diagnosis. Faded and long course of odontogenic inflammatory processes, unsuccessful leaded anti-inflammatory therapy always affray concerning actinomycosis.

The diagnostic of actinomycosis lighten the appearance focus softening, covered by reddish, with bluish color skin, line of fistula tracts.

Clinical diagnosis of actinomycosis should be supported by the examinations of the separate, diagnostic skin- allergenic reaction with the actinolysate, X-ray and pathomorphological examinations.

Cytological exanimations of the colored smear permits to find out the process character, to establish the presence of mycelium actinomycetis , secondary infection, and also according to cell`s composition judge about the reactive ability of organisms.

Treatment. The treatment of the actinomycosis of the maxilla-facial and neck region should be complex and be composed from immunotherapy (actinolysatotherapy and vaccinotherapy), stimulating and surgical treatment.

Actinolysat - is an medication, which represents the product of spontaneously lyse bouillon aerobic cultures of pathogenic actinomycetis, detached from the pathological material during the peoples actinomycosis.

Actinomycetic polyvalent vaccine (APV) – is a new medication, offered for actinomycosis treatment.

During often aggravation of the process, tendency to the expansion apply massive courses of antibiotics in relation to microflora sensibility: ocsyciline, eritromecine, oleandomecine, fusidine.

The surgical treatment of the actinomycosis consists in:

- 1) Teeth extraction, which were portal of entry of actinomycetic infection;
- 2) Opening of the actinomycetic focuses in the perignathic tissues.

The good effect gives iodine electrophoresis, sol.Lidazi, ultrasound, lasar rays on the cicatrices, and rudimentary infiltrates during the actinomycosis.

Prognosis. Prognosis during the actinomycosis of maxilla-facial region in many cases is productive (advantageous).

Tuberculosis.

It is possible the penetration of the tuberculate infection through damaged mucosa membrane of mouth cavity or during tissues inflammation of mouth cavity and jaws.

Clinical picture.

In the maxilla-facial region it is usual to distinguish primary and secondary tuberculate affection.

Primary tuberculate affection usually is formed in the skin region of the mouth cavity mucosa membrane, lymph nodes of maxilla-facial region.

On skin or on the mouth cavity mucosa membrane a some weeks after penetration of the tuberculate infection forms papulous, bullate or pustulous formation, which is opening out with the formation of painful ulcer with abrupt, mine edges and floor, fulfilled by the grainy granulations yellowish or pinkish color.

Primary affection of lymph nodes of maxilla-facial region appears during the entry of tuberculate infections through teeth, tonsils, mucosa membrane of mouth cavity and nose, face skin during their inflammation or damage.

Secondary skin tuberculosis - scrofuloderma occurs predominantly at children and is localized in the skin of submandibular, submental, cervical, parotid regions. Skin over the focus is sharp drawn and on its surface are seen separate fistulous tracts or ulcers of incorrect form, filled by abundant granulations. On the skin you can be seen the “phenomena of apple jelly” – change of the liupom color during the diascopy. In the mouth cavity, during the gingiva affection, the process can spread on the alveolar bone. The process flow is slow. As bosselations, as ulcers, can be many months and years.

The affection of the mucosa membrane of mouth cavity by miliary-ulcerous tuberculosis happens during the difficult tuberculosis form of lungs and throat. Mucosa is affected in the result of entry in the mouth cavity of the significant number of tuberculous microbacterion with expectoration, during the lowering of organism`s reactivity.

More often the process is localized on the cheek mucosa, especially across the line of teeth articulation on the gingiva and tongue mucosa.

The disease more often proceeds in chronic form and is accompanied by subfebrile temperature, general asthenia, appetite loss.

Maxilla tuberculous appears secondary in the result of spread of tuberculate mycobacteria hematogenic or lymphogenic from to the organs, from breathing organs and alimentary organs.

Diagnosis.

Diagnosis of the tuberculous affection of maxilla – facial region tissues has special difficulties. In the case if the process spreads on the bone, it should be differentiated with a banal inflammation, actinomycosis and syphilis, and also with malignant neoformations.

Maxilla-facial region tuberculous diagnostics consist from range of methods. First of all this is tuberculinodiagnostics, which allows establishing the presence of tuberculate infection in the organism. Tuberculin solutions are used according different methods (Mantoux test, Pirke, Coha).

The treatment of patients consists from the complex apply of streptomycin, ftivazide. Teeth with the affected by the tuberculous periodont can be extracted.

Syphilis.

Syphilis is a chronic infectious venerean disease, caused by treponema pallidum (spirohaete), which can affect all organs and tissues also maxilla-facial region.

This disease has 4 periods: syphilis I, syphilis II, syphilis III and syphilis IV. After implementation of different medications for syphilis treatment (iodine medications, bismuth, arsenic, penicillin) the clinical picture of disease and its separate manifestations significantly changed. The tissue pathology of the maxilla-facial region is seen in particular during first third syphilis stages, and also separate disorder are seen during the inborn syphilis.

Primary syphilis (Syphilis I) is characterized by the appearance on the mucosa and also in mouth cavity of primary syphiloma or hard chancre. The primary syphilitic focus usually appears on the red edge of lips, lips mucosa, cheek, gingiva, tonsils and on the tongue.

The disease begins from the limited space of mucosa inflammation, which in thickened until the cartilaginous consistence. For the primary affection of the mucosa of mouth cavity is characteristic increasing and induration regional lymph nodes, predominantly of submandibular, parotid, submental.

Secondary syphilis (syphilis II) more often affects mucosa of the mouth cavity and has an aspect of pustulous or roseolous elements. Their lovely localization is on tonsils, tongue, neck mucosa and lip mucosa.

The papule formation is characterized by the appearance of the sharp limited space of hyperemia of the mucosa with infiltrate of its foundation.

Tertiary syphilis (syphilis III) is characterized by the formation of such named - gumma. Gumma represents infectious granuloma, which includes lymphocytes, plasmatic and monocytoïd cells with the significant development of blood vessels. The third syphilis should be differentiated as: mucosa syphilis, periostenium syphilis, and maxilla bony tissue syphilis. Between the gummatous affections of mouth cavity mucosa the process localization in the soft palate the has a range of peculiarities. In the bone originates gradual tissue healing with the formation of rough, solid, often astringent cicatrices, after degradation of the gumma. In the bone develops hyperostosis, exostosis, namely along the edges of bony defects.

Diagnosis.

Diagnosis of the syphilitic affection of the mouth cavity, teeth and jaws represents known difficulties, as has a range of features similar with other specific processes – actinomycosis, tuberculous, premalignant affections of mouth cavity, malignant neoplasms.

Treatment.

The treatment of the syphilis of the mouth cavity and jaws is divided into general and local. More effective is therapy by bicillin in combination with antibiotics.

Simultaneously with the general treatment is leaded local treatment. Its consists in ablation of syphilitic elements, exulceration of fistulous tracts by different antiseptic solutions, more often by the 2% of chloramines solution.

Topic № 4

Odontogenic inflammation of the maxillary cavity (Sinusitis)

Pathogenesis

Sinusitis appears during the penetration of the infection in a maxillary cavity. Depending on character, ways of infection penetration, pathogenesis, and sinusitis are distinguished as:

- Odontogenic;
- Rhinogenous;
- Hematogenic;
- Traumatic;
- Allergic.

More often odontogenic sinusitis appears in the result of infections spread from the inflamed periodont (usually upper small and big root teeth, rarer – canine), in other words from the focuses of acute or chronic periodontitis. The possibility of such infection spread is explained by the range of circumstances, on the first place foremost amongst is topography-anatomical adjacency of apices specified teeth to the mucosa membrane of the supramaxillary cavity floor. Launch their development on the 10th week of people live, supramaxillary cavity increase gradually, comes up to 15-40 cm². In the case of its overdeveloping the cavity floor descends in the alveolar crest.

Usually the distance between apices of small and big root teeth and floor of supramaxillary cavity fluctuates from 0 till 10-12 mm, in the result of which at many people osseous septum between the cavity mucosa membrane and teeth apices is insignificant thin or is absent entirely. In such cases the teeth apices are covered only by mucosa membrane directly.

In the case of the osseous septum between the periodont and mucosa membrane of supramaxillary cavity is expressed sufficient, as often as not it is *destroyed in the granulation or granulomatous periodontitis process*.

Clinical picture

Odontogenic sinusitis according to its clinical course could be acute, subacute and chronic, and also represent the aggravation of inflammatory inflammation.

G.N. Marchenko (1966) presupposes the following clinical classification:

1. Closed form: a) sinusitis due to of chronic periodontitis and b) sinusitis due to suppuration of odontogenic cysts which are grown in the supramaxillary cavity.

2. Opened form: a) perforated sinusitis and b) sinusitis, developed as aggravation of the chronic osteomyelitis of the alveolar crest or maxilla body.

By the nature of pathomorphological changes odontogenic sinusitis could be divided into catarrhal, purulent, polypous and purulent-polypous.

Acute odontogenic sinusitis

Clinical picture

Acute odontogenic sinusitis appears as the result of acute purulent or aggravated periodont, acute osteomyelitis and cysts suppuration. Quite often is represents the aggravation of the chronic sinusitis.

During the acute inflammation of the supramaxillary cavity patient complaints are come to the following: pain and heaviness in the corresponding face part; the pain irradiates in frontal, cervical and temporal region and also in the upper teeth, simulating a pulpitis; corresponding nasal parts congestion (blocking) and smell weakness; defluvium of the slim from the naris of the sick part (especially during the forward head bend) and breathing difficulty, general atony, body temperature rising from 37.5 till 39 C°; sleep violation.

Diagnostics

Objectively it is observed, painful with slightly lubricous skin during the palpation; the mucosa membrane of the nose cavity is hyperemic and edematous; under the middle concha are present purulent exudates. Percussion on one-three teeth on the sick part causes the pain (one or several from them are gangrenous and disturbed). Percussion along the malar bone causes the pain also. X-ray picture of the cavity: becomes clear sharp shadowing; on the X-ray picture of the alveolar crest are seen the phenomenon of a chronic periodontitis, cystogranuloma or suppurated cyst; the structure of osseous septum is impaired between the focus of inflammation at the tooth apex and floor of the supramaxillary cavity.

During the centesis of supramaxillary cavity through the lower nasal duct or through the transitory fold of mucosa membrane could be got the purulent exudates, situated in the cavity.

In the blood – leucocytosis, increase of ESR, number of stab leucocytes.

Treatment

The treatment of the acute odontogenic sinusitis is conservative and pursues aims: to create the free outflow of an exudates from the supramaxillary cavity, to eliminate the tooth focus of infection and to trample the infection in the supramaxillary cavity.

During the sinusitis take place the sensitization of the patient's organism, are prescribed desensitizing drugs (intravenously 10% of calcium chloride solution; inside - dimedrol).

Preventive measures

The preventive measures of the sinusitis consist in timely treatment of teeth diseases, periodontium, maxillary cysts and osteomyelitis.

Chronic odontogenic sinusitis

Clinical picture

Chronic odontogenic sinusitis is developed from the chronic periodontitis or is outcome and aggravation of the acute sinusitis. Complains could be absent, but as a rule patient complain on feeling of heaviness in the head, in the front and temple region, nasal stuffiness, smell violation, lowering of the employability; sometimes is observed putrefactive – malodorous smell from the nose, but during the delay of the exudates outflow from a cavity appears symptoms of acute sinusitis. Objective data: purulent discriminate through ostium maxillarae; mucosa membrane of the nose is edematous and hyperemic sometimes. Chronic sinusitis takes toll on the state of optic nerve on the affection part; from the functional violation are observed: decrement in visual activity within 0.5-3.0 dioptries. On the X-ray picture of the additional nasal cavities the supramaxillary cavity in the sick part is clouded in that degree in what is expressed edema and polypous growth of its mucosa membrane. If in the cavity the purulence is present on the X-ray picture, made in the vertical position of a head, will be observed a distinguished boundary of the liquid level. From the afterextraction fistula could appear red granulomatous or grey polypous growth.

Diagnostics

Symptoms of the odontogenic sinusitis with perforation of the supramaxillary cavity floor could be in this correlation: headache – at 43.6% patients, pain and feeling of heaviness in the region of upper maxilla – 80.7%, passage of air from the mouth cavity in the nose cavity – at 67.9%, passage of liquid – at 46.1%, defluvium from the fistula after tooth extraction- at 67.1%, defluvium from the nose – at 23.1%, edema of the nose cavity and hypertrophy of concha of cranium – at 33.3%.

Differential diagnosis

Sometimes occur significant difficulties during the leading of differential diagnostics between rhinogenous and odontogenic sinusitis.

Chronic odontogenic sinusitis should be differentiated with the *chronic allergic inflammation* of the membrane mucosa of the supramaxillary cavity.

Odontogenic chronic sinusitis should be differentiated with the *malignant neoplasm* of the membrane mucosa of the supramaxillary cavity.

Treatment

The treatment of a chronic odontogenic sinusitis pursues the following aims:

- 1) To end the following infection of the supramaxillary cavity and eliminate the changed mucosa membrane;
- 2) To end the entry in the organism of inflammation products and microorganism life activity.

If such treatment will be unsuccessful, the operation is made, the aim of which is elimination of polypous cavity mucosa membrane and creation of a wide inosculation between it and lower nasal duct. Thanks to this is provided free outflow of exudates from the supramaxillary cavity.

It is necessary to irrigate 5-6 times supramaxillary cavity (through the perforation hole in the tooth fossa) by 6ml of 0.5% Novocain solution, in which are dissolved: any proteolytic ferment (chymopsin, chymotripsin, tripsin in the quantity of 20 mg). The surgical intervention should be used after described preliminary pharmaceutical treatment.

Anesthesia during the maxillary sinusotomy: palatal, incisival and tuberal regional anesthesia are combined with a plexus-anesthesia through transitory fold.

Surgery technique. Many surgeons use the Kolduel-Lyuk method, in other words open the supramaxillary cavity by the horizontal discission in buccal cavity and trapanize it in the region of canine fossa. For this on the frontal wall are made a range of perforation holes, that in the following are connected among themselves by the help of fissure bur.

Main stages of the surgery intervention according to Kolduel-Lyuk are: resection of the anterolateral wall, elimination from the supramaxillary cavity of a purulent, polyps, membrane mucosa and formation of wide inosculation of the cavity with lower nasal duct. The membrane mucosa and periostenium are incised in the mouth anteroom lower transitory fold from the canine to the second molar. It is formed trapezoidal flap according to Vasmund – Neiman-Zaslavsky.

It is performed the curettage of supramaxillary cavity.

Treatment of the perforated sinusitis

In some cases can be a self-active closing of perforated hole in the socket of extracted tooth. This can occur in three cases:

- 1) In the case if in the cavity there are no any debris (tooth root) and inflammatory changes;
- 2) In the case of its acute inflammation;
- 3) In the case of aggravation of the chronic sinusitis, but without polypous phenomenon.

In the second and third cases are necessary ablutions of the supramaxillary cavity (every day, 6-10 days) by the furacilin solution (1:5000) and introduction in the cavity 1.00.00 UN of penicillin in 10 ml 0.5% of Novocain solution.

More often the opening of the perforated hole is performed by buccogingival, rarer by palatal flap. If the perforated hole is not big is enough to “refresh” its edges, mobilize tissues from the vestibular and palatal parts, and to put interrupted stitches.

Topic № 5

Diseases of salivary glands

Diseases of the salivary glands include different processes according to etiology and pathogenesis:

- 1) reactive -dystrophic;
- 2) inflammatory;
- 3) traumatic;
- 4) tumorous;
- 5) tumor-like.

In the present time for making diagnosis and the stage of salivary glands disease, and also for supervision over process dynamics greater meaning has cytological examination of a secretion swab, examination of the secretion function of salivary glands, ropiness of a saliva, X-ray and radioisotope methods.

For specification of a debris in the main excretory duct of submandibular gland is made X-ray picture of a mouth cavity floor (intraoral), and during the assumption of presence of debris in an intraadenous canals of the salivary gland is made X-ray examination in different extraoral view.

The more significance during the specification of character changes in a salivary gland has sialography – the contrast study of salivary glands ducts for X-ray diagnostics. After insertion of canula in the duct by the syringe is administered around 1-2 ml of heated oil or water solution of a contrast material till the appearance of a slight tenderness and is made X-ray picture.

Reactive-dystrophic changes of salivary glands (sialosis).

In response to general diseases: hormonal, inflammatory, tumorous, collagenoses, lymphomatosis, granulomatosis and other, salivary glands react by swelling, violation of the function or combinations either changes. Among reactive-dystrophic processes in the salivary gland define the Mikulicz diseases and Sjogeren syndrome.

Mikulicz disease.

For disease is characteristic enlargement of salivary and lacrimal glands. In some cases, Mikulicz disease courses against the background of lymphomatosis or granulomatosis. Actual reasons of the disease are insufficient clear.

Pathological anatomy. During the lymphomatosis are defined the proliferation of accumulation of lymphoid tissue, is observed the empty of intraadenous part of ducts of salivary glands.

Clinical picture. The disease begins from the gradual swelling of salivary glands, in the first place from the both parotid, then submandibular, sublingual salivary and lacrimal glands. The disease develops slowly, for years, the edema achieves a significant sizes sometimes. The function of gland in the initial and clinical apparent stages is not violated. The general state of patients does not suffer.

During the external examination of a patient is seen edema in the region of affected salivary glands, the skin color under the swelling is not changed. During the palpation increased glands in the beginning of disease are soft and in the advance stage of disease becomes solid and torose. It is observed the narrowing of palpebral fissure in the external third by means of increasing of salivary glands.

On sialogram during the Mikulicz disease is observed the sharp narrowing excretory ducts parotid salivary gland. In the beginning stage of disease there is no filling of minor ducts, this is associated with plerum of ducts in the hilus of lobes (places of proliferative lymphoid tissue). This phenomenon gradually grows, the gland size increases.

Prognosis. In the case of restricted form of lymphomatosis or granulomatosis prognosis is favourable.

Treatment is included in the usage of X-ray therapy, which in the case of restricted forms of lymphomatosis and granulomatosis give a quite good results.

Sjogren-Gougerot syndrome

More often affects women in the age older than 40 years old.

Clinical picture. Patients complain on the sharp dryness in the mouth, impossibility to talk and eat without frequent humidifying of a mouth; especially is difficult the meal of dry food.

The disease is characterized by the range of symptoms:

- xerostomia;
- recurrent swelling of parotid glands;
- xerophthalmia and xeratoconjunctivitis;
- dry laryngotracheitis;
- chronic polyarthritis.

The disease is chronic, lasts by years, leading to atrophy of salivary and lacrimal glands, and by this is explained dryness of mouth cavity and conjunctivitis.

During the face examination is observed:

- edema of the parotid regions;
- dryness of lips, which is covered by barks and disobeys epithelium.

In the mouth cavity the mucosa membrane is dry powdered by folds and fissures, hyperemic.

Kneading of the parotid glands with the aim to obtain saliva from ducts is unsuccessful.

Diagnosis. Sialography detects the destruction of gland parenchyma; typical picture of the ducts arborization is not seen; as they are partially obliterated, and partially augmented. Introduced in the gland **sol.Iodolipoli** is effused back extremely slowly, which indicates about the sharp lowering excretory function of gland.

During the cytological examination is observed flexible exfoliation of the epithelium of excretory duct, but some patients have a deep epithelium affection as excretory ducts, as gland parenchyma; the presence of scaphoid and ciliary cells, layers of inflammation changed epithelium and exudates cells.

The picture of the chronic polyarthritis is quite typical: small joints are affected, in some could be observed sclerosis.

The treatment is elaborated deficiently. The propitious result can be achieved in the case of usage of short procaine blocks or salivary glands galvanization.

The treatment for such patient is necessary in collaboration with oculist and therapist.

Acute parotitis (epidemic parotitis)

This is acute viral disease, which is characteristic by inflammation of big salivary glands. Usually are affected parotid glands, rarer submandibular and sublingual salivary glands. In main cases disease affects children, sometimes adults, and rarer women.

Etiology. The disease causative agent is filterable virus. The infection proceeds by airborne way by objects.

Clinical picture. The incubation period is 2-3 weeks. Three forms of clinical flow:

- 1) mild form of a disease;
- 2) middle form of a disease;
- 3) severe form of a disease.

Also are distinguished an uncomplicated and complicated course of a process. Three tender point: in front of ear hircus, at upper mastoid bone, under the creana of mandibular.

During *the mild form of a disease* the clinical features are expressed weakly, body temperature do not arise. Swelling of parotid salivary gland is almost without pain; from their ducts is discharged transparent saliva in moderate quantity. More often than not is affected only one parotid gland. The edema and pain disappears during one week.

During the *middle form of a disease* after (3-2 days) prodromal stage is observed discomfort, small appetite, rigor, headache, painless in the region of neck or joints and limb muscles, sometimes a small temperature rising, dryness in the mouth cavity, tender swelling of a one parotid salivary gland. Usually after 1-2 days the second salivary gland is swelling, following body temperature rising till 37.5-38 C. The edema increases very quick. When this happens one part has more changed then other.

Some patients have hyperemia of mouth mucosa membrane and entrance of parotid duct. Salivation is usually deepened. In 2-3 days the inflammation begins to remit.

During *the severe form of a disease* after expressed prodromal phenomenon is swelled parotid gland, rarer two. Fast comes inflammation in the circle. The edema is localized in the beginning along the external surface of the mandible branch, speared upwards till the eyesocket level, behind approaches to mastoid and lowers till the maxilla angles, sometimes till the clavicle. The skin under above the swelling has normal color, but is tense. During the involving of the sublingual, submandibular glands, the swelling spreads on the neck. Increased parotid salivary gland pushes aside outside ear lobe, squeezes and sometimes significantly narrows external acoustic duct. Sometimes the mouth opening is difficult. More often develops catarrhal stomatitis, is observed the reddening of the mucosa membrane of pharynx, regions of the mouth of parotid duct. The duct is palpated as band. It is observed sharp diminution or even ceasing of salivation from the swelled gland. In rare instances, particular in the initial stage of disease the salivation can be raised. During the purulent-necrotic process from the duct exudes the pus. The body temperature can arrive 39-40 C. On the 5th-6th day the body temperature gradually falls. The swelling falls but can begin the abscess formation.

Complication:

– affection of the nervous system: meningitis, encephalitis, sometimes with the paralysis of cranial and craniocerebral nerves, changing of visual, oculomotor, abductor, facial and threshold – snail-like nerve, and also mental disorder;

- Orchitis, mastitis, pancreatitis, nephritis;
- Can be lethal outcomes in the case of developing of a purulent-necrotic process in the gland, affection of the nervous system.

Diagnosis:

- Epidemiological anamnesis;
- Changes of glycemia level in the blood and quantity of diastasis in the blood and urine.

- The diagnosis is approved by the release of the epidemiological parotitis virus, by reaction of complement connection, by reaction of agglutination braking.

Differential diagnosis:

- False parotitis of Herzenberg, as complication of the wisdom tooth eruption difficulty with the one part lymph nodes affection put in the capsule of the parotid salivary gland. During the false parotitis the salivation is not observed.
- Parenchymatous parotitis, chronic flow, aggravation, usually this is one part disease.
- Mikulicz disease affects all salivary and lacrimal glands, also differs by the chronic flow.

Complications: gland fusion, cheek necrosis by the type of salivary fistula formation, cicatrices' contracture of mandible.

Treatment is performed symptomatic and consists in care for sick and aggravation prevention. Bed regime 7 days.

- Physiotherapy procedures
- Rinsing, irrigation
- Hot compresses
- Antibiotics for prevention 5-7 days
- During the suppuration - the section, antibiotics and prolongation of general treatment.

The section are made taking into consideration direction of branches of facial nerve, dissect by the scalpel only skin and subdermal base, then by the hemostatic forceps is foliated capsule and glandular tissue.

Rational is the combination of a section, which edges the angle of mandible, with the section under the zygomatic arch. Both sections are connected one with other in the gland thickness. In the case if there are features of the fusion of a gland –chymotrypsin, at the same time to underrun the salivary gland duct.

According to character infections can be:

- 1) Banal, non specific (purulent);
- 2) Specific (virus, tuberculous, actinomycetic, syphilitic).

There are:

- 1) Acute epidemiological parotitis;
- 2) Acute non epidemiological (typhus, measles, flu, scarlatina, polyarthritis), surgical interventions in the abdominal cavity, exhaustion and dehydration of the organism, dryness.

Etiopathogenesis of acute parotitis:

- 1) Neurogenic or neuroendocrine violations of the salivary glands functions, appears in the result of surgical interventions on stomach, esophagus or bowels; after ovariectomy during arthritis.
- 2) Inflammatory inflammations, ways: stomatogenic, hematogenic, lymphogenic and contact.

Non epidemiological parotitis, differentiation:

- 1) Catarrhal form;
 - 2) Purulent form;
 - 3) Gangrenous form.
- Phlegmon of the parotid region, the purulence exudates is absent from the duct.
 - Lymphadenitis, there is no purulence exudates.

Chronic parenchymatous parotitis.

Clinical picture.

Complaints of the patients depends from the disease stage. In the beginning painless swelling, sometimes increases during ingestion; in same case by patients are observed the sensation of smack of purulence in the mouth. The gland edema achieves big sizes sometimes, has a strict borders. The disease usually affects one gland, and lasts by years, gives periodic aggravations and simulates the picture of epidemiological parotitis.

Objective: during the palpation is defines increased solid –bulged and painless gland. During its massage from the duct is segregated saliva with the purulence admixture or fibrinous clots in smart quantity.

In the beginning of disease on contrast sialograms are seen duct deformation of II-IV order, but later – the death of these ducts and adjoin glands spaces, on the picture are seen characteristic cavities filled by contrast substance. These cavities lose their contours, interflow with each other:

- 1) Diagnostics. In the Inflammatory process can die entire gland parenchyma and be substituted by fibrous tissue. At the same time the main excretory duct, lost the tone, sharply widen and takes a flakes-like shape.
- 2) X-ray sialographical examination:
 - For the initial stage of disease it is characteristic a quick growth of radioactivity of affected gland during first 30 minutes – cytological;
 - In the stage of expressed features, more slow accumulation radioactive connection in gland;
 - In the advanced stage of disease on the radiosialogram the affected gland radioactivity is low.

The aggravation of chronic parenchymatous parotitis appears in the autumn time usually and is characterized by the stabbing pain (during meal) in the affected gland; edema increase, body temperature increase till 38C, appears limitation of mouth opening.

The aggravation remits (under the influence of warm or by oneself), but after some time repeats again.

Objectively: the edema of parotid region, painful and bulging of a gland during the palpation, skin tensity.

The mucosa membrane of the cheek is good; the excretory duct of a gland is palpated in the view of tension bar; mouths of the duct gape, during the gland massage from the duct segregates thickened saliva with the admixture of fibrous impurities and purulent.

Complications:

- The phlegmon of parotid masticatory region;
- Violation of gland functions.

Pathological anatomy.

Edema and expressed angiomas in interlobular layers, widening of a separate excretory ducts. Around them in some areas there are lymphohistiocytic infiltrations. In some

places the development of fibrous tissue. In lumens of separate ducts are seen pinkish structureless masses.

Differential diagnosis:

- Interstitial sialadenitis;
- Sialodochitis (clinic, X-ray, cytological)
- Epidemiological parotitis;
- Tumors.

Treatment: It is highly difficult disease even in the initial stage of disease, as died parenchyma of gland is not possible to regenerate. The aim of treatment is to suspend the disease progressing and its aggravation. For this it is defined the character of microflora which is segregated from the duct and sensitivity to antibiotics. Then rinse the excretory duct by orbital probe, moreover daily rinse the duct by solution of corresponding antibiotics.

The introduction of the solution in the duct is made slowly, till the appearance of pressure sense (but not pain!) in the duct region. Then it is introduced antibiotics with 2 ml of an anesthetic and the duct is massaged again, free it from the introduced antibiotics and mucus-purulent-masses until the getting of a clean saliva, then one more time is introduced antibiotic and is left in gland. Such treatment is daily until the clinical recovery.

- Also is used the chymotrypsin solution (splits the fibrous clots, dilute the thickened saliva), for necessary concentration in the flacon with chymotrypsin is added 5 ml isotopic NaCl solution;
- Anti-inflammatory action has iodine, it is better to introduce it after aggravation, during the improvement of salivation;
- Potassium-iodate solution(beginning from 2% solution 1 tablespoon 3 times a day, raise the concentration till 10% and lead this course of treatment during 2.5 months);
- Injections of galantamine (1 ml 0.5%) or pyrogenal (25 injection by individual scheme);
- Galvanization of gland (daily, 30-40 days);
- X-ray therapy (by/Gr with interruption 2-3 days, total doze 5-8 Gr);

Rarely, in the case of unsuccessful conservative treatment, it is resorted to total or partial excision of parotid salivary gland, providing safety to facial nerve.

The treatment of aggravation of chronic parenchymatous parotitis is directed to prevention of phlegmon affection of parotid –mastication region:

- UHF (ultra high frequencies);
- Intramuscular antibiotics (sensitivity ;
- Hexamethylenetetramine with phenyl salicylate or 2% solution of K Iodinol.

Hexamethylenetetramine is prescribes, taking into consideration that after absorption it exudates from the organism not only by kidney with urine, but also by salivary glands with saliva. The mechanism is following: after absorption in bowels it meet in the inflammation zone acid medium and by means of it fall to formic aldehyde and ammonia ($(\text{CH}_2)_6\text{N}_4 + 6\text{H}_2\text{O} = 6\text{HCHO} + \text{NH}_3$).

In the inflamed gland, from the hexamethylenetetramine is distinguished a substance - formic aldehyde, which is active antiseptic means, providing anti-inflammatory action.

Phenyl salicylate get into contact with alkali content of nutrition canal is distinguished into salicylic acid and phenol; these substances soaked and pass through liver, partial get into contact with glycogen, sulphuric acid. A part of phenol, do not manage to get into tandem connections penetrates into a greater blood circulation, including - in the inflamed salivary gland, to which provide an antiseptic action.

Prognosis. In the case if described treatment of aggravation do not lead to decline and leads to phlegmon inflammation, it is indicated dissection of tissues and the same treatment as during acute purulent parotitis.

Chronic interstitial parotitis

Clinical picture.

Patients complain come to cosmetic discomfort: they make anxious swellings in the gland region. In the past, patients marked tingling and gravity in the gland which transient to light nagging pain. The in cold time the grade of gland increase increases.

Chronic interstitial inflammations are seldom. In the base of disease is reactive growth of interlobular tissues without destruction of glandular substance.

More often are affected both glands, the skin above what remain unchanged. By palpation is defined painless, soft consistence, increased gland. During the massage from its duct exudes clean saliva, but in poor quantity, as gland function lowers. In the aggravation period is aching pain in the affected gland.

Diagnostics.

By the help of sialography is defined narrowing of excretory ducts, which are not good contoured. In the stage of expressed features is observed a significant oppression of saliva release, is observed narrowing of excretory ducts.

Treatment.

X-ray irradiation for 0.6-0.9 Gr through very 2-3 days, total from 6 to 8 Gr.

- Anti-sclerotic means
- For improvement of gland function - galantamine, in usual dozes.

Pathological anatomy.

Friable connective tissue in interlobular layers with the edema and angiomatosis; blood vessels are widen and filled by blood. Lymphahistiocytic infiltrates are in layers. Parenthetic gland is replaced by lymphoid elements. In the lumen of ducts are observed eosinophil masses. By fields is developed fibrous tissue and fat tissue.

Differential diagnosis.

- Chronic parenchymatous sialadenitis;
- Sialodochitis;
- Tumors;
- Epidemiological parotitis.

Actinomycosis of salivary glands

More often are affected submandibular, rarer – parotid salivary glands.

Clinical picture. Patients complain to swelling and indurations of separate parts of salivary gland. During the actinomycosis of a parotid salivary gland troubles the difficult mouth opening. During the examination the swelling is matted together with cyanotic skin integuments, which have characteristic indrawn. The process courses slow. During the formation on the mouth skin or mucosa membrane of the fistulas, could be identified in the separable, actinomycetes and their druses.

On a sialogram of the gland is seen the narrowing of gland ducts in the affection region, in abscessation places appears cavities of invalid form.

Tuberculosis of salivary glands

The tuberculous affection can be rare. Infection spreads to the salivary glands by lymphogenic and hematogenic way.

Clinical picture. It is affected parotid or submandibular salivary gland. The process develops slowly, sometimes by years. Of an area appears painless swelling or of all salivary gland. During the palpation in the gland is felt separated indurated areas. The tuberculous affection of salivary glands is necessary to differentiate with the malignant neoplasms (it is characteristic a permanent process growth, remission absence).

Treatment – specific (after phthisiatrician consultation).

Syphilis of salivary glands

It is observed rarely, in the second period of syphilis. It is affected parotid salivary gland predominant.

Clinical picture. Appears painless solid swelling of one or several areas of salivary gland with the following quick their mollities (fluctuation). Sometime there are cankering and appears fistula.

During the syphilitic sialadenitis salivary gland increase slowly, compresses, seals with surrounding tissues, their mobility is limited.

The diagnosis is clarified on the base of anamnesis (syphilis disease), blood examination (Wasserman reaction), and histological examination.

Treatment. Specific.

Chronic calculous sialadenitis (Ptyalolithiasis)

For ptyalolithiasis is characteristic a chronic inflammation of salivary glands with the formation of ptyaloliths in their ducts. More often ptyaloliths are formed in the ducts of submandibular glands, but can be in parotid, and sometimes in sublingual and small salivary glands. More often of ptyalolithiasis are sick men of middle age. Calculus can be formed in the main excretory duct or in the ducts of I, II, III order, they conditionally are named "gland calculus". Number and size of calculus can be different, from the size millet grain till the chicken egg. The calculus color is usually hoary or yellow. On the calculus fracture is seen lamination, the centre of the calculus usually are foreign bodies or fungus colony.

Calculus formation in the ducts or in the salivary glands depends from the violations of mineral turnover in the body, as often during this is observed accretion of calculus in the cholic and uretic ways.

Clinic picture. Ptyalolithiasis is manifested by different symptoms depending on the stage of process. (A.V. Klementov).

The initial stage (without clinical manifestations of inflammations) flows usually insensibly for the patient. In the stage patient complains to colicky pains and swelling in the region of salivary gland during ingestion, which quick, in 20-30 minutes disappear. During the bimanual palpation in some cases managed to feel a bit increased salivary gland, during the calculus placement in the main duct is palpating their thickening.

In the case of increase in the gland of chronic inflammation features becomes more frequent its aggravation, at the same time appears the features of acute sialadenitis. In the region of salivary gland and along its ducts appears pains and swelling, which growth rapidly.

In the cellulose which surrounds duct or gland. Appears tissue infiltration, develops a purulent process – abscess or phlegmon.

Patients complains on the acute pain in the region of one or another salivary gland, swelling, temperature rising till 37,5 – 37,8 C, general uneasiness.

Pains increase during ingestion.

During the examination of a patient, inside of inflammatory processes in soft tissues, thrust oneself attention the localization of the process in the duct or gland region, reddening and ostium of entrance duct, absence of exude from it or pus release.

During the ptyalolithiasis in the advanced stage, during the clinical features of chronic inflammation, patients complain on the permanent swelling in the gland region, stinging pains,

especial during the ingestion of soul food. During the patients interview is found out the bigger duration of disease (from 3 till 30 years). Patients mention often short-term process aggravations.

Objectively during the patient examination can be seen the swelling in the gland region (submandibular, parotid).by the palpation is defined solid sometimes bulging, painless salivary gland and increase of lymph nodes. Bimanual palpation (by two hands- from the mouth cavity and from the outside) gives the opportunity to define solid formation along the duct or in the middle section solid and increased submandibular salivary gland (calculus). Saliva segregation is sharply decreased in comparison with symmetric gland on 1 – 1,3 ml.

Diagnosis of ptyalolithiasis is approved by the X-ray examination. The calculus can be defined on the X-ray picture of mouth floor cavity or on the lateral mandible body projection. For definition of gland ducts state and definition of non X-ray contrast calculus is reasonable to make sialogram.

Treatment of ptyalolithiasis depends from the disease stage. In the initial and advanced stage is indicated the calculus removal, during the irreversible changes in the ducts and glands is indicated - gland removal.

Fistula of salivary gland

Fistula of parotid salivary gland and its duct appears in the result of hurt, rarer as the outcome of inflammation process of gland (saliva calculus, neoplasms, and suppurations), sometimes a result of purulent and other processes in the cheek region and parotid-mastication region (phlegmon, noma, syphilis, cancer and other).

Fistula of submandibular salivary gland is observed very seldom. It appears in the result of epithelization of a wound canal at salivary ducts cell`s and skin epithelium`s cost. More stable fistula appears during hurt of a main excretory duct.

Fistula of parotid salivary gland it is usual to divide into the fistula of main duct and intraadenous ducts.

Clinical picture. Patients complain on the permanent defluvium of saliva from the fistula duct on the facial skin surface, increased during the ingestion. On the facial skin integuments, which are without a break wetted by saliva, often is observed the irritation.

During the examination of a patient is managed to find out fistulous tract corresponding to place of affection. From the punctuate entrance, on the fundus of choanoid crypt is segregated a light liquid drop by drop.

For definition of duct patency it is probed by thin flexible probe.

Treatment. The choice of one or another method of treatment in the case of main duct fistula and parotid salivary gland depends from a range of peculiarities.

Methods of treatment if salivary glands fistulas can be divided into three groups:

- 1) Surgical methods of treatment of salivary fistulas;
- 2) Methods, based on the diminution of salivation, spray through the fistula of amnyctic medicaments or cauterly of fistula duct, which cause через adhesive process (adjuvant methods);
- 3) Methods of retiremet of parotid salivary gland function by its denervation or radiation of X-rays.

PATIENT MEDICAL RECORDS
Of patient Lebedev Vladimir Alexeevich

Clinic diagnosis:

- a) Prior disease: acute purulent periostitis of maxillary bone;
- б) Associated disease: multiple carries;
- в). Complications of the prior disease: — .

OVERVIEW

- 1. Lebedev Vladimir Alexeevich
 - 2. Age: 3 years, date of birth: on the 28 of April 2003 year.
 - 3. Sex: masculine
 - 4. Nationality: russian
 - 5. Place of residence: c. Ijevsk, 17-b Shkolinaya str.
 - 6. Place of work: -----
 - 7. Day of illness: on the 26 of May 2006 year.
 - 8. Date to clinic admission: on the 27 of May 2006 year.
- Follow-up data: on the 30 of May 2006 year.

ANAMNESIS

**1. THE ANAMNESIS OF A REAL DISEASE
(ANAMNESIS MORBI)**

Complaints during the admission. Complains of the severe pain in the region of upper gingiva, increasing during the gingiva palpation by the upper incisors which prevent mastication; body temperature rising till 37.6-38⁰ C; general weakness, trouble.

On the follow-up day there are complaints of insignificant weakness.

1.2. Development during the disease. During 5 days before admission to the clinic was sick by flu; the disease proceeded difficult, the body temperature was on the level of 38-39,5⁰ C, the child was debilitated, loosed weight; toward evening of the 25 of May the patient state increased, the body temperature normalized. On the night of the 26th/27th the child becomes restless, sleep bad. Morning on the 27th of May becomes apparent the edema and painfulness of the upper gingival in the incisors region, an edema of upper lip, body temperature 37.8⁰ C. They were become in the Clinical hospital N 2; the child was admitted in the dental department. In the same day were made the surgical treatment: were extracted right medial and lateral incisors, made prosection and drainage of the appeared in the upper gingival region apostasies. The state of the patient in the same day improved: pains reduced, body temperature normalized. The drainage was taken away on the 29th of May.

**2. LIFE HISTORY
(ANAMNESIS VITAE)**

Was born and live in Chisinau. In physical and psychical development the child is not behind. All products and all courses of supplemental feeding were introduced at term. The nutrition is regular, different, the caloric value is middle. Diseases in past: at the age of 2 years was ill by rubeola; was all by ARVI (acute respiratory viral infection), grip.

Housing conditions are satisfying. He visits kindergarten N 71. The hereditary background: mother has a chronic periodontitis, abnormal occlusion (progenia); matrimonyc grandfather has a chronic periodontitis. Relatives there were not tuberculosis, syphilis, alcoholism, psychical diseases and malignant neoplasms.

Allergic background: pharmaceuticals and alimentary products intolerance was not educed. The blood transfusion was not made.

PHYSICAL EXAMINATION

THE GENERAL EXAMINATION OF THE PATIENT

The general state of the patient is satisfactory. The location is active. The consciousness is clear. The walk is firm. The attitude is straight. The facial expression is usual. The head form is unchanged. There is no square, tower skull, the craniomalacia is absent. Cerebral cranium prevails over facial.

Palpebral fissures are placed symmetrical. The mucosa sclera there is conductivities of the physiological color. Apples of eye are of right form.

Nystagmus is absent.

Nasal dorsum: there is no attenuation; the deflection of nasal septum, defects, and the saddle nose are absent. Wings of nose in the respiratory act do not participate. The nasolabial folds are symmetric. Lips angles are on the one level; there are no fissures, bridoe and rash. Lips are of physiological color.

The neck is of middle sizes. Vessels blood fillings is normal; there are no torticollis, movement restrictions; there are no Stok`s collar and Venus necklace. Thyroid gland is not palpated. Body smell and expiratory air is physiological. The body built is right. The body type is normosthenic. The nutrition is satisfactory. Subcutaneous fat is 1.5 cm in the region of theatears, on the lateral abdominal wall on the omphalus level, in the region of the lower angle of shield bone, on the inner surface of femus, shoulder; at the breast bone edge – 1cm; the distribution is equal.

Skin integuments are of physiological color, there are no rash, ulcers, cicatrices and bed sores. Collateral perfusion is absent.

Nails are of physiological color, there are no exfoliation and striation. There are no nails in the nature of “hour`s glases” and fingers in the nature of “clubbed fingers”.

The skin palpation: the turgor is preserved, humidity is in norm, the skinn stiffness on the back of hand is preserved.

Lymphonodus: parotid, proper cervical, postaural, supraclavicular, retromuscular, popliteus are not palpating; submandibular are increased till 7-8 mm, painless of the soft-elastic consistence, with the smooth surface, are flexible and with the surrounding tissues are not soldered together.

Bones are painless, not distorted; the bearing function is not disturbed. Articulation: the movement in the full force, painless, the skin under the articulation is not changed. Muscles are painless; functions are preserved, the muscle force is symmetrical downed. The vertebral column has a physiological arcuation, painless; the movement in the full force. Hand fingers tremor is absent.

Edema on the face, sacral bone and cnemis there are not.

THE STADY OF RESPIRATORY SISTEM

1. General surveying of breast. The breast is normosthenic: the anteroposterior size is smaller than lateral, the supra- and infraclavicular fossa are expressed moderate, feather have moderate transverse duct, intercostals spaces are expressed, shoulder-neck angle is blunt, Ludwig angle is not expressed, the epigastric angle is right; bladebones are contouring soft; along the full vertical of extent of the body breast region is equal to abdominal.

Bothe parts of breast participate in the breathing process, the arrearage of one half from other there is no. There is the combined type of breathing. The breathing rhythm is right. The breathing movements of the middle depth; the frequency of breathing movements is 28 times / minute. The objective signs of apnea: the change of frequency and depth of breathing, the participation of the auxiliary musculature and wings of nose in the breathing process, acrocyanosis, orthopnea – are absent.

2. **Palpation.** Resistance of the breast is normal. The painfulness along the intercostals nerves, muscles, feathers there are no. The vocal fremitus is not changed, is performed equal on the symmetrical parts of the breast. The sensation of pleura friction during the palpation is not observed. The excursion of the breast during the breathing composes 1 cm, the maximal excursion is 4 cm.

3. The percussion of the breast.

A. Topographic percussion:

The definition of the upper borders of the lungs:

Identification line	Right lung	Left lung
Parasternal	V rib	—
Mid-clavicular	VI rib	—
Anterior axillary	VII rib	VII rib
Midaxillary	VIII rib	VIII rib
Posterior axillary	IX rib	IX rib
Scapular	X rib	X rib
Paravertebral	On the level of the spine of XI dorsal vertebra	On the level of the spinal of XI dorsal vertebra

The identification of the active flexible of lungs along lines:

Identification line	Right lung	Left lung
Mid-clavicular	3 cm	—
Midaxillary	4 cm	4 cm
Scapular	3 cm	3 cm

The height of the lang apex: in front (relative to clavicular) – 2 cm right and left; backward – on the level of spine of VII of cervical vertebra right and left. Traube`s space is free.

Б. The qualitative percussion: under the symmetrical lungs palight rts the percussion sound is pulmonary is not changed.

4. **Auscultation.** Under the symmetrical lung parts is listen puerile respiration. The side breathing murmurs: rales, the sound of pleura friction, crepitation are absent. Bronchophony is not changed is listen in the type of not clear gabble.

THE STUDY OF THE BLOOD CIRCULATORY SYSTEM

1. General surveying.

The general surveying of the heart area: cardiac hump is absent; the apex beat is not defined. The negative apex beat there is no. The hart hump is absent. Epigastric pulsation is absent.

The examination of large vessels: the pulsation of the temporal arteries, carotid shudder, the swelled of neck veins, the venous pulse is not observed. The “worm” syndrome, the Quincke's capillary pulse, the widening of subcutaneous veins on the breast are absent, the varix dilatation on the lower limb are absent. The Alfred Musset's sign is absent.

2. The palpation. The apex beat is palpated in the 5th intercostals space in 1 cm to the outside from the left and mid-clavicular line; rhythmic, restricted (square space 2 cm²) of the middle force and height. The cardiac impulse is not defined.

The bruissement sign, pericardial friction are absent. Retrosternal pulsation of aorta is not palpated.

The pulse is of the same force on the both hands. Right (pulsus regularis), hard (pulsus durus), full (pulsus plenus), great (pulsus magnus), quick (pulsus celer). Pulse rate is 98 /minute.

3. Heart percussion.

The identification of borders of the relative absolute heart dullness:

<u>Borders</u>	<u>Относительная тупость</u>	<u>Абсолютная тупость</u>
Right	The right edge of the sternal	Left edge of the sternal
Left	To 1 cm to the outside from the left cardiac-clavicular line	To 0,5 cm inward from the left mid-clavicular line
Upper	2 rib	3 rib

The wideness of the vascular fascicle – 3,5 cm.

4. Auscultation.

A. Heart auscultation:

I point: There are auscultated 2 tones; tones are rhythmic, increased; 1st tone according to volume are equal to the 2nd tone. It is auscultated blowing systolic noise.

II point: There are auscultated 2 tones; tones are rhythmic, increased: 1st tone according to the volume is equal to the 2nd tone.

III point: There are auscultated 2 tones; tones are rhythmic, increased; 2nd tone is more loud then 1st tone.

IV point: there are auscultated 2 tones; tones are rhythmic, increased; 2nd tone is louder then 1st tone. There is observed the accent of the II tone in the IV point.

The secondary aortic area (Botkin-Erb point): there are auscultated 2 tones; tones are rhythmic, increased; the 2nd tone is louder then 1st tone.

The changes in the tones configuration neither the point there are no auscultation. The quail rhythm and gallop rhythm are no auscultate. The pericardial fremitus is absent.

B. Vessels uascultation:

On carotid and subclavicular arteries there are auscultated 2 tones. Traube tone, the double Vinogradov-Dyurozie noise during the auscultation of femoral artery are absent. Nun`s murmur is absent during the auscultation of jugular vein.

THE STUDY OF ALIMENTARY SYSTEM

1. The examination of the abdomen in the vertical and horizontal position. The abdomen form is usual, the omphalus is mired. The extended skin veins, chromatism, the cyanotic areas are absent. Peristaltic and anti-peristaltic movements of the stomach and intestinal tract are not observed.

2. Superficial abdomen palpation in the vertical and horizontal position. Local and generalized abdominal tenderness are not observed. Local and General strain are absent. Hernia holes, the dehiscences of rectus muscles of abdomen, masses are absent. Ascite by the fluctuation method is not defined. The increases of the organs of abdominal region are not observed.

3. Deep methodical palpation according to Obraztov-Strajesko.

The parts of the intestinal tract are without any peculiarities.

A lesser curvature of stomach is not palpated. The bigger curvature of stomach is palpated for 2 cm higher the omphalus in the type of painless ruga of soft-elastic consistence with the smooth surface; pylorus is not palpated.

Liver palpation according to Obraztov. The lower edge of the liver emerges from under the costal arch for 1 cm; soft, with the smooth surface, acute, straight, and painless. Pulsation during the palpation of the liver is not observed.

The palpation of the gall bladder. The gall bladder is not palpated.

Palpation of the pancreas. The pancreas is not palpated. Te painfulness in the Shoffar triangle is not observed. The painfulness in the Dejarden point is absent.

The palpation of the lien in the horizontal position and right lateral decubitus (accoding to Sali). The lien is not palpated.

5. Abdomen percussion. Free fluid in the abdomen is not defined. The symptom of Obraztov is negative.

Percussion sizes of the liver according to Kurlov:

- 8 cm – along the right middle - clavicular line;
- 7 cm – along the frontal middle line;
- 5 cm – along the left costal arch.

The percussion sizes of lien: lateral – 5 cm, transverse – 4 cm.

6. Auscultation. It is auscultated murmurs in all parts of intestinal tract. The peritoneal murmur over the liver, lien is not auscultated.

EXAMINATION OF THE URINE ORGANS

1. General surveying. General surveying of the lumbar region: bulge, reddening of skin integuments are absent.

2. Palpation. Kidneys palpation according to Obraztov ans Botkin: kidneys are not palpating.

Palpation of the urinary bladder: is not palpated.

The palpation across of renal ducts: is painless.

3.Percussion. The Pasternatkyi symptom is negative. The percussion of the urinary bladder: the percussion sound over the urinary bladder is tympanal.

STATUS LOCALIS

Face is symmetric. Skin integuments are of the physiological colour, rash and cicatrices are absent. Nasolabial, mental folds are not expressed. The Corners of lips are one the one level; there are no fissures, bridoes and eruptions. Lips are of the physiological color.

Regional lymph nodes: occipitalis, postaural, aural, hinder and frontal jugular, submental, supraclavicular do not palpated; submandibular are increased till 7-8 mm, painless of the soft – elastic consistence with the smooth surface, flexible with the surrounding tissues are not glomerated.

Both temporal-mandibular joints are painless, skin over ligatures is not changed, bulging is absent. The active and passive movements in the full volume are painless.

Emergent points of the trifacial nerve are painless.

Lips are of the physiological color, fissures and eruptions are absent.

The mucosa of mouth cavity atrium is pink, calculus and hemorrhage are absent. Frenulum of upper and lower lips are of the physiological color, the entirety of the mucosa is preserved, the hemorrhage and calculus there are no. On the upper gingival, on 5 mm higher of alveolus of the right medial and lateral incisors is post surgical horizontal discission of the length 1 cm covered by pink granulation tissue; there is no drainage, the serous drainage is low. The gingival mucosa membrane is a bit congested in the discission region.

Stenon ducts from the right and left side are opened on the check mucosa membrane on the level of the upper 2nd molars; the mucosa membrane around entrances are of the physiological color; there is no any edema; during the massage of the parotic salivary glands from the right and left side from entrances Stenon ducts free effuses transparent, without color saliva of the serosal character.

The tongue lolls out along the middle line and has a white calculus; papillas are expressed, there are no teeth indentations.

The mucosa membrane of the mouth floor cavity is of the physiological color, there are no calculus and hemorrhages. Sublingual elevation are well expressed, the mucosa membrane is of the physiological color. The mucosa membrane of the sublingual papillas is not congested, the entrances of Vartanov ducts from the left and right side during the massage of the submandibular salivary glands free effuses colorless, transparent saliva of the serosal-mucic character. The mucosa membrane of the soft and hard palate are of the physiological color, there are no calculus and hemorrhages. There is observed insignificant hyperemia of faucial pillars.

The hinder part of the throat is smooth, shine of the physiological color; there are no calculus and hemorrhages.

Tonsils are of the physiological color, there are no purulent plugs, lacuna apostasies.

Teeth formula:

V	IV κ	III	II	I		I	II κ	III	IV	V κ
V	IV	III	II	I		I	II	III	IV	V
	κ								κ	

Sockets of the extracted teeth are closed by the pink granulations, there is no secretion.

CLINICAL EXCLUSION:

- a). The main disease: acute purulent periostitis of the right maxilla bone;
- б). Intercurrent diseases: multiple caries;
- в). Affection of the main disease: — .

TREATMENT

1. The surgical treatment. The local infiltration anesthesia by the 1% solution of the trimecaine. It is carried out the prosection of the apostasies by the transection of the gingival mucosa membrane and periostenium; the purulent cavity is drained by the rubber strip. «Causative» teeth: medial and lateral right upper incisors are removed.

2. Pharmaceutical treatment.

- 1) Sulfanilamides:
Rp.: Tab. Sulfadimethoxini 0,5 N. 6
D.S. Should be taken by mouth 2 tablets in the 1st day, then by 1 tablet per day drink after a big quantity of water.
- 2) Nonnarcotic analgetics:
Rp.: Tab. Analgini 0,5 N. 10
D.S. Should be taken by mouth ½ of tablets 2 times per day.
- 3). Nitrofurans derivatives:
Rp.: Tab. Furacilini 0,02 ad usum externum N. 10
D.S. Dissolve 1 tablet in 100 ml of the water, by the received solution rinse the mouth cavity 2 times per day.
- 4). Antihistamine products:
Rp.: Tab. Suprastini 0,025 N. 10
D.S. Should be taken by mouth ½ of tablets 2 times per day.

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