

**MINISTRY OF HIGHER EDUCATION, SCIENCE AND INNOVATION OF
THE REPUBLIC OF UZBEKISTAN
MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN
TASHKENT STATE DENTAL UNIVERSITY**

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DISEASES OF THE PERIODONTIUM AND ORAL MUCOSA

Textbook

For master's students of medical higher education institutions,
Faculty of Dentistry

Field of knowledge: **900000 – Healthcare and Social Security**

Field of education: **910000 – Healthcare**

Educational direction: **70910101 – Dentistry (by specialties)**

Tashkent – 2026

ISBN

UDK

BBK

and

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Introduction

The Resolution of the President of the Republic of Uzbekistan, Sh.M. Mirziyoyev, "On the Establishment of the Tashkent State Medical University" No. PQ-149, issued on April 22, 2025, assigned prospective tasks to the Tashkent State Medical University and its branches. According to this resolution, the university is the leading higher education institution for training highly educated specialists, scientific personnel, and improving their qualifications in the medical field. This necessitates a review of the scientific and methodological foundations of specialized higher education. Based on this resolution, the tasks include developing state educational standards, curricula, and programs in the medical field, writing textbooks and methodological guidelines, as well as proposing new effective treatment methods based on modern medicine and improving existing ones. Furthermore, doctors will need retraining and qualification improvement based on the developed methodological guidelines and textbooks. It is worth noting that the measures and actions outlined in the resolution are among the most urgent tasks in carrying out reforms in the medical field in our country. The shortage of textbooks written in the Uzbek language is not a secret to anyone. Moreover, they often do not meet the requirements of modern specialized education and do not encompass all parts of dentistry. Considering the above-mentioned shortcomings, we hope that this textbook will meet the requirements of specialized higher education. Specifically, the textbook describes diseases of the oral mucosa that are common in Uzbekistan. The textbook mainly provides information about oral cavity diseases that dental practitioners frequently encounter in their daily activities. It also describes lesions of the oral mucosa in infectious and somatic diseases, the understanding of which opens a wide and clear path to the diagnosis of dental diseases. Indeed, for dental practitioners directly engaged in practical work, it is more important to identify and differentiate the changes in the oral cavity caused by the diseases covered in the textbook. Clinical signs and laboratory indicators serve as the main criteria in clinical diagnostics. Without identifying the pathogenetic factor (or factors) that played the main role in the development of the disease, a positive result from the

administered treatment cannot be achieved. When describing the clinical signs of diseases, we focused mainly on the changes in the periodontium and the oral cavity. Dental practitioners must first thoroughly understand these signs. Changes found in the periodontal tissues and internal organs and systems, depending on the nature of the disease, are further clarified by relevant specialists. Therefore, we have tried to present the information that dentists need to master in simple, clear, and precise language. This textbook also provides information on modern methods for the diagnosis and treatment of a number of periodontal tissue and oral cavity diseases that Master's degree students need to master.

PERIODONTAL DISEASES

Anatomic and Physiological Structure of Periodontal Tissue

The term periodontium originates from the English term "periodontal disease" – diseases of the periodontium (periodontium). The free gingiva ends at the gingival papilla. It is close to the tooth surface and is separated by the gingival sulcus. The main part of the free gingiva consists of collagen fibers, and elastic fibers are also present. The gingiva is well-innervated, containing various nerve endings (Meissner's corpuscles, thin fibers entering the epithelium and belonging to receptors that sense pain and temperature). The adherence of the marginal gingiva to the tooth surface and its resistance to mechanical impacts are explained by the turgor of the tissues, i.e., their internal pressure, the presence of high molecular weight substances, and also adhesion. The gingiva is formed by stratified squamous epithelium and a specific mucous lamina propria, with a poorly expressed submucosa. In a normal state, the gingival epithelium is keratinized, and keratohyalin substances are present in the cytoplasm of its cells. Glycosaminoglycans play an important role in the protective function of the gingival epithelium, especially in preventing the penetration of infection and toxins into the internal tissues. Acidic glycosaminoglycans (chondroitin sulfate, hyaluronic acid, heparin) are high molecular weight compounds that are important in the trophic functions of connective tissues, as well as in the processes of tissue regeneration and growth. The most common locations for acidic glycosaminoglycans are in the tissue papillae and the basal membrane, and they are found in smaller amounts in the stroma. In the periodontium, acidic glycosaminoglycans are located in the walls of blood vessels and along the direction of collagen fibers. Neutral glycosaminoglycans (glycogen) are detected in the gingival epithelium, located mainly in the cells of the prickle layer, and they decrease with age. Neutral glycosaminoglycans are also present in the endothelium of blood vessels and within leukocytes. Ribonucleic acid (RNA) is present in the cytoplasm of epithelial cells and in plasma cells. The presence of sulfhydryl groups was detected in the keratinized epithelial cells of the upper layer. In gingivitis and periodontitis, with edema and loss of intercellular

connections, they disappear. Currently, there is definite information about the important role of hyaluronic acid and the hyaluronidase system in regulating the permeability of capillary-tissue structures. Hyaluronidase, produced by microorganisms (tissue hyaluronidase), depolymerizes glycosaminoglycans and breaks the bond of hyaluronic acid with proteins (hydrolysis), thereby sharply increasing tissue permeability and losing protective properties. Therefore, glycosaminoglycans protect periodontal tissues from the effects of bacterial and toxic agents. The most common cells in the connective tissue of the gingiva are fibroblasts, less frequently histiocytes and lymphocytes, and even less frequently mast cells and plasma cells. Mast cells in normal gingiva are mainly located around blood vessels.

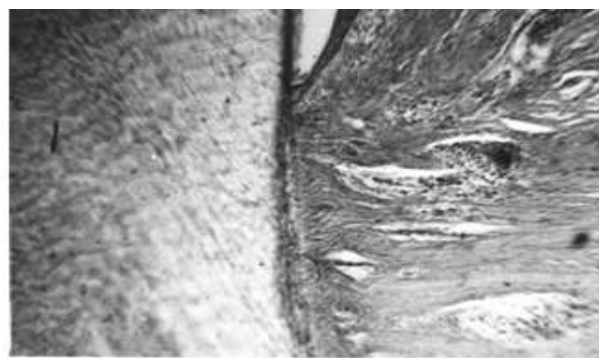
Gingiva. The free (interdental) and alveolar (attached) gingiva are distinguished. The marginal part of the gingiva is also separated. The part located between the teeth is called the free gingiva. It consists of the labial and lingual papillae, which form interdental papillae, resembling a triangle facing the incisal (occlusal) surfaces of the teeth. The attached gingiva is the part surrounding the alveolar ridge. It changes from the external surface of the alveolar ridge to the skin part along the tooth, and to other parts of the oral cavity. The alveolar gingiva is attached to the tissues of the upper jaw bones and protects against stress.

Gingival-Tooth Connection. The epithelium of the gingival papilla consists of the sulcular epithelium and the junctional epithelium (or attachment epithelium). The sulcular epithelium is a stratified squamous epithelium, and the junctional epithelium is located in an intermediate state between the stratified squamous epithelium and the gingival epithelium. There are many similarities between the junctional and gingival epithelia, but histologically they are completely different. The junctional epithelium is composed of several layers of elongated cells positioned parallel to the tooth surface. Radiographic studies show that the cells of the junctional epithelium contain proline and are renewed every 4-8 days, which is much faster than the renewal of gingival epithelial cells. Electron microscopic examination showed that the surface cells of the junctional epithelium have numerous

hemidesmosomes, which are connected to the apatite crystals of the tooth and attached through a thin granular layer of organic material (40-120 nm). Gingival Sulcus is a crevice between the healthy gingiva and the tooth surface, which is detected by careful probing. The depth of the gingival sulcus is usually less than 0.5 mm, and its base is located at the point where the epithelium organically attaches to the tooth surface.

Blood Supply of Periodontal Tissues

Periodontal tissues are supplied with arterial blood through the maxillary artery, which is a branch of the external carotid artery. The teeth of the upper jaw and the surrounding tissues receive blood from the pterygoid branches of the maxillary artery, while the teeth of the lower jaw and the surrounding tissues receive blood from the branches of the inferior alveolar artery. One or more dental branches exit the inferior alveolar artery for each interalveolar perpendicular bundle, which in turn gives branches to the periodontium and the cementum of the root. These branches form a network and unite through anastomoses. In the marginal periodontium, near the enamel-cementum junction, a vascular loop is noticeable, which connects with the gingival and periodontal vessels through anastomoses. The presence of arteriovenous anastomoses in periodontal tissues indicates the absence of terminal-type arteries in them.



**Figure 1. Collagen fibers in the circular ligament of the tooth.
Microphotograph.**



Figure 2. Blood supply of periodontal tissue (in the area of the upper jaw teeth).

The microcirculation system includes structural components: arteries, arterioles, precapillaries, capillaries, postcapillaries, venules, veins, and arteriovenulo-venular anastomoses. Capillaries are the thinnest-walled vessels of the microcirculation system, through which blood passes from the arterial to the venular segment. It is the capillaries that are responsible for delivering oxygen and other nutrients to the cells. The diameter and length of capillaries, as well as the thickness of their walls, vary significantly in different organs and depend on their functional state. On average, the internal diameter of a normal capillary is 3-12 micrometers. The network of capillaries forms the capillary bed. The capillary wall is composed of cells (endothelium and pericytes) and special non-cellular structures (basal membrane). Capillaries and the surrounding connective tissue, as well as the lymphatic network, ensure the nutrition of periodontal tissues and perform a protective function (Figure 5). The state of capillary permeability is of great importance in the development of pathological processes in periodontal tissues.



Innervation of Periodontal Tissue

The innervation of the periodontium is carried out by the branches of the second and third divisions of the trigeminal nerve. In the alveolar socket, the bundles of the dental nerve divide into two: one goes towards the pulp, and the other goes to the surface of the periodontium and towards the gingiva, moving parallel to the main nerve of the pulp. The periodontium has many thin, parallel-running myelinated and unmyelinated nerve fibers (Figure 3). At various levels of the periodontium, myelinated fibers branch and thin out as they approach the cementum. Free nerve endings located between the cells are present in the periodontium and gingiva. The

main trunk of the periodontal nerve moves parallel to the cementum of the root, from the intercorneal space towards the intercorneal arch. The abundance of nerve receptors allows the periodontium to be considered an extensive reflexogenic zone, suggesting the possibility of transmitting nerve impulses from the periodontium to the heart, gastrointestinal tract organs, and others.

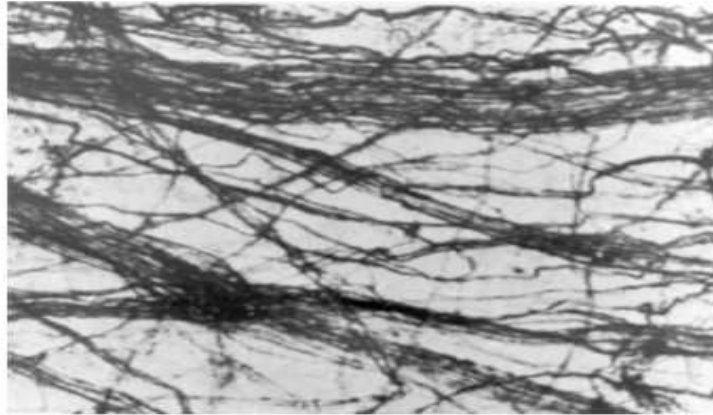


Figure 3. Nerve fibers of the periodontium.

The network-like structure of the lymphatic vessels plays an important role in the periodontal system, especially in its diseases. Healthy gingiva contains small, thin-walled lymphatic vessels of irregular shape. They are mainly located in the subepithelial connective tissue. During inflammation, the lymphatic vessels sharply dilate. Cells of inflammatory infiltrate are detected in the lumen of the vessels and around them. In the process of inflammation, lymphatic vessels help to remove interstitial material from the site of damage.

Age-Related Changes in Periodontal Tissues

The involutional changes in periodontal tissues, mainly, have practical significance. Understanding them helps the doctor in diagnosing periodontal diseases. Tissue aging is viewed as a complex and not fully studied general medical problem. They are, first and foremost, associated with changes in the genetic apparatus of the cells around the tooth, a decrease in their metabolism, and a reduction in the intensity of physical-protective processes. Age-related changes are mainly associated with changes in vessel walls, collagen, enzyme activity, immunobiological reactivity, and a decrease in the transport of nutrients and oxygen, which leads to the predominance

of cellular breakdown processes over regenerative processes. Age-related changes observed in the gingiva include a tendency toward hyperkeratosis, thinning of the basal layer, atrophy of epithelial cells, homogenization of fibers in the subepithelial layer, a decrease in the number of capillaries, dilation and thickening of vessel walls, a reduction in collagen content, loss of glycogen in the cells, a decrease in the amount of lysozyme in the prickly layer, and dehydration of the gingival tissues.

In the bone tissue, the number of Sharpey's fibers in the cementum decreases, hyalinization increases, the activity and number of proteolytic enzymes increase, bone marrow cavities widen, cortical plates thicken, osteon canals widen and become filled with fatty tissue. Bone tissue destruction during aging may also be associated with a decrease in the anabolic effect of sex hormones, which suggests a relatively greater activity of glucocorticoids. Age-related changes in the periodontium are characterized by the loss of intermediate network fibers, the loss of some collagen fibers, and a decrease in cellular elements. From a clinical and radiological perspective, involutional changes in periodontal tissues manifest as gingival atrophy, loss of root cementum coverage (in the absence of periodontal pockets - PC), and inflammatory changes, osteoporosis (especially post-menopausal), osteosclerosis, narrowing of the periodontal space, and hypercementosis. The age-related changes described above are accompanied by a reduction in the resistance of cellular and tissue elements to the effects of local factors (trauma, infections).

Microbial Flora of the Oral Cavity

According to modern concepts, the following are present on the enamel surface:

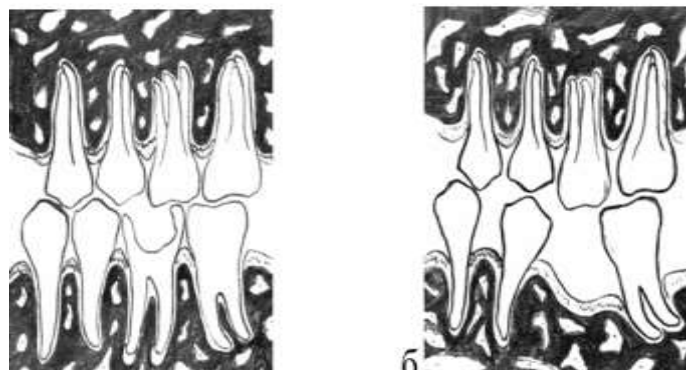


Figura 4. Cuticle – the reduced epithelium of the enamel.

Pellicle – an organic polymer film that forms upon contact of saliva with the enamel. Dental Plaque is composed of microorganisms and a small amount of organic substance, which is detected by Scanning Electron Microscopy (SEM) and immunofluorescent microscopy.

The stages of dental plaque formation are:

Stage 1 – within 1-4 hours after cleaning the teeth;

Stage 2 – formed during 4-5 days. For people with "high natural sanitation," the microbial population of the plaque may persist for a long period in this state, where microbial ratios are maintained even without systematic tooth cleaning.

Stage 3 (Established Plaque) – from 6-7 days and longer. Dental plaque is a typical example of a biofilm—a symbiotic community of species of microorganisms that forms in the presence of salivary fluid.

The number of bacterial cells in the oral fluid ranges from 50 million to 5 billion, and in dental deposits (plaque), there are more microbes—from 10 billion to 1,000 billion per gram of material. Currently, the total number of microorganisms in the oral cavity reaches 600-800 species, most of which belong to anaerobes. Periodontal diseases can develop under the influence of local factors (microorganisms of dental calculus, occlusal trauma) as well as the combined effect of local and general (endogenous) factors, where changes in the body's reactivity are significant. The following factors play a key role in the etiology of periodontal diseases: Plaque and dental calculus: their metabolic products and condition; Factors in the oral cavity: factors that enhance or diminish the pathogenetic potential of microorganisms and their metabolic products;

Factors controlling the metabolism of oral tissues: those that determine their response to pathogenic influences. The development of gingivitis is caused by the activity of microorganisms, influenced by the microbial plaque, which consists of their endotoxins, exoenzymes, and antigen-dependent substances. The number and species composition of bacteria, as well as their metabolic products and the degree of tissue resistance, determine the type (catarrhal, ulcerative, hypertrophic) and severity of inflammation, as well as the development of periodontitis and the speed

of periodontal tissue damage. Furthermore, it is appropriate to cite the definition of inflammation provided by one of the greatest authorities, Academician A.M. Chernykh: "Inflammation is a reaction to tissue damage that has arisen in the course of evolution as a response of the organism to changes; it consists of complex, phased changes in the microcirculation system of the circulatory system and connective tissues, carried out ultimately with the aim of isolating and eliminating the harmful agent, as well as restoring (or replacing) the damaged tissues." Thus, inflammation is an initial, sometimes short-lived, reaction to subcellular tissue damage. This reaction can end in prolonged tissue damage or complete recovery. It is probable that bacterial enzymes and toxins directly damage periodontal tissues. Metastases, impaired permeability of the histo-hematic barriers, perivascular edema, diapedesis of blood components, aggregation of erythrocytes, embolism, and finally, thrombosis of blood vessels. Hypoxia phenomena develop. All of this affects the structure of periodontal tissues. Due to the impaired supporting function of collagen and elastic fibers in the periodontium, tooth mobility and pain when biting on the teeth appear. Subsequently, in addition to edema, the destruction of collagen fibers is also observed. The integrity of the epithelial lining is disrupted in the area of the tooth-gingiva border, and as a result of the formation of periodontal sulci and the activity of microflora, inflammatory phenomena develop. Then, inflammatory-destructive processes lead to the resorption of the alveolar bone. Thus, traumatic factors can be the cause of localized, but not generalized, damage to the periodontium, but the effect of the microbial factor cannot be denied either. Furthermore, the excessive loading of certain teeth significantly accelerates the development of general processes, intensifying inflammatory and atrophic processes in periodontal tissues, leading to the loss of the ligamentous apparatus, and ultimately, the loss of teeth. Functional insufficiency (hypofunction) can also be a cause of periodontal disease. Insufficiency of the chewing function is a consequence of modern civilization. Well-processed, soft food does not place a full load on the periodontal tissues, as a result of which atrophic processes may develop. Furthermore, such food leads to the formation of dental calculus, which often causes

severe inflammation of the periodontium. Another type of functional insufficiency is local, for example, the absence of antagonist teeth, in cases of open bite, and so on. Insufficiency of the function of the masticatory apparatus reduces the resistance of periodontal tissues to various external factors (microorganisms, trauma), promotes the accumulation of dental calculus, reduces local blood circulation, and thus impairs tissue metabolism. This, in turn, leads to the development of destructive processes in the periodontium.

Atherosclerotic Damage to Blood Vessels. The influence of this factor is studied based on the concept of A.I. Evdokimov, who viewed periodontitis as a dystrophic process resulting from atherosclerotic changes. This theory is still confirmed by some scientists today, as it corroborates the fact known from epidemiological studies regarding the age-related increase in periodontitis and atherosclerosis. Atherosclerotic changes in periodontal tissues can, of course, play a significant role in the development of dystrophic processes in the periodontium. However, new data and the reevaluation of the cholesterol-based nature of atherosclerosis require some corrections.

Periodontitis of Neuro-Dystrophic Nature. It is known that atrophy-degenerative changes in periodontal tissues often arise due to poor blood supply to the tissues. But even with good blood supply, the tissue may lose its ability to assimilate external substances. Here, neuro-trophic factors play a role. The weakening of physical and chemical defense mechanisms can lead to the development of dystrophic processes in the periodontium. These manifest as increased permeability of blood vessels, decreased blood circulation, hypoxia, and metabolic disturbances in the tissues.

Factors Affecting the Epidemiology of Periodontal Diseases:

Social factors (age, gender, race, socio-economic status);

Microbial dental film, occlusal trauma, defects in fillings and prosthetics, orthodontic treatment; Harmful habits (non-compliance with oral hygiene, smoking, betel chewing); Systemic factors (hormonal changes of teeth, adolescence, pregnancy, menopause, etc.);

Drug therapy (steroid drugs, immunosuppressants, oral cyclosporine, etc.).

Methods for Examination and Diagnosis of Periodontal Diseases

Patient examination begins with studying the anamnesis of life and illness. The patient's complaints, the reason for consulting the doctor, profession, chronic diseases, diet, presence of harmful habits, and so on are clarified. In studying the dental status, attention is paid to the condition of the oral vestibule and, first of all, to the correct attachment of the labial frenulum, the depth and correct formation of the vestibule. Considering that local factors play a large role in the onset of inflammation in marginal periodontal tissues, it is necessary to mention the nature of tooth placement (anomalies of position, congenital partial adentia, presence of diastemas and tremas). Early removal of teeth, especially molars, often leads to the displacement of adjacent teeth, extrusion of antagonists, and subsequent development of traumatic situations (traumatic nodes according to Kurlyandsky) due to the overloading of their periodontium. Attention is paid to existing prostheses, as their incorrect fabrication often leads to tooth mobility. In normal swallowing, the tongue should not rest on the anterior teeth. Disruption of the swallowing act can be the sole cause of the forward protrusion of the anterior, especially the lower teeth, which leads to disruption of articulation and, consequently, excessive strain on the periodontal tissues and the development of a pathological process. Among the various types of bite pathologies, deep incisal overlap holds a special place, where pathological changes typically begin in the periodontium of the anterior teeth of the upper and lower jaws. Similar changes also appear, though much less frequently, in open and other anomalous bites. When collecting anamnesis data, special attention should be paid to bruxism. Inflammatory phenomena in the gingiva are assessed based on the following signs: hyperemia, edema, bleeding, desquamation, ulceration. In addition to these signs, atrophic and hyperplastic processes are also noted. In the medical history, the presence of supragingival dental calculus, plaque (film), and subgingival dental calculus is meticulously recorded. To detect dental plaque, they are stained with methylene blue using the Schiller-Pisarev test. Special tablets with erythrosine are also used for this purpose; these dissolve in saliva and stain the dental deposits.

Among the special examination methods, it is necessary to point out the Schiller-Pisarev test, radiological test, capillary microscopy, functional research methods, and determination of special indices. The Schiller-Pisarev test (crystal iodine 1 g, potassium iodide 2 g, distilled water 40 ml) is a method for vital staining of gingival glycogen, the amount of which increases when the gingiva is chronically inflamed. More intense staining of the gingiva after applying the solution indicates the presence of inflammation. This test can also be used after treatment—a positive test indicates the need to continue anti-inflammatory therapy. The determination of the resistance of gingival capillaries (according to Kulazhenko) is based on the principle of registering the time of hematoma formation in the gingiva. The diameter of the vacuum tip and the magnitude of the reduced pressure are constant. After creating a vacuum in the dilution system (residual pressure 20-40 mm Hg when the atmospheric pressure is 720-740 mm Hg), the sterile tip is placed on the gingiva, and the clamp is removed from the rubber hose, after which the tip is pressed against the gingiva. The time it takes for hematomas to appear indicates the stability of the gingival capillaries. Hematomas normally form in the frontal section of the jaws in 50-60 seconds, and in other sections, in a somewhat longer time. Vacuum diagnostic hematomas have a therapeutic effect, supporting enzymatic activity and stimulating the restoration of tissues in the area of the pathological process. This effect is more pronounced when vacuum hematomas are recreated 4-6 times each session until the condition of the gingival capillaries normalizes. With the development of the process, the following examination methods are currently used: panoramic enlarged radiography and orthopantomography. These methods allow a more accurate and objective assessment of the condition of the bone tissue of the alveolar processes of the jaws in periodontal diseases compared to the intraoral radiography method. In periodontitis, radiological examination reveals inflammatory changes (resorption) in the bone tissue. They begin with damage to the cortical plates and progressively continue with the destruction of the cancellous bone, often resulting in the appearance of bone pockets. Periodontosis is radiologically characterized by sclerotic changes in the jaw bone tissue; the height of the interalveolar septa may be

uniformly reduced. This, on the one hand, allows the study of bone tissue changes in the area of all tooth groups, and on the other hand, provides information about the symmetry of the damage. Pocket depth is measured using a special graduated WHO probe, pins, contrast solutions, and the degree of mobility is measured according to Platonov. 3 degrees of tooth mobility are distinguished.

In Degree I, mobility not exceeding 1 mm is observed in the buccal-lingual (palatal) or labial-lingual (palatal) directions relative to the crown of the adjacent tooth;

Degree II - mobility exceeding 1 mm in the same directions. Mobility also appears in the palatal-distal direction;

Degree III - the tooth is mobile in all directions; the tooth may tilt towards the adjacent edentulous area, and so on.

Probing pins have an advantage over solutions and plastic materials. The safest are gutta-percha pins. Pins are inserted into the pocket in the axial direction until elastic resistance occurs, which is considered the bottom of the pocket. In the case of bone pockets, the pin is inserted up to their base. The use of computed tomography is considered promising for studying the nature of periodontal pockets, especially for studying pocket topography. These changes in pocket depth are recorded on the dental formula using Arabic numerals around the symbol of each tooth, and the degree of its mobility is recorded with Roman numerals. Thus, the degree of periodontal damage is determined based on data from radiographic examination, pocket depth, and mobility measurements. Regarding the morphological examination of biopsy material (gingival biopsies), it is very important for making a more accurate diagnosis, and in some cases (eosinophilic granuloma, rheumatic diseases, collagenoses, idiopathic forms of periodontal lesions), it is one of the main diagnostic methods. It is advisable to use bacteriological and cytological examination methods suitable for the conditions of periodontology offices or departments (studying the microflora of gingival pockets), as well as determining leukocyte emigration according to the Yasinovsky method before, during, and after treatment. The passage of neutrophil leukocytes through the oral mucosa indicates their participation in the process of phagocytosis. The M.A. Yasinovsky method is

used to assess the migratory activity of leukocytes. Exfoliative cytology, as a method for dynamically observing the course of a pathological process, can be successfully used in periodontology (ulcerative gingivitis, periodontitis). Material for smear-imprints is collected using rubber strips. The staining of the preparation depends on the goal and tasks of the study: for general examination, it is carried out according to the Giemsa-Romanovsky method, and for determining the distribution of nucleic acids and glycogen, special methods are used, such as those by Brachet and MacManus. The preparations are examined under an immersion system microscope. Quantitative and qualitative composition of cellular elements (neutrophil leukocytes, epithelial cells, lymphocytes, plasma cells), and the distribution of glycogen and nucleic acids in the cells are used as cytomorphological and cytochemical evaluation criteria.

Periodontal Index (PI). The Periodontal Index (PI), developed by Russel (1956), is currently widely used to study the prevalence and intensity of periodontal tissue damage. The condition of the periodontium in each tooth is determined by a score from 0 to 8, taking into account the degree of gingival inflammation, tooth mobility, and the depth of the periodontal pocket. In doubtful cases, the lowest possible score is assigned (Table 1). All scores are added and divided by the number of existing teeth. The obtained PI is assessed as follows:

0.1 - 1.0 - initial and stage I of the disease;

1.5 - 4.0 - presence of destructive changes characteristic of stage II of the disease;

4.0 - 8.0 - stage III of the disease.

The condition of periodontal tissues can also be taken into account using the Papillary-Marginal-Alveolar Index (PMA). The PMA index can only be used to study initial changes in the periodontium, which is why it is called the gingivitis index. Inflammation of the papilla (P) is scored as 1, inflammation of the gingival margin (M) as 2, and inflammation of the alveolar gingiva (A) as 3. The numerical value of the PMA index is the sum of the scores of the marginal periodontal condition of all existing teeth and is always expressed as an integer. Parma (1960) proposed his modification to express the PMA index value as a percentage. The

PMA index according to Parma (in percent) is calculated as follows: The sum is formed by adding up all the highest scores of the marginal periodontal condition for each tooth; the number of teeth is taken as 24 for ages 6 to 11, 28 for ages 12 to 14, and 30 from age 15 onwards.

A number of indices have also been proposed to assess the hygienic condition of the oral cavity [Fedorov Yu. A., Volodkina V. V., 1964; Green, Vermillion, 1960]. The hygienic condition of the oral cavity, as well as its dynamics, is assessed using the Fedorov-Volodkina Hygiene Index as follows. The Schiller-Pisarev solution is applied to the vestibular surfaces of the teeth. Depending on the amount of plaque, different areas of the tooth surface are stained with different intensities. The plaque is evaluated quantitatively and qualitatively. Quantitative evaluation (K) is carried out using a five-point system: if the entire surface of the crown is stained - 5 points, 3/4 - 4 points, 1/2 - 3 points, 1/4 - 2 points, not stained - 1 point. The calculation is performed according to the following formula:

$$K_{cp} = \frac{\sum K}{n},$$

where I_h - is the hygienic index of the intensity of dental plaque; $\sum K$ - is the sum of the index values on all teeth; n - is the number of teeth (6).

G. N. Pakhomov (1974) modified the Fedorov-Volodkina index, increasing the number of teeth to 12:

$$\begin{array}{c|c} 6 & 1 \\ \hline 6 & 3 & 2 & 1 \end{array} \quad \begin{array}{c|c} 1 & 6 \\ \hline 1 & 2 & 3 & 6 \end{array}.$$

Quantitative and qualitative evaluation is carried out as in the Fedorov-Volodkina method.

OHI-S Simplified Hygiene Index. This index [Greene, Vermillion, 1969] involves studying dental plaque on the vestibular and oral surfaces of 6 adjacent teeth or 1-2 teeth from other groups (molars, premolars, incisors) of the lower and upper jaws.

Soft dental plaque is detected using a probe. If it covers no more than 1/3 of the tooth crown surface, its amount is scored as 1, the presence of plaque on 1/2 of the crown surface as 2, and on 2/3 as 3. If there is no plaque on all examined tooth surfaces, 0 is assigned. When debris is unevenly deposited, the surface with more debris is evaluated. A more accurate index is obtained when the arithmetic mean values of the two or four surfaces examined are taken. Then the numerical values of the dental plaque are added and divided by the total number of examined teeth, i.e., 6. The fraction obtained from the division is the simplified hygienic index of the oral cavity. In normal condition (ideal hygienic state), it should not exceed 1.0; if the index is greater than 1, it is an indicator of poor hygienic condition of the oral cavity. The individual index is determined up to one tenth, and the group index (i.e., by age, gender, background pathology, bite pathology, etc.) up to one hundredth. The WHO currently recommends the new Community Periodontal Index of Treatment Needs (CPITN) for epidemiological studies. This index is notable because it allows for an accurate determination of treatment tactics when a small number of examinations are conducted in groups of individuals. It uses assessment criteria that are understandable to the doctor—the presence of gingival bleeding, dental calculus, periodontal pocket, and its depth.

Functional Methods. These methods are increasingly used in periodontology. Rheoperiodontography is often performed in the blood supply system of the periodontium. The method is based on measuring changes in the electrical resistance of the studied tissues associated with the pulse dynamics of blood filling as a result of heart activity, which allows determining the condition of the arterial and venous segments of the vascular bed based on the analysis of the graphical representation of peripheral hemodynamics. The graphical recording of pulse changes in the electrical resistance of periodontal tissues is called a rheoperiodontogram, which has qualitative and quantitative characteristics. The qualitative analysis of the rheoperiodontogram includes a visual description of the curve shape, the presence and location of dicrotic and additional waves, which allows the detection of functional changes (vasoconstriction, vasodilation) in the tonic tension of the

vascular wall. Quantitative analysis allows the determination of amplitude-time parameters of the rheographic curve. These include: Rheographic Index (RI), Vascular Tone Indicator (VTI), Peripheral Resistance Index (PRI), Elasticity Index (EI). Rheoperiodontography can be used both in the diagnosis of periodontal diseases and in monitoring the effectiveness of treatment measures. Functional tests with nicotinic acid, nitroglycerin, and temperature stimuli are performed to differentiate functional changes in periodontal vessels from structural changes. Regional blood circulation of periodontal tissues can also be studied using the photoplethysmography method. Its difference from rheography is that when a light beam passes through the periodontal tissues, the pulse oscillations of their optical density—the photoplethysmogram (FPG)—are recorded.

The functional state of the periodontal microcirculatory bed can be examined using the biomicroscopy method. This method allows for the visual observation of angioarchitectonics and the determination of the functional state of the arteriolar and venular segments of the microcirculatory bed of the gingival mucosa, as well as the nature of blood flow in the microvessels. Photography with subsequent processing of the photonegatives allows the determination of microvessel diameters. The study is performed using a contact microscope with 100-fold magnification and a depth of n. In recent years, the polarography method—the study of oxygen tension levels in the tissue—has become quite widespread in periodontology. This method is based on the reduction of oxygen at a polarizing platinum electrode when a constant electric current with a voltage of 0.6-0.8 V passes through the tissue. The electrode consists of an open platinum sensor with a working surface of 0.3 mm, which is fixed to the surface of the gingival mucosa, i.e., using the contact method. The contact polarography method has several advantages over the previously used needle methods: the method is physiological and does not damage the mucous membrane. The exoosteometry method can be used in periodontology to study the functional state of bone tissue. It is based on measuring sound conductivity, which depends on the density of the bone tissue. The time taken for the ultrasonic pulse to pass through the bone is recorded. The method is very sensitive to changes in bone structure,

which depends on the physical loads applied to the bone and the destructive processes occurring in it during periodontal diseases. With the development of osteoporosis, the osteometry index increases.

The study of the pH of saliva in various pathological conditions of the oral cavity has certain diagnostic significance in diseases of the periodontium and oral mucosa. This test allows for the correct selection of a particular drug, including the alkaline rinsing method. The study of the composition of gingival fluid opens up great opportunities for researchers. Its composition depends on the degree of inflammation expressed. The fluid is collected from the gingival pockets of the upper frontal teeth. If necessary, general clinical examination methods should be consulted (blood, urine, glucose-ultrasound, etc.) and examinations by specialists (gastroenterologist, cardiologist, endocrinologist, therapist, etc.) should be conducted.



Classification of Periodontal Diseases

Currently, there are hundreds of classifications of Periodontal Diseases in Periodontology. Such diversity is explained not only by the existence of various forms of periodontal diseases but also by the lack of a unified principle in systematization. The clinical manifestations of the disease, pathomorphology, etiology, pathogenesis, as well as the nature and extent of the process are used as the main criteria. The multiplicity of various classifications of periodontal diseases is also explained by the varying location of specific mechanisms of damage to the periodontium and the lack of clear knowledge about the cause-and-effect relationships of periodontal diseases with diseases of various organs and systems.

The main categories used by periodontologists in systematizing periodontal diseases are related to the clinical form of the disease and the nature of the pathological process, and its stage (severity). The clinical forms of the disease are: Gingivitis, Periodontitis, Periodontosis, and Periodontoma.

Gingivitis

The group of gingivitis includes the following independent forms of marginal periodontal damage: Catarrhal, Hypertrophic, Ulcerative, Atrophic. The first 3 forms of gingivitis, which are most common in clinical practice, are discussed here.

Catarrhal Gingivitis. It is more common in the clinic than others. Inflammation can be localized in the marginal periodontium of 1-2 teeth or be generalized. The characteristics of the location of the inflammatory process in the marginal periodontium largely depend on the etiological factors. Local causes lead to or contribute to the appearance of localized gingivitis. Generalized symmetric lesions of the periodontium, located in the area of the upper jaw and the anterior teeth of both jaws in adolescence, indicate the predominance of general factors (diabetes mellitus, cardiovascular diseases, gastrointestinal tract lesions, etc.) rather than local factors in their development. Gingivitis with pigmentation at the gingival margin, which occurs in some Southern peoples—Arabs, Africans, etc.—in leukoses. Catarrhal gingivitis in adults often proceeds as a chronic inflammatory process. Patients complain of bleeding from the gums during eating and brushing their teeth, an unpleasant taste and odor from the mouth. Upon objective examination, edema and hyperemia of the gingival margin and interdental papillae, bleeding from the tip of the papillae when pressed and probed at their base, the presence of dental plaque, and a positive iodine reaction are detected. The general condition of the patients is unchanged, except in cases of acute or exacerbated chronic gingivitis. Radiological examination usually reveals no pathological changes in the bone tissue of the alveolar processes of the jaws. The severity of gingivitis depends on the combination of general changes in the body and the extent to which the gums are involved in the pathological process.

Hypertrophic Gingivitis. It is mainly localized in the area of the anterior teeth of the upper and lower jaws. In adults, this disease is less common than in adolescence. Hormonal changes (juvenile gingivitis, gingivitis of pregnant women), drug preparations—taking Difenin (5,5-diphenylhydantoin), cyclosporine, long-term use of oral contraceptives, Vitamin C deficiency, blood diseases (leukemic reticulosis)

are important in the etiology of hyperplastic gingivitis. Among local factors, bite pathology and irrational prosthetics should be mentioned. Hypertrophic gingivitis in pregnant women occurs in 45-49\% of examined women. It develops if the periodontium was inflamed before pregnancy and if the hygienic condition of the oral cavity is poor. Hypertrophic gingivitis begins in almost 40\% of patients suffering from epilepsy who take Hydantoin (Difenin). Clinically, this process is characterized by a significant increase. The papillae of the frontal teeth are firm when palpated, edema and hyperemia are faintly expressed, and depend on the hygienic condition of the oral cavity. The histological picture is without any changes. Hypertrophic gingivitis can manifest in specific diseases such as leukemic reticulosis. Its appearance is explained by the weakening of the general reactivity of the body and, as a result, the activation of local microflora due to toxic and autoimmune changes. Phenomena occurring in the oral cavity in leukoses are characterized by being very diverse (hemorrhage, desquamation, ulcerative-necrotic changes, gingival hyperplasia). Hypertrophic gingivitis occurring in leukemic reticulosis is characterized by aching, bleeding, significant, and relatively rapid enlargement of the gingival papillae.

Two types (stages) of hypertrophic gingivitis are distinguished: edematous and fibrous gingivitis.

The edematous form (stage) is clinically characterized by enlargement of the gingival papillae, a bluish tinge, a shiny surface, spontaneous or induced bleeding, pain during eating and brushing teeth, and aesthetic disturbances.

In the fibrous type of hypertrophic gingivitis (fibrous hyperplasia), the gingival papillae grow, are firm when palpated, do not bleed, and have false pockets. No change in the color of the papillae is observed, and accumulations of supragingival calculus are detected. Hypertrophic gingivitis is called mild when gingival hypertrophy reaches at most 1/3 of the tooth crown. Moderately severe proliferative gingivitis is characterized by a somewhat more significant deformation of the gingiva: at most 1/2 of the tooth crown is deformed; when it is severe, the gingiva covers 2/3 or all of the tooth crown.

Ulcerative Gingivitis. In the origin of ulcerative gingivitis, the altered reactivity of the organism and the reduced resistance of the marginal periodontal tissues to autoinfection of the oral cavity, especially to Gram-negative bacteria, are of primary importance. Gingivitis often manifests in adolescence after general illnesses (influenza, tonsillitis). Stress can be of great importance in the development of ulcerative gingivitis. The gingiva of the upper and lower jaws are affected; less often, the disease is localized only in one jaw. Difficult eruption of wisdom teeth can play a provocative role. Subjective symptoms: unpleasant odor from the mouth, pain in the gums, especially when eating, bleeding. Upon objective examination, diffuse ulcers are found at the gingival margin, the tips of the papillae are cut off, the ulcer surface is covered with a fibrinous coating, and bleeding begins when the coating is removed; the presence of dental calculus and plaque is characteristic. Often, body temperature rises, regional lymph nodes enlarge and become painful, headache, malaise, insomnia appear; appetite is lost, digestion is disturbed, and there is a foul odor from the mouth. Changes in the number of blood cells and increased ESR are detected. It must be remembered that ulcerative gingivitis can be observed in systemic diseases. The severity of ulcerative gingivitis depends both on the extent of the inflammatory process and the intensity of the clinical manifestations mentioned above. In ulcerative disease, in addition to streptococci and staphylococci, fusospirochetes are also found in gingivitis, which indicates impaired resistance of periodontal tissues; the role of protozoa (gingival amoeba, trichomonas) is particularly large in the appearance of necrotic changes in the connective tissue structures of the periodontium. Catarrhal and ulcerative gingivitis of mild severity can regress without complications, but defects in the junctional epithelium and post-necrotic defects of moderate and severe severity can remain irreversible. The neck of the teeth may become exposed after a past ulcerative gingivitis.



Figure 4. Hypertrophic gingivitis.

Periodontitis

Periodontitis is an independent nosological form of periodontal disease, which can be localized in the area of one or more teeth or have a generalized nature.

Localized Periodontitis. It develops under the influence of local causes: filling material or arsenic paste entering between the teeth, occlusal trauma (bite pathology, premature removal of molars, etc.), physical trauma (post-traumatic osteolysis of the bone), poorly made prostheses, and others. Localized periodontitis is common in clinical practice, but its generalized form is a much more serious problem.

Generalized Periodontitis. It always begins with catarrhal gingivitis and is characterized by a breach of the integrity of the tooth-gingiva junction, destruction of the attachment, bone tissue resorption, and the formation of a periodontal pocket. Subjectively, periodontitis manifests as bleeding gums, tooth mobility of varying degrees, pain sensations, hyperesthesia of the tooth necks (symptoms may not be persistent), and pus discharge from the pocket. Objective examination notes gingival bleeding, the presence of subgingival calculus and plaque, tooth mobility, pus discharge when the gingival margin is pressed with an instrument, and a positive Schiller test result. However, the main sign of periodontitis is the presence of a periodontal pocket.

The severity of periodontitis is primarily determined by three leading symptoms: the depth of the periodontal pocket, the degree of bone tissue resorption, and the resulting tooth mobility. These signs form the basis for choosing surgical and orthopedic treatment. Mild Periodontitis is characterized by a periodontal pocket depth of up to 3.5 mm, mainly in the interdental area, and initial destruction of the bone tissue of the interdental septa: widening or disappearance of the cortical plates, phenomena of osteoporosis, and an insignificant reduction in the height of the interdental septa (less than 1/3); the teeth are not mobile, do not shift, but there is induced bleeding. The patient's general condition is usually not disturbed in mild periodontitis. Moderately Severe Periodontitis is characterized by: pocket depth reaching up to 5 mm; bone tissue of the interdental septa is resorbed from 1/3 to 1/2; pathological mobility of teeth of degree I-II, shifting may occur.

Severe Periodontitis is characterized by: pocket depth greater than 5-6 mm, pathological mobility of teeth of degree II-III, bone tissue resorption greater than 2/3, sometimes complete resorption of the alveolar septum; shifted teeth; pronounced traumatic articulation; significant tremas (gaps); defects in the dental arches are present. Pus discharge and abscess formation are often observed in moderate and severe periodontitis. The radiological appearance of periodontitis depends on the stage of the process. The initial stages are characterized by osteoporosis and destruction of the cortical plates and interalveolar septa.

Subsequently, blunting (or cupping) of the septal crests is noted. An important radiological sign of rapidly progressing periodontitis in adults is the frequent appearance of foci of destruction, leading to the formation of bone pockets. The contours of the bone tissue of the alveolar processes have a characteristic festoon-like "eaten away" appearance, and subgingival calculus accumulates. The structure of the bone tissue in other parts of the jaw is unchanged. There is no bone osteosclerosis characteristic of periodontosis. Inflammation-related changes in periodontitis can spread in three main directions. After the cortical plate of the interdental septum bone is destroyed, inflammatory-resorptive phenomena spread to the cancellous bone—this is the most common direction. Such a condition is

radiologically determined as the blunting (destruction) of the crest of the interdental septum (2). Spread towards the periodontal ligament (3) also initially involves resorption of the bone compact plate, followed by destruction of the cancellous substance, but the resorption continues in a vertical direction, forming bone pockets and abscesses. The third path of inflammation spread to the periosteum is clinically manifested as: Subsequent resorption of the bone tissue. All the aforementioned variations of periodontitis are observed in the clinic. Furthermore, a combination of the indicated variants is also possible.



Figure 5. Generalized Periodontitis.

Parodontosis

Parodontosis is relatively rare in clinical practice, occurring in 1-8% of patients. The absence of inflammatory phenomena and periodontal pockets in this form, along with good tooth fixation, minimal dental deposits, gingival recession, exposure of tooth necks, and the presence of wedge-shaped defects, are characteristic of this disease. In addition to the hyperesthesia of the necks to various stimuli, patients complain of itching in their gums. Differential diagnosis is conducted taking into account anamnestic data, the specific features of the clinical manifestations, and primarily the radiological picture. Radiological examination allows for the detection of sclerotic changes in the bone tissue (narrowing of bone marrow spaces, fine-cellular appearance of the bone). Atrophic processes in this tissue lead to a uniform decrease in the height of the interdental septa while the cortical plates are preserved.

From a radiological point of view, changes in the bone when an inflammatory process is present should be considered secondary destructive changes. Mild Periodontitis is said to be present when the neck or root of the teeth is exposed up to $\frac{1}{3}$; on the radiograph, the height of the interdental septum is reduced up to $\frac{1}{3}$. In moderately severe periodontitis, the tooth root is exposed up to $\frac{1}{2}$, and the interdental septum is reduced up to $\frac{1}{2}$. The teeth may be mobile (Degree I). In severe periodontitis, exposure of the root greater than $\frac{1}{2}$ and a reduction in the height of the interdental septa of $\frac{1}{2}$ or more are observed, and tooth mobility may be present.



Figure 6. Periodontitis.

Periodontolysis (Idiopathic Diseases with Progressive Lysis of Periodontal Tissues)

These diseases, due to the unique features of their clinical manifestations and prognosis, do not fit the nosological forms listed above. This group includes Papillon-Lefèvre Syndrome, neutropenia, immunodeficiency states (agammaglobulinemia, etc.), diabetes mellitus, especially juvenile or established diabetes, eosinophilic granuloma, Letterer-Siwe and Hand-Schüller-Christian diseases (Histiocytosis X), and others. For most diseases in this group, the following signs are leading: Progressive intensification of significant processes involving the destruction of all periodontal tissues: gingiva, periodontium, and bone tissue,

accompanied by tooth loss within 2-4 years. The relatively rapid formation of a periodontal pocket, pus discharge, tooth migration, and dystopia are observed.

Specific changes in the radiological picture: The predominance of rapidly progressing resorption of bone tissue, forming lacunae, and the complete dissolution of the bone tissue in a relatively short time. In the bone tissue, osteolysis processes dominate, which apparently begin due to the activation of osteocyte enzyme systems, a significant disturbance of metabolism, and specific microangiopathies (for example, in diabetes mellitus). In syndromes like palmoplantar keratosis and neutropenia, children initially lose their deciduous teeth, and subsequently, as permanent teeth erupt, they also lose them. Usually, in such cases, it is possible to identify one or another form of pathology characteristic of neutropenia, diabetes mellitus, and others, accompanied by other clinical manifestations.

Eosinophilic Granuloma. It is also observed in children. Eosinophilic granuloma is encountered in the clinic in diffuse and focal forms [Segelnik L. N., 1961; Kolesov A. A. et al., 1976]. In the oral cavity, gingivitis, periodontal pockets, and tooth mobility are observed. It is characteristic that the phenomena occurring in the oral cavity are often the initial and sole symptoms of the disease in its early stages. Radiological examination reveals osteoporosis and resorption of bone tissue; in the focal form of the disease, the process is localized and gives a significant compensatory reaction (sclerosis at the edges of the rarefaction focus). The pathomorphological picture is characterized by the presence of reticular cells, accumulation of eosinophils, and lymphomacrophage infiltration.

Letterer-Siwe Disease. In its advanced stage, it is characterized by fever and pallor of the skin. Radiological examination shows osteoporosis, and diffuse or focal resorption of the interalveolar septa and the body of the jaw.

Hand-Schüller-Christian Disease. Also belongs to the reticulohistiocytoses. It is clinically manifested by diabetes insipidus, exophthalmos, the appearance of tumor-like formations in the bones, and delayed growth and development. The clinical picture of generalized periodontitis is detected during the period of pronounced

symptoms of oral pathology. Radiological examination reveals destruction of the bone tissue of the alveolar processes of the jaws.

Papillon-Lefèvre Syndrome. Characterized by a progressive osteolysis of the bone of the alveolar process of the jaws, which stops with tooth loss. The etiology is unknown. It is inherited in an autosomal recessive pattern. The syndrome is accompanied by hyperkeratosis and desquamation of the palms and soles. The clinical and radiological signs of changes in periodontal tissues correspond to the picture of generalized periodontitis. Histological examination reveals an infiltrate filling the entire gingiva, consisting mainly of plasma cells, and intense fibrosis of the bone marrow area with inflammatory infiltrate. Treatment is symptomatic.



Figure 7. Periodontolysis.

Periodontomas

Periodontomas include gingival fibromatosis, epulis, periodontal cyst, and other tumors of the periodontal tissues. This section of the classification covers diseases that require surgical intervention, and detailed information about them is presented in the textbook on surgical dentistry.

Gingival Fibromatosis. It is more common in adults. The etiology is unknown. It belongs to the group of genetic diseases. The disease is characterized by a slow progression and the appearance of firm, non-tender, irregular growths upon palpation, which are localized along the entire alveolar process or in the area of certain, often frontal, teeth. The color of the gingiva is unchanged. Histologically,

gingival fibromatosis is characterized by the presence of dense connective tissue, a scarcity of blood vessels, and the absence of cellular infiltration. Radiologically, phenomena of osteoporosis, and sometimes the destruction of the interdental septa, are found. Treatment is surgical.

Epulis. The formation of a mushroom-shaped growth resembles the hypertrophy of the interdental gingival papilla in terms of clinical manifestations. It is located on a stalk, which ensures the relative mobility of the epulis. It is observed in both adults and children. Fibrous, vascular, and giant cell epulides are distinguished. The final diagnosis is made after histological examination. Epulis is often detected in pregnant women and may partially regress after childbirth. Limited phenomena of osteoporosis are characteristic of the radiological picture. Treatment is surgical, always involving electrocoagulation; in case of recurrence, the tumor is removed along with one or two adjacent teeth.

Periodontal Cyst. It occurs when the primary disease proceeds in a chronic manner; in this case, the epithelium completely covers the inner surface of the pocket, displacing the periosteum. The process is initially asymptomatic, and subsequently, as the cyst enlarges, a bulge appears. On the radiograph, a rarefied area of bone tissue with a clear, rounded shape is visible; pathological changes in the apical periodontium of the teeth may be absent, which is confirmed by testing the electric excitability of the pulp. The contours of the periodontal cyst on the radiograph may be indistinct because it is superimposed on the image of the jaw body. Therefore, orthopantomography provides the best result. Treatment is surgical, with additional fixation using a pre-prepared temporary plate.



Figure 8. Periodontoma.

Treatment of Periodontal Diseases.

Treatment is based on the principle of a maximum individualized approach to each patient, taking into account data on their general and dental status. In this regard, treatment is always complex, employing both local and systemic therapy, which are inseparable from each other. Depending on the nature, cause, and mechanism of progression of the process, treatment can be:

Etiotropic (all periodontal diseases initiated under the influence of local causes); Pathogenetic (periodontitis, periodontosis); Symptomatic (idiopathic forms of periodontal disease); Or a combination of pathogenetic and symptomatic treatment elements. Treatment should be aimed not only at eliminating the inflammatory process in the periodontium but also at strengthening the general condition of the body and increasing its defensive capabilities.

Local Treatment. Regardless of the form and stage of the pathological process in the periodontium, local treatment begins with the meticulous removal of dental deposits. Dental calculus is removed using scalers or manually, for which sharp excavators and curettes are used. The removal of dental calculus is concluded with antiseptic treatment of the gingival margin. Furthermore, treatment involves eliminating defects in fillings and prosthetics, such as: removing excess filling material between the teeth, deep subgingival crowns, incorrectly designed bridge prostheses, adjusting or making new removable prostheses, timely prosthetics, and

treating various types of bite pathologies (crowding of lower frontal teeth, open and deep bite, etc.).

Gingivitis. Local treatment of gingivitis is determined by the specific features of the clinical picture (catarrhal, ulcerative, hypertrophic gingivitis).

Catarrhal Gingivitis. Treatment consists of applying the following therapeutic measures. First, the patient is taught oral hygiene skills. Dental calculus and plaque are removed; Rutin (Vitamin P), Ascorbic Acid (Vitamin C), Vitamins B, A, E are prescribed (Vitamins C, P, B can be administered using electrophoresis). Medications include 1-3% solutions of resorcinol, 2-10% solutions of zinc chloride, the Bulgarian preparation "Pinocadinen," Furin; 0.05-0.1% chlorhexidine solutions. Chlorophyllipt (a preparation made from eucalyptus leaves), 0.2% salvin solution (a preparation made from sage), romazulan, propolis tincture (1:10), 5% butadion ointment, solcoseryl, mundisal-gel, local applications of Vikasol and internal use (in cases of continuous bleeding); gingival massage, orthodontic and orthopedic treatment (according to indications), and controlling oral hygiene using anti-inflammatory and anti-exudative rinses and pastes (Rotocan, Chlorhexidine) are beneficial. It should be noted that generalized forms of catarrhal gingivitis are treated much worse. Eliminating the causes that contribute to their appearance and progression is the main and necessary condition for successful therapy.

Hypertrophic Gingivitis. In the edematous stage of hypertrophic gingivitis, the choice of treatment method depends on the etiological factors. After the removal of dental deposits, it is advisable to resort to sclerosing therapy, i.e., injections of a 50-60% glucose solution, administered into the tip of the papilla, 0.1-0.2 ml each time, with a 1-2 day break, for 3-8 times. If there is no effect after 2-3 injections, other means must be used, such as steroid ointments in the form of therapeutic dressings, for which they are mixed with artificial dentin powder; ointments containing lysozyme and heparin are successfully used. Resorcinol in 20-30% solution on a swab, Prospidin in 30-50% ointment form, zinc chloride (10-25% solutions), plantain juice, Pinocadinen are used as keratolytic agents. Orthodontic treatment is indicated if bite pathology is present. Rinses with anti-edema effect are prescribed

for home use: sodium chloride solution and chamomile decoction, and nitrofuran series preparations.

Ulcerative Gingivitis. Considering that the microbial factor is of primary importance in the etiology of ulcerative gingivitis, the success of its treatment largely depends on the local application of antibacterial preparations. Treatment begins with antiseptic treatment of the gums using hydrogen peroxide, potassium permanganate solution (1:1000), 0.05\% solution of etacridine lactate (Rivanol), 0.05-0.2\% solution of chlorhexidine, furacilin, etc. The interdental spaces are treated with swabs, after which careful removal of dental deposits begins. Appliance anesthesia in the form of aerosols or ointments based on pyromecaine or lidocaine helps to succeed. Antibiotics or their application in combination with corticosteroids in the form of applications, pastes, ointments, and therapeutic dressings give good results. Ointments "Fastin-1" and "Fastin-2" containing furacilin, synthomycin, anesthesin, as well as 3\% oktatin ointment with fungicidal and fungistatic effects, and Solcoseryl under a dressing are successfully used. Applications of the enzymes Trypsin, Chymotrypsin, preferably in combination with antibiotics, as well as Lysozyme, are indicated. Local therapy is concluded by applying a therapeutic hardening dressing. Prescribing Metronidazole (Trichopol, Flagyl) with chlorhexidine under a dressing gives a good result. Trichopol is used according to the following scheme: 0.25 g orally for 10 days (first 4 days, 0.25 g 3 times a day, the remaining 6 days, 0.25 g 2 times a day). In the treatment of ulcerative gingivitis, polyvitamins (especially in spring) and desensitizing therapy (calcium chloride, sodium thiosulfate, etc.) and general stimulating therapy (when the process is prolonged) are necessarily prescribed. Among other preparations, keratoplastic agents should be prescribed: rosehip and sea buckthorn oil, carotolin, ingalipt, aloe liniment, 10\% methyluracil ointment. Herbal preparations (romazulan, sage leaf, snake knotweed rhizome, St. John's wort, oak bark), Rotocan; combined aerosol preparations containing anesthesin, lidocaine or trimecaine, as well as methyluracil, streptocide, urea, etc.; applications of Solcoseryl-adhesive give good results. It has analgesic, anti-inflammatory, and keratoplastic effects. Ingalipt in aerosol form,

stabilized with a surfactant, containing norsulfazol, streptocide, thymol, eucalyptus and mint oils, alcohol, sugar, and glycerin, has proven effective in treating ulcerative gingivitis. Before irrigation, it is advisable to rinse the mouth with furacilin solution and remove the necrotic plaque. The preparation should be kept in the oral cavity for 5-7 minutes after irrigation. Lysozyme applications are also indicated. Among imported preparations, Mundisal-gel is prescribed for dressings. Laser therapy and a complex of vitamins along with microelements should be administered regularly. The diet should include fermented milk products, raw eggs, and fresh raw vegetables. The following sequence of therapeutic measures provides good results in ulcerative gingivitis: Appliance anesthesia; Applications of proteolytic enzymes (trypsin, chymopsin, lysoamidase) to the gums; Treatment with hydrogen peroxide using swabs in each interdental space; Chlorhexidine (0.03-0.3\% solutions) held in the mouth for 2-3 minutes; Trichopol under a dressing with Solcoseryl-gel; Laser therapy; Adherence to oral hygiene.

Gingivitis against the background of radiation sickness. Radiation damage to the oral cavity in the form of gingivitis can occur both when radiation directly affects the gum and in radiation sickness. Treatment of gingivitis occurring during radiation sickness should begin with sanitation of the oral cavity to quickly eliminate foci of infection, as gingivitis becomes very dangerous during radiation damage. If erosions are present, oily emulsions with steroids (hydrocortisone, etc.), vitamins, antibiotics with metacil are used. Dicin is indicated in case of persistent bleeding. Treatment of gingivitis in hormonal disorders begins with sanitation of the oral cavity, regardless of the stage of the process. Careful oral care is important.

Periodontosis. In periodontosis, it is necessary to use preparations and methods aimed primarily at improving blood circulation and the regenerative capacity of periodontal tissues. For this purpose, electrophoresis of Vitamin C and Vitamin B is prescribed, as well as all types of massage and laser therapy.

Periodontitis. Local treatment in periodontitis depends on the severity of the process and its individual characteristics, but anti-inflammatory therapy is always carried out before other therapeutic procedures. It is always performed after very

careful removal of plaque and dental calculus. In mild degrees of damage, antiseptics, glucocorticoids combined with antibiotics, keratolytic and fibrolytic agents are used: resorcinol (20-30\% solutions), maraslavin, 25\% zinc chloride solution, 0.1\% novoimanin solution, 1\% iodinol solution. In addition, antibiotics are used in combination with corticosteroids and metacil, ortofen is used with ethonium, biseptol-canesten-methyluracil paste is used, and physiotherapeutic treatment methods are employed, which are recommended according to strict indications and only after inflammatory phenomena have been eliminated. Other preparations used for local treatment of periodontitis include indomethacin ointment, nitrofurantoin preparations, 0.2\% sanguinarine solution, 10\% dibunol emulsion (as an antioxidant), 0.1\% novoimanin solution, biseptol, 1\% sodium mefenamate solution. 1\% dioxidine solution, 0.01\% miramistin solution; cliostom, levosin ointment. To normalize microcirculation in periodontal tissues and eliminate inflammatory phenomena in microvessels, 2\% troxevasin gel is prescribed by application (for mild and moderate periodontitis) under a dressing or film for a course of 8-10 procedures; lingezin under a dressing. In cases of deepened periodontal pockets and increased osteolysis, similar anti-inflammatory measures are taken, and surgical methods of treatment—curettage, gingivotomy, radical operations—are widely used. The emerging occlusal trauma requires orthopedic treatment, selective grinding, splinting, and prosthetics. In diseases of autoimmune genesis (kidney diseases, rheumatism and rheumatoid conditions, anemias, etc.), the presence of a constant focus of chronic periodontal infection and intoxication, where delaying tooth extraction can cause irreparable harm to the patient, is an absolute indication for the extraction of such teeth. Depulpation of teeth as a treatment method for periodontitis does not give significant clinical results, but this method is widely used before surgical and orthopedic procedures in periodontosis and periodontitis, when there is a risk of developing pulpitis and periodontitis; in the presence of certain bone pockets and when the electric excitability of the tooth pulp is reduced.

Histiocytosis. Local therapy includes removing dental plaque under appliance anesthesia, curettage of pockets, extraction of teeth in the terminal stage, and orthopedic treatment.

General Treatment

Periodontitis. For general treatment, vitamin therapy and primarily Rutin (Vitamin P) with Vitamin C are used, especially in periodontal diseases with bleeding gums in adolescence. As for other vitamins, there is currently no reliable clinical data on their use in periodontology. Immunity-stimulating and immunomodulating therapy (timalin, sodium nucleinate, timogen, levamisole, etc.) should only be used in periodontal diseases in conjunction with the relevant specialist. Antihistamine preparations (sodium thiosulfate, dimedrol, pipolfen, suprastin, tavegil) are used in periodontal diseases accompanied by an allergic component. Antibiotics are prescribed according to strict indications: when the process intensifies, especially when an abscess appears, and before and after surgery. Among antibiotics, Randomycin, Rovamycin (3-4 capsules a day) or Lincomycin 0.5 g (500,000 IU) 2 times a day in capsules 2 hours after or 2 hours before meals, with a course of 7-12 days, should be preferred. Lincomycin is rapidly absorbed and accumulates in bone tissue. Penicillin and erythromycin do not accumulate in high concentrations in the periodontal pocket when administered orally, and therefore their use is inappropriate; Doxycycline, Minocycline, Tetracycline, Amoxicillin, Metronidazole accumulate, which ensures their concentration in the periodontal pocket is 5-7 times higher than in the blood serum. Changes in the function of local protective factors, expressed in a significant deviation from the norm of the level of lysozyme, secretory immunoglobulin IgAs, and IgG immunoglobulins in the saliva of patients with periodontitis, have been identified. These indicators, as well as the qualitative and quantitative composition of the periodontal pocket microflora, taking into account the clinical picture, can be used both to assess the effectiveness of complex treatment of periodontal diseases and for prognostic purposes. Immunostimulating therapy is usually not performed in a polyclinic setting; T-activin, Levamisole, sodium nucleinate, and others are prescribed in the hospital.

Gingivitis. General treatment of gingivitis is carried out in conjunction with an endocrinologist, hematologist, allergist, or other specialist, taking into account the underlying disease (hormonal changes in hypertrophic gingivitis, blood diseases, allergic nature of the disease, disturbances in vitamin balance, and other factors).

Histiocytoses. Treatment of histiocytoses is carried out thoroughly after a general clinical examination in conjunction with other specialists, especially when hormonal preparations, protein anabolizers, antibiotics, and desensitizing therapy are being prescribed.

Surgical Treatment Methods

Surgical methods are widely used in the complex treatment of periodontal diseases, as eliminating periodontal pockets without surgical intervention is practically impossible. The following types of operations are used:

Curettage.

Removal of gingival papillae.

Gingivectomy (partial, total).

Vestibuloplasty and frenulum displacement.

Gingival plastic surgery for recession.

Gingivotomy.

Flap surgeries with the use of biogenic materials (collagen hydroxyapatite granules, tricalcium phosphate, brefocost, ostim-100, etc.) or imported preparations such as Biofix, Resolut, Bio-OSS, etc., in the form of membranes.

Even in rapidly progressing periodontitis, complex therapy can lead to stabilization of the process. Periodontal operations are performed in the operating block of the polyclinic. Gingivosteoplasty and vestibule formation are preferably performed in a hospital setting. 2 days a week are allocated for surgical procedures. Before surgery, caries and its complications are treated, supragingival plaque is removed, anti-inflammatory therapy is performed, and pus discharge from the periodontal pocket

is eliminated. Pre-operative preparation also includes immobilization of mobile teeth, selective grinding and depulpation of teeth (according to indications), premedication (seduxen 1-2 tablets 1-1.5 hours before the operation), and Trichopol (0.25 g 2-3 times for 3 days before the operation).

Curettage (scraping). It has been used in therapeutic practice for a long time. It is performed to eliminate periodontal pockets, the depth of which should not exceed 4-5 mm, as visual control of all manipulations is impossible in deeper pockets. The goal of curettage is to obtain a clean wound surface. During the operation, blood fills the pocket cavity and turns into a blood clot, which has a positive effect on the process of wound healing. The blood clot turns into connective tissue, the pocket scars, and collagen fibers grow into the newly formed root layer.

Curettage technique. After infiltration anesthesia with 1-2\% lidocaine solution, the periodontal pocket is treated in the area of no more than 3 teeth at a time. Subgingival calculus and pathologically altered cement are removed with sharp curettes, excavators, and files. All surfaces of the tooth—vestibular, approximal, and oral—are treated sequentially. When removing dental plaque, the instrument is directed from the root apex towards the tooth crown. Then the softened cement is removed until a dense consistency is reached, after which the root surface is definitively treated with a scaler; the pocket cavity is irrigated with a furacilin solution (1:5000) from a syringe to remove hard debris. Then treatment of the pocket base begins. Granulations at the bottom of the periodontal pocket are carefully scraped off with sharp instruments. If bone is detected, it is also treated.

The final stage—deepithelialization of the gingival pocket wall—is the most crucial and requires caution and skill from the doctor in performing such an intervention. The gum is pressed against the tooth with the thumb, a sharp curette is inserted to the bottom of the pocket, and under finger control, by moving the instrument towards the tooth crown, the epithelial lining is cut, thereby removing the barrier to the gum's attachment to the tooth. Then the gum is pressed against the tooth, and a protective dressing is applied. The patient is instructed on the rules of oral hygiene, a gentle diet is prescribed, and rinsing with alkaline and hypertonic solutions is

recommended. Probing the pocket is prohibited. The gingival dressing is changed every other day and kept for another 5-7 days. It is not recommended to introduce medications into the pocket so as not to disrupt the process of new connective tissue attachment and bone regeneration. Possible complications. After curettage, an inflammatory reaction may begin in the periodontal tissues, causing pain upon percussion, swelling, and sometimes lymphadenitis. Complications often arise due to rough curettage. Frequent rinsing with a salt and soda solution (one teaspoon of each per glass of water) and rinsing with a solution of Lugol (1/2 teaspoon per glass of water) or herbal infusions (sage, chamomile, etc.) are recommended. Hypersensitivity (hyperesthesia) to temperature and tactile stimuli appears after the removal of pathologically altered cement and the surface layer of dentin. Treatment consists of prescribing fluoride preparations (applications of 1-2% solutions of sodium fluoride, fluorlac, fluoride-containing gels).

Contraindications. Thin gingival walls of the pocket, the presence of bone pockets. Removal of gingival papillae (Gingivectomy). Performed for hypertrophic gingivitis and gingival fibromatosis. The operation is performed under infiltration (intrapapillary) anesthesia. An incision is made at an angle of $30-45^\circ$ to the base of the gingival sulcus, after which the overgrown tissue is removed, dental calculus is removed with sharp excavators, the wound surface is coagulated, and a gingival dressing is applied, which is changed every other day until complete epithelization.

Gingival papillae can be removed using a diathermocoagulator.

In cryodestruction of gingival papillae, a cryoprobe is inserted into the gingival sulcus, and the apparatus is turned on for 3-10 seconds; the cryoprobe is removed after thawing. Cryonecrosis begins after 24-48 hours, and regeneration of the damaged area begins after 6-12 days. Managing the post-operative period is important in cryosurgery. Protective therapeutic dressings are not applied during the first 2-3 days after the operation, until the necrotic part of the gum sloughs off.

Good oral care is necessary during the first 2 days. To accelerate lysis, proteolytic enzymes with antibiotics are prescribed, and after the necrotic tissues slough off,

medications that stimulate metabolic processes and regeneration in the gum (solcoseryl, mundisal-gel, oily solutions of vitamin A and A+E, kerotolin, sea buckthorn oil) are prescribed under a dressing. After cryosurgery, the wound heals 2-3 times slower than when other methods are used.

Splinting and Prosthetics. The unification of a group of teeth into a single block using special orthopedic appliances is called splinting. In this way, the mobility of individual teeth is reduced, and the chewing load is evenly distributed across all remaining teeth. Various designs of removable and non-removable splints exist. According to the duration of action, they are divided into temporary and permanent. Besides immediate prostheses, wire splints, as well as various designs made of fast-curing plastics, can be used as temporary prostheses.

Permanent Splints. Permanent splinting and prosthetics are carried out to achieve long-term immobilization of mobile teeth and an even distribution of the chewing pressure over the remaining teeth and alveolar processes.

Temporary Splints

These splints are placed for a short period—from several weeks to several months. Splinting is performed to immobilize teeth during therapeutic and surgical treatment, which improves conditions for tissue healing and consolidation of the therapeutic effect. Temporary splints are also used after orthodontic treatment, i.e., as retention appliances. Furthermore, such splinting helps preserve the remaining teeth during the period of preparation of the oral cavity for prosthetics. For this purpose, so-called immediate splints can be used as temporary splints, which allow for the restoration of soft tissues, the unity and integrity of the dental arches, the elimination of aesthetic defects, and the normalization of chewing and speech function. Non-removable and removable constructions can be used as permanent splints. Non-removable splints include cap splints, intradental para-pulpal, double and pin splints, soldered semi-crowns, equatorial or full crowns, bar splints, and inlay splints combined with equatorial crowns. The latter four types of splints are used to immobilize the posterior teeth. Non-removable splints provide reliable fixation of mobile teeth and equalize vertical, anteroposterior, and transversal occlusal functional loads (forces).

They normalize the chewing function and do not impair speech. The disadvantages of these splints include the necessity of preparing the hard tissues of the teeth. Furthermore, non-removable splints worsen the hygienic status of the oral cavity. The manufacture of some types of such splints (pin, intradental) requires complex equipment, tools, and materials. Splints made of soldered crowns provide reliable stabilization of mobile teeth but irritate the tissues when they touch the gums, aggravating the course of the process and hindering therapeutic treatment. The use of these splints should be limited. The displacement of the crown edge under the gingival margin is unacceptable. Constructions that leave the necks of the teeth free and do not interfere with therapeutic and surgical treatment should be preferred.

Removable Splints. Removable splints do not have such disadvantages, but they cannot provide rigid immobilization of mobile teeth in all three directions: vertical, transversal, and mesiodistal. When these splints are used, the autonomous vertical movement of the teeth is possible, which to a certain extent reduces their splinting properties. Nevertheless, removable splints with retaining elements relieve mobile teeth from mesiodistal and transversal (horizontal) occlusal forces, which have a more harmful and destructive effect on periodontal tissues and the course of the pathological process. In this regard, some constructions of removable splints and splint-prostheses are quite widely used in dental practice. Removable splints include splints with a multiple-clasp incorporated into the design; cast metal caps for the anterior teeth; a cast splint for the entire dental arch with a bar, claw-like overlays, or forming elements for immobilizing the anterior teeth.

Prosthetics

Prosthetics of teeth in periodontosis and periodontitis is performed with bridge-like (non-removable) and removable prostheses of various designs. Often, splinting elements (splint-prostheses) are included in the design of such prostheses. The use of cantilever non-removable prostheses, porcelain overlays, all-cast metal-ceramic, and metal-acrylic prostheses is limited in periodontal diseases and tooth mobility. Among removable prostheses, clasp prostheses with various fixing elements, including all-cast plates (in Kennedy Class IV defects), are widespread. Such

prostheses can replace defects of various lengths and topographies. The design of clasp prostheses includes multiple-clasps, occlusal rests, claw-like, and other fixing elements. In periodontal diseases, the indications for including both fixing elements and occlusal rests in the design of clasp prostheses are broadened to distribute the chewing pressure as much as possible over the remaining teeth and reduce their overloading in centric, anterior, and lateral occlusions. When clasp prostheses are used, the gingival margin is left free both on the vestibular and oral sides, which allows for unimpeded therapeutic treatment when necessary. The use of prostheses with such a design allows for the grafting of artificial teeth after loose teeth have been extracted. Splinting of the remaining teeth and prosthetics of dental arch defects in periodontitis and periodontosis can also be carried out by combining non-removable and removable splint-prostheses. In this case, the main requirements remain: to ensure reliable immobilization of mobile teeth, to eliminate overloading of the periodontium, to distribute the load to the teeth and the prosthetic bed mucosa, to eliminate additional irritation of the marginal periodontal tissues by splint-prostheses, and to create optimal conditions for therapeutic and surgical treatment.

Prognosis. All forms of Gingivitis often have a good prognosis. Stabilization of the process with supportive therapy and adherence to oral hygiene should be considered successful treatment for generalized periodontitis. Rapid progression (continuous bleeding, increasing tooth mobility, increasing depth of exudation from the periodontal pocket) is observed in patients with severe diseases: blood disorders, metabolic disorders; adverse effects of medications (hydantoin, cyclosporine), syndromes related to hereditary causes (Down syndrome), Papillon-Lefèvre Syndrome, etc.; and rapidly progressing periodontitis in adults. Treatment fails if patients do not comply with oral hygiene rules.

Anatomy and Histology of the Oral Mucosa

Anatomy of the Oral Mucosa (OM). The oral cavity is divided into the oral vestibule and the proper oral cavity. The oral vestibule is bounded anteriorly by the lips and cheeks, and posteriorly by the vestibular surfaces of the teeth and the alveolar processes of the jaws. The proper oral cavity itself is bounded anteriorly by the teeth

and alveolar processes, superiorly by the hard and soft palate, and inferiorly by the floor of the mouth. The mouth is a collection of organs and tissues: the tongue, lips, teeth, salivary glands, periodontium, and jaws. The oral vestibule and the proper oral cavity are lined with mucous membrane. The Oral Mucosa (OM) is frequently subjected to mechanical trauma from hard foods, temperature changes, biting, and microorganisms. Consequently, one of the main functions of the oral mucosa is protection. This is ensured by the constant renewal of the multilayered epithelium and its high regenerative capacity. The surface of the mucosa is moistened with saliva, produced by the salivary glands. Saliva moistens and softens food, and to some extent prevents mechanical damage to the mucous membrane. Furthermore, saliva contains antimicrobial substances, antibodies, nerve system factors, and epidermal growth factor. The mucous membrane participates in the body's immune reactions.

Relief of the Mucous Membrane. The surface of the oral mucosa is mostly smooth, but there are transverse folds on the hard palate. The epithelium of the oral mucosa is stratified squamous. Its thickness varies greatly in different areas. The tendency of the epithelium to keratinize in humans is observed in the area of the hard palate and gingiva, along the "white line" where the teeth meet in the cheeks, as well as on the upper surface of the tongue—in the filiform papillae. The oral cavity is the initial part of the human digestive system. According to the distinct local characteristics of the structure, the oral mucosa is divided into three types: Masticatory, Lining (Covering), and Specialized. This division is very convenient because it allows for connecting the structural features with the function performed by different parts of the oral cavity. It is used for the consumption of food products and their primary processing (including mechanical grinding during chewing and the initial stage of digestion, during which the polysaccharides in the food are broken down under the action of amylase and maltase present in saliva). As a result, a bolus of food is formed, which enters the esophagus through the pharynx. The oral cavity participates not only in the process of eating but also in the processes of breathing and speech. The epithelium is stratified squamous, with a thickness of up to 500 μm

(micrometers). The deepest layer of cylindrical or cuboidal cells located on the basement membrane is called the basal layer. The protoplasm of the cells in this layer is often characterized by basophilia depending on the content of ribonucleic acid. Next is the prickle cell layer, consisting of several rows of polygonal cells with lighter protoplasm and intercellular bridges. Approaching the surface, these cells flatten and become squamous, and they are constantly shed and mixed with saliva. The basis of epithelial regeneration and the transitional folds consists of the submucosa. The epithelium of the mucous membrane of the hard palate and gingiva, i.e., the parts most susceptible to mechanical stress during chewing, is keratinized. Above the prickle cell layer is the granular layer, consisting of elongated cells whose protoplasm contains keratohyalin granules. The superficial "horny" layer consists of several rows of fully keratinized and anuclear cells.



Histology of the Oral Mucosa (OM)

The oral mucosa, unlike other mucous membranes of the human body, has a number of features. Due to the increased regenerative capacity of the OM, it is resistant to the effects of physical, thermal, chemical factors, and the introduction of infection. The oral mucosa is covered with stratified squamous epithelium with a thickness of 180-600 μm , consisting of several cell layers, and is characterized by the absence (or weak development) of the muscular layer, as well as the absence of a submucosa in some places; in such cases, the mucous membrane lies directly on the bone (in the gingiva and hard palate) or on the muscles (in the tongue and soft palate) and is firmly fused with the underlying tissues. The pink color of the mucous membrane is characterized by the presence of numerous capillaries. The OM consists of 3 layers—epithelium, propria lamina (lamina propria), and submucosa. The epithelium is stratified squamous, with a thickness of up to 500 μm . The deepest layer of cylindrical or cuboidal cells located on the basement membrane is called the basal layer. The protoplasm of the cells in this layer is often characterized by basophilia depending on the content of ribonucleic acid. The next layer is the prickle cell layer, consisting of several rows of polygonal cells with lighter cytoplasm and

intercellular bridges. Fibrous structures are represented by collagen and argyrophilic fibers, with the most collagen fibers being located in the mucous membrane of the gingiva and hard palate. Cellular elements are mainly represented by fibroblasts, macrophages, cells, plasma cells, and histiocytes (immobile macrophages).



Figure 9. Epithelium, Lamina Propria, and Submucosa.

The cells of the prickle cell layer flatten and become squamous as they move towards the surface; they are constantly shed and mixed with saliva.

Stratified Squamous Keratinized Epithelium

The schematic representation of the stratified squamous keratinized epithelium of the oral mucosa can be expressed as follows: its own lamina propria, basement membrane, prickle cell layer, granular, and horny layers of cells. The epithelium of the hard palate and gingiva mucosa is the most susceptible to mechanical stress during chewing and therefore becomes keratinized. The process of keratinization is particularly pronounced in the epithelium of the hard palate. Above the prickle cell layer is the granular layer, consisting of elongated cells whose protoplasm contains keratohyalin granules. The superficial "horny" layer is formed by several rows of fully keratinized and anuclear cells. The basal layer (stratum basale) is formed by keratinocytes, melanocytes, Merkel cells, and Langerhans cells. Keratinocytes have a prismatic shape. Organelles and keratin intermediate tonofilaments are detected in their cytoplasm. The basal layer contains the root cells of the keratinocyte differon

and dividing keratinocytes, due to which the physiological renewal (regeneration) of the epithelium occurs.

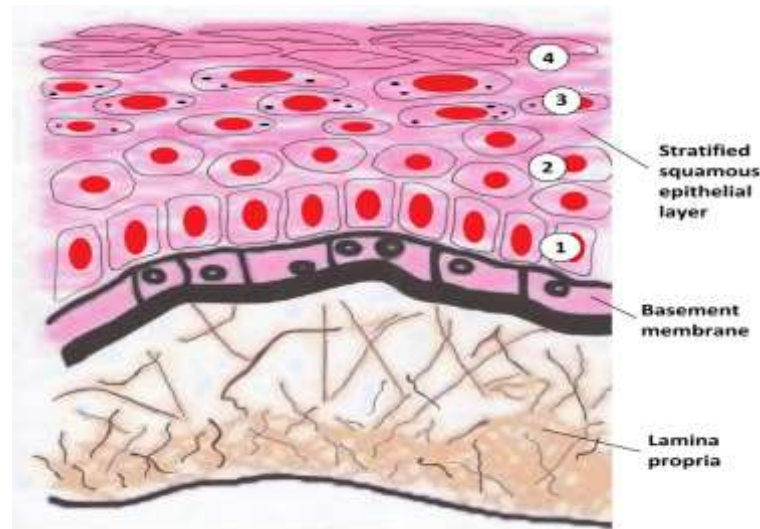


Figure 10. Diagram of the structure of stratified squamous keratinized epithelium.

The cells of the prickly cell layer flatten and become squamous as they move towards the surface; they are constantly shed and mixed with saliva.

Stratified Squamous Keratinized Epithelium

The schematic representation of the stratified squamous keratinized epithelium of the oral mucosa can be expressed as follows: its own lamina propria, basement membrane, prickly cell layer, granular, and horny layers of cells. The epithelium of the mucous membrane of the hard palate and gingiva is the most susceptible to mechanical stress during chewing and therefore becomes keratinized. The process of keratinization is particularly pronounced in the hard palate epithelium. Above the prickly cell layer is the granular layer (stratum granulosum), consisting of elongated cells whose protoplasm contains keratohyalin granules. The superficial "horny" layer (stratum corneum) is formed by several rows of fully keratinized and anuclear cells. The basal layer (stratum basale) is formed by keratinocytes, melanocytes, Merkel cells, and Langerhans cells. Keratinocytes have a prismatic shape. Organelles and keratin intermediate tonofilaments are detected in their cytoplasm. The basal layer contains the stem cells of the keratinocyte differon and dividing

keratinocytes, due to which the physiological renewal (regeneration) of the epithelium occurs.

The prickle cell layer (stratum spinosum) is represented by keratinocytes forming 5-10 layers. These cells are large in size, polygonal in shape, and connected to each other by desmosomes. Keratin synthesis and the formation of tonofilaments from it are developed in their cytoplasm; these are united into bundles—tonofibrils. Keratinosomes or lamellar granules are formed in the cells of the prickle cell layer. They are bounded by a membrane cluster containing lipids and hydrolytic enzymes. The granular layer (stratum granulosum) forms 2-4 layers of flattened keratinocytes, in which a number of proteins are synthesized: keratin, filaggrin, involucrin, keratolinin. Filaggrin participates in the aggregation of keratin tonofilaments, forming an amorphous matrix between them. They combine with proteins, polysaccharides, and lipids and are released when the disintegration of the nuclei and organelles of keratinocytes begins there. As a result, a complex compound—keratohyalin—is formed. Under a light microscope, it is detected in the form of large basophilic granules. Involucrin and keratolinin form a protein layer under the plasmalemma, protecting it from the action of hydrolytic enzymes. The number of keratinosomes in keratinocytes gradually increases; they are released into the intercellular spaces via exocytosis. The lipids in the keratinosomes form a cementing substance that creates a water-impermeable barrier in the epidermis.

The horny layer (stratum corneum) consists of keratinocytes that have completed their differentiation and lost their fragment structure, called the horny scale. They have a flat polygonal shape, arranged in columns. The scale is covered with a thick shell containing the protein keratolinin. The inner part of the scale is filled with longitudinally oriented keratin fibrils. Due to the processes of proliferation, differentiation, and movement from the lower layers, a number of mature scales that have lost their cellular structure are replaced by new ones during the process of keratinization. Due to these processes, the composition of keratinocytes is renewed. Bundles of collagen fibers are most densely located in the mucous membrane of the gingiva and hard palate, which are subjected to the greatest mechanical stress—

chewing. The epithelium is connected to its lamina propria through the basement membrane, which consists of a dense network of argyrophilic fibers closely connected with the cytoplasmic processes of the basal layer cells of the epithelium. The processes of proliferation and keratinization constantly occur in the keratinized epithelium. The described type of keratinization is called orthokeratosis (Greek orthos - true). In some areas of the epithelium where the greatest pressure occurs during chewing (hard palate, gum), a type of keratinization called parakeratosis (Greek para - about) occurs. With parakeratosis, there are cells on the surface of the formation that contain keratin, but they retain pyknotic nuclei, remnants of organelles, and the granular layer is poorly expressed. Parakeratosis in the oral cavity is considered a physiological phenomenon.

Stratified Squamous Non-Keratinized Epithelium

The mucous membrane in the area of the cheeks (excluding the line of tooth closure), lips, soft palate, transitional folds of the oral vestibule, as well as the floor of the mouth and the lower surface of the tongue do not keratinize. The stratified squamous non-keratinized epithelium consists of 3 layers: 1) basal, 2) prickly cell, 3) surface. However, the layer of non-keratinized epithelium in the oral cavity is often much thicker than the layer of keratinized epithelium. The basal layer is structurally similar to the basal layer of the keratinized epithelium. The prickly cell layer is represented by large polygonal epithelial cells. Tonofilaments located in the cytoplasm do not form large bundles and are scattered. Keratinosomes are rounded in shape. Keratohyalin accumulates in the superficial layers of the prickly cell layer. The surface layer is formed by flattened cells containing cytokeratin filaments. The cell plasmalemma is thickened. Cell nuclei are usually dark. The cells of the stratified squamous non-keratinized epithelium participate in the production of substances with antimicrobial effects (calprotectin, etc.). The basement membrane is located between the epithelium and the lamina propria of the oral mucosa. The lamina propria mucosae can be divided into 2 layers: a) papillary layer, formed by loose fibrous connective tissue, and b) reticular layer, represented by dense fibrous unformed connective tissue. The height of the papillae and the degree of expression

of these layers vary, and sometimes the layers are not expressed. The muscular layer of the mucosa (lamina muscularis mucosae), which separates the lamina propria from the submucosa, is usually absent in the oral cavity.

The submucosa (tela submucosa). Due to the absence of the muscular layer, the lamina propria, without a sharp boundary, transitions into the submucosa, consisting of loose connective tissue. The submucosa is not found in the area of the hard palate, the dorsal surface of the tongue, and the gingiva. In these areas, the mucous membrane is fused with the layers of connective tissue located between the muscles or the periosteum of the corresponding bones. The submucosa often accumulates fat cells and small salivary glands.

Epithelial regeneration in the non-keratinized layer occurs due to cell division in the deep layer. Such an epithelial structure is characteristic of the mucous membrane of the cheeks, lips, soft palate, transitional folds of the oral vestibule, as well as the floor of the mouth and the lower surface of the tongue. The epithelium of the listed areas of the oral mucosa does not keratinize. It should be noted that a specific feature of the epithelial cells of the human oral mucosa is their large size, which is especially noticeable in the mucous membrane of the cheeks and lips. The thickness of the epithelium varies in different areas. The epithelium on the lips and cheeks is the thickest; thin epithelium covers the lower surface of the tongue. Bundles of fibers of the orbicularis oris muscle lie in the thickness of the lips, which is responsible for closing the mouth and protruding the lips forward. The walls of the cheeks contain the buccinator muscle, which is responsible for pulling the corners of the mouth to the sides and pressing the cheeks to the teeth. The red border of the lips has a stratified squamous epithelium, which is a zone of keratinization, and wide capillary loops of the vessels come very close to the surface and shine through the epithelium. The stratified squamous epithelium of the red border of the lips keratinizes, and this process is accompanied by the formation of eleidin, an intermediate product of the transformation of keratohyalin into keratin. The mitotic activity of the oral mucosa is higher than that of the skin epidermis, and mitoses are particularly pronounced in the non-keratinized areas of the mucosa. These changes are also detected in

pathological processes. Therefore, the degree of mitotic activity of the epithelium is one of the severity indicators of the protective processes of the oral mucosa. Histochemically, it has been found that the oral epithelium can accumulate large amounts of glycogen, most of which is in the epithelium of the lips, cheeks, soft palate, transitional folds, and tongue. In the epithelium of the hard palate and gingiva, glycogen is either normally absent or present in insignificant amounts. Thus, glycogen is most abundant in the non-keratinized parts of the epithelium. Probably, glycogen plays an important role as an energy source or plastic material for protein synthesis—keratin. Therefore, it is rapidly consumed and is not detected in keratinized areas. The presence of glycogen is a characteristic feature only of the human oral mucosa epithelium. Nucleic acids also play an important role in the vital activity of the epithelium; their largest amount is present in the cells of the epithelial growth zone, which proves the intensity of metabolism in these cells.

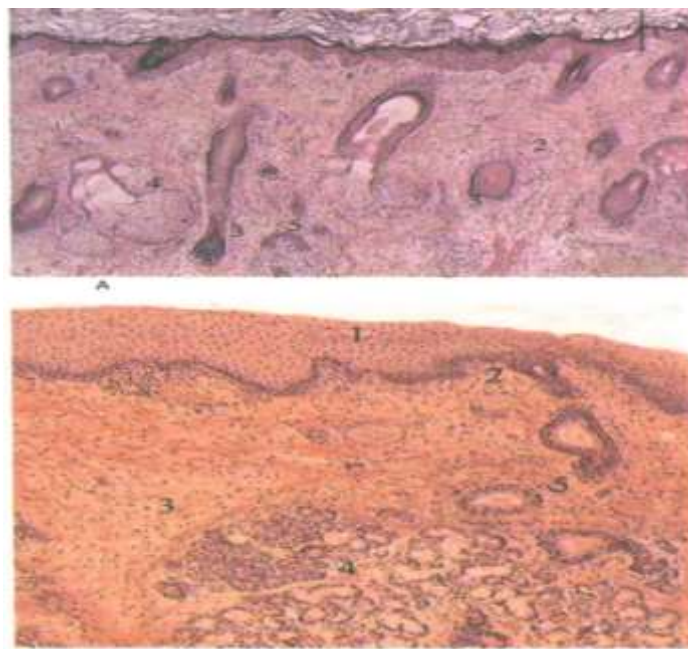


Figure 11. A-Skin of the lip; B-Mucous membrane of the lip.

The connective tissue, which is the basis of the OM epithelium, infiltrates the epithelium and forms the lamina propria (propria layer). It consists of dense connective tissue and forms numerous protrusions—papillae—that interdigitate with the epithelium. Blood vessels that nourish the epithelium and nerves pass through them. The papillae increase the contact surface between the lamina propria and the epithelial layer, which improves metabolism between them. The height of

the connective tissue protrusions is proportional to the thickness of the epithelium. The highest ones are observed in the area of the lips, cheeks, and the back and tip of the tongue. The lamina propria transitions into the submucosa without a sharp boundary. In some areas of the oral cavity, the submucosa is completely unexpressed (tongue, gingiva, hard palate, and lateral parts of the palatine raphe). Here, the mucous membrane is fused with the intermuscular connective tissue (in the tongue) or with the periosteum (in the hard palate and gum). The submucosal layer consists of loose connective tissue, where fat tissue is also present along with bundles of collagen fibers. This layer is absent in the mucous membrane of the tongue, gingiva, and hard palate. The interdental papillae of the gingiva are formed by loose connective tissue, through which capillaries and sensory branches of nerve endings pass. The epithelium of the hard palate and gingiva mucosa has a pronounced keratinization. Above the prickle cell layer is the granular layer, consisting of elongated cells with keratohyalin granules in the protoplasm. The superficial layer of the hard palate epithelium is formed by several rows of fully keratinized and anuclear cells. The process of epithelial keratinization is protective in nature and is observed in areas of the OM that receive large chewing pressure.

It has been established that the number of leukocytes in the underlying connective tissue is low in the horny layer and in the non-protected area of the mucous membrane; the number of leukocytes is very high, which is the protective function of the OM. The epithelium is closely connected to the lamina propria of the mucous membrane through the basement membrane and consists of numerous protrusions (papillae) embedded in the epithelium. Epithelial renewal occurs due to cell division in the deep layers. The epithelium of the mucous membrane of the cheeks, lips, soft palate, transitional folds of the oral vestibule, the floor of the mouth, and the lower surface of the tongue does not keratinize. The mucous membrane of the floor of the mouth, transitional folds, lips, and cheeks has a well-developed submucosa and is loosely attached to the underlying tissues, which ensures their mobility.

Structural Features of the Tongue Mucosa

The mucosa of the tongue differs in structure from other areas; it is firmly attached to the intermuscular connective tissue, and the submucosa is absent. The dorsum of the tongue is covered with irregularly keratinized stratified squamous epithelium; the mucous membrane here is thickened and rough. The lamina propria forms high protrusions—lingual papillae—covered with epithelium.

There are 4 types of papillae on the surface of the tongue: filiform, fungiform, circumvallate, and foliate. Taste buds are located in the mucous membrane of the tongue—the human taste organs. Food particles dissolved in saliva come into contact with taste receptors, passing through small openings in the epithelial mucosa—the taste pores. The taste bulb has an oval shape and consists of the following types of cells: Sensory epithelial cells are elongated, spindle-shaped cells. Mitochondria have microvilli on their apical surface. Between the microvilli is an electron-dense substance with a high content of specific receptor proteins: sweet-sensitive, acid-sensitive, salt-sensitive, and bitter-sensitive. Sensory nerve fibers approach the lateral surface of the sensory epithelial cells and form receptor nerve endings. Supporting cells are curved spindle cells that protect and surround the sensory epithelial cells. Basal epithelial cells are poorly differentiated cells for the restoration of cells 1 and 2. These cells are renewed every 10 days. The apical surfaces of the taste bud cells form taste pits that open into the taste pores. Substances dissolved in saliva enter the taste pits, are absorbed by the electron-dense substance between the microvilli of the taste-sensitive cells, and act on the receptor proteins of the cell membrane, which leads to a change in the electric potential difference between the inner and outer surface of the plasmalemma, i.e., the cell enters a state of excitation, which is governed by the nerve endings.

Filiform papillae on the surface of the tongue: 1-Epithelium; 2-Lamina Propria.

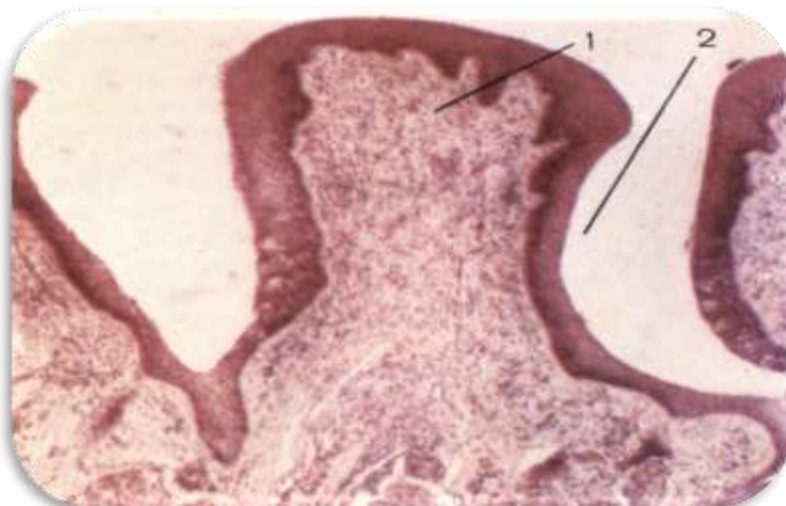
Filiform papillae are the most numerous and have a tightly packed cone shape, which gives the tongue a velvety appearance. Filiform papillae perform a tactile (mechanical) function. The epithelium covering the filiform papillae is stratified squamous keratinized epithelium; the shedding of the surface layer of the filiform papillae epithelium is an expression of the process of physiological regeneration.

When the function of the digestive organs is disturbed, in inflammatory, especially infectious diseases, the shedding of the surface layer of the filiform papillae epithelium is inhibited, and consequently, the tongue becomes "coated".



Figure 12. Fungiform Papillae of the Tongue: 1-Epithelium; 2-Lamina Propria.

Fungiform papillae are less numerous than filiform papillae. This papilla is covered with stratified squamous non-keratinized epithelium. Fungiform papillae are organs of taste, and the majority of them are located at the tip of the tongue.



**Figure 13. Circumvallate Papillae of the Tongue:
1-Papillae; 2-Circumvallate (Groove/Trench)**

Circumvallate papillae are 8-12 in number and are located at the border between the root and the body of the tongue, close to each other, forming a Roman letter V shape.

They are also organs of taste and protrude above the mucosal surface. Each papilla is separated from the surrounding mucosa by a deep trench (groove), where the small serous-proteinaceous glands of Ebner open. The papillae epithelium contains taste buds, which contain a large amount of acetylcholinesterase and acid phosphatase, highlighting their significance in taste transduction.



Figure 14. Foliate Papillae on the Lateral Surface of the Tongue:
1-Epithelium; 2-Lamina Propria.

Foliate papillae are 2-5 mm long, shaped like 3-8 parallel folds, and are located on the lateral surface at the base of the tongue. The papillae epithelium also contains taste buds. Numerous small salivary glands are located in various areas of the Oral Mucosa (OM). According to their secretory nature, they are divided into mucous, serous, and mixed. Most small salivary glands are located in the submucosa of the lips and soft palate. In addition, the excretory ducts of the three major salivary glands—the parotid, submandibular, and sublingual salivary glands—open onto the surface of the OM.

Physiology of the Oral Cavity

Saliva is the secretion of the salivary glands into the oral cavity. It is necessary to distinguish between the saliva secreted by the major salivary glands (parotid, sublingual, and submandibular) and mixed saliva, or oral fluid, which is the saliva collected from the excretory ducts mixed with the microflora, gingival fluid,

breakdown products of plaque microflora waste, food debris, and leukocytes that migrate with the saliva.

Saliva is produced by the parotid, sublingual, and submandibular glands, and the small salivary glands located in the submucosa of the lips, tongue, and palate. Small salivary glands are absent in areas subject to strong mechanical stress during chewing. The salivary glands consist of terminal sections and excretory ducts; saliva is mainly formed in the secretory terminal sections and undergoes secondary changes in the duct system. The secretion of all small and large glands forms saliva. Approximately 1.5 liters of saliva are produced and secreted into the oral cavity per day. In addition to water, mucus, and protein, saliva contains 0.2\% inorganic substances, mainly salts of calcium, potassium, and sodium, the concentration of which is several times higher than in the blood. It contains trace elements—iron, copper, manganese, nickel, lithium, and others. Organic substances are mainly represented by albumins, globulins, and enzymes. Various vitamins are also present in saliva. The following main groups of proteins are distinguished in oral fluid: Proline-rich proteins bind with streptococci, and also protect the OM by binding food tannins from harmful effects, give saliva viscosity, and contribute to the formation of the dental pellicle. Histidine-rich proteins exert a bactericidal effect and affect glucose transport and glycolysis reactions in microorganisms (*Str. mutans*, *Candida albicans*). Tyrosine-rich proteins prevent the nucleation and growth of Ca and P salts and participate in the restorative protective function in healthy teeth during the formation of tooth enamel. Albumins—mainly enter the saliva from the gingival fluid; their number increases with stomatitis, diseases of the gastrointestinal tract, and other diseases.

Salivary glycoproteins determine viscosity.

Mucin moistens the OM and teeth, protects them from damage, and is capable of adsorbing onto the surface of teeth, forming an insoluble film. It has high viscosity, is elastic, and participates in the formation of pellicles.

The activity of more than 10 enzymes is detected in mixed saliva, which are divided into 3 groups by origin:

From the parenchymal salivary glands;

Formed during the enzymatic activity of bacteria;

Formed as a result of the breakdown of leukocytes in the oral cavity.

More than 50 enzymes related to hydrolases, transferases, lipases, and isomerases have been found in saliva. Among salivary enzymes, α -amylase should be highlighted, which partially hydrolyzes carbohydrates in the oral cavity, converting them into dextrans and maltose. Salivary lysozyme is of particular interest, which has a bacteriolytic, anti-inflammatory, antihistamine, and hemostatic effect, enhances the action of antibiotics, and accelerates reparative processes in the oral tissues. The "immune" bodies in saliva ensure the local immunity of the oral cavity, especially S-IgA (Secretory Immunoglobulin A), which prevents bacteria from adhering to epithelial cells, making their colonization on the mucous membrane difficult. Immunoglobulin IgM is present in saliva in smaller quantities, is synthesized locally, and is selectively secreted. Immunoglobulin IgG comes from the blood serum through the gingival fluid, as well as with the secretion of the parotid salivary glands. The absolute IgG content can change significantly with local inflammatory processes and increased vascular permeability. Saliva entering the oral cavity from the excretory ducts of the salivary and mucous glands mixes with shed epithelial cells, microorganisms, neutrophilic leukocytes, and sometimes lymphocytes, turning into oral fluid. The composition of saliva also changes depending on the composition of the consumed food, the influence of the external environment, and the state of the body. Normally, the reaction of the oral fluid is slightly alkaline (pH $\approx$ 6.9). The value of the hydrogen index changes depending on the nature of the pathological process in the oral cavity: the saliva reaction becomes acidic in infectious diseases. Changes in the composition of saliva lead to the appearance of dental calculus, which in turn leads to the onset of gingivitis.

Sensory Function of the Oral Cavity

The OM contains sensory receptors that provide taste, pain, cold, heat, tactile, and other sensations. Taste buds are mainly embedded in the papillae of the tongue.

Tactile sensitivity is most pronounced on the red border of the lips and especially on the tip of the tongue. Tactile receptors provide information about the consistency and shape of the food substance, regardless of its chemical properties. Pain sensitivity is less expressed and is better in the palatine arches, soft palate, and oral vestibule. Temperature sensitivity varies in different parts of the mucous membrane; for example, it is completely absent on the floor of the mouth and the gingiva. The perception of cold receptors is better developed than that of heat receptors. Depending on which areas of the OM are irritated, corresponding reflex changes occur, for example, vascular reactions. Thus, when the taste buds are irritated by sweet substances, their vessels dilate, and bitter substances cause them to constrict. The nature of the excitation of oral receptors affects gas exchange and muscle function. The physiological properties of the OM are also manifested in the regenerative capabilities of the epithelium. A large number of squamous epithelial cells are shed during the day. The renewal of epithelial cells occurs as a result of mitosis of the cells of the basal and prickle cell layers. The rate of renewal of the OM epithelium exceeds the proliferation rate of the epidermis. It is known that wounds in the oral cavity heal much faster than skin wounds. The increased regeneration of the OM is due to the early appearance of glycogen in it, an increase in the RNA content, and the accumulation of acid mucopolysaccharides. The faster healing of the OM compared to the skin is also explained by the presence of poorly differentiated cellular elements in the mucous membrane. OM fibroblasts are less differentiated than skin fibroblasts and have the appearance characteristic of young cells. Blood clotting factors and fibrinolytic enzymes play a leading role in reparative processes. The physical strength of the OM and its turgor are determined by its ability to withstand pressure, compression, and stretching. The hydrophilicity of the mucous membrane, its physical tension, elasticity, durability, and ability to be stretched determine its turgor, which does not change and does not depend on the thickness of the submucosa, but undergoes age-related changes. The stretching ability of the OM depends on the state of elastic and collagen fibers. Its physical strength in newborns is less than in young and mature individuals. The buffering

capacity of the OM is its ability to neutralize the action of acids and alkalis and quickly restore the pH of the oral environment. This depends on the presence and thickness of the horny layer and the secretion of the salivary glands and changes during pathological processes in the oral cavity. The buffering capacity (the ability to neutralize acid and alkali) is provided by three main systems: bicarbonate, phosphate, and protein. Bicarbonates provide 80\% of the buffering property of saliva; their concentration in saliva increases in direct proportion to the secretion rate. The buffering capacity can vary and depends on the nature of the diet, the time of day, and the state of the gastrointestinal tract. The second most important is the phosphate system, and the third is the protein system. Saliva consists of 99.42\% water and 0.58\% organic and inorganic substances. Inorganic components in saliva include calcium salts, phosphates, potassium and sodium compounds, chlorides, bicarbonates, fluorides, thiocyanates, and others. The concentration of Na in saliva is usually much lower than in the plasma of the rest of the gland, but increases with an increase in the rate of salivation. The OM has an assimilative capacity that varies in different areas and for different substances. This property is used for taking some medicines; for example, validol is better absorbed by the mucous membrane on the floor of the mouth. It should be remembered that healthy mucous membrane absorbs medicinal substances faster than pathologically altered one. Thus, the barrier function of the OM is determined by various anatomical and functional factors. These include: the unevenness of keratinization, the metastatic activity and high regenerative capacity of epithelial cells, the activity of metabolic processes, the accumulation of glycogen, the presence of numerous cellular elements in the lamina propria of the mucous membrane and the migration of leukocytes into the oral cavity, the bactericidal action of salivary components, the selective absorption capacity, and the physical strength of the OM, and others.

Sensory Function of the Tongue

The sensory function of the oral mucosa causes the emergence of sensations that arise under the influence of various environmental effects. Of the simple five senses—sight, hearing, touch, taste, smell—only taste and touch belong to true

sensory functions. OM receptors are divided into somatosensory (tactile, thermal, cold) and chemoreceptors (gustatory). The study of OM receptors showed that tactile receptors give nerve impulses that are registered after a short latency period. These impulses spread quickly through the nerve fibers.



Figure 15. Weber's Compass (Aesthesiometer)

Determining Tactile Sensitivity

Tactile receptors provide information about the consistency and shape of the food substance upon contact, regardless of its chemical properties. Discriminatory sensitivity (spatial touch sensitivity) is determined using Weber's compass (aesthesiometer) as the minimal distance between two points at which these stimuli are perceived separately. Tactile receptors are most numerous on the tip of the tongue (discrimination threshold – 1 mm), and least numerous on the back of the tongue (discrimination threshold – 2.4 mm). The legs of the compass are moved, and the legs touch the tip of the tongue with equally strong pressure. At the same time, the subject is asked how many touches they feel. If the answer is "one," the legs of the compass are moved another 1 mm apart until the subject clearly perceives two touches. The distance at which the subject felt two touches is measured with a ruler. This will be the threshold of excitation. This is followed by impulses from temperature receptors, which report the "warmth" level of a particular food substance to the central nervous system. Impulses from the chemoreceptors of the tongue appear the latest. The receptor parts of the sensory systems of the tongue

represent a strong reflexogenic zone, from which reflex reactions of various body systems begin.

The tongue, as a sensory organ, is a polymodal sensory formation. The mucous membrane of the tongue is a complex receptive field that provides the perception of taste, temperature, and pain. The fact that the tongue is a muscular organ plays an important role in the sensory function. In humans, it is highly mobile, easily changing its shape and position in the oral cavity. Due to such motor activity, the tongue is the first to come into contact with the stimulus. Furthermore, the tongue moves food in the oral cavity during chewing and provides the acts of chewing, swallowing, and sucking. The participation of the motor component in taste perception is confirmed by the presence of contractile elements in the taste buds and even in the taste papillae. The tongue is a multifunctional organ because it not only performs a sensory function but also participates in the functions of digestion and speech formation. One of the functional elements of the tongue is the sensory functional element of the taste organ (taste papillae). With the normal activity of the tongue receptors, the following reflexes are correctly formed:

Salivary reflexes;

Secretion of gastric juice;

Secretion of digestive enzymes by the pancreas;

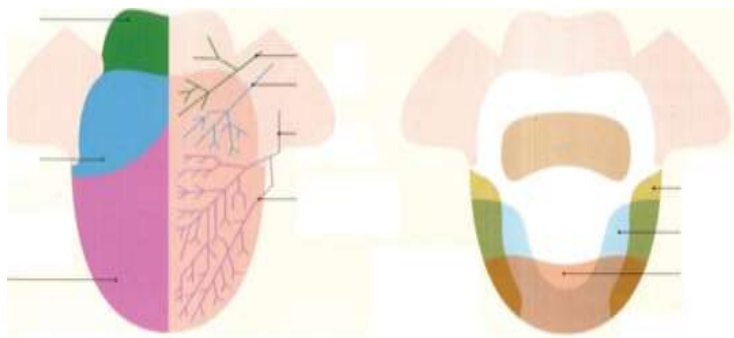
Initiation of chewing and swallowing movements.

The influence of tongue receptors on the cardiovascular system is described: when rinsing the oral cavity with solutions of sweet substances, the vessels reflexively dilate, and sour and bitter excretions constrict the vessels. Under the influence of impulses from the tongue receptors, metabolism, muscle tone, and the function of hematopoietic organs change. The receptors for tactile sensitivity on the tongue are the filiform papillae. They have a conical appearance, are tightly packed, and therefore the surface of the tongue has a velvety appearance. The epithelium covering the filiform papillae keratinizes. The shedding of the surface layer of the filiform papillae epithelium is an expression of the physiological process of regeneration. In case of impaired function of the digestive organs and inflammatory

processes, a "coating" on the tongue appears, which slows down tactile perception and adversely affects the systemic reactions of the body. There are no taste buds in the filiform papillae. Temperature receptors are mainly located in the anterior part of the tongue. The central part of the posterior surface of the tongue does not perceive the effect of cold or heat. Foliate papillae, in the form of 3-8 parallel folds, are located at the base of the lateral surface of the tongue. These papillae contain taste buds.

Mechanism of Taste Sensitivity

Sweet or bitter substances, soluble in saliva, enter the pores of the taste buds, interact with the glycocalyx, and are adsorbed on the cell membrane of the microvilli, where the receptor proteins for sweet and bitter tastes are embedded. Under the influence of salts and acids, the concentration of electrolytes near the taste cell changes. In any case, the permeability of the taste cell membrane increases, the movement of ions occurs, and as a result, a receptor potential is formed.



Taste zones of the tongue

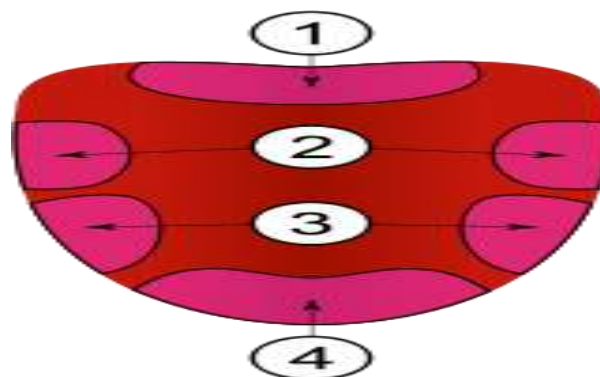


Figure 16. Taste Graph:

1-Bitter, 2-Sour, 3-Salty, 4-Sweet.

The receptor potential propagates across the cell to its base. A mediator (acetylcholine, serotonin, and a hormone-like substance) is formed in the taste cell, which leads to the appearance of a generator potential in the nerve fiber at the receptor-afferent synapse. Under the influence of a qualitatively different taste stimulus, an impulse of a certain frequency is generated in the nerve fiber, which determines the sensation of different tastes. Conduction of Excitation to the Brain. Taste receptor cells do not have axons. Information is transmitted to the endings of sensory fibers via transmitters. These fibers originate from the ganglion cells of the cranial nerves VII (the facial branch called chorda tympani) and IX (glossopharyngeal). The first excitation from a sensory fiber reveals an optimal response to a single stimulus but gives a significantly smaller response to other taste stimuli.

Oral Cavity Microflora

The oral cavity is the main gateway for microorganisms from the external environment throughout life. The constancy of the microflora is ensured by the antagonistic effects of some microbes on others, and the bactericidal properties of saliva. The microflora of different parts of the oral cavity varies and changes with age. Streptococci are the characteristic flora of the smooth surfaces of the oral mucosa (palate, cheeks, gingiva); vibrios and the fusospirochete complex are associated with the presence of teeth. The species composition of the oral microflora includes aerobic and anaerobic microorganisms. The concentration of aerobic and facultative bacteria in one milliliter of saliva is 10,000,000, and anaerobes is 100,000,000. Among cocci, typical streptococci are present in saliva, which accounts for 306 out of 409 strains of streptococci isolated from saliva. Along with saprophytic cocci, α -streptococci and coagulase-positive staphylococci are isolated from the oral cavity. Enterococci (group D streptococci) are considered permanent inhabitants of the oral cavity. Microbes specific to saliva are confined only to the oral cavity and the pharynx. Escherichia coli is observed in saliva only in cases of dysbiosis when the body's resistance is reduced. The oral cavity often contains fusiform bacteria in symbiosis with spirochetes, which, in turn, are in

symbiosis with staphylococci and streptococci. Streptococci play an important role in maintaining the stability of the oral microflora; under stress, the balance between the constant flora of the cavity is disturbed, and the reproduction of accidental flora is noted. Thus, bacterial antagonism is an important factor in the antibacterial defense of the OM. Some viruses also saprophytize in the oral cavity, such as herpes simplex viruses, which can be carried by about 60\% of people. With various disturbances in the physiological state of the oral cavity, representatives of unstable flora, including pathogenic species, can persist and multiply in it, leading to various diseases. In 40-50\% of cases, Candida fungi (*C. albicans*) are found in the oral cavity of healthy people, which usually do not cause harm, but with excessive reproduction (e.g., with uncontrolled use of antibiotics), they can lead to candidiasis or dysbiosis. The oral microflora also contains protozoa (representatives of the *Entamoeba*, *Trichomonas* genera).

Immune Properties of the Oral Mucosa

Immunity is a system of factors that ensure the body's internal defense against exo- and endogenous biological stimuli. The body has several protective "lines of defense": non-specific immunity factors (paleoimmunity); specific immunity (lymphocytic immunity), which in an evolutionary sense implies previous acquisition by the organism. Their purpose is the primary recognition of microorganisms and damaged cells. The main methods of neutralizing and destroying foreign substances are phagocytosis, extracellular cytolysis, cytotoxic reactions of NK cells, and the cytolytic action of complement. The first barrier to the antigen route is the mucous layer covering the epithelial cells. In addition to mucins, it contains non-specific antibacterial defense factors (including lysozyme, lactoferrin, defensins, myeloperoxidase, low molecular weight cationic peptides, complement components, etc.). Neutrophil granulocytes and macrophages are also present here, mainly migrating from the bloodstream. However, there is evidence of a population of immobile macrophages permanently located in the mucous membrane. These cells pass through the epithelial cells to reach the mucosal surface and destroy microorganisms using phagocytosis and other mechanisms. With

sufficiently rapid neutralization and removal of foreign substances, the development of specific immune reactions is not supported and subsides.

If this parameter is not possible, the "second" line of defense, or immunity itself, is included in the process. A specific feature of mucosal immunity is the ability of lymphocytes to migrate and concentrate at the site of antigen penetration. In children, when the barrier function of the oral mucosa is disturbed, the production of lysozyme is reduced, which leads to incomplete phagocytosis in macrophages and results in the chronicity of the inflammatory process. Furthermore, the rapid course of inflammatory processes in the oral cavity is caused by the activation of the oxidative metabolism of polymorphonuclear neutrophils. These factors should be considered when selecting immunomodulators. The integrity of the mucous membranes is the best guarantee of reliable body defense. The damaged surface of the epithelial layer is easily colonized by bacteria, which are capable of multiplying under conditions of weakened protective factors.

Components of Saliva:

Lysozyme (muramidase) is an enzyme with bactericidal activity, present in many cells, tissues, and secretory fluids of the human body, such as leukocytes, saliva, and tear fluid. Together with other components of saliva, such as secretory immunoglobulins A (sIgA), it helps destroy microorganisms in the oral cavity, which allows their number to be limited. Under the action of lysozyme, the bacterial cell turns into a spheroplast, which is lysed by osmotic pressure. Lactoferrin is a protein capable of binding iron, which has bacteriostatic activity. By binding iron, it prevents its use in bacterial metabolism, which leads to the cessation of microorganism reproduction. Lactoferrin is present in gingival fluid secretion and is secreted locally by polymorphonuclear neutrophils. sIgA plays the most important role in the local immune defense of the mucous membranes. They inhibit the ability of viruses and bacteria to adhere to the surface of the epithelial layer, preventing the penetration of pathogens into the body. They are secreted by the plasmocytes of the submucosa and the cells of the lamina propria of the tonsils. Saliva contains

significantly more sIgA than other immunoglobulins: for example, the IgA/IgG ratio in saliva secreted by the parotid glands is 400 times higher than in blood serum.

Cellular Elements. The cellular elements of the non-specific defense of the oral cavity are mainly polymorphonuclear neutrophils and macrophages. Both types of cells are found in saliva.

Secretory Elements: Derivatives of Macrophages. Macrophages induce certain factors of inflammatory spread or chemotaxis for inflammatory agents. Derivatives of Polymorphonuclear Neutrophils. Polymorphonuclear neutrophils trigger a chain of oxidation-reduction reactions (oxidative metabolism). Superoxide ions, hydroxyl radicals, and atomic oxygen are found in saliva, which are secreted by cells during immune conflicts and enter the oral cavity directly, where they cause the death of the foreign cell captured by phagocytes. In this case, the local inflammatory process may worsen due to the aggressive effect of free radicals on the cell membranes of the gingiva and periodontium. Derivatives of T-helper lymphocytes (CD4). Although CD4 lymphocytes are a factor of specific cellular immunity, they stimulate the non-specific immunity of the oral cavity, secreting a number of substances, the main ones being: Interferon γ —an active inflammatory mediator that promotes the formation of Class II histocompatibility antigens on the membranes necessary for the interaction of immunocompetent cells (HLA system).

Interleukin-2—a stimulator of the local immune reaction that acts on T-lymphocytes (increasing the secretion of immunoglobulins), T-helper lymphocytes, and cytotoxins (repeatedly enhancing local cellular defense reactions).

Lymphoid Tissue. Besides the lymph nodes located outside the oral cavity that "serve" its tissues, it contains four lymphoid formations that differ in their structure and functions. The palatine and lingual tonsils are the only lymphoid formations of the oral cavity with the classic structure of lymphoid follicles, consisting of peripheral B and T cells. Plasmocytes and lymphocytes of the salivary glands are involved in sIgA synthesis. The gingiva contains lymphoid clusters formed by lymphocytes, macrophages, and polymorphonuclear leukocytes, which play a major role in immune conflict with dental plaque bacteria. Thus, the main purpose of the

oral cavity lymphoid tissue is primarily the synthesis of sIgA and the antibacterial protection of the salivary glands.

Cellular Elements of Specific Mucosal Immunity:

T-lymphocytes. Depending on their specialization, T-lymphocytes are capable of repeatedly increasing the local immunity to the appearance of a foreign agent or directly destroying the foreign agent itself.

Plasmocytes (and B-lymphocytes). They play a major role in the synthesis and secretion of immunoglobulins, which are effective only with the participation of T-lymphocytes and helper cells (phagocytes). Mast cells. Mast cells, which are strong inducers of the local inflammatory reaction, play a secondary role in combating infections of the oral mucosa. Immunoglobulin G IgG. Small amounts of IgG enter the oral cavity with the bloodstream, but they can also be synthesized directly by plasmocytes after specific stimulation. They then enter the sites of immune conflict—the submucosa or the lamina propria. Immunoglobulin M IgM. IgM enters the oral cavity by the same routes as IgG, with IgM appearing faster during an immune conflict. They are less effective than IgG but have a significant immunostimulatory effect on the local lymphatic system. The hypersecretion of IgA in saliva allows this class of immunoglobulins to be considered the most important in the local immune defense of the oral cavity. In addition, the less noticeable but important role of IgA produced by plasma cells and entering the site of immune conflict with the bloodstream should be noted.



Functions of the Oral Mucosa

Protective:

Mechanical: Protection of the organs of the oral cavity and food structures from direct damage; high regeneration rate.

Immunological: The oral mucosa participates in ensuring local immunity. It prevents microbial invasion due to the barrier properties of the epithelium, its constant cleansing, and the presence of immunocompetent cells and IgA.

Chemical: Protection against chemically active components of the food substance.

Reflex: Participation in ensuring protective reflexes.

Secretory: Chemical processing of food (initial stages of carbohydrate and protein digestion are performed by salivary enzymes). Moistening of food, forming the food bolus and moving it to the pharynx. **Excretory:** Saliva formation. **Participation in Speech Formation:** By moistening the organs of the oral cavity with saliva.

Sensory: Taste perception in the papillae of the tongue. The mucous membrane is rich in receptors (cold, heat, pain, taste, and tactile) and is a reflexogenic zone for the glands of the gastrointestinal tract. **Assimilative:** It is capable of absorbing organic and inorganic substances (used for sublingual administration of medicines).

Buffering: Rapid restoration of pH occurs if necessary. **Immune:** The mucous membrane is a source of antigens entering the body, which, with adequate mechanisms of three-cell cooperation, allows maintaining an adequate titer of antibodies to these antigens (this function is performed by the tonsils).

Thermoregulation: One of the mechanisms for increasing heat transfer by the body is associated with increased ventilation of the oral mucosa, which is rich in hemocapillaries (it does not play a leading role in humans). The functioning of the oral mucosa is closely linked to the structural and functional features of its organs.

The following organs are distinguished in the oral cavity:

Lips.

Gingiva.

Floor of the mouth.

Tongue.

Cheeks.

Hard and soft palate.

Tonsils.

The oral mucosa performs most of its functions in conjunction with the organs of the oral cavity. Therefore, pathological processes occurring in the oral mucosa or one of its organs affect the functioning of its other formations and the digestive tract as a whole. **Structural Features of the Organs of the Oral Cavity** **Lips:** Closed lips cover the oral opening and limit the transverse slit of the mouth (Rima oris), the ends of which are called the corners of the mouth (anguli oris). The orbicularis oris muscle

(musculus orbicularis oris) is the basis of the lips. Lip surfaces: External (skin surface, pars cutanea). Transitional (intermediate, red surface, red border, pars intermedia). Internal (mucous, pars mucosa). The external-skin part of the lip is covered with stratified squamous keratinized epithelium, and the internal part of the lip is covered with stratified squamous non-keratinized epithelium characteristic of the oral cavity.

The basis of the lip is formed by the orbicularis oris muscle. The submucosa is well-developed, where adipose tissue, numerous vessels, and the terminal sections of the labial glands (glandulae labialis) are located. The labial glands are complex alveolar-tubular glands of the mixed muco-serous type.

Cheeks:

The skin part of the cheeks is covered with thin skin with well-developed subcutaneous fat. A specific feature of this part is the abundance of elastic fibers, which form rich accumulations of interconnected three-dimensional networks under the epidermis. This explains the possibility of strong stretching of the cheeks without deformation. The middle layer of the cheeks is represented by the striated skeletal muscle tissue of the buccinator muscle. The mucous part of the cheeks has different structures in different parts. Between the skin and the buccal mucous membrane is the buccinator muscle (m. buccalis), which forms its basis. The submucosa is well-expressed, where adipose tissue is evident, ensuring the mobility of the cheeks. Buccal salivary glands are also present, whose excretory ducts open on the buccal mucosa. In addition, the parotid papilla (parotid cushion) is present on the right and left sides of the buccal mucosa at the level of the upper second large molar, where the excretory duct of this gland opens. The buccal mucous membrane is covered with stratified squamous non-keratinized epithelium. The development of leukoplakia can occur on the buccal mucosa under the influence of negative factors. This is a chronic disease of the oral mucosa based on the process of hyperkeratosis, leading to the thickening and keratinization of the mucosal epithelial layer and the loss of its transparency. Leukoplakia is more common in men after the age of 30-40. Its causes can be smoking, hot and spicy food, alcohol, chewing tobacco (nas),

chronic mucosal trauma (prostheses, biting the cheeks, sharp edges of teeth). The localization sites of leukoplakia are the gingival mucosa along the line of tooth closure, the corner of the mouth, as well as the dorsal and lateral surfaces of the tongue. Prolonged exposure of the mucous membrane to negative factors can often lead to malignancy.

Functions of the Cheeks: 1. Participation in Food Intake: Participation in sucking; ensuring the hermetic closure of the oral cavity. 2. Protection (relative to the organs of the oral cavity): Protection from drying out; mechanical protection; chemical (due to the secretion of buccal glands—maintaining pH and electrolyte concentration); participation in protective reflexes and voluntary emptying of the oral cavity; immunological (secretion of immunoglobulins by buccal salivary glands). 3. Participation in Speech Formation. 4. Receptor field. 5. Participation in forming facial expressions. 6. Resorption (due to the large area and permeability of the mucous membrane). 7. Communicative and aesthetic function (due to the skin section).

Gingiva (Gum): The gingiva is part of the oral mucosa that covers the alveolar processes and the alveolar part of the mandible and directly surrounds the teeth. The gingiva has three sections: gingival papilla, marginal gingiva, and alveolar gingiva: anatomically. The free gingiva or gingival papillae are triangular in shape, located in the spaces between adjacent teeth, loosely adhere to the tooth surface, and are separated from it by the gingival sulcus. The main mass of the free gingiva tissue consists of collagen fibers containing elastic fibers. The marginal part of the gingiva is attached to the neck of the tooth, where the fibers of the circular ligament of the tooth are interwoven. Together with other fibers, it is designed to protect the periodontium from mechanical damage. The alveolar gingiva is the part of the gingiva that covers the alveolar process (firmly fused with the periosteum of the alveolar processes of the jaws) and is therefore immobile. It has a wavy surface due to the alternation of elevated sections and grooves. The grooves correspond to deep epithelial ridges attached to the lamina propria of the mucous membrane. The gingival crevice (sulcus) is a slit-like space between the tooth surface and the

adjacent free edge of the gingiva. Normally, its bottom is located in the cervical part of the enamel or at the cemento-enamel junction of the tooth. There is a clinical and anatomical gingival crevice. The clinical crevice is always deeper than the anatomical one—1-2 mm. If the depth of the gingival sulcus exceeds 1-1.5 mm, the formation of a gingival pocket is mentioned.

The formation of gingival fluid occurs in the zone of the gingival crevice. The mucous membrane covering the gingiva is stratified squamous keratinized epithelium with a thickness of 255 μm , containing a granular layer with keratohyalin located in its cytoplasm. The keratinization of the gingival epithelium is considered a protective function due to frequent mechanical, thermal, and chemical irritation. The epithelium in the marginal gingiva lacks a horny layer. Due to its good blood supply and thinness, the gingival epithelium is prone not only to increased traumatization but also to rapid regeneration.

The gingival crevice serves as the main source of leukocytes found in saliva and forming salivary bodies. In the initial period after migration, most of these cells (70-99%) not only remain viable but also possess high functional activity. In pathology, the number of migrating leukocytes can significantly increase. Healthy gingiva contains small thin-walled lymphatic vessels of irregular shape. They are mainly located in the subepithelial connective tissue base. With inflammation, the lymphatic vessels sharply dilate. Cells of the inflammatory infiltrate are detected in the lumen of the vessels, as well as around them. In a state of inflammation, lymphatic vessels contribute to the removal of interstitial material from the mucous membrane.

Palate The palate forms the roof of the oral cavity, consisting of the hard palate (anterior two-thirds) and the soft palate (posterior one-third). The hard palate (palatum durum) is a bony partition separating the oral cavity from the nasal cavity, serving as both the roof of the mouth and the floor of the nasal cavity. The soft palate (palatum molle) is a fold of mucous membrane with a muscle-fibrous base that separates the oral cavity from the pharynx. The base of the hard palate is bony (palatum osseum), covered by a mucous membrane. The bony body is formed by the palatine processes of the maxillae anteriorly and the horizontal plates of the palatine

bones posteriorly. The mucous membrane has a pale pink color. The incisive papilla (papilla incisivum) is located anteriorly to the central incisors. In the anterior 1/3 of the palate, 2-6 transverse palatine folds (plicae palatini) run towards the midline suture.

The soft palate is a continuation of the hard palate. Its anterior part is located in a nearly horizontal plane, while the posterior part is the palatine veil (velum palatinum), which hangs down and ends with the palatine uvula (uvula palatine). Both surfaces of the uvula are covered with stratified squamous epithelium. Laterally, the palatine veil passes into two folds (arches): the posterior—the palatopharyngeal arch (arcus palatopharyngeus) descends to the posterior wall of the pharynx; the anterior—the palatoglossal arch (arcus palatoglossus) goes to the lateral edge of the root of the tongue. The soft palate is a duplication of the mucous membrane where muscles are located. Its upper surface faces the nasal cavity and is covered with simple ciliated (ciliated) epithelium. The lower surface of the soft palate is a continuation of the hard palate mucous membrane and is covered with stratified squamous non-keratinized epithelium. The pharynx/isthmus of the fauces (fauces) is the opening connecting the oral cavity with the pharyngeal cavity. It is bounded superiorly by the posterior edge of the soft palate and the uvula, laterally by the palatine arches, and inferiorly by the superior surface of the root of the tongue. The size and shape of the pharynx depend on the degree of contraction of the soft palate and tongue muscles. The lymphoid ring (Pirogov–Waldeyer's ring) is located around the pharynx. The mucous membrane of the hard palate is a typical example of masticatory-type mucosa, directly fused with the periosteum of the palatine bones and is therefore immobile. It is covered externally with stratified squamous keratinized epithelium, beneath which there is a distinct lamina propria containing thick bundles of collagen fibers and forming long, narrow papillae. A submucosa is present underneath.

Tongue. The tongue is an unpaired muscular organ of the oral cavity, covered with a mucous membrane. It participates in the acts of chewing, swallowing, taste and

tactile sensations, and the formation of speech sounds. The following parts are distinguished in the tongue:

- a) Tip of the tongue (apex linguae);
- b) Body of the tongue (corpus linguae);
- c) Root of the tongue (radix linguae).

The upper surface of the tongue is called the dorsum (dorsum linguae). The mucous membrane of the tongue contains epithelial-covered protrusions—the lingual papillae (papillae linguales). In the root area, papillae are absent, and there is lymphoid tissue—the lingual tonsil (tonsilla lingualis). Filiform papillae (papillae filiformes) and conical papillae (papillae conicae) cover the anterior two-thirds of the tongue's dorsum and provide pain, temperature, and tactile (general) sensitivity. The filiform papillae are densely packed next to each other (500/cm²) in a specific order (following the terminal line) on the back of the tongue.

Fungiform papillae (papillae fungiformes) have a rounded shape and are mainly located on the tip and body of the tongue. Foliate papillae (papillae foliatae) are located on the lateral surface of the tongue. Circumvallate or vallate papillae (papillae vallatae) are located at the root of the tongue, forming a "V" shape. Taste buds are located in the epithelium of the fungiform, foliate, and circumvallate papillae. They provide taste perception. The septum of the tongue (septum linguae) is a thin, sometimes perforated, fibrous plate located in the muscle mass of the tongue along the midline plane. It divides the tongue into two symmetrical halves. On the dorsal surface of the tongue, it corresponds to the median sulcus of the tongue (sulcus medianus linguae). The relief and structure of the mucous membrane of the tongue are not uniform on its different surfaces. The surface of the lower surface of the tongue's mucous membrane is unspecialized, while the upper part is specialized. The lower surface of the tongue is covered with a mucous membrane, which includes a thin, stratified squamous non-keratinized epithelium and a lamina propria that protrudes with low papillae and contains a significant amount of elastic fibers, ensuring its rapid adaptation to constant deformation during tongue movements.

The submucosa, which provides the mobility of the mucous membrane, is deeper but limited due to its firm attachment to the muscle base of the organ. Mixed sublingual salivary glands are present in the anterior parts of the lower surface of the tongue. The upper, dorsal (back), and lateral surfaces of the tongue are covered with a mucous membrane, which includes a stratified squamous partially keratinized epithelium and a lamina propria, which are firmly connected to the underlying muscle tissue. Filiform papillae (papillae filiformes) are the most numerous and smallest, evenly distributed over the upper surface of the tip and body of the tongue. They have the appearance of conical protrusions about 2 mm high, located in rows parallel to each other and the terminal sulcus separating the root and body of the tongue. Filiform papillae are covered with epithelium, the thin horny layer of which forms pointed processes facing the pharynx. The thickness of the horny layer decreases from the tip of the papilla to its base. The connective tissue base of the papillae is characterized by a high content of collagen fibers, blood vessels, and nerve fibers; the function of the papillae is tactile. In old age, the number of filiform papillae on the dorsal surface of the tongue decreases, which is exacerbated by insufficient dietary intake of iron or B vitamins.

Foliate papillae (papillae foliatae) are well-developed only in early childhood; in adults, they are rudimentary or absent. They are located on each of the lateral surfaces of the tongue at the border of the root and body in an amount of 3-8. They are formed by parallel folds of the mucous membrane, separated by crevices into which the excretory ducts of serous salivary glands open. Taste buds are present in the epithelium on the lateral surfaces of the papillae. Function: perceives sour, sweet, and salty tastes. Fungiform papillae (papillae fungiformes) are smaller and lie singly among the lower filiform papillae; they are especially numerous at the tip of the tongue. These papillae reach a height of 2 mm and resemble a mushroom with a narrow base (foot) and an expanded top (cap). The connective tissue base of these papillae is highly vascularized; the blood in their vessels shines through the thin non-keratinized epithelium, giving the papillae a red color. Taste buds are constantly found in the epithelium of the papillae's tip. Function: gives a response to sour,

sweet, and salty tastes. Circumvallate papillae (papillae vallatae), or trench-surrounded papillae, are the largest (diameter 3 mm and height 1 mm); 6-15 in number, they are located within the V-shaped (terminal) sulcus and do not protrude above its surface. Each papilla is surrounded by a vallum (thickening of the mucous membrane), separated from it by a deep furrow, at the bottom of which the excretory ducts of serous salivary glands (Ebner's glands) open.

The secretion of these glands helps wash out the furrows, and it contains the enzyme lipase. Function: sensitive to bitter taste.

Taste buds (caliculi gustatoriae)—chemoreceptors located in the epithelium of the tongue (2000 in total—half of which are in the circumvallate papillae), soft palate, and pharynx—represent the peripheral part of the taste analyzer (organum gustus). Taste buds are compact clusters of 40-60 cells belonging to three main types: taste (sensory), sustentacular (supportive), and basal. Taste (sensory) cells are spindle-shaped, narrow, light, highly prismatic cells with a light nucleus in the basal part. Well-developed organelles are located in the cytoplasm around the nucleus: agranular endoplasmic reticulum, mitochondria, primary and secondary lysosomes. Microvilli are present on the apical surface, which are the adsorbents of taste stimuli. Specific receptor proteins are embedded in the primembrane layer of their cell membrane. Between the microvilli, there is an electron-dense substance with a high content of specific receptor proteins—sweet-sensitive, acid-sensitive, salt-sensitive, and bitter-sensitive. Up to 50 unmyelinated nerve fibers approach the lateral surface of the subepithelial cells and form receptor nerve endings. The apical surfaces of the taste bud cells form taste pits that open into taste pores. Substances dissolved in saliva enter the taste pits, are adsorbed by the electron-dense substance between the microvilli of the subepithelial cells, and affect the receptor proteins of the cell membrane, leading to a change in the electrical potential difference between the inner and outer surfaces of the cell membrane, the generation of excitation, which is captured by the nerve endings. Each cell senses several types of taste stimuli. The perception of salty and sour taste is ensured by the interaction of Nations or protons with ion channels on the outer cell membrane of the sensory cells. The perception

of sweet and bitter taste is provided by more complex mechanisms, including membrane receptors, the G-protein system, and secondary messengers. The final result of the action of any substances on sensory cells is the depolarization of their membrane. The epithelium is stratified, squamous, and non-keratinized, covering the surface of the tonsil and extending into the lamina propria of the mucous membrane, forming crypts that are 35-100 in adults and 30-70 in children, short and weakly branched. The ducts of mucous salivary glands open at the bottom of the latter, whose terminal sections are located in the submucosa. The lingual tonsil is part of the lymphoepithelial pharyngeal ring, which also includes other lymphoepithelial organs—the tonsils surrounding the entrance to the pharynx: palatine, pharyngeal, and tubal. The tonsils perform a protective function, which is ensured by lymphocytes, plasmocytes, antigen-presenting cells, and macrophages interacting with the epithelium. The lower surface of the tongue is covered with a mucous membrane, which includes a thin, stratified squamous non-keratinized epithelium and a lamina propria that protrudes with papillae and contains a significant amount of elastic fibers, ensuring its rapid adaptation to constant deformation during tongue movements. The submucosa, which provides the mobility of the mucous membrane, is deeper but limited due to its firm attachment to the muscle base of the organ. Mixed sublingual salivary glands are present in the anterior parts of the lower surface of the tongue. Their terminal sections, in the form of compact clusters, are located in the depth of the lamina propria and the submucosa and often penetrate the connective tissue layers between the bundles of muscle tissue. The submucosa is absent on the dorsum of the tongue. In the upper half of the tongue, between the muscle body and the lamina propria of the mucous membrane, there is a strong connective tissue plate, a kind of aponeurosis of the tongue. It is most developed in the area of the V-shaped terminal sulcus, where it is represented by a network of intertwined bundles of collagen and elastic fibers.

Lingual Glands: Mucous salivary glands lie at the root of the tongue, serous glands in the area of the vallate papillae, and mixed salivary glands at the tip.

I. Mucous and Mixed:

Anterior (glandula lingualis anterior), paired (under the inferior longitudinal muscle, closer to the tip, up to 7 ducts open on the lower surface of the tongue);

Posterior, numerous (glandulae tonsillae lingualis), located in the posterior half of the tongue, with ducts opening on the back and near the circumvallate papillae. They are all simple tubular or alveolar-tubular.

II. Serous Glands. They are located in the area of the papillae vallatae foliatae.

Floor of the Mouth (Og'iz Bo'shlig'ining Tubi)

The floor of the oral cavity, or its lower wall, is formed by a complex of soft tissues located between the tongue and the hyoid bone. The floor of the mouth is covered with a mucous membrane anteriorly and partially laterally to the tongue, between it and the gingiva of the mandible. A number of folds are formed at the junction of the mucous membrane: Frenulum of the tongue (a vertical fold of the mucous membrane) runs from the lower surface of the tongue to the floor of the oral cavity. Anteriorly, it reaches the oral surface. Sublingual folds (ridges) run along the sides of the tongue. At the medial ends of the ridges, tubercles, the sublingual papillae, are formed, where the ducts of the sublingual gland and the major sublingual duct open. Anteriorly, the ducts of the small incisive salivary glands are located behind the teeth under the mucous membrane near the mandible.

Functions of the Floor of the Oral Cavity: Absorption (the mucous surface plays the most important role as a route for administering a number of drugs—nitroglycerin). Reflex (in the clinic, validol is used when acting on cold receptors). Epithelium of the floor of the oral cavity: Stratified squamous non-keratinized (thin layer). The lamina propria of the mucous membrane contains numerous blood and lymphatic vessels, which ensure the easy absorption of a number of substances. The mucous membrane in the lower part of the oral cavity has a well-developed submucosa, consisting of loose connective and adipose tissue. The submucosa is represented by loose fibrous connective tissue, adipose tissue, and the excretory sections of the minor salivary glands. The mucous membrane is easily gathered into folds because it is weakly attached to the underlying tissues.

Salivary Glands (So'lak Bezlari)

Classification of Salivary Glands:

I. Minor: Labial, buccal, palatine, lingual, tonsillar (located in the thickness of the mucous membrane of the respective areas where their ducts open).

II. Major: Parotid, submandibular, sublingual.

III. By Nature of Secretion:

Serous (Protein): Parotid salivary glands, minor glands of the buccal mucosa, soft palate.

Mucous: Labial, palatine, buccal glands.

Mixed: Submandibular salivary glands, sublingual salivary gland.

By Mechanism of Secretion, all salivary glands are merocrine (synonym: eccrine), meaning secretion does not involve the destruction of cells or the separation of the apical part of the cytoplasm.

By Location:

Glands of the Oral Vestibule: Molar, buccal, labial, parotid salivary glands.

Glands of the Oral Cavity Proper: Submandibular salivary glands, sublingual salivary gland, lingual, hard and soft palatine glands.

Functions of Salivary Glands: Salivary glands perform the function of mixed secretion glands, but they mainly exhibit exocrine activity. The following groups of functions are distinguished:

Main function: Secretory (exocrine, endocrine);

Excretory: Excretion of various organic and inorganic substances—excretates that must be removed from the cell when accumulated. For example, carbon dioxide, uric and lactic acids, water, microelements, ions.

Reabsorption.

Functions of Saliva:

Protective, including immune: Protection from mechanical damage (softening food); contains antimicrobial substances (lysozyme, etc.), immunoglobulins A;

Cleansing function;

Buffering properties (against pathogenic microorganisms);

Mineralizing function (trophic for enamel); epidermal growth factor (regulation of mitosis in the oral cavity epithelium); thermoregulatory (primarily against food).

Excretory.

Digestive.

Regulation of water-salt homeostasis (1.0-1.5 L per day).

Endocrine: Production of biologically active substances (BAS), hormone-like substances, namely parotin (an insulin-like substance), etc. Nerve growth factor, epidermal factor, epithelial growth factor are also produced here. Under the influence of these factors, the growth of mammary glands increases, the growth of endothelial cells of skin, kidney, and muscle vessels, and the thickening of the skin occur. Participation in speech formation (optimizes articulation).

Participation in taste perception—dissolves food chemicals for direct contact with taste buds. Ensures reabsorption, i.e., used for sublingual administration of drugs dissolved in saliva.

Composition of Saliva: Saliva is the secretion of the salivary glands released into the oral cavity. It is necessary to distinguish between saliva collected from the excretory ducts and mixed saliva or oral fluid. Mixed saliva is the total secretion of all salivary glands, as well as oral detritus, microflora, contents of the gingival sulcus, gingival fluid, waste products of plaque microflora, decomposition of leukocytes migrating from the mucous membrane and secreted with saliva, food debris, etc. Saliva is a mixture of the secretions of three pairs of major, as well as numerous minor, salivary glands (800 to 1500 mL per day).

The secretion released from the excretory ducts of the salivary glands mixes with epithelial cells, food particles, salivary bodies (i.e., neutrophil leukocytes, sometimes lymphocytes), mucus produced by mucous glands, and microorganisms. Such saliva mixed with various inclusions is called oral fluid. It is opaque and has a viscous consistency. The composition of oral fluid may vary depending on the quality of food, the state of the body, and the influence of various environmental factors. The composition of salivary gland secretion is about 98-99% water, and the rest is dry residue (mineral anions of phosphates, bicarbonates, chlorides, iodides,

bromides, fluorides, sulfates). Saliva contains cations of calcium, sodium, potassium, magnesium, iron, and microelements: copper, nickel, lithium, etc. The concentration of inorganic substances such as iodine, calcium, potassium, and strontium is many times higher than in the blood.

Organic substances are mainly represented by proteins (albumins, globulins, enzymes). A large part of the protein substances accounts for enzymes: amylase, maltase, hyaluronidase (involved in the breakdown of polysaccharides); nucleases (break down nucleic acids and nucleoproteins); alkaline and acid phosphatases (split inorganic phosphate from organic compounds), lipase, peptidase. However, the role of these enzymes in the overall balance of enzymatic reactions of the gastrointestinal tract is small. Saliva also contains endocrine factors produced by the salivary glands, which are partially released not only into the blood but also into saliva. These are insulin-like growth factor (growth factor), lymphocyte, nerve, and epithelial growth factor, kallikrein (which causes vasodilation), renin (which causes vasoconstriction and increases aldosterone secretion by the adrenal gland), parotin (which reduces calcium content in the blood), epithelial growth factor, thymosyn-transforming factor. In addition, saliva contains nitrogen-containing components—products of excretion and filtration (urea, ammonia, creatinine, free amino acids, gamma-aminoglutamate, taurine, phosphoethanolamine, oxyproline, vitamins). The antimicrobial function of saliva is provided by substances such as lysozyme, lactoferrin, and peroxidase. Immunoglobulins enter saliva from two sources: as a result of local synthesis by plasma cells and through transudation via the gingival sulcus, which is the main source of leukocytes entering the oral cavity from the blood. Saliva is hypotonic relative to plasma (pH 6-7.4), which is optimal for α -amylase function. An acidic shift in the pH value of oral fluid from 6.2-6.0 turns it from a liquid saturated with calcium and phosphorus into an unsaturated one. Alkalinization has the opposite effect—the formation of tartar begins.

Classification of Oral Mucosa Diseases

To systematize pathological conditions (diseases) of the Oral Mucosa (OM), E.V. Borovsky and A.L. Mashkillayson proposed in 1984 to group them based on the etiological or pathogenetic factor:

I. Traumatic Lesions: Occurring under the influence of mechanical factors, high and low temperatures, radiation, adverse meteorological factors (meteorological cheilitis, cracks on the lip), chemicals, etc. Forms of manifestation: hyperemia, erosion, ulcers, hyperkeratosis (leukoplakia).

II. Infectious Diseases:

Involvement of the OM in acute and chronic infectious diseases (measles, scarlet fever, chickenpox, tuberculosis, syphilis, leprosy, etc.).

Infectious and parasitic diseases of the OM and lips proper:

Viral (herpes, HIV infection, papillomas, etc.);

Fusospirochetosis;

Bacterial (streptococcal and staphylococcal, gonorrhea, etc.);

Fungal (candidiasis, actinomycosis, etc.).

III. Allergic and Toxic-Allergic Diseases:

Contact allergic stomatitis, gingivitis, glossitis, cheilitis (from drugs, plastics, and other materials used in dentistry, dyes, toothpastes, elixirs, and other chemicals that contact the OM or the red border of the lips, ultraviolet rays);

Tolerance and widespread toxic-allergic lesions (from drugs, food substances, and other allergens entering the body through various routes);

Dermatoses with lesions on the OM of toxic-allergic genesis (erythema multiforme exudativum, Stevens–Johnson syndrome, Lyell's syndrome, primary systemic vasculitis, including Wegener's syndrome).

IV. Diseases with an Autoimmune Component in Pathogenesis:

Recurrent aphthous stomatitis, including scarring aphthae;

Behçet's syndrome, including Touraine's major aphthae;

Sjögren's syndrome;

Dermatoses with lesions on the OM (pemphigus, bullous pemphigoid, systemic lupus erythematosus, systemic scleroderma).

V. Skin-Mucosal Reaction: Lichen planus.

VI. Changes in the OM during Exogenous Intoxication.

VII. Changes in the OM and Red Border of the Lips in Pathology of Various Organs and Systems and Metabolic Disorders:

In visceral and endocrine pathology;

With hypo- and avitaminosis;

For diseases of the blood and hematopoietic organs;

With pathology of the nervous system;

During pregnancy.

VIII. Congenital and Genetically Determined Diseases:

Nevuses and epithelial dysplasia: vascular nevuses, including Sturge-Weber syndrome, verrucous and pigmented nevuses, epidermoid cyst, Fordyce's disease, white spongy nevus (soft leukoplakia, "cheek biting," etc.), hereditary benign intraepithelial dyskeratosis;

Folded and rhomboid glossitis;

Glandular cheilitis;

Dermatoses with OM lesions: bullous epidermolysis, atopic dermatitis (cheilitis), psoriasis, Darier's ichthyosis, Peutz–Jeghers–Touraine syndrome, congenital pachyonychia, anhidrotic epithelial dysplasia.

IX. Precancerous conditions, Benign and Malignant Tumors:

Obligate precancerous conditions: Bowen's disease, verrucous precancer, limited hyperkeratosis of the red border of the lips, abrasive precancerous cheilitis, Manganotti's cheilitis;

Facultative precancerous conditions: Leukoplakia, keratinizing papilloma and papillomatosis, keratoacanthoma, cutaneous horn, erosive-ulcerative and hyperkeratotic forms of red and discoid lupus erythematosus, post-radiation cheilitis;

Benign tumors;

Cancer.

The examination of patients with diseases of the oral mucosa is a complex study, including anamnesis and objective examination. The final diagnosis, the choice of a rational treatment method, the assessment of the disease course, and the determination of its prognosis are supplemented by data from necessary auxiliary methods (laboratory, functional, immunological, instrumental, etc.).

Interviewing the patient is an important stage, sometimes sufficient for diagnosis. However, one must keep in mind the subjective nature of pain perception by a particular patient. In most cases, the doctor needs additional examination methods based on physical, chemical, histological, microbiological, and other methods.

However, an accurate diagnosis and correctly planned treatment are only possible taking into account the patient's general condition. In this regard, there is a general and special anamnesis. General Anamnesis aims to determine the degree of influence of general diseases on the condition of the organs and tissues of the oral cavity. It must be remembered that many changes in the oral cavity, especially diseases of the oral mucosa, may be the result or manifestation of concomitant diseases. It is necessary to identify the patient's current general condition, the diseases they have experienced, and their connection with changes in the oral cavity. Attention should be paid to diseases that may limit anesthesia (cardiovascular diseases), bleeding tendencies, endocarditis, and the presence of allergies. The survey allows identifying pain in the mouth during speech, eating, and swallowing, which is often associated with pathology of the trigeminal, glossopharyngeal, or superior laryngeal nerves, the pterygopalatine ganglion, the presence of glossitis, aphthae, erosions, and ulcers on the mucous membrane. Diction disorders may occur due to mucosal defects, scarring, cleft palate, macroglossia, or errors in the manufacturing of dental prostheses. Bad breath is characteristic of ulcerative-necrotic gingivitis, periodontitis. Complaints of burning, paresthesia, and changes in taste sensations are observed with stomatalgia and glossalgia. To identify diseases in the patient that will affect the planned treatment, an oral survey is conducted or standardized questionnaires are used, which are filled out and signed by the patient. Special Anamnesis aims to collect information about complaints related to the maxillofacial

region. At the same time, you can ask questions, but it is better to allow the patient to first describe their complaints. When and how the initial signs of the disease appeared, what the causes of their occurrence were, and what the nature of the pain was. It is necessary to determine the localization, duration, intensity, and nature of the pain, as well as the factors that cause it. This information is important for diagnosis, but it should be remembered that these are subjective sensations, which must subsequently be confirmed or refuted by objective studies. With a disease of the oral mucosa, the main complaint is usually an indication of the appearance of redness, sores, or ulcers. Sometimes they report burning and dry mouth. The subsequent examination should be aimed at identifying the existing element (primary or secondary lesion), which will allow for a correct diagnosis.

Objective Examination

External Examination

As in other types of examination, the external examination requires a systematic approach. The examination begins with an assessment of the patient's general appearance, identifying asymmetry, and swelling. When complaining of diseases of the oral mucosa and the appearance of any lesion elements, the skin should be examined. Then, the submandibular and cervical lymph nodes are palpated to determine their size, firmness, mobility, and pain upon palpation. The color of the facial skin and the condition of the red border of the lips should be noted. Pay attention to the corners of the mouth (presence of cracks, crusts) and the inner surface of the lips. Normally, it is slightly lobulated, which is due to the presence of minor salivary glands in the mucous and submucous layers.

Examination of the Oral Cavity

The oral cavity is examined using an instrument set including a mirror, angled probe, tweezers, and excavator. The oral vestibule is examined with the jaws closed using a mirror. The examination starts with the buccal mucosa. Along the line of tooth closure in the posterior part, yellowish nodes, sometimes in the form of clusters, are found. These are sebaceous glands (Fordyce granules), which are sometimes mistaken for a pathological element. They are better seen when the mucous

membrane is stretched. The papilla of the excretory duct of the parotid salivary gland, which opens at the level of teeth 17 and 27, is also sometimes mistaken for a pathological formation. The buccal and labial area of the gingiva is examined starting from the upper right posterior part and then moving to the left. They descend to the mandible on the left and move in an arch to the right. Then the lingual and palatine areas of the gingiva are examined: on the maxilla from right to left, and along the mandible from left to right. Changes in color, the appearance of growths, and swelling of various shapes and consistency may be noted in the gingiva. The firmness, mobility, and all types of papillae of the tongue are assessed. Changes in color, vascular patterns, and the relief of the floor of the oral cavity are noted. The isthmus of the fauces is examined with the mouth wide open and the head tilted back. The hard and then the soft palate are examined using a dental mirror.



Additional Examination Methods for the Oral Mucosa

Stomatoscopy is used for the detailed examination of areas of the oral mucosa to differentially diagnose lesion elements, study the bottom of erosions, ulcers, the surface of verrucous growths, papules, plaques, etc. Diagnostic efficacy increases with the staining of the mucous membrane, for example, with Lugol's solution (2%) or toluidine blue (1%). Photostomatoscopy involves photographing the lesion using specialized devices.

Schiller–Pisarev Test and Svrakov's Iodine Number.

Method of study: Dry the examined area of the gingival mucosa, isolate it from saliva, and treat it with a cotton swab soaked in a solution containing 1 g of crystalline iodine, 2 g of potassium iodide, and 40 mL of distilled water. The color of the gingiva changes depending on the intensity of the inflammation. With a healthy periodontium, the gingival mucosa turns a straw-yellow color. Under the influence of chronic inflammation in the gingiva, the amount of glycogen, which stains brown with iodine, sharply increases, which changes from light brown to dark brown depending on the degree of the inflammatory process. By intensity, a negative sample (straw-yellow staining), weakly positive (light brown), and positive (dark

brown) are distinguished. The areas of the sample should be indicated in the patient's history before treatment.

The test must be performed in the same way on the same sites after the course of treatment or during the test. This allows you to compare the test results and evaluate the effectiveness of anti-inflammatory treatment. For objectivity, the Schiller–Pisarev test can be expressed numerically (Svrakov's Iodine Number) by calculating the color of the papillae at 2 points, the color of the gingival margin at 4 points, and the color of the alveolar process at 8 points. The total sum of scores should be divided by the number of teeth on which the study was conducted (usually 6). Mild inflammatory process—up to 2.3 points; moderate inflammatory process—from 2.67 to 5.0; severe inflammatory process—from 5.33 to 8.0.

Luminescent Methods. Luminescent diagnostics of OM diseases are based on the ability of tissues and cells to change color under the influence of UV light (UFL). There are two types of fluorescence:

Primary or intrinsic fluorescence of the substance; Secondary, caused by the action of luminescent substances (fluorescein, fluorochromes, eosin, tryptaflavin, etc.).

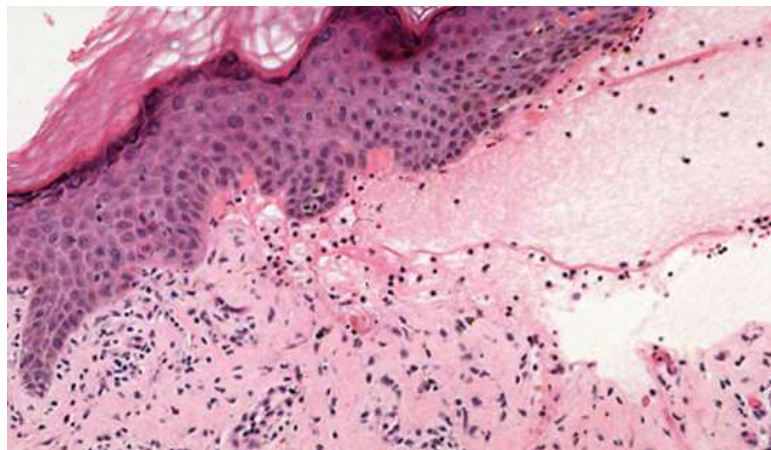
For the luminescent research method, OLD-41 devices, microscopes equipped with a quartz lamp (UFL source) with a filter made of dark violet glass—a Wood's lamp—are used. The study is carried out in a dark room, after the eyes adapt to the darkness, placing the lamp 20-30 cm from the surface being studied. The tongue of a healthy person in UFL glows from orange to red. Moreover, in some people, such a glow is observed over the entire tongue, while in others—only at the tip of the tongue, which may indicate hypovitaminosis B₁₂. With leukoplakia, a bright blue glow is observed. A dull glow with a yellowish tinge is characteristic of the phenomena of keratinization of the filiform papillae on the back of the tongue. Traumatic OM lesions luminesce with a dull bluish-violet color in Wood's light. In discoid lupus erythematosus, foci of hyperkeratosis have a white-snow-blue glow. Areas of leukoplakia-related hyperkeratosis have a cloudy white color in simple form. Clearly hyperkeratotic areas glow yellow or dirty yellow. Foci of congested hyperemia on the red border of the lips have a dark violet color, and hyperkeratotic

scales are blue-white. Luminescence is of particular importance in the presence of malignancy sites in the OM for diagnosis. Malignant areas of the OM have a blue-white glow combined with areas of red glow. In addition to the typical visual luminescent assessment of the OM, the following are also used:

Luminescent cytological examination;

Immunofluorescence examination.

Cytological diagnosis is widely used in the study of mucous membrane diseases. Material can be collected in various ways. The Yasinovsky test, which studies the amount of leukocyte migration into the oral cavity and exfoliated epithelium, involves a series of sequential rinses, followed by counting living and dead blood cells—leukocytes. Typically, 90-150 leukocytes (of which 20\% are dead) and 25-100 epithelial cells are found.



Cytological and Histological Examination Methods

Figure 17. Cytological Examination.

Histological methods are closely related to cytological studies. Sampling of tissues is performed through biopsy or excisional biopsy. After fixation, preparations are obtained via thin and ultra-thin sections, followed by staining of the cellular structural elements. Biopsy is performed under local anesthesia with a scalpel, needles of various designs, and trephine dissectors, observing the rules of asepsis and antiseptics. The collected biopsy material must include not only the affected area but also clinically normal tissue. The excised tissue is immediately placed in a fixative solution (ethyl alcohol or 10\% formalin solution) and sent to a pathomorphological laboratory. Examination of the preparations using light

microscopy methods is a reliable source of information about morphological changes in the mucous membrane. Bacteriological Examination Methods include the analysis of microbial and fungal flora taken from the affected area. Often, the imprint smear method is used for material sampling, but scraping, smear, and other methods can also be used. When collecting the material, the following rules must be followed: The ulcerative surface should be cleaned with a gauze swab, the material should be taken from the depth of the ulcer, and immediately sent to the laboratory. For the detection of *Candida* species of fungi, the material for laboratory testing is the whitish plaque on the mucous membrane. After fixation and staining, bacterioscopy is performed, i.e., the microflora is visually identified by characteristic staining patterns. It is also possible to study the activity of bacterial growth and their sensitivity to drugs. Infection of animals in experiments is used to study the pathogenic activity, infectious, and other properties of microorganisms. Allergological studies are carried out in case of impaired immune status (in vivo patch tests, blood cell counts, tests with a standard set of allergens). They collect an allergological history, paying special attention to heredity burdened with allergic diseases and the patient's past and present tendency to allergies. Determine the patient's reaction to food, smells, medications, household and industrial chemicals, as well as contact with animals. Provocative and parenteral tests are excluded from the arsenal of examination methods, as they are associated with a potential risk of complications. An obligatory assessment of the patient's individual reaction to the drug must be performed during the initial administration of medications (most often anesthetics), especially for parenteral administration. A sensitivity test is also performed if the patient has a history of allergic reactions to other drugs. In addition, in patients wearing prostheses, when subjective sensations or objective changes occur in the oral mucosa, the level of metals in the blood, electrical currents in the oral cavity, and reaction to plastic parts and other materials are determined. Complete Blood Count (CBC). The analysis includes determining the amount of hemoglobin, the number of erythrocytes and leukocytes, the color index, and the leukocyte formula. Blood examination is an important additional method that should

be performed in every patient with a disease of the oral mucosa. Absolute indications for this method include necrosis of the mucous membrane in the oral cavity, non-healing ulcers, as well as all cases where a disease of the blood-forming organs is suspected.

Biochemical Blood Examination. Examination for glucose level is performed in cases of suspected diabetes mellitus (dry mouth, chronic recurrent candidiasis, periodontal disease, etc.).

Kavarik test (Aldrich test)– used to check tissue hydrophilicity and detect latent edema.

Histamine Test – used to determine sensitivity to histamine, providing information about capillary permeability and the allergic status of the organism.

Kavetskiy (Bazarnova) Test – performed with a trephine needle to determine phagocytic activity and regenerative properties of tissues.

Rotter Test – performed to determine the saturation of the organism with ascorbic acid.



Pathological Changes and Morphological Elements of Oral Mucosa Lesions

Damage to the oral mucosa is expressed by a violation of its relief, color, and integrity. In the diagnosis of mucosal diseases, it is especially important to be able to distinguish between degenerative processes of the epithelium.

Spongiosis (Sponginess) – the accumulation of fluid between the cells of the prickle cell layer. The accumulation of fluid stretches the protoplasmic bridges between the cells and fills the resulting spaces, leading to the formation of vesicles. **Ballooning Degeneration** – the bonds between the cells of the prickle cell layer are broken, and the cells lie individually or in groups in the resulting exudate, taking on a "balloon" appearance.

Acantholysis – a degenerative change in the prickle cell layer, characterized by the destruction of the intercellular protoplasmic bridges.

Acanthosis – enlargement of the cells of the prickle cell layer.

Hyperkeratosis – excessive keratinization, resulting from the over-keratinization of cells or a violation of the shedding of the horny layer.

Parakeratosis – a violation of keratinization characterized by incomplete keratinization of the surface cells of the prickle cell layer.

Papillomatosis – overgrowth of the papillary layer of the oral mucosa.

Primary elements that appear independently and secondary elements that appear in place of primary elements are distinguished. Furthermore, monomorphic forms of lesions-consisting of a single type of primary element—and polymorphic forms-consisting of different types of primary elements—are distinguished. At the same time, one element itself can take on various appearances during its development, which is called false polymorphism.

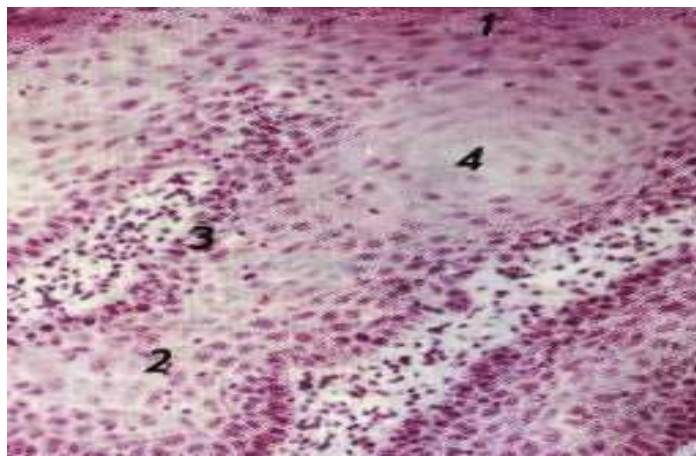


Figure 18

Parakeratosis

Acanthosis

Polymorphism and egg complex of the basal and prickle layers

Keratinization of prickle layer cells

Primary Morphological Elements

A. Infiltrative

Spot– a change in the color of the oral mucosa. It can appear in various forms: large and small, scattered and localized, temporary and persistent. A spot is characterized by not being palpable. Spots are divided into vascular and pigmented (dyschromatic), which result from the accumulation of coloring substances in the mucous membrane. Vascular spots are caused by inflammation or temporary reflex dilation of blood vessels. Inflammatory spots are red with varying intensity; they

disappear when pressed with a finger and reappear shortly after. Numerous and small spots are called roseola, while large ones are called erythema.

Spots resulting from the persistent dilation of superficial blood vessels without inflammation or from tumors formed from them are called telangiectasias. Spots on the skin and mucous membrane can also result from the rupture (breakage, increased permeability) of the blood vessel walls—these are hemorrhagic spots. Such spots do not disappear when pressed and have a different color depending on how long ago they appeared. Pinpoint hemorrhages are called petechiae, numerous small ones are called purpura, and large ones are called ecchymoses. Pigmented (Dyschromatic) spots result from an increase or decrease in melanin pigment, and sometimes from its complete absence.

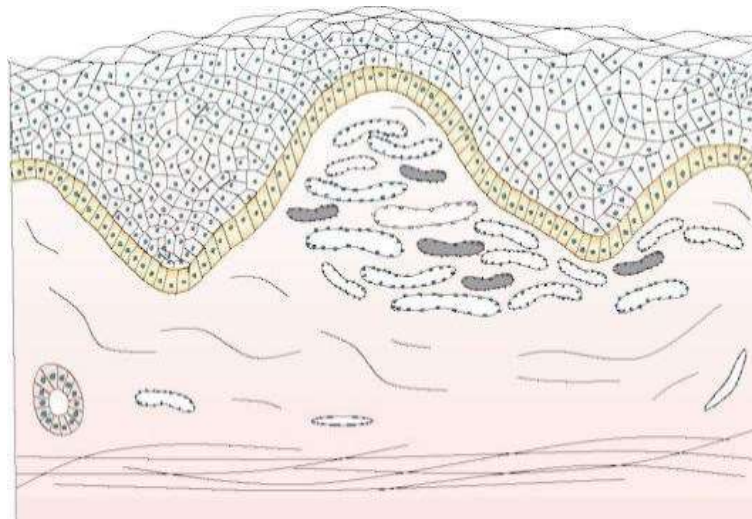


Figura 19

Papule .A papule is a non-cavitary lesion that arises due to **acanthosis** (thickening of the prickle cell layer of the epidermis) and is elevated above the mucous membrane/skin layer. Its surface can be **flat (yassi)**, **conical (konus)**, or **spherical (shar)**, while its borders may be **round (yumaloq)** or **polygonal**. Its color and consistency vary. It is the size of a **millet seed** or larger, and they can merge with each other to form **plaques (pilakchalar)**. Papules heal without leaving a scar (**iz qoldirmay bitib ketadi**).

Nodule.A nodule is a dense lesion that originates from the submucosal/subcutaneous layer. It is often palpated as a slightly painful, dense, round, fetus-like formation. As

it develops, it enlarges and becomes elevated above the mucous membrane/skin layer. The nodule has the potential to ulcerate and suppurate/fester.

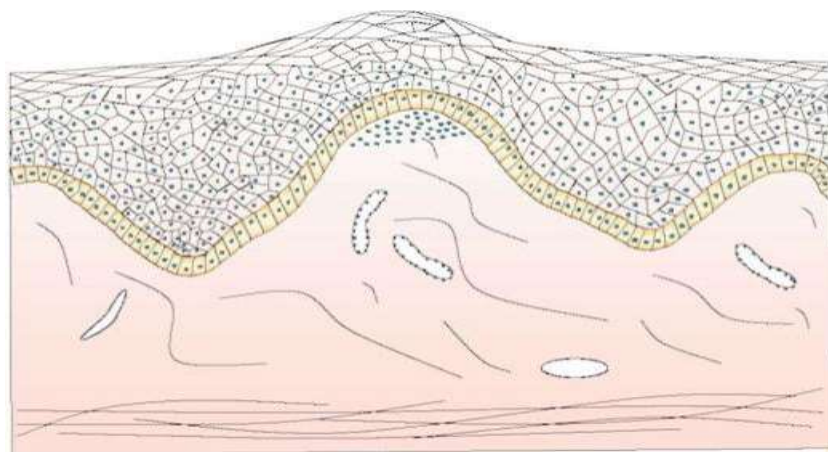


Figura20: Tubercule and Papule

The provided text defines a dermatological lesion called Tubercle

A tubercle is a non-cavitary, infiltrative lesion that involves all layers of the mucous membrane (or skin) and is elevated above the surface of the mucous membrane. It usually appears in clusters and rapidly disintegrates. In its place, ulcerated surfaces covered with granulation and vegetation are formed.

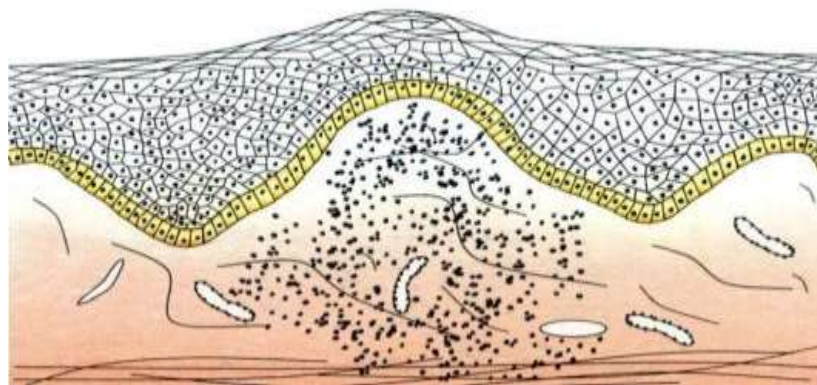


Figure 21. Tubercle

The text describes an Exudative Vesicle, which is a type of primary skin lesion.

B. Exudative Vesicle A vesicle is a cavitory element that forms as a result of the accumulation of fluid within a circumscribed cavity. It is located in the spinous layer and is characterized by a floor and a cover/roof that is elevated above the surface of the mucous membrane/skin. The cover/roof easily ruptures due to mechanical irritation/trauma. A vesicle is up to 2 mm in size.

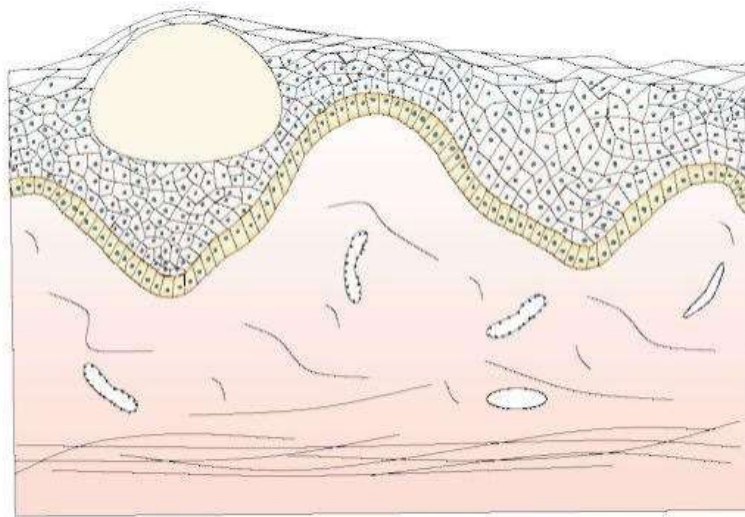


Figure 22: Vesicle

The text describes a Bulla, which is distinguished from a vesicle .

Bulla. A Bulla is a cavitory lesion that differs from a vesicle in its larger size and the fact that fluid can accumulate under the epithelium as well. If the fluid is located only intraepithelially, its cover/roof, which is composed of spinous layer cells, ruptures quickly. However, the cover/roof of a subepithelial bulla is stronger and remains intact for several days.

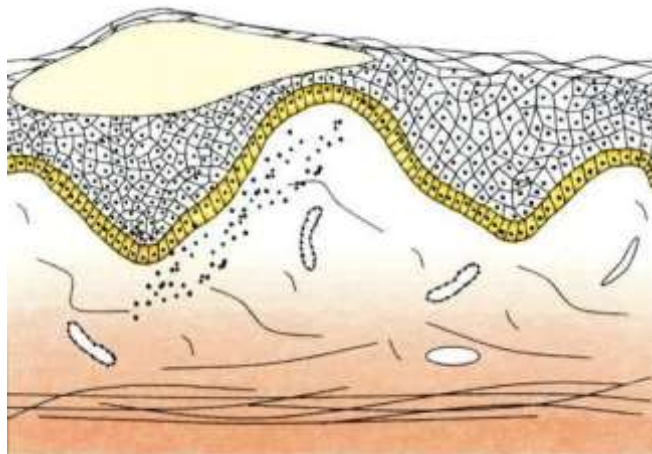


Figure 23: Bulla

The text describes a Pustule, which is another type of primary skin lesion.

Pustule A pustule is a cavitory lesion filled with pus . Primary and secondary ulcers are present. Pustules are located at various depths, meaning they can be superficial or deep.

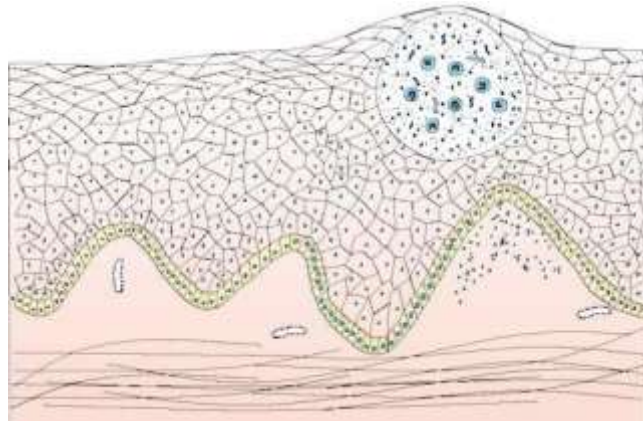


Figure 24: Pustule

The text describes a Cyst, which is also a type of skin or mucous membrane lesion. Cyst A cyst is a cavitory lesion lined on the inside with epithelium and surrounded by a connective tissue membrane. Cysts can be of epithelial or retention (retinsionniy) origin. The latter (retention cysts) are formed due to the blockage of the excretory ducts of small mucous or salivary glands. Epithelial cysts have a connective tissue wall lined with epithelium. The content of the cyst can be serous, seropurulent, or hemorrhagic. Retention cysts are located on the mucous membrane of the lips, palate, and cheeks, and are filled with a transparent content, which becomes purulent if infected/inflamed.

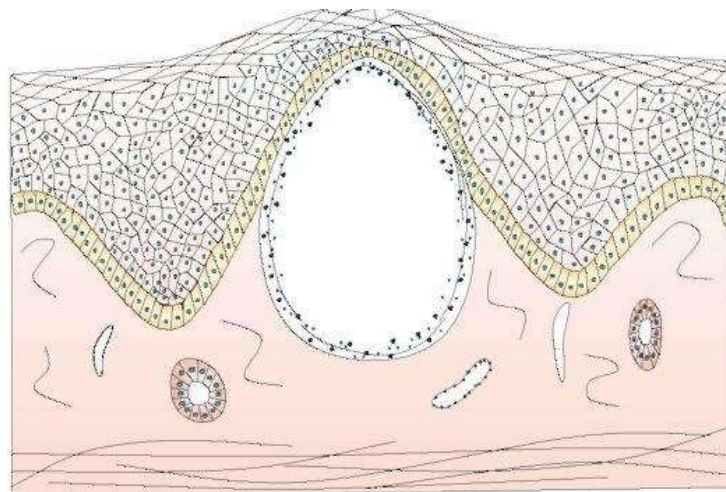


Figure 26 cyct

The text describes a dermatological lesion called a Wheal. Wheal. A wheal is a non-cavitory lesion that arises as a result of sharply circumscribed edema of the papillary layer (of the dermis/submucosa). It is elevated above the mucous membrane/skin layer and has a flat, whitish or reddish

appearance, with a size ranging from 0.2 to 1.5 cm. It can be of exogenous or endogenous origin.

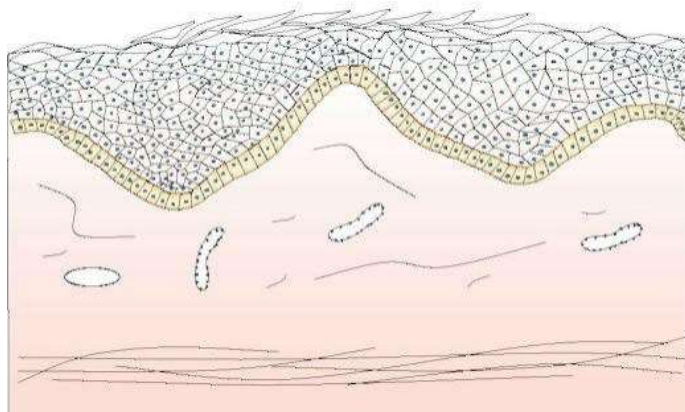


Figure 25 Wheal

The text shifts from describing primary lesions to Secondary Morphological Elements, starting with Erosion. Secondary Morphological Elements. Erosion

An erosion is a defect of the superficial layer of the epithelium; thus, after healing, it does not leave any scar. Erosion results from the rupture of a bulla, vesicle, the destruction/dissolution of papules, or traumatic injury. When a bulla breaks, the erosion follows its contours. In the oral cavity, erosive surfaces can form without a prior vesicle, such as erosive papules in syphilis, the erosive-ulcerative form of lichen planus and lupus erythematosus . A superficial defect of the mucous membrane caused by mechanical trauma is called excoriation

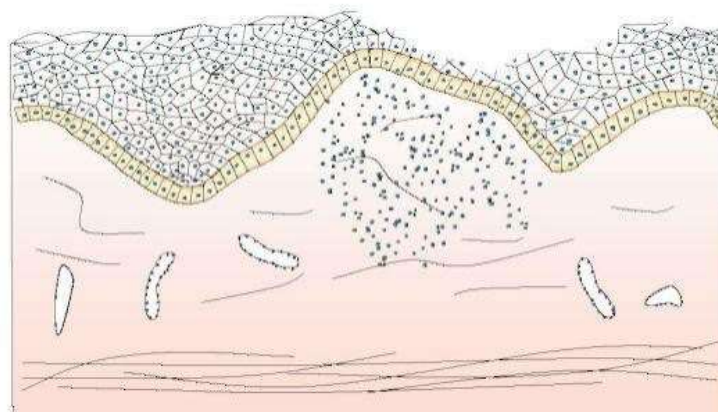


Figure 27 Erosion

The text describes a specific type of secondary lesion, known as an Aphtha.

Aphtha

An aphtha is a circumscribed necrosis of the epithelium. It has a yellowish-gray color and a round shape. Its size is 0.2–0.5 cm and larger, and it is surrounded by a bright-red inflammatory border. It heals without leaving a scar

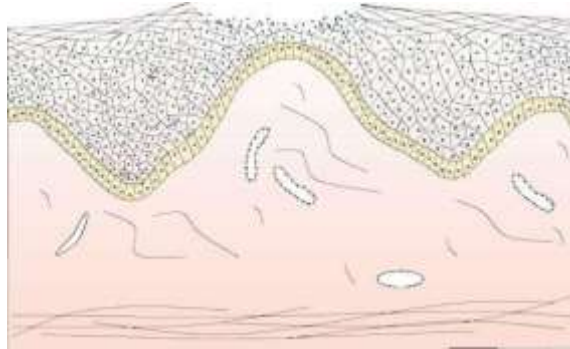


Figure 28: Aphtha

The text describes an Ulcer , which is a significant secondary morphological element.

Ulcer An ulcer is a defect within the connective tissue layer of the mucous membrane. The healing of an ulcer is accompanied by scarring. Since the formation of ulcers is characteristic of a number of pathological processes, it is necessary to evaluate all features of the lesion to determine their nature: the condition of the borders/edges , depth , shape, and the condition of the surrounding tissues. Knowing their characteristics also facilitates differential diagnosis. The edges of the ulcer may be undercut and overhanging the base, vertical, or saucer-shaped . The edges and base of the ulcer can be soft or hard. Often, the elements of the primary pathological process are preserved along the borders of the ulcer. Sometimes, the ulcer spreads to (and even destroys) the underlying tissues (muscles, bone). It should be noted that the clinical evaluation of the ulcer alone is sometimes not enough to clarify the diagnosis of the disease. This requires a full range of laboratory investigations as well as a general examination of the patient.

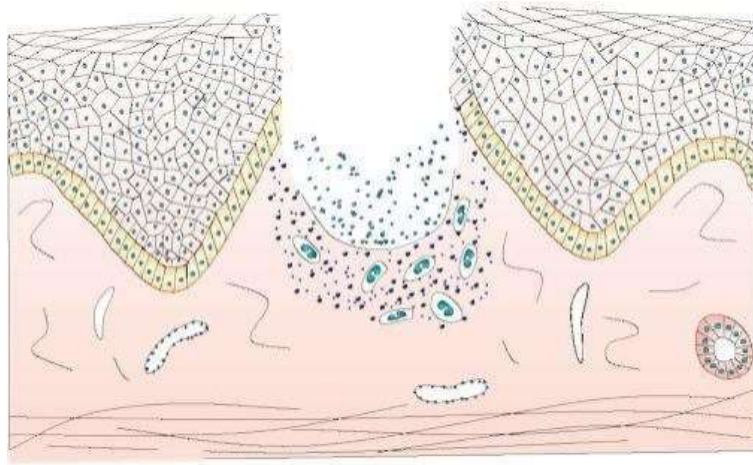


Figure 29: Ulcer

The text describes the final secondary morphological element: Scar.

Scar. A scar is an area of connective tissue that appears at the site of a defect, injury, or pathological process of the mucous membrane. A scar consists mainly of collagen fibers, covered by a thin layer of epithelium that lacks epithelial appendages. There are hypertrophic and atrophic types. Atrophic scars are formed after the healing of elements related to tuberculosis, syphilis, or lupus erythematosus. Scars appear in place of some primary and secondary elements.

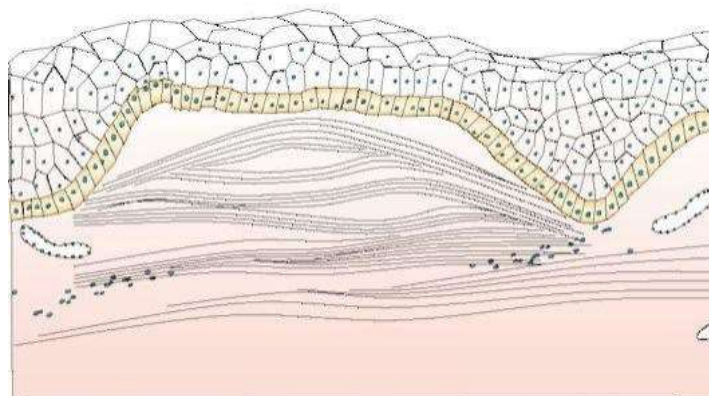


Figure 30: Scar

The text continues describing Secondary Morphological Elements, introducing Scale and Crust.

Scale. A scale is a shedding plate of keratinized epithelium.

Crust. A crust is dried exudate formed after the opening of a bulla, vesicle, or pustule. A crust is a combination of coagulated tissue fluid and blood plasma,

necrotic blood cells, and epithelial cells. When the crust is forcibly removed, an erosive or ulcerative surface is revealed, and after it falls off naturally, a site of regeneration, a scar, or scar atrophy appears.

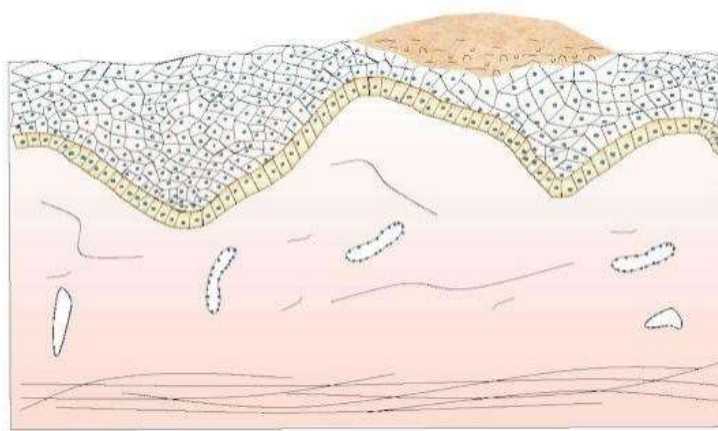
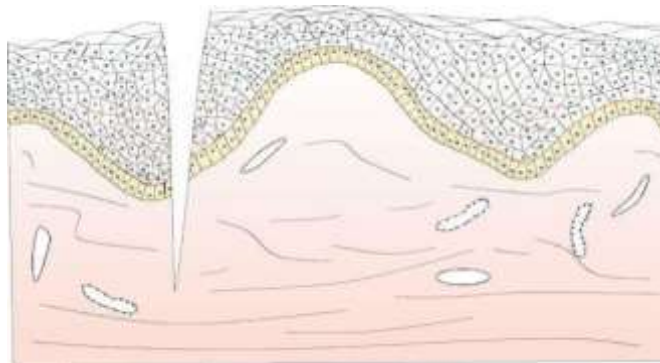


Figure 31: Crust

Crack -A fissure is a linear tear from the red border of the lips, which occurs with excessive dryness or loss of elasticity, inflammation - Often fissures form in natural locations - Observed in transitional folds and areas prone to stretching. The deep fissure spreads to the connective tissue of the lamina propria and heals with the formation of a scar. There are surface and deep cracks. A superficial rupture is located inside the epithelium and is treated without a scar.



Crack

ABSESS - a pus-filled cavity formation.

Atrophy - thinning of the mucous membrane.

Pigmentation - a change in the color of the tissue remaining at the site of inflammation. Damage to the oral mucosa can be acute (short-term strong mechanical impact) or chronic (long-term mild impact). Acute mechanical injury can occur in everyday life, at work, or as a result of a dentist's negligence during treatment. Trauma can be accompanied by a violation of the integrity of the mucous membrane (open wound) and preservation of its integrity (hematomas).

Hematomalacia can be caused by impact on the mucous membrane or by its bite. Blood enters the tissue, pain is felt in this area, the pain disappears in 1-3 days, and a dark bluish hematoma forms in its place. When the epithelium is damaged, painful erosion occurs, with infiltration and localized signs of inflammation in the surrounding mucous membrane. If this area is not re-injured, it quickly epithelializes. In cases of severe pain, local analgesics (lidocaine, trimecaine, anesthetics in solution, gel, ointment form) can be used.

to prevent secondary infection, it is advisable to treat the mouth with antiseptics (lysozyme, furacillin, etc.). In addition, agents that accelerate epithelialization are used.

Traumatic injuries

The oral mucosa is in direct contact with the external environment and is constantly subjected to various mechanical, thermal, chemical, microbiological, physical, and other factors. If these damaging substances do not exceed the damaging limit of the oral mucosa, it does not change due to its protective function. However, when exposed to stronger stimuli, changes occur in the oral mucosa, the nature of which depends on the type, intensity, and duration of the traumatic factor. The degree of development of these changes is also determined by the localization of the influence of an external factor, the state of reactivity of the body, individual features of the structure of the mucous membrane, etc. Since the main role in the clinical picture of traumatic injuries of the oral mucosa belongs to the irritating type, changes in the

mucous membrane caused by the action of mechanical, thermal, radiation, and chemical factors should be considered separately.

Mechanical damage

This injury can be acute when the mucosa is damaged by a short-term but significant factor and is characterised by chronic-weak prolonged exposure. Acute mechanical injury

Acute mechanical damage

Acute mechanical injury (trauma mechanicum acutum) - bite or instrumental injury - is rare. It manifests itself as a hematoma (intracranial bleeding without disrupting the integrity of the epithelium), erosion, or ulcer. Pain with internal bleeding is noted at the site of the lesion, which quickly disappears (after 1-3 days), but when the integrity of the epithelium is damaged, a painful erosive or infiltrating ulcer forms. A secondly infected wound can turn into a long-term incurable wound. Diagnosis, as a rule, is not difficult - the cause is determined from the anamnesis.

Treatment. The amount of medical assistance depends on the depth and extent of the damage. In the presence of hematomas, erosions, and small ulcers, careful antiseptic treatment of the affected area and the entire oral cavity is sufficient. For this purpose, rinse with antiseptic solutions (potassium permanganate 1:5000; 0.5% hydrogen peroxide solution and other medications), the use of agents that stimulate epithelialization (vitamins A, E, carotolin, rose oil, jelly, solcoseryl ointment, etc.). Sutures are used for deep wounds.

Chronic mechanical injury

Chronic mechanical trauma (travma mechanicum chronicum) is widespread. A traumatic factor can be sharp edges of the teeth, bridge and removable prostheses, dental calculus, bad habits (biting the mucous membrane), etc. The clinical picture and course of the process largely depend on the localization of the lesion (presence or absence of the submucosal base), the patient's age, the presence of secondary infection, and the strength of the irritating factor. Chronic irritant damage to the mucous membrane occurs more often in the elderly. This is facilitated by a decrease in the turgor of the mucous membrane, friction of the hard tissues of the teeth, loss

of teeth, and a decrease in bite height due to displacement. In the elderly, the regeneration process slows down, which leads to slow healing of the damaged mucous membrane. In chronic mechanical trauma, changes in the mucous membrane may not bother the patient for a long time, but sometimes discomfort, mild pain, and swelling may occur. Examination of the mucous membrane may reveal catarrhal inflammation (edema, hyperemia), disruption of its integrity (erosion, ulcers), proliferative changes (hypertrophy of gingival papillae, gingival margins), hypertrophy of papillomatous papillae, and increased leukocytosis (leukoplakia). These changes can occur together with each other. It should be remembered that simultaneously with mechanical damage, the mucous membrane affects the microflora of the oral cavity, which often affects the clinical picture of the disease.

Catarrhal inflammation. This is often accompanied by chronic trauma and manifests as hyperemia, edema with infiltration. The development of these changes depends on the strength and duration of the stimulus. In this case, the process is possible when exudation accumulates (initially serous, then purulent).

Among the irritating properties and factors leading to damage to the oral mucosa, prostheses should be highlighted. They are inadequate stimuli. The removable prosthesis transmits chewing pressure to the mucous membrane, delaying the self-cleaning of the oral cavity, which leads to a disruption of the established balance between various microorganisms, altering the analytical function of mucosal receptors. The occurrence of inflammation of the mucous membrane under the prosthesis depends not on one, but on the influence of several factors (traumatic, allergic, infectious).

Inflammation of the mucous membrane of the prosthetic arch can be focal in the form of point hyperemia or large hyperemic spots and is diffuse, often covering the entire surface of the prosthetic bed. Against the background of inflammation and swelling of the mucous membrane, spotted hemorrhages, erosions, as well as hyperplasia of the mucous membrane in the form of large granules may appear. Changes in the mucous membrane can also be observed when using permanent

dentures. Even in well-made crowns, the condition of the gums is disrupted - self-cleaning is difficult, and conditions are created for retaining food residues.

The course of catarrhal inflammation can be acute or chronic. Acute inflammation lasts 7-14 days and resolves quickly when the damaging property is eliminated. Superficial wounds and erosions of the mucous membrane with purulent exudate are observed. Destruction of the damaging agent and subsequent treatment leads to the rapid elimination of liquidation. Without treatment, chronic localized focal purulent inflammation is observed, consisting of a purulent mass. As a result, a traumatic wound is formed.

Traumatic ulcer. Traumatic ulcers, also called decubital, are located at the sites of injury, most often on the tongue, lips, and cheeks along the line of closure of the teeth. Wounds are usually painful, especially when eating or talking. Often this is a single ulcer. The clinical picture of a traumatic wound is diverse and depends on the condition of the wound, the body's reactivity, and the influence of the oral microflora, which gives it a damaging character. With the prolonged presence of the ulcer, its edges and base thicken. (due to the predominance of the phenomenon of proliferation) However, the edges of the ulcer are hyperemic, painful on palpation, the lower part is covered with a necrotic coating. Depth changes - up to the muscle layer. Local lymph nodes are enlarged, mobile, painful on palpation. Traumatic ulcers can be complicated by fusospirochetosis or candidiasis; if prolonged (2-3 months), they can progress to a malignant process.



Figure 33 . Decubital wound on the tongue.

Comparative diagnosis. Traumatic ulcers should be differentiated from cancer, tuberculosis, and syphilitic ulcers, as well as from chronic ulcerative necrotic gingivostomatitis of Vincennes and trophic ulcers.

Removal of the traumatic factor also serves the purposes of differential diagnosis. The formation of the ulcer within a few days indicates its traumatic origin.

A cancerous ulcer is characterized by a high density of the edges and wall, often with edge dehiscence. After the elimination of this damaging factor, the wound does not heal. A cytological or histological examination can reveal atypical epithelial cells. The tuberculous ulcer has more pronounced cut edges, the fundus is covered with a coarse-grained yellowish coating, epithelialization is not observed after removal of the pathogen. When examining wound scrapings, epithelioid cells and giant Langhans cells are detected, and when staining tuberculosis using the Nelson method, mycobacterial tuberculosis is detected. The patient's general condition, as a rule, worsens. For clarification of the diagnosis, the patient is referred to a phthisiologist.

Usually, a hard chancre differs from a traumatic wound by a dense infiltrate surrounding the wound, smooth edges, enlargement and densification (scleradenitis) of smooth lower, local lymph nodes. The diagnosis is confirmed when a pale spirochete is found in the excretory ulcer. Wassermann's reaction to syphilis and other reactions are positive 3-4 weeks after the onset of the ulcer. Elimination of the injury does not significantly affect the course of the hard chancre.

Secondary complications of traumatic ulcers with fusospirochetous lead to the formation of a greenish-gray plaque, with scrapings containing numerous spirochetes and spindle-shaped rods. Trophic ulcers are distinguished from traumatic ones by a slower course, mild signs of inflammation (areactive ulcers), and a sharp deterioration in general condition (often cardiovascular disease). The traumatic factor can be very weakly expressed, and after its elimination, if general treatment is not carried out, such ulcers are not amenable to healing.

Treatment. In the presence of traumatic ulcers, elimination of the damaging factor, antiseptic treatment of the ulcer, and rinsing of the oral mucosa are prescribed. For

severe wound pain, the use of painkillers is recommended. Necrotic tissues are carefully removed mechanically using proteolytic enzymes under anesthesia. Keratoplastic agents are prescribed (vitamins A and E; rose oil; jelly, solcoseryl ointment, actovegin, etc.). Burning the wound is strictly prohibited. Complete sanitation of the oral cavity is carried out. In traumatic injuries with removable prostheses, the appearance of hyperemia, erosions, or ulcers quickly resolves after prosthesis repair.

Hyperkeratosis. Prolonged wearing of a removable prosthesis can lead to leukoplakic changes - hyperkeratosis.

Lobular fibroma (denture). Sometimes prolonged damaging exposure to the edge of the prosthesis can lead to the development of denture fibroma. Initially, erosions or superficial ulcers appear. If the wound is not eliminated, fibrous tissue gradually develops at the base of the ulcer, a lobular fibroma grows - a dense, slightly painful tumor-like formation is observed, which is affected by the edge of the prosthesis. In the area corresponding to the edge of the prosthesis, a fold with a superficial linear ulcer or erosion is formed. Chronic damage to the denture edge can lead to the formation of several folds parallel to the denture edge. Lobular fibroma can persist for several years.

Treatment. This consists of correcting the prosthesis or discontinuing its use; After 2-3 weeks, the lobular fibroma heals, and a new prosthesis is made.

Papillomatous hyperplasia. Damage to the oral mucosa under a removable prosthesis (often on the upper jaw) is usually called prosthetic stomatitis (a combination of mechanical, chemical, and infectious factors). It manifests as hyperemia - diffuse or observed only around the openings of small salivary gland ducts in the posterior part of the palate. With prolonged wearing of a low-quality removable prosthesis, papillomatous hyperplasia develops against the background of diffuse hyperemia - with a soft granular bright red surface. Bone tissue of the alveolar process is absorbed locally, the alveolar ridge is soft and mobile ("hanging"). In some cases, angular cheilitis develops simultaneously. In addition to

chronic trauma, the influence of the Candida group fungus plays a role in the occurrence of such conditions.

Treatment. This consists of stopping wearing an incomplete prosthesis and making it better; if multiple Candida zambrugs are detected, anti-zambruge treatment is indicated.reatment. This consists of stopping wearing an incomplete prosthesis and making it better; if multiple Candida zambrugs are detected, anti-zambruge treatment is indicated. It is of great importance to carefully care for dent.

Typical bite of the mucous membrane. Usually biting or sucking lungen, lips, and less often tongue is rare among neuropaths, mainly observed in young (high school students and students) children.ypical bite of the mucous membrane. Usually biting or sucking lungen, lips, and less often tongue is rare among neuropaths, mainly observed in young (high school students and students) children. This mayypical bite of the mucous membrane. Usually biting or sucking lungen, lips, and less often tongue is rare among neuropaths, mainly observed in young (high school students and students) children. This may be imperceptible or conscious by patients. The habit of constantly biting the mucous membrane leads to chronic injuries of a specific type, which can be located along the line of tooth chipping. The mucous membrane swells, spots with a whitish, matted, scaly surface are observed in the form of large, indistinct limited areas. The epithelium is unevenly desquamated, giving the affected surface an uneven appearance due to the many tiny fragments of the epithelium.The epithelium is unevenly desquamated, giving the affected surface an uneven appearance due to the many tiny fragments of the epithelium.

This modified epithelium is removeThe epithelium is unevenly desquamated, giving the affected surface an uneven appearance due to the many tiny fragments of the epithelium.

This modified epithelium is removed by scraping. There is usually no pain, in severe cases painful erosion can occur. Pathogistologically, parakeratosis events are identified.

Comparative diagnosis. Such a change should distinguish the mucous membrane from leukoplakia, chronic candidiasis, White gubchatius nevus and leukoeema.

The cessation of the bite leads to the disappearance of all pathological changes in a short time. Candida fungi are not found when mucous membranes are scraped. The cessation of the bite leads to the disappearance of all pathological changes in a short time. Candida fungi are not found when mucous membranes are scraped.

The white sponge nevus differs in that it The cessation of the bite leads to the disappearance of al

Chemical damage

Chemical damage (trauma ximicum) can be both acute and chronic. Acute injuries are caused by exposure to the mucous membrane of chemicals in a very harmful concentration. hemical damage (trauma ximicum) can be both acute and chronic. Acute injuries are caused by exposure to the mucous membrane of chemicals in a very haemical damage (trauma ximicum) can be both acute and chronic. Acute injuries are caused by exposure to the mucous membrane of chemicals in a very harmful concentration. Most often, this happens at home, at work, during an accidental injury or attempt to commit suicide, as well as during an appointment with a dentist. Recently, dentists have abandoned many incinerators, but in practice there are strong chemicals that require careful handling. Burns of the oral mucosa can occur in contact with acids, alkalis, when using a mixture of arsenic paste, phenol, formalin, silver nitrate, resorcin farmolin, when wearing a prosthesis from insufficiently polymerized plastic.

As a result of the consumption of high-concentration chemicals, it is necessary to distinguish between Burns and an allergic reaction, for example, plastic, amalgam, low-concentration chemicals. Burns and an allergic reaction can be combined.s a result of the consumption of high-concentration chemicals, it is necessary to distinguish between Burns and an allergic reaction, for example, plastic, amalgam, low-concentration chemicals. Burns and an allergic reaction can be combined. Sometimes patients, trying to get rid of toothache on their own, put alcohol, acetylsalicylic acid and other substances on the tooth, and on the mucous membrane eaten along the way, which leads to Burns.

Clinical picture. Acute pain with Acute Burns, as a rule, occurs immediately and is localized in areas where chemicals enter. The clinical picture depends on the nature and quantity of the damaging substance, the duration of its action. Acid burns lead to the appearance of coagulation necrosis on the mucous membrane—a dense film of Brown (from sulfuric acid), yellow (from nitric acid) or white-gray (from other acids) color becomes characteristic. The main tissues in which the plaques are located are destroyed against a pronounced inflammatory background with maxillary connected edema and hyperemia.

Burns with alkalis lead to colliquial necrosis of the mucous membrane, no dense film is formed, necrotic tissues acquire a gelatinous consistency. The acid is deeper than the burn.

Burns with alkalis lead to colliquial necrosis of the mucous membrane, no dense film is formed, necrotic tissues acquire a gelatinous consistency. The acid is deeper than the burn. Burns with alkalis lead to colliquial necrosis of the mucous membrane, no dense film is formed, necrotic tissues acquire a gelatinous consistency. The acid is deeper than the burn. Necrosis in alkaline burns can occur on all layers of soft tissue, especially on the gums and hard palate. Burns, especially extensive, cause severe suffering to the patient. Within days of the rejection of necrotic tissue, erosive or scarred surfaces that gradually heal are exposed. In mild cases, alkaline burns can only lead to a catarrhal inflammatory process.

Treatment. After the chemical has fallen into the Oral mucosa, it must be removed as soon as possible. Treatment. After the chemical has fallen into the Oral mucosa, it must be removed as soon as possible. If such a burn happened in front of the doctor, you should immediately treat it. After the chemical has fallen into the Oral

mucosa, it must be removed as soon as possible. If such a burn happened in treatment. After the chemical has fallen into the Oral mucosa, it must be removed as soon as possible. If such a burn happened in front of the doctor, you should immediately start rinsing the oral cavity (rinsing, watering) with a weak solution of the neutralizing agent. If the doctor is absent, then it is washed off with water, and then quickly washed off the necessary neutralizing solution. In acid burns, soapy water, 1% lime water, water-burnt magnesia, 0.1% ammonia peanuts (15 drops per glass of water) are used. Alkalis are neutralized with 0.5% solutions of citric and acetic acids (1/4 teaspoon of acid per hundred kan water), 0.1% hydrochloric acid solution (10 drops per glass). Alkalis are neutralized with 0.5% solutions of citric and acetic acids (1/4 teaspoon of acid per hundred kan water), 0.1% hydrochloric acid solution (10 drops per glass). In this way, further penetration of chemicals into tissues can be stopped.

To reduce the absorption of concentrated silver nitrate Solutions, a 2-3% sodium chloride solution or Lugol solution is used, insoluble silver compounds are formed. To reduce the absorption of concentrated silver nitrate Solutions, a 2-3% sodium chloride solution or Lugol solution is used, insoluble silver compounds are formed. When burned with phenol, the mucous membrane is treated with 50% ethyl alcohol or castor oil. Further treatment of patients with chemical burns is carried out in accordance with the type of treatment of the acute nonspecific inflammatory process: pain relievers are used (1% trimecaine yeritma, trimecaine with hexamethylenetetramine, 1-2% lidocaine yeritma, etc.). Further treatment of patients with chemical burns is carried out in accordance with the type of treatment of the acute nonspecific inflammatory process: pain relievers are used (1% trimecaine yeritma, trimecaine with hexamethylenetetramine, 1-2% lidocaine yeritma, etc.), oral baths and weak solutions of antiseptic agents in the form of rinses, drugs that accelerate epithelization (1% citral yeritma in Peach oil contains vitamins A and ye, aevit, rose oil, sea buckthorn,

etc.) are used. High-calorie puree food that does not bother, cold (ice cream), multivitamins are recommended inside. Surgical intervention with wide scars is indicated



Figure 34. Chemical burn

Physical injuries

Acute injuries are caused by short-term exposure of the mucous membrane to high doses of hot water, hot steam, fire (thermal damage), electric shock and ionizing radiation. Acute injuries are caused by short-term exposure of the mucous membrane to high doses of hot water, hot steam, fire (thermal damage), electric shock and ionizing radiation. Burns with hot steam or water initially cause acute pain, the intensity of which quickly decreases, a feeling of roughness of the mucous membrane appears. Acute catarrhal stomatitis develops. The epithelium of the oral mucosa is partially or completely matsified; after severe burns, it is broken into layers or bubbles appear, which quickly crack, under which erosion or superficial wounds are found.

Treatment after severe burns, it is broken into layers or bubbles appear, which quickly crack, under which erosion or superficial wounds are found.

Treatment. It is carried out in the same way as in non-specific acute inflammatory processes.

Prevention. Prevention of such injuries is an expression from the performance of sanitary and hygienic work, compliance with safety measures

Galvanism. Galvanic flow in the oral cavity is observed in the presence of various metals (prostheses and fillings). Galvanism. Galvanic flow in the oral cavity is observed in the presence of various metals (prostheses and fillings). The presence of various metals and Galvanism. Galvanic flow in the oral cavity is observed in the presence of various metals (prostheses and fillings). The presence of various metals and large microcurrents does not always cause pathological changes, but in some people even small currents can cause them. Conditionally, up to 10 microflows in the oral cavity are the norm. Their growth is caused by a significant (compared to the norm) amount of solution formed when the technology of prosthetic production is broken. The largest microcurrent is formed by the following substances: gold-amalgam, steel - solute, amalgam-solute and amalgam-steel. The largest microcurrent is formed by the following substances: gold-amalgam, steel - solute, amalgam-solute and amalgam-steel. Galvanic flow plays a certain role in the occurrence of inflammation. The largest microcurrent is formed by the following substances: gold-amalgam, steel - solute, amalgam-solute and amalgam-steel. Galvanic flow plays a certain role in the occurrence of inflammatory processes of Oral mucosa, especially leukoplakia and KPL, as well as in the exacerbation of existing pathological processes. In many patients with leukoplakia and KPL, the resolution of the process or a more favorable course of the disease is caused by the replacement of some prostheses with others, which leads to a decrease in the size of the lesion microcurrents. The effect occurs after 1-4 months. It should also be remembered about the possibility of increasing the sensitivity of the mucous membrane to Mercury, Silver and other substances. The effect occurs after 1-4 months. It should also be remembered about the possibility of increasing the sensitivity of the mucous membrane to Mercury, Silver and other substances.

In some patients, galvanic flow causes unpleasant subjective sensations, is observed on the tongue or other areas of the mucous membrane without obvious objective changes.

The appearance of complaints is consistent with the installation of prostheses or metal fillings. Galvanic flow in the oral cavity exceeds 10 mkA. The appearance of complaints is consistent with the installation of prostheses or metal fillings. Galvanic flow in the or



Photo 35. Galvanism

Papillary hyperplasia of palatine mucosa

Treatment. This consists of stopping wearing an incomplete prosthesis and making it better; if multiple Candida fungi are detected, antifungal treatment is indicated. Careful care of dentures, cleaning them with a toothbrush with powder or paste, is of great importance. At night, it is recommended to place prostheses in weak hydroxide solutions (2% borax or sodium bicarbonate solution).

A typical bite of the mucous membrane. Typically, biting or sucking the cheeks, lips, and less frequently the tongue is rare among neuropaths, mainly observed in young children (high school pupils and students). This may be imperceptible or conscious by the patients. The habit of constantly biting the mucous membrane leads to a specific type of chronic trauma, which can be located along the line of dentition. The mucous membrane is edematous, with large, blurred, macerated spots with a cortical surface in the form of indistinctly limited areas. The epithelium is unevenly desquamated, has an angular appearance due to numerous small fragments of the epithelium, giving an uneven surface. This modified epithelium is removed by scraping. Usually, there is no pain, and in severe cases, painful erosions may appear. Pathohistologically, phenomena of parakeratosis are detected.

Comparative diagnosis. Such changes in the mucous membrane should be differentiated from leukoplakia, chronic candidiasis, white spongy nevus, and leukemia.

The cessation of occlusion leads to the disappearance of all pathological changes in a short time. Candida fungi are not found when scraping the mucous membranes.

White spongy nevus is characterized by its manifestation in early childhood and subsequent growth, often of a familial nature; the mucous membrane of the cheeks appears thickened, deeply covered, and spongy.

Treatment. This consists of stopping cheek bites.

Chemical damage

Chemical damage (trauma ximicum) can be both acute and chronic. Acute injuries are caused by exposure to the mucous membrane of chemicals in a very harmful concentration. hemical damage (trauma ximicum) can be both acute and chronic. Acute injuries are caused by exposure to the mucous membrane of chemicals in a very hahemical damage (trauma ximicum) can be both acute and chronic. Acute injuries are caused by exposure to the mucous membrane of chemicals in a very harmful concentration. Most often, this happens at home, at work, during an accidental injury or attempt to commit suicide, as well as during an appointment with a dentist. Recently, dentists have abandoned many incinerators, but in practice there are strong chemicals that require careful handling. Burns of the oral mucosa can occur in contact with acids, alkalis, when using a mixture of arsenic paste, phenol, formalin, silver nitrate, resortsin farmolin, when wearing a prosthesis from insufficiently polymerized plastic.

As a result of the consumption of high-concentration chemicals, it is necessary to distinguish between Burns and an allergic reaction, for example, plastic, amalgam, low-concentration chemicals. Burns and an allergic reaction can be combined.s a result of the consumption of high-concentration chemicals, it is necessary to distinguish between Burns and an allergic reaction, for example, plastic, amalgam, low-concentration chemicals. Burns and an allergic reaction can be combined. Sometimes patients, trying to get rid of toothache on their own, put alcohol,

acetylsalicylic acid and other substances on the tooth, and on the mucous membrane eaten along the way, which leads to Burns.

Clinical picture. Acute pain with Acute Burns, as a rule, occurs immediately and is localized in areas where chemicals enter. Acute pain with Acute Burns, as a rule, occurs immediately and is localized in areas where chemicals enter. Acute pain with Acute Burns, as a rule, occurs immediately and is localized in areas where chemicals enter. The clinical picture depends on the nature and quantity of the damaging substance, the clinical picture. Acute pain with Acute Burns, as a rule, occurs immediately and is localized in areas where chemicals enter. The clinical picture depends on the nature and quantity of the damaging substance, the duration of its action. Acid burns lead to the appearance of coagulation necrosis on the mucous membrane—a dense film of Brown (from sulfuric acid), yellow (from nitric acid) or white-gray (from other acids) color becomes characteristic. The main tissues in which the plaques are located are destroyed against a pronounced inflammatory background with maxillary connected edema and hyperemia.

Burns with alkalis lead to colliquial necrosis of the mucous membrane, no dense film is formed, necrotic tissues acquire a gelatinous consistency. The acid is deeper than the burn. Burns with alkalis lead to colliquial necrosis of the mucous membrane, no dense film is formed, necrotic tissues acquire a gelatinous consistency. The acid is deeper than the burn. Burns with alkalis lead to colliquial necrosis of the mucous membrane, no dense film is formed, necrotic tissues acquire a gelatinous consistency. The acid is deeper than the burn. Burns with alkalis lead to colliquial necrosis of the mucous membrane, no dense film is formed, necrotic tissues acquire a gelatinous consistency. The acid is deeper than the burn.

Treatment. After the chemical has fallen into the Oral mucosa, it must be removed as soon as possible. Treatment. After the chemical has fallen into the Oral mucosa, it must be removed as soon as possible. If such a burn happened in front of the doctor, you should immediately start treatment. After the chemical has fallen into the Oral mucosa, it must be removed as soon as possible. If such a burn happened in front of the doctor, you should immediately start treatment. After the chemical has fallen into the Oral mucosa, it must be removed as soon as possible. If such a burn happened in front of the doctor, you should immediately start rinsing the oral cavity (rinsing, watering) with a weak solution of the

neutralizing agent. If the doctor is absent, then it is washed off with water, and then quickly washed off the necessary neutralizing solution. In acid burns, soapy water, 1% lime water, water-burnt magnesia, 0.1% ammonia peanuts (15 drops per glass of water) are used. Alkalis are neutralized with 0.5% solutions of citric and acetic acids (1/4 teaspoon of acid per hundred kan water), 0.1% hydrochloric acid solution (10 drops per glass). Alkalis are neutralized with 0.5% solutions of citric and acetic acids (1/4 teaspoon of acid per hundred kan water), 0.1% hydrochloric acid solution (10 drops per glass). Alkalis are neutralized with 0.5% solutions of citric and acetic acids (1/4 teaspoon of acid per hundred kan water), 0.1% hydrochloric acid solution (10 drops per glass). In this way, further penetration of chemicals into tissues can be stopped.

To reduce the absorption of concentrated silver nitrate Solutions, a 2-3% sodium chloride solution or Lugol solution is used, insoluble silver compounds are formed. When burned with phenol, the mucous membrane is treated with 50% ethyl alcohol or castor oil. Further treatment of patients with chemical burns is carried out in accordance with the type of treatment of the acute nonspecific inflammatory process: pain relievers are used (1% trimecaine yeritma, trimecaine with hexamethylenetetramine, 1-2% lidocaine yeritma, etc.). Further treatment of patients with chemical burns is carried out in accordance with the type of treatment of the acute nonspecific inflammatory process: pain relievers are used (1% trimecaine yeritma, trimecaine with hexamethylenetetramine, 1-2% lidocaine yeritma, etc.), oral baths and weak solutions of antiseptic agents in the form of rinses, drugs that accelerate epithelization (1% citral yeritma in Peach oil contains vitamins A and Ye, aevit, rose oil, sea buckthorn, etc.) are used. High-calorie puree food that does not bother, cold (ice cream), multivitamins are recommended inside. Surgical intervention with wide scars is indicated.



Figure 34. Chemical burns

Physical injuries

Acute injuries are caused by short-term exposure of the mucous membrane to high doses of hot water, hot steam, fire (thermal damage), electric shock and ionizing radiation. Acute injuries are caused by short-term exposure of the mucous membrane to high doses of hot water, hot steam, fire (thermal damage), electric shock and ionizing radiation. Burns with hot steam or water initially cause acute pain, the intensity of which quickly decreases, a feeling of roughness of the mucous membrane appears. Acute catarrhal stomatitis develops. The epithelium of the oral mucosa is partially or completely destroyed; after severe burns, it is broken into layers or bubbles appear, which quickly crack, under which erosion or superficial wounds are found. Treatment after severe burns, it is broken into layers or bubbles appear, which quickly crack, under which erosion or superficial wounds are found.

Treatment. It is carried out in the same way as in non-specific acute inflammatory processes. After severe burns, it is broken into layers or bubbles appear, which quickly crack, under which erosion or superficial wounds are found.

Prevention. Prevention of such injuries is an expression from the performance of sanitary and hygienic work, compliance with safety measures

Galvanism

Galvanic flow in the oral cavity is observed in the presence of various metals (prostheses and fillings). Galvanic flow in the oral cavity is observed in the presence of various metals (prostheses and fillings). The presence of various metals and large microgalvanic flow in the oral cavity is observed in the presence of various metals (prostheses and fillings). The presence of various metals and large microcurrents does not always cause pathological changes, but in some people even small currents can cause them. Conditionally, up to 10 microflows in the oral cavity are the norm. Their growth is caused by a significant (compared to the norm) amount of solution formed when the technology of prosthetic production is broken. The largest microcurrent is formed by the following substances: gold-amalgam, steel - solute, amalgam-solute and amalgam-steel. The largest microcurrent is formed by the following substances: gold-amalgam, steel - solute, amalgam-solute and amalgam-steel. Galvanic flow plays a certain role in the occurrence of inflammation. The largest microcurrent is formed by the following substances: gold-amalgam, steel - solute, amalgam-solute and amalgam-steel. Galvanic flow plays a certain role in the occurrence of inflammatory processes of Oral mucosa, especially leukoplakia and KPL, as well as in the exacerbation of existing pathological processes. In many patients with leukoplakia and KPL, the resolution of the process or a more favorable course of the disease is caused by the replacement of some prostheses with others, which leads to a decrease in the size of the lesion microcurrents. The effect occurs after 1-4 months. It should also be remembered about the possibility of increasing the sensitivity of the mucous membrane to Mercury, Silver and other substances. The effect occurs after 1-4 months. It should also be remembered about the possibility of increasing the sensitivity of the mucous membrane to Mercury, Silver and other substances.

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Photo 35. Galvanism

Damage with light. Light disease (morbus radialis) develops as a result of the action of ionizing radiation on the entire body or large parts of it: chest, abdomen, pelvis. Damage with light. Light disease (morbus radialis) develops as a result of the action of ionizing radiation on the entire body or large parts of it: chest, abdomen, pelvis. Damage with light can cause any type of ionizinge.

Acute radiation sickness (morbus radialis) develops after a single radiation of the body in large doses (1-10 Gr or 100-1000 rad). Acute radiation sickness (morbus radialis) develops after a single radiation of the body in large doses (1-10 Gr or 100-1000 rad). In the first period-the period of primary reactions, which occurs shortly after irradiation (1-2 hours later) acute radiation sickness (morbus radialis) develops after a single radiation of the body in large doses (1-10 Gr or 100-1000 rad). In the first period-the period of primary reactions, which occurs shortly after irradiation (1-2 hours later) and lasts up to 2 days, dryness (or saliva) appears in the oral cavity, the taste and sensitivity of the mucous membrane decreases. The mucous membrane of the mouth and lips swells, hyperemia appears and blemish bleeding may occur. In the second, latent period, which lasts from several hours to 2 weeks, all these phenomena pass. The period of pronounced clinical signs (the third period) is the onset of the disease. Against the background of a sharp deterioration in the general condition, changes in the oral cavity reach their maximum. The period of pronounced

clinical signs (the third period) is the onset of the disease. Against the background of a sharp deteriorationThe period of pronounced clinical signs (the third period) is the onset of the disease. Against the background of a sharp deterioration in the general condition, changes in the oral cavity reach their maximum. A burning sensation appears, the mucous membrane becomes anemic and becomes dry. Clinical signs of Ray stomatitis are observed.Changes in blood vessels and blood composition lead to the development of hemorrhagic diathesis. Rapidly developing autoinfection, especially to rot, due to a sharp decrease in tissue resistance. Thus, the clinical picture of radiant stomatitis consists mainly of hemorrhagic syndrome and ulcerative necrotic process.hus, the clinical picture of radiant stomatitis consists mainly of hemorrhagic syndrome and ulcerative necrotic process. The latter is most clearly manifested in damaged areas (filler, tartar) and the accumulation of microflora before the diThus, the clinical picture of radiant stomatitis consists mainly of hemorrhagic syndrome and ulcerative necrotic process. The latter is most clearly manifested in damaged areas (filler, tartar) and the accumulation of microflora before the disease, that is, first on the edge of the gums and tonsils, and then on the lateral surfaces of the tongue, on the palate.Damage can be more pronounced due to secondary radiation in areas adjacent to the mucous membrane of metal prostheses and fillings. The mouth, mucous membrane and mucous membrane of the face swell. Gum suckers, loose, then the gum edges undergo necrosis. The bone tissue of the alveolar process resorbs, the teeth become loose and fall out.um suckers, loose, then the gum edges undergo necrosis. The bone tissue of the alveolar process resorbs, the teeth become loose and fall out. Most necrosis of the mucous membrane does not have sharp boundaries, the inflammatory reaum suckers, loose, then the gum edges undergo necrosis. The bone tissue of the alveolar process resorbs, the teeth become loose and fall out. Most necrosis of the mucous membrane does not have sharp boundaries, the inflammatory reaction of the surrounding tissues is weakly expressed. The bottom of the wounds is covered with dirty gray necrotic plaque with a rotten smell. In severe cases, necrosis can spread from the mucous membrane to the lower soft tissues and bone, with sequestration, light necrosis of the bone occurs,

there is a fracture of the jaws. This is facilitated by the spread of infection from chronic periodontitis and foci of periodontitis. This is facilitated by the spread of infection from chronic periodontitis and foci of periodontitis.

The tongue swells, becomes covered with plaque, cracks, bleeding and necrosis appear, of this is facilitated by the spread of infection from chronic periodontitis and foci of periodontitis.

The tongue swells, becomes covered with plaque, cracks, bleeding and necrosis facilitated by the spread of infection from chronic periodontitis and foci of periodontitis.

Chronic radiation sickness (morbus radialis chronicus) develops as a result of prolonged exposure to low doses of radiation throughout the body or an important part of it. Chronic radiation sickness (morbus radialis chronicus) develops as a result of prolonged exposure to low doses of radiation throughout the body or an important part of it. The oral cavity is sensitive to the effects of ionizing chronic radiation sickness (morbus radialis chronicus) develops as a result of prolonged exposure to low doses of radiation throughout the body or an important part of it. The oral cavity is sensitive to the effects of ionizing radiation, so changes in the oral cavity at the beginning of the disease can be particularly pronounced. Dryness gradually increases due to damage to the salivary glands. Permanent catarrhal gingivitis occurs, which can later develop into a wound. Erosion can occur in the folds of the groove from the vestibular side, followed by aphthous-type wounds, which then penetrate into the gums and lips. Erosion can occur in the folds of the groove from the vestibular side, followed by aphthous-type wounds, which then penetrate into the gums and lips. Glossalgia can develop, and then with swelling of the tongue, glossitis, cracks in the tongue, cracks appear.

Treatment. Both general and local therapy is carried out for radiation injuries. Both general and local therapy is carried out for radiation injuries. The scope of therapeutic measures varies depending on the degree and stage of the treatment. Both general and local therapy is carried out for radiation injuries. The scope of therapeutic measures varies. Both general and local therapy is

carried out for radiation injuries. The scope of therapeutic measures varies depending on the degree and stage of the disease, the localization of jarokhat, the state of various organs and systems. General treatment involves the use of drugs that slow down light reactions — radioprotectors (cystamine, bathylene, etc. reducing the radiosensitivity of the body, corticosteroids, antihistamines (suprastin, tavegil, fencarol), antibacterial drugs (broad-spectrum antibiotics). Substances that restore the functions of the blood system (vitamins B6 and B12, folic acid, sodium nucleinate, pentoxyl, coamide), prevention of hemorrhagic diathesis (rutin, vicasol, calcium gluconate), multivitamins and substances that carry out blood transfusions are also used.

Neoplasms of the oral mucosa

In the treatment of jarochates in the facial jaw socket, methods of nurtherapy are used: remote, short-focus and interstitial. In the treatment of jarochates in the facial jaw socket, methods of nurtherapy are used: remote, short-focus and interstitial. In addition to the treatment of jarochates in the facial jaw socket, methods of nurtherapy are used: remote, short-focus and interstitial. In addition to the affected tissues, normal tissues, including the mucous membrane of the mouth, also fall into the radiation field. The effect of its reaction to radiation is very common, depending on the speed of the dose, the dose of single and total absorbed radiation, the duration of exposure, the biological characteristics of the body, as well as the condition of the oral cavity before irradiation. The clinical picture of the radiation response of the mucous membrane develops gradually — from hyperemia and swelling to the appearance of erosion. This reaction has its own characteristics in different areas of the mucous membrane. The clinical picture of the radiation response of the mucous membrane develops gradually — from hyperemia and swelling to the appearance of erosion. This reaction has its own characteristics in different areas of the mucous membrane. The first clinical signs on the mucous. Then the mucous membrane becomes dull, loses its brightness, thickens and folds appear that is, when scraping, the surface layer is not removed. This is due to increased keratinization of the epithelium. Then the mucous membrane becomes dull, loses its brightness, thickens and folds appear

that is, when scraping, the surface layer is not removed. This is due to increased keratinization of the epithelium. Some affected areas are similar to leukoplakia or QYaT. If the dose of radiation is increased, the molten epithelium is first rejected in some places, desquamation, erosions appear, covered with sticky necrotic caraches — a focal-film radiomucosite, then the epithelium is rejected in large areas, the erosions merge and the focal radiomucosite becomes a drainage-film radiomucosite. If the dose of radiation is increased, the molten epithelium is first rejected in some places, desquamation, erosions appear, covered with sticky necrotic caraches — a focal-film radiomucosite, then the epithelium is rejected in large areas, the erosions merge and the focal radiomucosite becomes a drainage-film radiomucosite. The mucous membrane of the soft palate has a high radiosensitivity, no branching is observed when irradiated, the reaction develops faster than in other parts of the oral cavity. In areas of the mucous membrane that usually undergo branching, the radiation reaction proceeds more positively and leads only to focal desquamation of the epithelium or one erosion. In areas of the mucous membrane that usually undergo branching, the radiation reaction proceeds more positively and leads only to focal desquamation of the epithelium or one.



Photo 36. The drainage film is radiomucosite.

On the side surface of the tongue, against the background of pronounced hyperemia, there is extensive erosion, covered with Necrotic Carache

Taste buds on the tongue die, resulting in a loss of taste. At first, sensations in the tongue can manifest as glossalgia, then taste disorders appear, and then its loss is detected. Radiation changes in the oral cavity are mostly reversible. After irradiation stops or during a treatment break, the mucous membrane quickly returns to relative normal. This period lasts 2-3 weeks. In a large dose (more than 50-60 Gy or 5000-6000 rad), irreversible changes in the salivary glands and mucous membranes (edema, hyperemia, telangiectasia, atrophy, radiation ulcers) can occur. In a large dose (more than 50-60 Gy or 5000-6000 rad), irreversible changes in the salivary glands and mucous membranes (edema, hyperemia, telangiectasia, atrophy, radiation ulcers) can occur. Oral hygiene must be performed before light therapy, since the reaction of the mucous membrane to the action of light is more severe in the unhygienic oral cavity. Due to trophic disorders, the reactivity of the mucous membrane to mechanical damage and infection decreases sharply. A swollen mucous membrane with an incomplete epithelial cap is easily damaged by the sharp edges of the dentures, which can lead to the appearance of sharply painful, long-lasting wounds.

Treatment. Preparing the oral cavity for radiation therapy is necessary to mitigate the radiation reaction during treatment and prevent its complications. Preparing the oral cavity for radiation therapy is necessary to mitigate the radiation reaction during treatment and prevent its complications. It is recommended to carry out therapeutic measures in the oral cavity in this sequence: All roots and teeth with periodontitis are removed under anesthesia, as well as sharply moving teeth are removed 3-5 days before the start of radiation therapy. All roots and teeth with periodontitis are removed under anesthesia, as well as sharply moving teeth are removed 3-5 days before the start of radiation therapy. The removal should be less

traumatic, after which the wound should be sutured, for faster healing. The removal of tartar on the surface of the gums and under the gums consists in the removal of gum and periodont tissue, curative peeling, treatment of caries teeth, removal of metal prtoes. The removal of tartar on the surface of the gums and under the gums consists in the removal of gum and periodont tissue, he removal of tartar on the surface of the gums and under the gums consists in the removal of gum and periodont tissue, curative peeling, treatment of caries teeth, removal of metal prtoes. To the removal of tartar on the surface of the gums and under the gums consists in the removal of gum and periodont tissue, curative peeling, treatment of caries teeth, removal of metal prtoes. To reduce the reaction of the mucous membrane before irradiation, it is recommended to rinse the oral cavity more often. Administration of adrenaline in an isotonic solution of sodium chloride (2: 100) or adrenaline under the skin, treatment of the mucous membrane with an emulsion of tezan, 5% dietone ointment. During light therapy, it is forbidden to smoke, drink alcohol, wear irritating food and removable dentures. Soft food should be non-irritating, high-calorie and fortified. oft food should be non-irritating, high-calorie and fortified. At the first manifestations of a light reaction, it is recommended to rinse your mouth with waoft food should be non-irritating, high-calorie and fortified. At the first manifestations of a light reaction, it is recommended to rioft food should be non-irritating, high-calorie and fortified. At the first manifestations of a light reaction, it is recommended to rinse your mouth with warm boiled water, a solution of potassium permanganate (1:5000). The oral cavity, especially the teeth and gums, is sanitized 4-5 times a day with tampons moistened with weak solutions of antiseptics. (0.5% hydrogen peroxide solution, 1:5000 furacilin solutions, 0.25% chloramine solution, 2% boric acid solution, 10% dimexide solution application), aerosol "Lioxa sol". In the middle of the radiant reaction, the oral cavity and gum pockets from the syringe are washed with solutions of weak antiseptics, the mucous membrane is lubricated with a 1% solution of citral in peach or other vegetable oil, applied with Le Vosin ointments, methyluracylic acid. n the middle of the radiant reaction, the oral cavity and gum pockets from the syringe are washed with solutionn the middle

of the radiant reaction, the oral cavity and gum pockets from the syringe are washed with solutions of weak antiseptics, the mucous membrane is lubricated with a 1% solution of citral in peach or other vegetable oil, applied with Le Vosin ointments, methyluracilic acid. For maxillary anesthetic, 1-2% solutions of trimecaine, lidocaine or 10% fatty solution of anesthetic are used. Surgical intervention, including tooth removal, at which time the paradontal pocket curettage karshi is indicated. You should give them up for months or years. Relative normalization of the mucous membrane occurs after 1-2.5 months.ou should give them up for months or years. Relative normalization of the mucous membrane occurs after 1-2.5ou should give them up for months or years. Relative normalization of the mucous membrane occurs after 1-2.5 montu should give them up for months or years. Relative normalization of the mucous membrane occurs after 1-2.5 months. In the case of a radiotherapy wound, in addition to general (vitamin therapy), local treatment is necessary. Rinsing the oral cavity with antiseptic melts and treating the light wound under anesthesia is carried out daily. Necrotic tissues are removed using solutions of proteolytic enzymes (lysoamidase, ronidase, Dncase, etc. For a long time (up to 1.5—2 months), ointments ("Iruksol", "Levosin", "Propolis", "Solkoseryl", "Actovegin", etc.or a long time (up to 1.5—2 months), ointments ("Iruksol", "Levosin", "Propolior a long time (up to 1.5—2 months), ointments ("Iruksol", "Levosin", "Propolis", "Solkoseryl", "Actovegin", etc.), fortified oils (sea buckthoor a long time (up to 1.5—2 months), ointments ("Iruksol", "Levosin", "Propolis", "Solkoseryl", "Actovegin", etc.), fortified oils (sea buckthorn, rose), galascorbine are applied to the wound. Long-term incurable light wounds that are not suitable for conservative treatment can then be radically cut in healthy tissues with plasticity. The surgical method is indicated in the case of local radiation (short-focus X-ray therapy, interstitial gamma therapy). With remote radiotherapy methods, the effectiveness of surgical treatment will be less. If necessary, radiation damage to the teeth and radiation osteomyelitis should be treated.

Leukoplakia (Leykoplakiya)

Leukoplakia is a chronic disease of the oral mucosa or the red border of the lips, accompanied by epithelial swelling and inflammation of the mucosa itself. Leukoplakia is observed not only in the oral cavity, but rarely in other mucous membranes (bladder, genitals, rectum, nose, esophagus, trachea). Leukoplakia is very rare in children. The disease is observed mainly in middle-aged and elderly people, more often in men, which is probably associated with the effects of a large number of substances with strong damaging properties on the oral mucosa (smoking, alcohol, mechanical injuries). Leukoplakia is considered a precancerous disease.

Etiology. Local factors (mechanical, thermal, chemical) play a major role in the development of leukoplakia. The most common cause of leukoplakia is smoking, in which the oral mucosa is exposed to substances that give thermal and chemical wound properties (ammonia and phenolic compounds, nicotine, resin derivatives). In the development of leukoplakia, the influence of weak, but long-standing damaging substances is significant: very hot or spicy food, strong alcoholic beverages, tobacco smoking, betel, naswar use (Naswar includes ash, tobacco, slaked lime, cottonseed oil or water), meteorological factors (hard insulation, cold, wind). Mechanical damage can be caused by sharp edges of teeth, roots of teeth, a large number of dental calculus, poor quality dentures, defects of teeth, anomalies in the position of individual teeth. Oral metal fillings with various metals.

Clinical presentation. The initial manifestations of leukoplakia are usually ignored, as subjective signs are absent. Leukoplakia usually begins with the appearance of opacity of the epithelium, branching on the surface, the branched surface is covered with gray elements. The lesion site appears on the unchanged mucous membrane, leukoplakia is typically located on the mucous membrane of the cheeks, along the line of closure of anterior teeth, in the corners of the mouth and on the red border of the lower lip. The posterior and lateral surfaces of the tongue are slightly less affected. Smokers are characterized by lesions of the palate, as well as foci of hyperkeratosis in the corners of the mouth, in "tappeiner smoker leukoplakia." The following forms of leukoplakia are distinguished: flat or simple, verrucous, and

erosive. These shapes can transition into each other. In the same patient, various forms of leukoplakia in different areas of the oral mucosa can be combined.

Flat or simple leukoplakia (leucoplakia plana) is common. This form usually does not cause subjective signs and is determined by chance. Flat or simple leukoplakia (leucoplakia plana) is common. This form usually does not cause subjective signs and is determined by chance. Sometimes patients complain of a feeling of gravity, a burning sensation and an flat or simple leukoplakia (leucoplakia plana) is common. This form usually does not cause subjective signs and is determined by chance. Sometimes patients complain of a feeling of gravity, a burning sensation and an unusual appearance of the mucous membrane. In the presence of wide ovens on the tongue, a decrease in taste sensitivity can be observed. On the mucous membrane in flat leukoplakia, the main morphological element is a hyperkerototic stain, a pale area of the epithelium with a clear outline. The examination reveals foci of hyperkeratosis of different shapes and sizes, in which the mucous membrane of the surrounding Sox does not rise upward, but there are clear boundaries of inflammation. The examination reveals foci of hyperkeratosis of different shapes and sizes, in which the mucous membrane of the surrounding Sox does not rise upward, but there are clear boundaries of inflammation. The examination reveals foci of hyperkeratosis of different shapes and sizes, in which the mucous membrane of the surrounding Sox does not rise upward, but there are clear boundaries of inflammation. The elements of flat leukoplakia resemble a burn of the mucous membrane with lyapis, a thin papyrus glued with paper or white plaque that cannot be removed even with intensive scraping. Branching can be of different intensity, so the color of the affected areas varies from light gray to intense White. The surface of flat leukoplakia is usually slightly rough and dry. There is a direct relationship between the shape, color, size of the branching sites and their location. The surface of flat leukoplakia is usually slightly rough and dry. There is a direct relationship between the shape, color, size of the branching sites and their location. Thus, in 85% of cases with hyperkeratosis of the mucous membrane in the

area of the corners of the mouth, jarochate symmetry is noted. The furnace has a triangular shape, with the base facing the corner of the mouth and the top facing the retromolar space. When leukoplakia is located on the mucous membrane of the lunges along the line of chipsification of the incisors, the jarochate elements are an elongated line, the continuity of which can be broken in certain places. When leukoplakia is located on the mucous membrane of the lunges along the line of chipsification, when leukoplakia is located on the mucous membrane of the lunges along the line of chipsification of the incisors, the jarochate elements are an elongated line, the continuity of which can be broken in certain places. When leukoplakia is located on the mucous membrane of the lunges along the line of chipsification of the incisors, the jarochate elements are an elongated line, the continuity of which can be broken in certain places. Leukoplakia on the red border of the lips looks like gray-white papyri paper of irregular shape. On the mucous membrane of the tongue, the hard palate and the lower part of the oral cavity, limited jarochate areas are formed, sometimes resembling wide stripes or hard spots. In addition, wider jarocats are observed. The relief of the mucous membrane, its turgor, is also reflected in the form of jarochat. The relief of the mucous membrane, its turgor, is also reflected in the form of jarochat. Thus, if leukoplakia develops against the background of a twisted tongue, the relief of the mucous membrane, its turgor, is also reflected in the form of the relief of the mucous membrane, its turgor, is also reflected in the form of jarochat. Thus, if leukoplakia develops against the background of a twisted tongue, the protruding areas branch, and the surface of the tongue resembles a stony coating. A similar appearance can be seen with a decrease in turgor and folds on lunges. If there is no clear fold on the tongue, flat leukoplakia looks like gray-white, slightly sinking spots with flattened tongue suckers. Under favorable conditions, flat leukoplakia can exist for years, decades and does not harm, does not develop and does not develop into more pronounced forms of the disease.



Figure 37.Flat leukoplakia.

The appearance of hyperkeratosis with clear boundaries on the lower lip

Tappeiner smoker's leukoplakia (leucoplakia nicotinic Tappeiner, nicotinic leukoceratosis of the palate) occurs in hyperkeratosis of the hard and soft palate mucosa with small red nodules - hyperplastic salivary glands and an enlarged outlet duct point. Tappeiner smoker's leukoplakia (leucoplakia nicotinic Tappeiner, nicotinic leukoceratosis of the palate) occurs in hyperkeratosis of the hard and soft palate. Tappeiner smoker's leukoplakia (leucoplakia nicotinic Tappeiner, nicotinic leukoceratosis of the palate) occurs in hyperkeratosis of the hard and soft palate mucosa with small red nodules - hyperplastic salivary glands and an enlarged outlet duct point. The mucous membrane of the hard palate and the adjacent part of the soft palate are mainly affected. Sometimes the tooth is involved on the edge of the gums. The mucous membrane in the affected area is gray-white, often blurred with folds. In this, basically, a red spot stands out on the back half of the hard palate –the cyst looks like small knots. They are formed by hyperkeratic masses due to clogging of excretory ducts.



Photo 38. Tappeiner's leukoplakia.

Hyperkeratosis of the mucous membrane of the hard and soft palate with small red nodules - a point of hyperplastic salivary glands and an enlarged excretory canal.

In smoker's leukoplakia, damage to the palate can be combined with the arrangement of elements on the mucous membrane of lunge, in the corners of the mouth, on the lower lip, etc.n smoker's leukoplakia, damage to the palate can be combined with the arrangement of elements on the mucous membrane of lunge, in the core



Photo 39.

Hyperkeratotic plates with an uneven surface protruding above the lunge mucosa

Verrucous leukoplakia (leucoplaria verrucosa) is the next stage in the development of flat leukoplakia. verrucous leukoplakia (leucoplaria verrucosa) is the next stage in the development of flat leukoplakia. This is facilitated by substances that have a local excitatory property: trauma with sharp edges of Verrucous leukoplakia (leucoplaria verrucosa) is the next stage in the development of flat leukoplakia. This is facilitated by substances that have a local excitatory property: trauma with sharp edges of teeth and dentures, biting areas of leukoplakia, smoking, eating hot and spicy food, microtoxias, etc. The main feature that distinguishes this form of leukoplakia from the flat form is branching, in which a significant thickening of the cornea is determined. The area of leukoplakia rises significantly above the level of the mucous membrane, and the color is sharply differentiated. During

palpation, hardness on the surface can be determined. The area of leukoplakia rises significantly above the level of the mucous membrane, and the color is sharply differentiated. During The area of leukoplakia rises significantly above the area of leukoplakia rises significantly above the level of the mucous membrane, and the color is sharply differentiated. During palpation, hardness on the surface can be determined. Patients usually complain of roughness and tightness of the mucous membrane, burning and feeling of pain during meals, sharpness. There are blyashkali and soggy forms of leukoplakia. The plaque shape of leukoplakia is limited to milk-white, sometimes with a straw-yellow plaque appearance that rises above the surrounding mucous membrane and has clear edges. The soggy type of varicose leukoplakia is characterized by dense sectional formations that sharply protrude above the level of the hard Gadir budir mucous membrane. The soggy type of varicose leukoplakia is characterized by dense sectional formations that sharply protrude above the level of the hard Gadir budir mucous membrane. The soggy form of leukoplakia has great potential for malignancy compared to the type of blyashkali. During palpation, the pathological elements are dense, painless and combined with the lower parts of the mucous membrane. The thickness of the raised areas of hyperkeratosis varies and is not noticeable during a simple examination. The slight elevation of the elements is almost undetectable in the polished section. When the leukoplakic furnace is large, it thickens, becomes dense, and it cannot be folded.



Figure 40. Erosive ulcerative leukoplakia.
erosion; branching area of the lunge mucosa

Erosive leukoplakia (leucoplakia erosiva) is actually a complication of simple or verrucous leukoplakia affected by trauma. Erosive leukoplakia (leucoplakia erosiva) is actually a complication of simple or verrucous leukoplakia affected by trauma. With erosive leukoplakia (leucoplakia erosiva) is actually a complication of simple or verrucous leukoplakia affected by trauma. With this form of leukoplakia, patients complain of pain, which is exacerbated by all types of factors (eating, speaking, etc.). Usually, against the background of ordinary or verrucous leukoplakia foci, erosion, cracks and deeper wounds appear. Erosions are difficult to epithelialize and often recur. Patients are especially concerned about erosion in the area of the red border of the lips. Under the influence of insolation and other annoying factors, they increase in size. The pain increases at the same time. Under the influence of insolation and other annoying factors, they increase in size. The pain increases at the same time. Pathohistological changes in leukoplakia are characterized by thickening of the epithelium due to increased keratinization. Under the influence of insolation and other annoying factors, they increase in size. The pain increases at the same time. Pathohistological changes in leukoplakia are characterized by thickening of the epithelium due to increased keratinization and granular layers. The keratin layer reaches considerable thickness, especially in the verrucous form of leukoplakia. In it, hyperkeratosis foci often alternate with parakeratosis foci. The granular layer of the epithelium in the field of Pathology has different degrees of development. The verrucous and erosive ulcerative form of leukoplakia is characterized by a pronounced thickening of the epithelium due to hyperkeratosis and acanthosis. The verrucous and erosive ulcerative form of leukoplakia is characterized by a pronounced thickening of the epithelium due to hyperkeratosis and acanthosis. Damaged surface of the mucous membrane diffuse chronic

inflammation in the connective tissue stroma is determined by the pronounced infiltration of its surface layers by lymphocytes and plasma cells, as well as the manifestation of sclerosis. The latter can end poorly and often recur with the appearance of cracks and erosions, which are most characteristic of the plaque form of leukoplakia. The latter can end poorly and often recur with the appearance of cracks and erosions, which are most characteristic of the plaque form of leukoplakia. Leukoplakia is included in the group of pre-cancer diseases, since in all its forms it undergoes a process of various degrees of risk with varying extentionality, spinocellular turns into cancer. Flat leukoplakia is malignant in 3-5% of patients, and in some the process of malignancy proceeds rapidly (1 - 1.5 years), in others the disease can exist for decades without turning into cancer. Most often, malignancy of the verrucous and erosive form of leukoplakia occurs (in 20-30% of cases). Most often, malignancy of the verrucous and erosive form of leukoplakia occurs (in 20-30% of cases). Clinical signs of malignancy are an increase in branching processes; a rapid increase in the size and density of the lesion; the surface under the plaque hardens.

The diagnosis is made by urgent cytological or histological examination.

Comparative diagnosis. It is necessary to distinguish between leukoplakia: Red flat iron, red runner, secondary syphilis, chronic hyperplastic candidiasis, clouding of the epithelium in the process of regeneration, Bowen's disease; mild leukoplakia, squamous cell carcinoma. It is necessary to distinguish between leukoplakia: Red flat iron, red runner, secondary syphilis, chronic hyperplastic candidiasis, clouding of the epithelium in the process of regeneration, Bowen's disease; mild leukoplakia, squamous cell carcinoma. Leukoplakia differs in the nature of the pathological elements of QYaT: in leukoplakia, this is a hyperkeratotic point or plaque; in QYaT, there are several papules on the mucous membrane of the mouth, which, as a rule, combine turbidly, and in some cases the skin has several papules. Leukoplakia never affects the

skin. leukoplakia never affects the skin. Signs of difference also include the absence of inflammatory phenomena in leukoplakia, and the presence of inflammation of the oral mucosa at QYaT. leukoplakia never affects the skin. Signs of difference also include the absence of inflammatory phenomena in leukoplakia, and the presence of inflammation of the oral mucosa at QYaT (in addition to the typical form). Leukoplakia differs from Red runner in the absence of bright erythema with typical contours, atrophy and characteristic skin damage. In the comparative diagnosis of leukoplakia with syphilitic papules, the following differences are taken into account: horny masses in leukoplakia are not removed by scraping, gray-white plaque covering syphilitic papules is easily removed, erosion occurs, many pale trepanomas are found on its surface. In the comparative diagnosis of leukoplakia with syphilitic papules, the following differences are taken into account: horny masses in leukoplakia are not removed by scraping, gray-white plaque covering syphilitic papules is easily removed, erosion occurs, many pale trepanomas are found on its surface. Leukoplakia does not contain infiltration typical of syphilitic papules. The Wasserman reaction and deposition reactions in secondary syphilis are generally positive. Unlike leukoplakia, bleaching caraches in chronic hyperplastic candidiasis are removed during scraping and a large number of fungi of the genus *Candida* are found in it in the form of spores or pseudomycelia. Like leukoplakia, bleaching caraches in chronic hyperplastic candidiasis are removed during scraping and a large number of fungi of the genus *Candida* are found in it in the form of spore-like leukoplakia, bleaching caraches in chronic hyperplastic candidiasis are removed during scraping and a large number of fungi of the ge.

Leukoplakia is distinguished from soft leukoplakia, which has a white-gray appearance. leukoplakia is distinguished from soft leukoplakia, which has a white-gray appearance. Unlike true leukoplakia, with mild leukoplakia leukoplakia is distinguished from soft leukoplakia, which has a white-gray appearance. Unlike true leukoplakia, with mild leukoplakia, this white-gray plaque is removed by scraping, as this is the result of paracatosis. Branching flat-cell cancer, unlike leukoplakia, has

a solid bottom and a characteristic Cytological and histological picture - the presence of atypical cells.

When leukoplakia settles on the red border of the lip, a differential diagnosis is made with limited precancerous hyperkeratosis. When leukoplakia settles on the red border of the lip, a differential diagnosis is made with limited precancerous hyperkeratosis. It is dist.

Treatment. The volume of therapeutic measures for leukoplakia is primarily determined by its clinical form, as well as the volume of jaroxat and the speed of development of the process. Treatment. The volume of therapeutic measures for leukoplakia is primarily determined by its clinical form, as well as the volume of jaroxat and the speed of development of the process. Once flat treatment. The volume of therapeutic measures for leukoplakia is primarily determined by its clinical form, as well as the volume of jaroxat and the speed of development of the process. Once flat leukoplakia has eliminated the annoying factors, quitting smoking can go back with the complete loss of hyperkeratosis foci. However, when smoking continues, leukoplakia develops again. The treatment of flat leukoplakia consists of the following steps: elimination of factors that contribute to local damaging properties; normalization of metabolic processes in the epithelium with the help of multivitamins and, above all, vitamin A.

Patients thoroughly sanitize the oral cavity by grinding the sharp edges of the teeth; amalgama fillings, if indicated, are replaced with plastic or cement. Patients thoroughly sanitize the oral cavity by grinding the sharp edges of the teeth; amalgama fillings, if indicated, are replaced with plastic or cement. When leukoplakia is located on the red border of the lips, patients should undergo treatment. Patients thoroughly sanitize the oral cavity by grinding the sharp edges of the teeth; amalgama fillings, if indicated, are replaced with plastic or cement. When leukoplakia is located on the red border of the lips, patients should undergo as little insolation as possible, while going out, the lips should be lubricated with a photoprotective cream "Ray" or hygienic photoprotective lipstick. It is very important to completely stop smoking, eating hot, spicy foods and alcohol. The risk

of smoking should be explained to the patient and, if necessary, sent for special treatment. The risk of smoking should be explained to the patient and, if necessary, sent for special treatment. In addition to eliminating the factors that characterize local damage, the treatment of inflammation. Vitamin A is prescribed inside (3.4% retinol acetate in oil or 5.5% retinol palmitate in oil) 10 drops 2-3 times a day for 1.5—2 months. Vitamin A is prescribed inside (3.4% retinol acetate in oil or 5.5% retinol palmitate in oil) 10 drops 2-3 times a day for 1.5—2 months. Local applications are carried out 3-4 times a day with these fatty solutions of vitamin A. Treatment lasts 1.5—2 months. The course of treatment is repeated 2-3 times a year. These therapeutic measures usually lead to the loss of leukoplakia or its stabilization. If candidiasis is detected with leukoplakia, anti-candidal treatment is carried out at the same time. Treatment of verrucous and erosive forms of leukoplakia is carried out like flat leukoplakia, if it does not lead to a positive result within 1 month (the transition of the form of verrucous to flat), surgical methods of treatment should be used: removal of the pathological focus in healthy tissues, followed by histological examination. Treatment of verrucous and erosive forms of leukoplakia is carried out like flat leukoplakia, if it does not lead to a positive result within 1 month (the transition of the form of verrucous to flat), surgical methods of treatment should be used: removal of the pathological focus in healthy tissues, followed by histological examination. Removal of leukoplakia sites can be done by electrocoagulation or cryodestruction. To prevent the process from becoming malignant, special attention should be paid to the treatment of erosions and ulcers on the surface of leukoplakia foci. To prevent the process from becoming malignant, special attention should be paid to the treatment of erosions and ulcers on the surface of leukoplakia foci. Treatment of the erosive form of leukoplakia to prevent the process from becoming malignant, special attention should be paid to the treatment of erosions and ulcers on the surface of leukoplakia foci. Treatment of the erosive form of leukoplakia, in addition to the above, involves the local use of drugs that stimulate the epithelization of the mucous

membrane (vitamins A, ye, oblipixa,namatak oil.) and in the presence of pain-local pain relievers (lidocaine, trimecaine, pyromecaine) are used. If erosion or wounds under the influence of drug treatment are not epithelialized Within 2 weeks, then with an urgent histological examination I will identify a pathological foci.f erosion or wounds under the influence of drug treatment are not epithelialized Within 2 weeks, thenf erosion or wounds under the influence of drug treatment are not epithelialized Within 2 weeks, then with an urgent histological examination I will identify a pathological foci. Patients with leukoplakia should be under constant medical supervision of a dentist every 3-4 months. In the slightest doubt about the possibility of malignancy, it is necessary to immediately carry out a biopsy, and then a histological examination, after which further treatment tactics should be solved.

Preventionrevention. To prevent leukoplakia, timely prevention of damage to the oral mucosa is considered: smoking, hot and spicy food, poor-quality dentures, sharp edges of the teeth, galvanic currents and other harmful characteristic.

Pashkov's soft

Soft leukoplakia B. M. Pashkov described mild leukoplakia in 1964, a disease that other authors also call "leukodema" (the surface of the mucous membrane is smooth) and "lunge bite" (the surface is rough). Mild leukoplakia (leucoplakia mollis) is a good quality lesion of Oral mucosa. It is characterized by periodic desquamation of the surface layers of the epithelium.ild leukoplakia (leucoplakia mollis) is a good quality lesion of Oral mucosa. It is characterized by periodic desquamation of the surface layers of the epithelium. Mild leukoplakia is obserild leukoplakia (leucoplakia mollis) is a good quality lesion of Oral mucosa. It is characterized by periodic desquamation of the surface layers of the epithelium. Mild leukoplakia is observed mainly in young people, mainly children and people under 30 years of age, more often in women and usually occurs without pronounced subjective sensations. It is mainly found during dental treatment or during preventive examination of the oral cavity. Sometimes patients complain of roughness, peeling of the mucous membrane, a feeling of thickened connective tissue. Often patients try to get rid of these sensations by biting high places, which often becomes a habit.

Etiology. The disease is hereditary, it is a type of epithelial dysplasia.

It is characterized by the presence of piling areas of a slightly swollen pasty mucous membrane, without inflammation and thickening of which the Cheras are not clear. The mucous membrane is gray-white. It is characterized by the presence of piling areas of a slightly swollen pasty mucous membrane, without inflammation and thickening of which the Cheras are not clear. The mucous membrane is gray-white. Most often, soft leukoplakia settles on the lunge, on the mucous membrane of the lips, less often on the tongue and gums. The lesion may be limited, located along the line of closure of the teeth on the mucous membrane, or diffuse, in the process of which almost the entire mucous membrane, and sometimes the red border of the lips, is involved. With the usual biting or sucking of lungen, lips, tongue, often observed in patients with age neuropathy, the epithelium along the line of tooth cipslaing is unevenly desquamated, with a hairy appearance due to numerous tiny pores. usual biting or sucking of lungen, lips, tongue, often observed in patients with age neuropathy, the epithelium along the line of tooth cipslaing is unevenly desquamated, with a hairy appearance due to numerous tiny pores. This altered epithelium is partially removed by scraping. In severe cases, painful erosion may occur when the epithelium is scraped.

Pathological anatomy. Histologically, paraceratosis, acanthosis have been recorded, at all levels of the thoracic layer there will be light cells whose cytoplasm is almost unpainted and whose nuclei are deformed (pyncnotised).athological anatomy. Histologically, paraceratosis, acanthosis have been recorded, at all levels of the thoracic layer there will be light cells whose cytoplasm is almost unpainted and whose nuclei are deformed (pyncnotised). In the layer of connective tissue, there is an expansion of small blood vessels, thickening of collagen, thinning of elastic fibers.

Diagnosis. What a challenge this hych does not cause.iagnosis. What a challenge this hych does not cause. The usual appearance of the mucous membrane and the

habit of constantly biting lunge, often pressing the mucous membrane between the teeth wi.



Figure 41. Mild leukoplakia.

The mucous membrane of the lunge, has a white color

Comparative diagnosis. The disease should be distinguished from: Flat leukoplakia, typical form of kyat, candidiasis, medicamentous stomatitis. Treatment comparative diagnosis. The disease should be distinguished from: Flat leukoplakia, typical form of kyat, candidiasis, medicamentous stomatitis.

Treatment. An oily solution of vitamin A, vitamins B, C is prescribed, and the oral cavity is sanitized. Treatment sometimes helps to eliminate the process, but often only temporary improvement is possible.

Kennon's white sponge nevus. For the first time in 1935, Cannon had a distinct clinical and histological structure, and "white gubchatius nevus" (naevus spongiosus Cannon). or the first time in 1935, Cannon had a distinct clinical and histological structure, and "white gubchatius nevus" (naevus spongiosus Cannon). described a good quality epithelial dysplasia of the so-called Oral mucosa. Many researchers believe that this disease is inherited in a hereditary and autosomal dominant type.

Clinical picture. The white sponge nevus can appear in early childhood or later, then grows and reaches its maximum development in adulthood. Clinical picture. The white sponge nevus can appear in early childhood or later, then grows and reaches its maximum development in adulthood. In addition, Clinical picture. The white sponge nevus can appear in early childhood or later, then grows and reaches its

maximum development clinical picture. The white sponge nevus can appear in early childhood or later, then grows and reaches its maximum development in adulthood. In addition, the disease may or may not change. Subjective signs are not observed. Patients may complain of an unusual appearance of the mucous membrane. The typical location of the white sponge nevus is the mucous membrane of the tongue. Leukoplakias are always symmetrically arranged. The mucous membrane of the tongue will look white or gray-white, slightly thickened, soft, spongy, with strong folds. In some cases, the crease of the mucous membrane is so pronounced that the folds hang over the oral cavity. The surface layers of the epithelium are removed by scraping. In some cases, the crease of the mucous membrane is so pronounced that the folds hang on the oral cavity. In some cases, the crease of the mucous membrane is so pronounced that the folds hang over the oral cavity. The surface layers of the epithelium are removed by scraping. However, similar injuries can occur on the mucous membrane of the genitals and rectum. Many authors consider white sponge nevus and soft leukoplakia to be different clinical forms of the same disease associated with leukoplakias.

Comparative diagnosis. It is performed with leukoplakia and candidiasis.

Treatment. This is the sanitation of the oral cavity, the elimination of local traumatic factors (grinding the sharp edges of the teeth, replacing poor-quality dentures, etc.). Treatment. This is the sanitation of the oral cavity, the elimination of local traumatic

Infectious Diseases

The mucous membrane of the oral cavity is almost always involved in the pathological process in infectious diseases. The mucous membrane of the oral cavity is almost always involved in the pathological process in infectious diseases. Therefore, many infectious diseases, both acute and chronic, have typical manifestations in Oral mucosa (scarlet fever, measles, syphilis, tuberculosis, HIV

infection, etc.). Knowing these manifestations greatly facilitates the diagnosis of infectious diseases. The nature and severity of lesions of infectious diseases Oral mucosa depends on the virulence of the pathogen and the state of macroorganism. This primarily applies to the state of the immune and endocrine systems, the heredity factor, etc. The human oral cavity contains the largest number of bacterial species compared to other lands in the body. This primarily applies to the state of the immune and endocrine systems, the heredity factor, etc. The human oral cavity contains the largest number of bacterial species compared to other lands in the body. In a healthy organism, the types and quantitative appearance of microflora practically do not change during all life activities. Due to the time of year, day, burning diseases, etc., there may only be some variability.

The main factor that ensures the types and quantitative stability of the oral microflora is saliva all its diverse properties (PH, viscosity, buffering properties of enzyme systems, ion potential, etc.) are characterized. The main factor that ensures the types and quantitative stability of the oral microflora is saliva all its diverse properties (PH, viscosity, buffering properties of enzyme systems, ion potential, etc.) are characterized. The composition and amount of microflora in the oral cavity is greatly influenced by somatic diseases, regular intake of drugs, especially antibiotics and corticosteroids, the state of the immune system, environmental factors, secondary adentia and the use of removable dentures.

All these factors lead to a violation of the composition and properties of microflora living in the oral cavity. All these factors lead to a violation of the composition and properties of microflora living in the oral cavity. The balance in microbial associations is disturbed, as a result of which the virulence of the resident microflora increases, will these factors lead to a violation of the composition and properties of microflora living in the oral cavity. The balance in microbial associations is disturbed, as a result of which the virulence of the resident microflora increases, which is a source of endogenous infection in the oral cavity, provoking acute and chronic inflammatory processes. The appearance of autoinfection can lead to a sharp decrease in the protective functions of the oral mucosa as a result of various

dystrophic processes, infectious diseases, acute or chronic trauma (mechanical, thermal, chemical).

Diseases of the oral mucosa caused by representatives of the resident microflora of the oral cavity are usually called endogenous infectious diseases or autoinfections. Diseases of the oral mucosa caused by representatives of the resident microflora of the oral cavity are usually called endogenous infectious diseases or autoinfections. These diseases of the oral mucosa caused by representatives of the resident microflora of the oral cavity are usually called endogenous infectious diseases or autoinfections. Diseases of the oral mucosa caused by representatives of the resident microflora of the oral cavity are usually called endogenous infectious diseases or autoinfections. These include candidiasis, fusospirochetosis, pustular and other bacterial infections. In acute infectious diseases, Oral mucosa is almost always involved in the pathological process. Viral, fungal or bacterial diseases affecting Oral mucosa (acute or exacerbation of chronic herpetic stomatitis, candidiasis, Vincent's ulcerative necrotic stomatitis, etc.) treated by dentists. Treatment of patients with damage to the oral mucosa in chronic infectious diseases (syphilis, tuberculosis, leprosy, AIDS, etc.) is carried out by doctors of other specialties (venerologists, pediatricians, infectious disease specialists).

Viral diseases

Viral diseases of the oral mucosa occupy an important place in the group of diseases of the mucous membrane in terms of their spread. There are several types of viruses in the oral cavity of an adult. Viral diseases of the oral mucosa occupy an important place in the group of diseases of the mucous membrane in terms of their spread. There are several types of viral diseases of the oral mucosa occupy an important place in the group of diseases of the mucous membrane in terms of their spread. There are several types of viruses ty

Herpes simplex is the most common viral disease caused by the herpes virus. Oral herpes (Herpes simplex). Herpes simplex is the most common viral disease caused by the herpes virus. According to its antigenic properties, herpes simplex viruses are divided into two main groups — Type 1 and type 2.

Type 1 viruses in the oral mucosa

And type 2 causes herpes on the mucous membrane in the genitals. Herpes simplex virus contains DNA. In the human body, it reproduces mainly in epithelial cells. type 2 causes herpes on the mucous membrane in the genitals. Herpes simplex virus contains DNA. In the human body, it reproduces mainly in epithelial cells. Entering the human body and leading to the manifestation of primary herpes infection, it mainly remains in a latent state throughout the life of a person or leads to a recurrence of the disease. Herpes simplex virus is found in 75-90% cases in adults. Primary infection usually occurs between the ages of 1 and 3, when the amount of antibodies obtained from the mother disappears or sharply decreases in the child's blood, and the body becomes more sensitive. Primary infection usually occurs between the ages of 1 and 3, when the amount of antibodies obtained from the mother disappears or sharply decreases in the child's blood, and the body becomes more sensitive. Most often, the primary infection of the child is ignored, but in some cases a clear manifestation of the disease in children — the so-called primary herpes. This disease is especially severe in the first months of a child's life, when General damage to the mucous membranes, skin, eyes and other organs occurs. In rare cases, primary herpes develops in adults who have not previously come into contact with the herpes virus. In rare cases, primary herpes develops in adults who have not previously come into contact with the herpes virus. After treatment from primary herpes, the infection may not persist, but may progress to a latent state and recur under the influence of various negative factors (hypothermia, stressful situations, sore throat, flu, pneumonia, diseases of the gastrointestinal tract, etc.). Herpes infection with Herpes simplex occurs through direct contact with the patient or the carrier of the virus, since the herpes virus is secreted from the saliva in people who do not have visible signs of infection; it is also transmitted through air drops. Herpes infection with Herpes simplex occurs through direct contact with the patient or the

carrier of the virus, since the herpes virus is secreted from the saliva in people who do not.

Acute herpetic stomatitis (stomatitis herpetica acuta). The disease was previously referred to as "acute aphthous stomatitis".cute herpetic stomatitis (stomatitis herpetica acuta). The disease was previously referred to as "acute aphthous stomatitis". Depending on the severity of the general concute herpetic stomatitis (stomatitis herpetica acuta). The disease was previously referred to as "acute aphthous stomatitis". Depending on the severity of the general ccute herpetic stomatitis (stomatitis herpetica acuta). The disease was previously referred to as "acute aphthous stomatitis". Depending on the severity of the general condition disorders and the prevalence in Oral mucosa, mild, moderate and severe forms of acute herpetic stomatitis are distinguished. The pathogenesis of the disease includes periods of incubation, prodromal (catarrhal), rash (progression of the disease), extinction, and clinical recovery (recovery). The disease is contagious for people who have not previously been infected with the virus. Primary herpes in newborns passes very difficult.

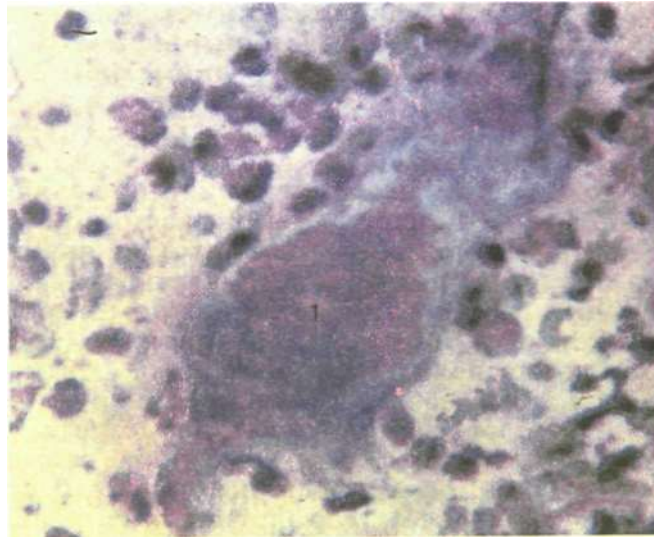
Clinical picture. The incubation period lasts from 1 to 4 days. In the incubation period and especially in the prodromal period, the jaw increases in the lymph nodes of the neck in severe cases.linical picture. The incubation period lasts from 1 to 4 days. In the incubation period and especially in the prodromal period, the jaw increases in the lymph nodes of the neck in severe



Figure 42. Acute herpetic stomatitis.

Multiple contoured aphthae and erosions on the hyperemic mucosa of the lower lip.

In the oral cavity, diffuse hyperemia, swelling of the mucous membrane first occurs, and then blisters (vesicles) containing a transparent exudate appear singly or in groups. In the oral cavity, diffuse hyperemia, swelling of the mucous membrane first occurs, and then blisters (vesicles) containing a transparent exudate appear singly or in groups. A rash of blisters is accompanied by the oral cavity, diffuse hyperemia, swelling of the mucous membrane first occurs, and then blisters (vesicles) containing a transparent exudate appear singly or in groups. A rash of blisters is accompanied by itching, a burning sensation and, in some cases, swelling of the oral mucosa. The number of vesicles can be from 2-3 to several dozen, located close to each other. The vesicles quickly open and round erosions form in their place. Erosions formed by the fusion of multiple vesicles have irregular polycyclic contours. Vesicles settled mainly on the hard palate, back of the tongue, gums, lunge, lip. Most often, the red border of the lips and adjacent areas of the skin are affected. Vesicles settled mainly on the hard palate, back of the tongue, gums, lunge, lip. Most often, the red border of the lips and adjacent areas of the skin are affected. Vesicles settled mainly on the hard palate, back of the tongue, gums, lunge, lip. Most often, the red border of the lips and adjacent areas of the skin are affected. keratoconjunctivitis, vesicular rashes on the face and vulvovaginitis are slightly less common. The procedure is accompanied by hypersalivation and severe pain. Since the pathological elements can appear within a few days, when examining the oral cavity, they can be observed at different stages of development. Diffuse gum disease is characterized by acute catarrhal gingivitis, more noticeable from the palate and tongue. Diffuse gum disease is characterized by acute catarrhal gingivitis, more noticeable from the palate and tongue. If oral care is insufficient, catarrhal gingivitis can develop into wound gingivitis, in the acute herpetic pr.



**Figure 43. Giant multinucleated cells (1) in recurrent herpes. Cytogram.
Micrography**

Maxillary lymphadenitis, which also occurs during the Prodromal period, lasts for another 5-10 days throughout the disease and even after erosion epithelization. axillary lymphadenitis, which also occurs during the Prodromal period, lasts for another 5-10 days throughout the disease and even after erosion epithelization. Nonspecific changes characteristic of the acute inflammaxillary lymphadenitis, which also occurs during the Prodromal period, lasts for another 5-10 days throughout the disease and even after erosion epithelization. Nonspecific changes characteristic of the acute inflammatory process are found in the blood. At the beginning of the disease, the salivary reaction is acidic, then a shift towards alkaline is determined (PH 5.8—6.4). As a result of reduced lysozyme content, interferon is lost. Histological changes in the mucous membrane of the mouth and skin in the Herpes simplex are characterized by the presence of intra-epithelial vesicles formed by balloon and lenticular degeneration of the cells of the thoracic floor, the phenomenon of acantholysis. istological changes in the mucous membrane of the mouth and skin in the Herpes simplex are characterized by the presence of intra-epithelial vesicles formed by balloon and lenticular degeneration of the cells of the thoracic floor, theHistological changes in the mucous membrane of the mouth and skin in the Herpes simplex are characterized by the presence of intra-epithelial

vesicles formed by balloon and lenticular degeneration of the cells of the thoracic floor, the phenomenon of acantholysis. DNA-containing appendages are found in degenerate epithelial cells. At the base of the mucous membrane, swelling of the sucker layer, enlargement of the lymph and blood vessels, infiltration of neutrophils and lymphocytes are observed. A cytological method is used to confirm the diagnosis of acute herpetic stomatitis. The study is carried out on the first 2-3 days of the disease, after the appearance of blisters. The Material is obtained by scraping from the erosion furnace or the contents of the bubbles. The study is carried out on the first 2-3 days of the disease, after the appearance of blisters. The Material is obtained by scraping from the erosion furnace or the contents of the bubbles. When dyeing the drug according to Romanovsky-Gimze, huge multicellular cells are detected. They differ in shape, color and size (30-120 microns in diameter) from acute polymorphism. The center of the cell is occupied by a dense conglomerate of nuclei (from 2-3 to several dozen). Nucleoli are undetectable. Nucleoli are undetectable. Cytological examination also identifies polymorphonuclear neutrophils at various stages of necrobiosis, layers of epithelial cells with polymorphism events, and scintia, iucleoli are undetectable. Cytological examination also identifies polymorphonuclear neutrophils at various stages of necrobiosis, layers of epithelial cells with polymorphism events, and scintia, in the form of macrophages. With acute herpetic stomatitis, the herpes virus can be isolated from the contents of the blisters in the first days of the disease, as well as during relapses. Antitela to the Herpes virus is not detected at the beginning of the disease, but with its development, the titer of the antitela increases. Immunofluorescence methods, skin tests with specific antigen, and serological reactions are used to diagnose acute herpetic stomatitis.

Comparative diagnosis Immunofluorescence methods, skin tests with specific antigen, and serological reactions are used to diagnose acute

herpetImmunofluorescence methods, skin tests with specific antigen, and serological reactions are used to diagnose acute herpetic stomatitis.

Comparative diagnosis. Other viral diseases (herpangina, oxime, vesicular stomatitis); and multi-form exudative erythema; and allergic diseases.

The difference from herpangina lies primarily in the location of the disease — with herpangina it is the area of the pharynx, as a result of which myalgia and dysphagia are often observed. The final diagnosis is confirmed by virological examination data. Comparative diagnosis with oxime disease is carried out taking into account the epidemiological situation and the usual skin diseases in this disease. The results of the methods of objective laboratory diagnostics of the presence of oxime are determined by: biological sampling, serological and virological research. Comparative diagnosis with oxime disease is carried out taking into account the epidemiological situation and the usual skin diseases in this disease. The results of the methods of objective laboratory diagnostics of the presence of oxime are determined by: biological sampling, serological and virological research.

In differential diagnosis with vesicular stomatitis, the results of virological research methods are decisive in diagnosis. Multichallic exudative erythema, unlike herpes simplex, is characterized by the presence of seasonal recurrence (spring and autumn). Multichallic exudative erythema, unlike herpes simplex, is characterized by the presence of seasonal recurrence (spring and autumn). Also, various potolgie elements appear: large subepithelial puffs, erythema on the mucous membrane of the mouth. Multichallic exudative erythema, unlike herpes simplex, is characterized by the presence of seasonal recurrence.

Treatment. Acute herpetic stomatitis, like a number of other infectious diseases, can be treated on its own. It lasts an average of 2-3 weeks. Treatment. Acute herpetic stomatitis, like a number of other infectious diseases, can be treated on its own. Acute herpetic stomatitis, like a number of other infectious diseases, can be treated on its own. It lasts an average of 2-3 weeks. Treatment tactics are determined by the severity of the disease and the period of its development. Treatment of acute herpetic

stomatitis includes the use of antiviral drugs from the first days of the disease and symptomatic therapy that can alleviate the patient's condition.

General therapy. In severe cases of the disease, antiviral drugs are used from the first days: bonafont 0.1 g 3-5 times a day with breaks of 1-2 days in 5-day cycles; atsiklovir 0.2 g 5 times a day for 5 days, for this purpose deoxyribonuclease (DNase) is used, which is 10-25 mg eneral therapy. In severe cases of the disease, antiviral drugs are used from the first days: bonafont 0.1 g 3-5 times a day with breaks of 1-2 days in 5-day cycles; atsiklovir 0.2 g 5 times a day for 5 days, for this purpose deoxyribonuclease (DNase) is used, which is 10-25 mg intramuscularly. times will be sent. powder in distilled water or isotonic sodium chloride solution for a pre-injection course of 6-10. Desensitization and anti-inflammatory therapy are performed. Antihistamine drugs (suprastin, tavegil, phencarol, etc.); sodium salicylate or acetylsalicylic acid (0.25 g 3 times a day, after meals), calcium gluconate (0.5 g 3 times a day) are used.odium salicylate or acetylsalicylic acid (0.25 g 3 times a day, after meals), calcium gluconate (0.5 g 3 times a day) are used. To increase the body's resistance, vitamins, especially C and P, and multivitamins are prescribed. In a hospitalium salicylate or acetylsalicylic acid (0.25 g 3 times a day, after meals), calcium gluconate (0.5 g 3 times a day) are used. To increase the body's resistance, vitamins, especially C and P, and multivitamins are prescribed. In a hospital setting, lysozyme is used for this purpose (7 mg 2-3 times a day for 150 days), prodigiosan is administered intramuscularly at 25-50 mcg per day with an interval of 4-7 days; the course of treatment consists of 3-6 injections. If there are complications with fusospirochetosis, metronidazole is prescribed orally. According to the instructions, cardiovascular drugs are used.

Local therapy. In the first days or even hours of the disease, the use of antiviral drugs is effective: leukocyte interferon, a solution of which is applied to Oral mucosa

6-7 times a day. Local therapy. In the first days or even hours of the disease, the use of antiviral drugs is effective: leukocyte interference therapy. In the first days or even hours of the disease, the use of antiviral drugs is effective: leukocyte interferon, a solution of which is applied to Oral mucosa 6-7 times a day. Ointments against the virus are successfully used: 2% tebrofen, 1-2% florenal, 1-5% chelepin, 3% gossipol liniment. These drugs are applied to the entire surface of the Oral mucosa after pre-treatment with proteolytic enzymes and antiseptics. Antiviral drugs should be applied to the entire surface of the mucous membrane and not only for the benefit of the affected areas, but also in addition to the therapeutic and prophylactic effect. Antiviral drugs should be applied to the entire surface of the mucous membrane and not only for the benefit of the affected areas, but also in addition to the therapeutic and prophylactic effect. It is carried out with a solution of 1% trimecaine, a solution of 1-2% pyromecaine, a solution of 1-2% lidocaine, aerosols "Lidestin", "Xylostesin", an anesthetic solution of 5-10% in Peach oil, etc. It is advisable to use a 0.2% solution of deoxyribonuclease or a 1% solution of lysoamidase, since these enzymes have proteolytic and anti-viral effects. It is advisable to use a 0.2% solution of deoxyribonuclease or a 1% solution of lysoamidase, since these enzymes have proteolytic and anti-viral effects. For antiseptic treatment of the oral cavity, warm solutions are used in the form of rinses or mouth t is advisable to use a 0.2% solution of deoxyribonuclease or a 1% solution of lysoamidase, since these enzymes have proteolytic and anti-viral effects. For antiseptic treatment of the oral cavity, warm solutions are used in the form of rinses or mouth baths: 0.25—0.5% hydrogen peroxide solution, 0.25% chloramine solution, furacilin solution (1:5000), 0.5% ethonium solution, 0.1% gossipol solution, etc. In the first days of the disease, rinsing the oral cavity every 3-4 hours is recommended to be treated. To stimulate Oral mucosa regeneration processes, fatty solutions of vitamins A, ye, carotolin, rose oil, solcoseryl ointment and jelly, 0.5-1% actovegin ointment and jelly, aerosols "Livian", "Speedian" are used. To stimulate Oral mucosa regeneration processes, fatty solutions of vitamins A, ye, carotolin, rose oil, solcoseryl ointment and jelly, 0.5-1% actovegin ointment and jelly, aerosols "Lo

stimulate Oral mucosa regeneration processes, fatty solutions of vitamins A, ye, carotolin, rose oil, solcoseryl ointment and. The consequence of acute herpetic stomatitis it is very favorable, recovery occurs after 1-3 weeks, erosion is epithelialized without traces, in the Sanated oral cavity and in the absence of timely treatment due to the addition of fuzospiroketosis can develop Vincent's ulcerative necrotic gingivostomatitis.

Prevention. Since acute herpetic stomatitis is a contagious disease, the patient should be isolated. Since acute herpetic stomatitis is a contagious disease, the patient should be isolated. Exclude in children's institutions the connection of healthy children withn.

Chronic recurrent herpes (herpes chronicus recidivans) is the most common form of endogenous infection caused by herpes simplex .hronic recurrent herpes (herpes chronicus recidivans) is the most common form of endogenous infection caused by herpes simplex.hronic recurrent herpes (herpes chronicus recidivans) is the most common form of endogenous infection caused by herpes simplex . It occurs at any age in people who have previously been infected with the herpes shronic recurrent herpes (herpes chronicus recidivans) is the most common form of endogenous infection caused by herpes simplex . It occurs at any age in people who have previously been infected with the herpes simplex virus and have anti-viral antibodies. The main role in the pathogenesis of recurrent herpes lies in factors that reduce cell immunity, such as hypothermia, viral infections, pneumonia, chronic stress, severe common diseases (leukemia, cancer, etc.). Exacerbation of chronic herpes in women is often associated with the menstrual cycle. Local factors that contribute to the onset of recurrence include trauma, increased insolation, and dryness of the red hoshia of the lips. Recidivism can occur at different frequencies, at different times of the year and does not have seasonality. Local factors that contribute to the onset of recurrence include trauma, increased insolation, and dryness of.



Image 44. Chronic recurrent herpes.

Clinical picture. Recurrence of chronic herpetic infection is characterized by a rash of single or bmr multiple vesicle rashes with a diameter of 1-2 mm on the hyperemic mucous membrane. linical picture. Recurrence of chronic herpetic infection is characterized by a rash of single or bmr multiple vesicle rashes with a diameter of 1-2 mm on the hyperemic mucous membrane. The procedure usually begins with linical picture. Recurrence of chronic herpetic infection is characterized by a rash of single or bmr multiple vesicle rashes with a diameter of 1-2 mm on the hyperemic mucous membrane. The procedure usually begins with a burning sensation, itching and sometimes mild pain at the site of the rash. Then there is mild hyperemia and swelling of the mucous membrane, small grouped bubbles appear against it. They crack very quickly, resulting in blisters and irregular-shaped bright red painful erosions with separate small fine-edged contours around. The erosions are then covered with white-yellow fibrinous caraches, hemorrhagic crusts on the red border of the lips and skin. Ends without scars on the 8th-10th day. he erosions are then covered with white-yellow fibrinous caraches, hemorrhagic crusts on the red border of the lips and skin. Ends without scars on the 8th-10th day. Most often, blisters appear in the area of the he erosions are then covered with white-yellow fibrinous caraches, hemorrhagic crusts on the red border of the lips and skin. Ends without scars on the 8th-10th day. Most often, blisters appear in the area of the border with the skin with red Hosia of the lips (herpes labialis), on the front parts of the hard palate, on the back of the tongue, lunges, on the wings of the nose. Recurrent herpes

in Oral mucosa mainly settles in areas where it usually branches. the location of the recurrent herpes in one place they are spoken in the form of fixed herpes. Sometimes the appearance of vesicles is accompanied by an enlargement of the lymph nodes under the jaw and under the tongue.

Comparative diagnosis. Recurrent aphthous stomatitis is done with allergic stomatitis , Streptococcus impetigo. Comparative diagnosis. Recurrent aphthous stomatitis is done with allergic stomatitis , Streptococcus impetigo.

Based on the peculiarities of the clinical picture of chronic recurrent herpes, as well as on the data of cytological studies of the contents of the vesicles and comparative diagnosis. Recurrent aphthous stomatitis is done with allergic stomatitis , Strepsed.

Treatment. This is aimed at increasing the level of specific and non-specific protection of the patient's body, which can suppress the persistent herpes simplex virus.reatment. This is aimed at increasing the level of specific and non-specific protection of the patient's body, which can suppress the persistent herpes simplex virus. To significantly prolong remission cycles and relieve the clinical picture of subsequent recurrence, levamizol (decaris) can be administered orally for 1-3 months, 150 mg 2 times a week, depending on the effectiveness of the treatment. To identify and eliminate foci of chronic infection in the body, including the oral cavity (periodontitis, tonsillitis, sinusitis, etc.), it is necessary to carry out a complete examination of patients with chronic recurrent herpes.identify and eliminate foci of chronic infection in the body, including the oral cavity (periodontitis, tonsillitis, sinusitis, etc.), it is necessary to carry out a complete examination of patieo identify and eliminate foci of chronic infection in the body, including the oral cavity (periodontitis, tonsillitis, sinusitis, etc.), it is necessary to carry out a complete examination of patients with chronic recurrent herpes.)• Local factors that contribute to the appearance of recurrence (chronic trauma, dry lips, chronic clefts of the lips) are eliminated. The use of a specific herpetic multivaxine gives good results. The drug is administered intradermally 2 times a week at a dose of 0.1—0.2 mg during the interstitial period. The course of treatment consists of 5 injections. The interval between the first two courses should be 2 weeks, between the next — 3-4 months.he

course of treatment consists of 5 injections. The interval between the first two courses should be 2 weeks, between the next — 3-4 months. The therapeutic effect is less ensured by deoxyribonuclease (the course of treatment consists of 5 injections. The interval between the first two courses should be 2 weeks, between the next — 3-4 months. The therapeutic effect is less ensured by the course of treatment consists of 5 injections. The interval between the first two courses should be 2 weeks, between the next — 3-4 months. The therapeutic effect is less ensured by deoxyribonuclease (DNA), which is injected intramuscularly at 10-25 mg, previously dissolving the powder in distilled water or isotonic sodium chloride solution. Intramuscular injections of gammaglobulin in the recurrent period are used 3 ml per day, at intervals of 3-4 days, for a course of 6 injections; the interval between courses is 2 months. Large doses of ascorbic acid are usually prescribed orally. Antiviral ointments are applied maxially from the first days of the disease, leukocyte interferon, its solution is recommended to apply damaged 5-6 times a day. Antiviral ointments are applied maxially from the first.

Encircling ironclad

The enveloping ferruginous Varicella is caused by the zoster virus. Encircling ironclad The enveloping ferruginous Varicella is caused by the zoster virus. This is due to the fact that the virus is the causative agent of two diseases that differ in clinical manifestation. Encircling ironclad The enveloping ferruginous Varicella is caused by the zoster virus. This is due to the fact that the virus is the causative agent of two diseases that differ in clinical manifestations: chickenpox and enveloping ferret. The Virus is characterized by neurodermatotropy. Enveloping iron is first seen as a recurrent infection against the background of a decrease in immunity, which developed as a result of the body's contact with the virus. The disease is caused by the activation of a virus that is in a latent state in the nerve ganglia. Ringworm, which is a fungal infection, affects children and adults, most often the elderly. Ringworm, which is a fungal infection, affects children and adults, most often the elderly. In most cases, the disease occurs against the background of a weakening of the body's protective forces as a result of pneumonia, blood diseases, neoplasms and other

immunocompromising diseases. The enveloping iron, as a rule, leaves a strong immune system. However, in people with severe chronic diseases (oncological, blood diseases), cases of its recurrence can be observed. The enveloping iron, as a rule, leaves a strong immune system. However, in people with severe chronic diseases (oncological, blood diseases), cases of its recurrence can be observed. The infection is transmitted through contact and air droplets. Herpes zoster most often occurs in the autumn-winter period, is characterized by fever, neuralgic pain and erythematous vesicular rashes on the skin and mucous membranes along the affected peripheral nerves. Some cranial and vertebrate nerve ganglia, often affected on one side. When affected by the first and third branches of the 3-Horned nerve, as well as the 3-Horned nerve node (herpetic ganglionite), blistering rashes appear on the oral mucosa. When affected by the first and third branches of the 3-Horned nerve, as well as the 3-Horned nerve node (herpetic ganglionite), blistering rashes appear on the oral mucosa. At the same time, it is affected by the first and third branches of the 3-Horned nerve, as well as the 3-Horned nerve node (herpetic ganglionite), blistering rashes appear on the oral mucosa. At the same time, it affects the corresponding areas of the skin. Elderly people develop enveloping iron, against the background of frequent severe diseases, cytostatic, corticosteroid therapy. It is characterized by generalized vesicular rashes resembling those of smallpox with distinct intoxication phenomena and chickenpox. It can also affect the cerebral cortex and lungs, which often leads to death.

Clinical picture. Usually, the disease begins with pain of different intensity at the site of the rash. Clinical picture. Usually, the disease begins with pain of different intensity at the site of the rash. mild pain, paroxysmal picture. Usually, the disease begins with pain of different intensity at the site of the rash. mild pain, paresthesia, nausea, burns, cuts, pain along the affected nerves are different from paroxysmal neuralgic pain. Pain increases under the influence of pests. At the same

time, tremors, restlessness, headaches appear, and the body temperature rises to 38-39. 1-5 days after the onset of pain in the affected area, swelling and hyperemia of the skin develops, against which grouped blisters with a diameter of 1-5 mm filled with transparent exudate appear. 5 days after the onset of pain in the affected area, swelling and hyperemia of the skin develops, against which grouped blisters with a diameter of 1-5 mm filled with transparent exudate appear. At the same time as skin damage, several vesicles also appear along the affected nerve in the hyperemied oral mucosa. In the oral cavity, the vesicles open quickly, forming single or single erosions covered with fibrinous Carache.

Blisters on the skin shrink and become covered with a crust or open as a result of injury with the formation of erosion. Blisters on the skin shrink and become covered with a crust or open as a result of injury with the formation of erosion. After erosion epithelialization in Oral mucosa and loss of scales on the skin, the process is completed, leaving the skin temporarily pigmented. The total duration of the disease is 2-3 weeks. The consequences are conical, however, older people, as well as those with severe diseases accompanied by a sharp decrease in immunity (leukemia, oncological diseases), can experience complications in the form of postgerpetic neuralgia, hyperesthesia and hyperpathy, which last for several months or years. The consequences are conical, however, older people, as well as those with severe diseases accompanied by a sharp decrease in immunity (leukemia, oncological diseases), can experience complications in the form of pThe consequences are conical, however, older people, as well as those with severe diseases accompanied by a sharp decrease in immunity (leukemia, oncological diseases), can experience complications in the form of postgerpetic neuralgia, hyperesthesia and hyperpathy, which last for several months or years. Most often, such phenomena are observed in the elderly, and the larger the patient, the higher the likelihood of developing postgerpetic complications.

Comparative diagnosis. Herpes zoster should be distinguished:

Acute herpetic stomatitis, rash, pemphigoid, allergic stomatitis;

in case of damage to the skin-from rojasimon inflammation.in case of damage to the skin-from rojasimon inflammation.In the diagnosis of Herpes zoster, the one-sided nature of the jaroxate, the absence of a faytalance recidiv, a clear symptom of pain and the location of vesicles in the arn case of damage to the skin-from rojasimon inflammation.

In the diagnosis of Herpes zoster, the one-sided nature of the jaroxate, the absence of a faytalance recidiv, a clear symptom of pain and the location of vesicles in the area innervated by the affected nerve are of great importance. For the final diagnosis, the following additional research methods are used: general clinical blood analysis, virological (separation of the virus from New vesicles, blood, brain fluid or throat rinsing), cytological.

Treatmenttreatment. The main tasks of treating enveloping ironate are to remove pain symptoms, prevent the appearance of blisters on the skin and mucous membrane, and prevent postgerpetic eatment. The main tasks of treating enveloping ironate are to remove pain symptoms, prevent the appearance of blisters on the skin and mucous membrane,reatment. The main tasks of treating enveloping ironate are to remove pain symptoms, prevent the appearance of blisters on the skin and mucous membrane, and prevent postgerpetic neuralgia. For this purpose, analgesics (analgin, baralgin), salicylates (acetylsalicylic acid 0.5 g 3-4 times a day), vitamins ganglerone, B, B12 are prescribed. Antiviral drugs are used, metisazone(0.6 g 2 times a day, after meals, for 6 days); bonafont (0.1 g 3-5 times a day, with breaks of 1-2 days in three 5-day cycles); Acyclovir (0.8 g 5 times a day for 5 days). Effective use of deoxyribonucleasacyclovir (0.8 g 5 times a day for 5 days). Effective use of deoxyribonuclease. The drug is administered intramuscularly at a dose of 25 mg every 4 hours ocyclovir (0.8 g 5 times a day for 5 days). Effective use of deoxyribonuclease. The drug is administered intramuscularly at a dose of 25 mg every 4 hours or 50 mg 2-3 times a day for 7-10 days. Rapid relief of the process is observed. Treatment with deoxyribonuclease also reduces the development of posttherpetic neuralgia.

Local treatment consists in the use of antiviral drugs from the first days of the disease (tebrofen, florenal, gossipol, megosine, oxolin, interferon).ocal treatment consists in the use of antiviral drugs from the first days of the disease (tebrofen, florenal, gossipol, ocal treatment consists in the use of antiviral drugs from the first days of the disease (tebrofen, flore).

Herpetic angina

Herpetic angina (herpangina, vesicular pharyngitis, aphthous pharyngitis) is an acute infectious disease caused by the **Coxsackie enterovirus** of group A, which occurs mainly in the summer months, mostly in children. Infection is transmitted by airborne droplets. Cases of disease in family members and even epidemics are expected.

Clinical PictureThe disease begins abruptly with a rise in body temperature to $37-38^{\circ}\text{C}$ in the first days of the disease, and by the third or fourth day it reaches $39-39.5^{\circ}\text{C}$. Patients complain of headache, pain when swallowing, general malaise, vomiting, and abdominal pain may be observed.

Upon examination of the oral cavity, hyperemia is detected on the soft palate, anterior arches, tonsils, and posterior wall of the pharynx, against which painful single and grouped vesicles filled with serous or hemorrhagic fluid are visible. The vesicles rupture very quickly, forming aphthae and erosions. As a result of the confluence of small erosions, large erosions with scalloped edges are formed. The pain of erosions is noted only in the first days of the disease, then they decrease or disappear completely. A slight swelling of the submandibular lymph nodes is observed, but they are painless. The disease often proceeds in a mild form, within 4-6 days, although more severe forms exist.

Treatment It consists of conducting general symptomatic therapy (salicylates, vitamins B, B2). Antiviral drugs, rinsing the mouth and throat with antiseptic solutions, and subsequently keratoplastic agents are applied locally in the first 2-3 days of the disease.

Acute Respiratory Viral Infections

Acute respiratory viral infections (ARVI) include influenza, parainfluenza, rhinovirus infection, respiratory syncytial infection, adenovirus diseases, and others. These diseases are caused by various types of RNA-containing viruses, except for DNA-containing adenoviruses. Infection is transmitted by airborne droplets. The disease is characterized by damage to various parts of the upper respiratory tract.

Changes in the oral mucosa in ARVI are non-specific and are located in the area adjacent to the pharynx. Hyperemia of the soft palate, uvula, palatine arches of the oropharynx, vascular spasm, plaque and desquamation of the epithelium on the tongue are noted. In some cases, the process extends to the mucous membrane of the posterior cheeks and tongue. The mucous membrane becomes granular due to inflammation of small lymph nodes. After a few days, hyperemia and granularity disappear, and distinct petechiae and hemorrhages appear. In some cases, a vesicular rash occurs. After 7-9 days, all phenomena disappear.

A severe course of respiratory viral infections can be complicated by the development of ulcerative necrotic gingivitis and Vincent's stomatitis, especially in an unsanitized oral cavity. In the same period, exacerbations of chronic recurrent herpes, periodontitis, and gingivitis are often observed. In the oral cavity, as a result of taking medications for ARVI, dysbiosis and candidiasis, or allergic drug stomatitis may develop.

Treatment Along with taking medications for acute respiratory infections, patients should carefully observe **oral hygiene**, rinse the mouth and pharynx with antiseptic solutions, and use drugs that increase the resistance of the oral mucosa to infection (e.g., Lysozyme, Interferon, UV radiation). If antibiotics are used (with complications), **prevention of candidiasis** in the oral cavity is carried out (prophylactic doses of antifungal drugs are prescribed).

Foot-and-Mouth Disease (Aphthae Epizooticae)

Foot-and-Mouth Disease (FMD), or epidemic stomatitis (aphthae epizooticae), is an acute viral infectious disease transmitted to humans from sick animals. It proceeds periodically with the appearance of vesicles and erosions on the oral mucosa and the skin between the fingers. The FMD virus affects domestic and wild animals (cows, goats, sheep, deer, pigs, etc.) and is excreted into the external environment by sick animals with saliva, milk, urine, and manure. The FMD virus is very stable in the external environment. Human susceptibility to it is low. Infection is usually transmitted alimentary when consuming raw dairy products, as well as meat that has not undergone culinary processing. Boiling and pasteurization of milk kill the virus. In addition, the disease can be occupational, transmitted by contact during the care of sick animals by veterinary personnel. The virus penetrates through damaged skin and the mucous membranes of the mouth, nose, and eyes. It is also transmitted through airborne dust.

Clinical Picture. There are three clinical forms of FMD: with skin damage, with oral mucosal damage, and with combined skin-mucosal damage. After an incubation period lasting from 2 to 10 days, the disease begins abruptly with chills, malaise, a rise in body temperature (to $38-39^{\circ}\text{C}$), muscle pain, lower back pain, and decreased appetite. After 1-2 days, patients develop photophobia, xerostomia, and a burning sensation; hyperemia and swelling of the mucous membrane develop. Vesicles with transparent contents (2-4 mm in diameter) appear on the lips, tongue, hard and soft palate, and cheeks, against a background of sharply hyperemic mucous membrane. Their number varies up to several hundred. Then their contents turn turbid yellow. After one or two days, the vesicles rupture, forming painful bright red erosions with polycyclic contours; erosions on the lips become covered with crusts. After the rupture of the vesicles, the body temperature usually decreases, but the patients' feeling of malaise increases. Excessive salivation occurs (up to 4-5 liters per day), swallowing even liquid food becomes difficult, the tongue enlarges, and speech is disturbed. Vesicles can also appear on the mucous membrane of the nose, conjunctiva, and genitals. Local lymph nodes are enlarged and painful. In most patients, in addition to the mucous membranes, the skin is also affected. FMD is characterized by the localization of vesicular rashes in the folds between the fingers and toes, which is accompanied by burning, itching in the area of the hands and feet. Erosions in the oral mucosa epithelialize after 3-5 days without leaving any trace, after which the general condition normalizes and a period of convalescence occurs, lasting 10-15 days. In severe FMD, rashes on the oral mucosa may recur, and maculopapular rashes may appear on the skin of the chest, neck, and back. There are also aborted forms of the disease, which are characterized by general malaise and single vesicles in the oral cavity and on the skin.

Differential Diagnosis. FMD should be differentiated from: Acute herpetic stomatitis, Chickenpox (vesicles are mainly located on the trunk and scalp); drug allergy, and erythema multiforme.

Diagnosis is based on the characteristic clinical signs of FMD (general infectious symptoms combined with vesicular stomatitis and skin lesions in the area of the nail

phalanges), taking into account the epidemiological history (contact with sick animals, consumption of raw milk), epizootological assessment, and laboratory results. Among the laboratory diagnostic methods, the biological test and serological reactions are most often used.

Treatment. Isolation and hospitalization of patients for at least 14 days from the onset of the disease are mandatory. Careful care of the patient is of great importance, liquid food is recommended 5-6 times a day. Local treatment is the same as with acute herpetic stomatitis.

Prognosis. Favorable. Full recovery and restoration of working capacity occur by the end of the 2nd week.

Prevention. The basis for the prevention of FMD in humans is the control of FMD in animals, carried out by the sanitary and veterinary service. Personnel in contact with sick animals should work in overalls. To prevent foodborne infection, milk should be boiled for 5 minutes or pasteurized for 30 minutes, and meat should be carefully heat-treated.

HIV Infection in the Oral Cavity

The Human Immunodeficiency Virus (HIV) is the causative agent of an infectious disease called HIV infection, which manifests as Acquired Immunodeficiency Syndrome (AIDS), characterized by a decrease in the overall resistance of patients to opportunistic microorganisms and an increased tendency to cancer.

The Human Immunodeficiency Virus (HIV) belongs to the group of retroviruses because they have an enzyme, reverse transcriptase, which allows the transfer of information from RNA to DNA. HIV destroys T4 lymphocytes and causes their death. As a result, the immune system is disrupted, and the virus-affected body loses resistance to opportunistic microflora, becoming susceptible to a number of non-specific infections and tumors, i.e., AIDS develops. The source of infection is an HIV-infected person or a virus carrier. Individuals with HIV infection include all those infected with the virus (regardless of clinical condition), as evidenced by positive serological tests: enzyme-linked immunosorbent assay (ELISA) confirmed

by immunoblotting (Western Blot), immunofluorescence reaction, or radioimmunological method, and virus isolation.

Following HIV infection, of infected individuals develop AIDS within the first 5 years, and about within 10 years. In HIV-infected individuals, the pathogen is detected in various biological fluids (blood, semen, vaginal secretions, breast milk, saliva, lacrimal fluid, sweat, etc.). However, infection is transmitted only through blood, semen, vaginal secretions, and breast milk. There are 3 known modes of HIV transmission: sexual, parenteral, and perinatal. HIV is most often transmitted sexually. Infection can be transmitted from an infected person to a sexual partner. By the parenteral route, infection is transmitted by the transfusion of infected blood or blood products, as well as by using needles, syringes, dental, or other instruments for puncturing the skin or mucous membranes of the mouth if these items are contaminated with blood. Transmission of HIV infection from a mother to the fetus or infant can occur before, during, or shortly after childbirth (through breast milk). Despite the fact that HIV is isolated from other biological fluids (saliva, lacrimal fluid, etc.), there is currently no data indicating other modes of transmission (respiratory, contact, food, etc.). People infected with HIV remain infected for life. Many of them may not have symptoms or signs of the disease for at least several years and therefore may not be aware of the presence of the infection. During this time, they are a source of virus transmission to other people.

The clinical signs of HIV infection are numerous and diverse. HIV infection can be divided into 4 stages, although not all of them are observed in every infected person. The acute period of HIV infection can develop one or more weeks after infection in the absence of a seropositive reaction to HIV, which is usually noted 6-12 weeks after infection. Typical clinical manifestations of this period are fever, lymphadenopathy, night sweats, skin rash, headache, and cough. Stage 1 of the disease is asymptomatic, or persistent generalized lymphadenopathy (PGL) develops, characterized by an increase in the size of lymph nodes greater than 1 cm in diameter in two or more sites outside the inguinal region. This condition lasts for about 3 months. As the disease progresses, persistent generalized lymphadenopathy

gradually undergoes reverse development. Stage 2 of the disease is called the "AIDS-related complex" (ARC). This complex is characterized by the presence of the following signs and symptoms: weight loss, diarrhea, anxiety, fatigue, drowsiness, loss of appetite, night sweats, neurological changes leading to memory loss, and peripheral neuropathies. These symptoms and signs often appear intermittently. This stage is also characterized by the presence of typical lesions of the skin and oral mucosa, such as hairy leukoplakia, herpes zoster, and exacerbation of chronic recurrent herpes on the oral and genital mucosa.

Stage 3 is called the "intermediate stage of the disease." Clinical manifestations in stage 3 are more likely to appear some time after the onset of early symptoms, but before the full detailed picture of AIDS. At this stage of the disease, oral candidiasis, hairy leukoplakia, acute herpetic stomatitis, or exacerbation of chronic herpes appear in the oral cavity. Pulmonary tuberculosis and alveolar pneumonia may develop. In Stage 4 of the disease (late stage of the disease), the severe clinical picture of AIDS fully unfolds. It is characterized by the development of inflammatory processes caused by opportunistic microflora and the formation of tumors such as Kaposi's sarcoma, the appearance of which is associated with the deficiency of the immune system caused by HIV. The most typical infections for AIDS include protozoal invasions (*Pneumocystis pneumonia*, toxoplasmosis), as well as fungal (systemic candidiasis, cryptococcosis), bacterial (atypical mycobacteriosis, salmonella septicemia), or viral (cytomegaly, papovavirus, etc.). Particularly severe forms of lymphoma, cachexia, and neurological disorders (neuropathy, memory loss, dementia) can also appear at this stage.

The first manifestations—the "markers" indicating the introduction of HIV, which exerts a destructive effect on the body's immunological protection—may be lesions of the oral mucosa.

Diseases of the oral mucosa associated with HIV infection include:

- Various clinical forms of Candidiasis.
- Viral infections (Herpes simplex, Herpes zoster, Papillomas).
- Hairy (villous) leukoplakia.

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- Ulcerative necrotic gingivostomatitis.
- Progressive form of Periodontitis (HIV-periodontitis).
- Kaposi's sarcoma.

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Diseases that should alert the dentist to the possibility of HIV infection in the patient include squamous cell carcinoma of the oral mucosa and lymphoma (non-Hodgkin's).

Candidiasis This is the most common intraoral sign of HIV infection. Acute pseudomembranous candidiasis is the most characteristic. In HIV-infected people, it lasts for a long time (for months), so the term "acute" loses its meaning. Clinical manifestations fully correspond to typical candidiasis. There is a grayish-white plaque, resembling curdled mass, on the oral mucosa. The plaque is easily removed with a spatula, and hyperemic oral mucosa is found beneath it. It is mainly localized on the mucous membrane of the cheeks, tongue, floor of the mouth, palate, and gums. Acute atrophic candidiasis resembles lesions of the oral mucosa with allergy, hypovitaminosis C, B1, B2, B6. The lesion is usually localized on the tongue in the form of hyperemia along its midline; filiform papillae atrophy.

Chronic hyperplastic candidiasis is less common, usually localized bilaterally on the mucous membrane of the cheeks, hard and soft palate. When the lesions are localized at the corners of the mouth, candidal angular cheilitis and cracks with pronounced epithelial hyperplasia occur. Over time, the cracks enlarge, harden, and become covered with crusts. Patients may not have pain or may have minor painful sensations and a burning sensation.

In the differential diagnosis of candidiasis, the symptoms of similar diseases should be taken into account: leukoplakia; lichen planus (LPL); allergic stomatitis; and traumatic injuries.

The diagnosis of candidiasis is based on the typical clinical signs and the results of a bacterioscopic examination of a scraping from the affected oral mucosa. The

presence of numerous fungi of the genus *Candida* in the form of spores and hyphae always confirms the diagnosis of candidiasis.

Viral Infections. Patients with HIV are characterized by lesions of the oral mucosa with Herpes simplex, which manifests in the form of frequent and painful exacerbations of recurrent herpetic stomatitis. Intraoral lesions are often accompanied by genital lesions. The lesions are atypical in location, prolonged, and painful. Vesicles on the tongue, soft palate, floor of the mouth, and lips quickly turn into erosions, which often transform into ulcers of large size (diameter \$0.5-3 cm\$). Ulcers can be crater-shaped with raised irregular edges and a bright hyperemic bottom, which can be covered with a grayish-white plaque. Without timely treatment, these lesions progress, their size increases, which subsequently leads to the spread of viral herpes infection to the visceral organs, increasing the severity of the clinical course and sometimes even leading to death.

The oral mucosa of HIV-infected patients is also often affected by Herpes zoster and viral warts (papillomas). They are often localized on the mucous membrane of the gums and hard palate.

Hairy Leukoplakia. is observed in HIV-seropositive patients and is considered a lesion associated with immunodeficiency and HIV infection. The presence of hairy leukoplakia is an indicator of HIV. According to recent literature data, \$98\%\$ of patients examined with hairy leukoplakia are HIV-seropositive. Manifestations of hairy leukoplakia can vary in size and localization, they can be unilateral or bilateral. Clinically, hairy leukoplakia appears as white folds or wrinkles that rise above the surface of the oral mucosa, hair-like in shape. Attempts to scrape the surface of the mucous membrane are unsuccessful. Hairy leukoplakia is most often localized on the lateral surfaces of the tongue, and can spread to its entire dorsal surface; less often it can manifest on the mucous membrane of the lips, cheeks, floor of the mouth, and palate.

Differential diagnosis of hairy leukoplakia is carried out with contact allergic reactions to dental materials, true leukoplakia and leukoplakia of the Tabak-Topper,

LPL, hyperplastic candidiasis, and lesions of the oral mucosa with galvanism phenomena.

Ulcerative Necrotic Gingivostomatitis A sign of HIV infection in the oral cavity is ulcerative gingivostomatitis. The disease begins acutely, suddenly, or imperceptibly with bleeding gums when brushing teeth. The acute course of ulcerative necrotic gingivitis can last 3-4 weeks, after which in many patients the disease transitions into a chronic form and recurs frequently. HIV-associated ulcerative necrotic gingivostomatitis is characterized by a progressive course leading to the loss of bone tissue and bone structures, often with sequestration.

In HIV-infected patients, an aggressive form of periodontitis with generalization of the process in periodontal tissues can be observed. Marked gum bleeding and hyperplasia are noted, tooth mobility increases, and diffuse destruction of alveolar bone tissue is observed.

Kaposi's Sarcoma. This is one of the most characteristic clinical manifestations of AIDS. The tumor is formed from lymphatic vessels. It often affects the skin of the hands and feet. This is one of the most characteristic clinical manifestations of AIDS. The tumor is formed from lymphatic vessels. It often affects the skin of the hands and feet. This is one of the most characteristic clinical manifestations of AIDS. The tumor is formed from lymphatic vessels. It often affects the skin of the hands and feet. This is one of the most characteristic clinical manifestations of AIDS. The tumor is formed from lymphatic vessels. It often affects the skin of the hands and feet. In HIV infection, Kaposi's sarcoma is described as an early symptom in 30% of cases. It is more common in whites and homosexuals than blacks and drug addicts. In the oral cavity, in most of the cases described, Kaposi's sarcoma settles on the palate, less often it is found on the mucous membrane of the gums. In the early stages, flat, purple, reddish or dark spots appear. They then darken, enlarge, often become lumpy, and bleed. On the mucous membrane of the mouth, a wound is observed more often than on the skin. Lesions in the oral cavity are painful during the wound stage. In the mucous membrane of the mouth, a wound is observed more often than on the skin. On the mucous membrane of the mouth, a wound is observed more often than on the skin. Lesions in the oral cavity are painful during the wound stage. Kaposi's sarcoma is similar in appearance to epulis when positioned above the gums. In the diagnosis of HIV infection, along with the symptoms described in the oral cavity, it is necessary to take into account other signs of the disease that complement the clinical signs of immunodeficiency: lymphadenopathy, pneumocystic pneumonia, unexplained weight loss, damage to internal organs and systems, including the nervous system, are damaged. When collecting Anamnesis, attention should be paid to the use of other drugs that can lead to head and side diseases, corticosteroids, cytostatics and disorders of the immune system. When collecting Anamnesis, attention should be paid to the use of other drugs that can lead to head and side diseases, corticosteroids, cytostatics and disorders of the immune system. The diagnosis of HIV infection can be made by identifying virus-specific antibodies in the laboratory. The most commonly used immunosorbent analysis (ELISA) is also immunoblotting (Western

blot) or indirect immunofluorescence methods. A certain role in the diagnosis of HIV infection is aimed at studying the immunological state, since in HIV patients the ratio of T - aids to t-suppressors decreases, the number of leukocytes and lymphocytes decreases, the amount of immunoglobulins increases, especially a and G. certain role in the diagnosis of HIV infection is aimed at studying the immunological state, since in HIV patients the ratio of T - aids to t-suppressoIn the oral cavity, Kaposi's sarcoma in most of the described cases is located on the palate, less often it is found on the mucous membrane of the gums. In the initial stages, flat, purple, reddish, or black spots appear. Subsequently, they darken, enlarge, often become nodular, and ulcerate. Ulceration is observed more often on the oral mucosa than on the skin. Lesions in the oral cavity become painful in the ulcer stage. When located on the gums, Kaposi's sarcoma resembles an epulis.

In the diagnosis of HIV infection, along with the described symptoms in the oral cavity, other signs of the disease that complement the clinical picture of immunodeficiency should be taken into account: lymphadenopathy, pneumocystis pneumonia, unexplained weight loss, damage to internal organs and systems, including the nervous system. When collecting the history, attention should be paid to past and concomitant diseases, and the use of corticosteroids, cytostatics, and other drugs that can cause disorders of the immune system.

The diagnosis of HIV infection can be established in the laboratory by detecting virus-specific antibodies. The most commonly used are the enzyme-linked immunosorbent assay (ELISA), as well as immunoblotting (Western blot) or indirect immunofluorescence methods. A certain role in the diagnosis of HIV infection is given to the study of the immunological status, since in HIV-infected patients the ratio of T-helpers to T-suppressors decreases, the number of leukocytes and lymphocytes decreases, and the content of immunoglobulins increases, especially A and G. However, these indicators also change with secondary immunodeficiencies of various etiologies, which reduces their value in the diagnosis of HIV infection.

Prevention of HIV infection. The most important part of effective daily HIV prevention is the application of traditional principles of sterilization and

disinfection. Prevention of HIV infection. The most important part of effective daily HIV prevention is the application of traditional principles of sterilization and disinfection. To prevent HIV transmission, it is prevention of HIV infection. The most important part of effective daily HIV prevention is the application of traditional principles of sterilization and disinfection. To prevent HIV transmission, it is enough to follow the recommendations to prevent infection with blood-borne infectious agents such as the hepatitis B virus.

The fight against HIV infection includes:

Precautions for injections and procedures involving skin puncture; Effective sterilization and disinfection. Precautions for injections and procedures involving skin puncture; Effective sterilization and disinfection. Since many dental procedures are invasive, they often cause bleeding and HIV is a blood infection, as a result of which the dentist is at risk of contracting HIV infection. Therefore, dentists, middle and small medical personnel should take precautions against blood and other body fluids (pus, saliva). Hands and other parts of the body that are contaminated with blood or other biological fluids should be thoroughly washed with soap and water. After removing the protective gloves, the hands should also be washed immediately. Hands and other parts of the body that are contaminated with blood or other biological fluids should be thoroughly washed with soap and water. After removing the protective gloves, the hands should also be washed immediately. Gloves should be worn when in direct contact with blood and other body fluids. During procedures in which splashes or suspensions of blood may appear (for example, the spread of aerosols from high-speed dental units and ultrasound machines), the eyes, nose and mouth should be protected with a mask and glasses or a plastic shield. During procedures in which splashes or suspensions of blood may appear (for example, the spread of aerosols from high-speed dental units and ultrasound machines), the eyes, nose and mouth should be protected with a mask and glasses or a plastic shield. To prevent injections, cuts and other injuries, it is

necessary to work very carefully with needles, scalpels, discs, endodontics and other sharp instruments. Disposable tools must be destroyed after use. If injuries with sharp tools are possible (for example, during cleaning), it is recommended to wear extra thick gloves and use the tools very carefully. It is advisable to use special ultrasonic devices for mechanical cleaning of small devices from contamination. Doctors With exudative skin lesions should not undergo invasive procedures and treat infected patients. Doctors With exudative skin lesions should not undergo invasive procedures and treat infected patients. An important condition for the Prevention of HIV infection through devices used to pierce the skin, mucous membranes and other invasive procedures is their reliable sterilization. Doctors With exudative skin lesions should not undergo invasive procedures and treat infected patients. An important condition for the Prevention of HIV infection through devices used to pierce the skin, mucous membranes and other invasive procedures is their reliable sterilization. HIV is very sensitive to traditional high-intensity sterilization and disinfection methods. Inactivated using methods developed to suppress other pathogens (e.g. hepatitis B virus). The most effective method of inactivating HIV is high temperature, so one of the most reliable methods for sterilizing instruments (including dental instruments) is high-intensity sterilization and disinfection based on heating (boiling, autoclaving, sterilization with dry hot air).

High-intensity disinfection can also be carried out using disinfectants: 2% glutaral solution (glutaraldehyde), 30% hydrogen peroxide solution. Moistening the cleaned instruments in these solutions for 30 minutes ensures a high disinfection intensity. For the treatment of medical tables, bormashins, crowns, and other surfaces around them, it is sufficient to wash them with a medium or low-intensity disinfectant solution containing chlorine-producing compounds, such as sodium hypochlorite, calcium hypochlorite, 4% chloramine solution, sodium dichloroisocyanurate, 4% chlorhexidine solution. The disinfecting ability of these chlorine-emitting compounds is measured by the presence of "active" chlorine in them (as a percentage). For example, sodium hypochlorite contains 5% "active" chlorine, calcium hypochlorite - 70%, chloramine - 25%. For the treatment of body

tissues, bactericidal antiseptics are used (70% ethyl alcohol, 70% isopropyl alcohol, 1% iodine solution, 10%). Disinfection of dental materials is carried out by cleaning them of saliva and blood (impression material, bite detection material, etc.). Before admission to the laboratory, it is necessary to clean and disinfect the plaster and intraoral orthodontic and orthopedic structures.

Vincent ulcerative necrotic stomatitis

Vincent's Ulceronecrotic Stomatitis (*Stomatitis Ulceronecroticans Vincenti*)

Vincent's ulceronecrotic stomatitis (*Stomatitis ulceronecroticans Vincenti*) has been described under various names: ulcerative gingivitis and ulcerative stomatitis, ulceromembranous stomatitis, fusospirochetal stomatitis, Plaut-Vincent stomatitis, "trench mouth," ulcerative film stomatitis, and others. According to modern classification, the disease is called "Vincent's Ulceronecrotic Stomatitis" or "Vincent's Stomatitis."

Etiology. Vincent's ulceronecrotic stomatitis is caused by the symbiosis of **fusobacteria** and **Vincent's spirochetes**. Under normal conditions, these microorganisms are representatives of the resident microflora of the oral cavity and are found in small quantities in all people with teeth. They are mainly found in the gingival sulcus, periodontal pockets, carious cavities, and crypts of the palatine tonsils. The number of fusobacteria and spirochetes sharply increases in an unsanitized oral cavity, with poor hygiene, and in periodontitis. The development of Vincent's ulceronecrotic stomatitis is associated with a sharp decrease in the body's resistance to infection, which can be caused by viral diseases (ARVI, herpetic stomatitis, pneumonia, etc.), vitamin deficiency, stress, overwork, and malnutrition. Ulceronecrotic gingivitis often complicates the course of severe widespread diseases (leukemia, agranulocytosis, pneumonia, infectious mononucleosis). It can appear as a complication of erythema multiforme exudativum (EMEE) and erosive allergic stomatitis.

The virulence of fusobacteria and spirochetes increases when the specific and non-specific defense mechanisms in the body are disturbed. Their number multiplies to such an extent that they become dominant over other microflora. The decrease in the

body's overall resistance negatively affects the resistance of the oral mucosa, which, being a reliable barrier to the introduction of infection, has its integrity disturbed. This always occurs in a poorly hygienic oral cavity, where the presence of local traumatic factors (sharp edges of teeth, prostheses, etc.) creates conditions for the introduction of fusobacteria and spirochetes. Therefore, Vincent's ulceronecrotic stomatitis often develops in people with unsatisfactory oral hygiene.

Clinical Picture

Patients are mainly young people (17-30 years old), predominantly male. Chilling/exposure to cold is often a precipitating factor in the development of Vincent's ulceronecrotic stomatitis, which may explain its occurrence in the autumn-spring period; the maximum incidence occurs from October to December.

Clinically, there is an acute and chronic course of Vincent's ulceronecrotic stomatitis, and its severity is differentiated into mild, moderate, and severe forms.

The disease begins acutely with a rise in body temperature to 37.5-38. Local lymph nodes are enlarged, thickened, painful on palpation, and maintain mobility. Patients complain of headache, general malaise, pain in the oral mucosa, difficulty eating and speaking, bleeding gums, hypersalivation, and foul odor from the mouth (fedor ex ore). The appearance of catarrhal inflammation in the oral mucosa quickly turns into ulceration. The process often begins on the gums, then moves to other areas of the mucous membrane. The gums are hyperemic, sharply painful, and bleed when touched. The epithelium on the gingival margin and the papillae between the teeth becomes dull and then necrotic. As a result, the gingival margin looks incised, with uneven pits, and its surface is covered with an easily removable grayish-yellow plaque. Subsequently, the affected gum margin is not fully restored and becomes deformed. The area of the lower eighth teeth is often affected, where necrosis quickly spreads to the buccal mucosa of the alveolar process and the retromolar area, sometimes causing trismus and pain during swallowing. Ulcers on the buccal mucosa can reach large sizes (up to 5-6 cm in diameter) and depth. Their edges are irregular and soft. The base is covered with a thick grayish-green necrotic plaque, which,

when removed, exposes the bleeding base of the ulcer. The mucous membrane around the ulcer is swollen and hyperemic.

In the presence of local traumatic factors in the oral cavity (roots of destroyed teeth, sharp edges of teeth, prostheses), other parts of the oral mucosa (hard, soft palate, arches, tonsils, tongue) may also be affected. Ulcers in the oral cavity can be single or multiple, of different sizes and depths. When the ulcer is localized on the hard palate, necrosis of all layers develops very quickly. The mucous membrane and bone are exposed. Angina of Simanovsky-Plaut-Vincent is, as a rule, unilateral and rare in dental practice. 2-3 weeks after the onset of ulceronecrotic stomatitis, the process usually resolves with complete epithelialization of the ulcerated surfaces. In rare cases, if treatment is not carried out or is ineffective, a chronic form of Vincent's ulceronecrotic stomatitis develops, in which general symptoms are not observed. Patients have persistent bleeding and pain in the gums, as well as a bad smell from the mouth. Upon careful examination of the gingival margin, exposed bone tissue is detected. Only certain groups of teeth are affected. In patients with chronic Vincent's ulceronecrotic stomatitis, the lymph nodes are hardened, slightly painful. When the disease lasts 4-8 months, the lymph nodes acquire a cartilaginous consistency.



Figure 45. Histological Picture and Diagnosis of Vincent's Ulceronecrotic Stomatitis

Pathohistological examination of the areas of the wound edges of the gums identifies two zones: superficial - necrotic and deep-inflammatory. Necrotic in the surface layers of the tissue, a different microflora is detected. Pathohistological examination of the areas of the

wound edges of the gums identifies two zones: superficial - necrotic and deep-inflammatory. Necrotic in the surface layers of the tissue, a different microflora is detected. (cocci, bacilli, Fusobacteria, spirochetes, etc. In the deep layers, Fusobacteria and Spirochetes strongly dominate the lower layers of connective tissue inflamed: they are swollen, the veins dilated. Formed blood elements are found in perivascular inflammatory infiltrate. In the same area of inflammation, only Spirochetes are found inside the intact tissue that penetrate between the epithelial cells.

In patients with ulcerative necrotic stomatitis of wensan, the cytological picture of scraping from the ulcerative surfaces of the mucous membrane corresponds to an unspecific inflammatory process. In patients with ulcerative necrotic stomatitis of wensan, the cytological picture of scraping from the ulcerative surfaces of the mucous membrane corresponds to an unspecific inflammatory process. In patients with ulcerative necrotic stomatitis of wensan, the cytological picture of scraping from the ulcerative surfaces of the mucous membrane corresponds to an unspecific inflammatory process. At the beginning of the disease, an excess of unstructured masses, a sharp predominance of neutrophils, is detected, mainly in a state of decay, and erythrocytes (due to heavy bleeding). In the second period of the disease, when Recovery begins, full-fledged large phagocytic cells, many macrophages, are detected, along with fragmented neutrophils. During the period of the onset of epithelization, layers of young epithelial cells appear, the number of Fusobacteria and Spirochetes decreases. During the period of the onset of epithelization, layers of young epithelial cells appear, the number of Fusobacteria and Spirochetes decreases. In the chronic course of wensan's ulcerative necrotic. During the period of the onset of epithelization, layers of young epithelial cells appear, the number of Fusobacteria and Spirochetes decreases. In the chronic course of wensan's ulcerative necrotic stomatitis, th.

Comparative diagnosis. Vensan's ulcerative necrotic stomatitis should first be distinguished from ulcerative jaxoxatallas in blood diseases (leukemia, agranulocytosis, infectious mononucleosis). Comparative diagnosis. Vensan's

ulcerative necrotic stomatitis should first be distinguished from ulcerative stomatitis in blood diseases (leukemia, agranulocytosis, infectious mononucleosis). In addition to clinical differences (whitening of the oral mucosa, bleeding, the presence of leukemic infiltrate).



Image 46. Acute ulcerative necrotizing stomatitis. Wound scraping image 46. Acute ulcerative necrotizing stomatitis. Fusospirochetosis. Wound deep decay Stage (2).

Ulcerative necrotic stomatitis differs from allergic stomatitis on the basis of Anamnesis data, features of clinical manifestations and the results of bacterioscopic examination. Ulcerative necrotic stomatitis differs from allergic stomatitis on the basis of Anamnesis data, features of ulcerative necrotic stomatitis differs from allergic stomatitis on the basis of Ulcerative necrotic stomatitis differs from allergic stomatitis on the basis of Anamnesis data, features of clinical manifestations and the results of bacterioscopic examination. A ulcerative necrotic process in the oral cavity similar to Vincent's stomatitis can occur with mercury intoxication. If contact with mercury is detected, a urine test is performed for the mercury content. It should be remembered that ulcerative necrotic damage to Oral mucosa can complicate the course of specific infections (venereal, HIV infection) or tumors of poor quality (cancer, sarcoma). In order to avoid diagnostic errors in these cases, it is necessary to carefully collect Anamnesis, take into account not only local, but also the general clinical picture of the disease and carry out the

necessary laboratory tests (clinical blood test, Wasserman serological reactions or HIV infection detection, cytological, bacterioscopic, pathohistological, etc.).

Treatment. The volume of measures for the treatment of wensan for ulcerative necrotic stomatitis is largely determined by the severity of its course.reatment. The volume of measures for the treatment of wensan for ulcerative necrotic stomatitis is largely determined by the severity of its course. The disease often occurs in young people, most of reatment. The volume of measures for the treatment of wensan for ulcerative necrotic stomatitis is largely determined by the severity of its course. The disease often occurs in young people, most of them are almost healthy, local treatment is decisive. The earlier and better the oral cavity is treated, the faster the patient's condition improves. High-quality treatment of Oral mucosa, the removal of traumatic factors is decisive in the further course and results of the disease.

Local treatment consists in the removal of local traumatic factors, necrotic tissues, exposure to microflora and stimulation of the regeneration processes of the oral mucosa.ocal treatment consists in the removal of local traumatic factors, necrotic tissues, exposure to microflora and stimulation of the regeneration processes of the oral mucosa. Treatocal treatment consists in the removal of local traumatic factors, necrotic tissues, exposure to microflora and stimulation of the regeneration processes of the oral mucosa. Treatment of the oral cavity is carried out under anesthesia. For this purpose, application and injection anesthetic with solutions of pyromecaine, trimecaine, lidocaine are used. After that, all substances that characterize mechanical jaro chats are removed: the sharp edges of teeth and dentures are smoothed, tartar and Carache are removed. Caries cavities should be treated with antiseptic solutions.aries cavities should be treated with antiseptic solutions. Removal of removable teeth should be done to the epithelization of the wound, since the removal of teeth in the infected oral cavity of a patient with ulcerative necrotic stomatitis will be accompanied by serious aries cavities should be treated with antiseptic solutions. Removal of removable teeth should be done to the epithelization of the wound, since the removal of teeth in the infected oral cavity

of a patient with ulcerative necrotic stomatitis will be accompanied by serious complications (alveolitis, periostitis, abscess, phlegmona). Cleaning wound surfaces from necrotic tissue is carried out with the help of proteolytic enzymes: trypsin, chymotrypsin, lysoamidase, deoxyribonuclease.

The entire oral cavity is treated with warm antiseptic solutions (0.5% hydrogen peroxide yeritma, 1% chloramine yeritma, 0.06% chlorhyexidine yeritma, 0.5% ethonium solution, 1:5000 potassium permanganate solution, etc.). The entire oral cavity is treated with warm antiseptic solutions (0.5% hydrogen peroxide yeritma, 1% chloramine yeritma, 0.06% chlorhyexidine yeritma, 0.5% ethonium solution, 1:5000 potassium permanganate solution, etc.). Milk pockets, wound surface, interdental spaces should be washed with a pressure stream. On the first visit, it is necessary to treat the entire oral cavity. Then daily processing is carried out. At the patient's home, rinses with warm antiseptics are prescribed: 3% hydrogen peroxide solution (2 tablespoons per glass of water), 0.25% chloramine solution, potassium permanganate solution (1:5000), 0.06% chlorhyexidine solution or other antiseptic preparations every 3-4 hours.

Local treatment with mild course of vensan's ulcerative necrotic stomatitis is sufficient. In more severe cases, general therapy is necessary. Local treatment with mild course of vensan's ulcerative necrotic stomatitis is sufficient. In more severe cases, general therapy is necessary. Local treatment with mild course of vensan's ulcerative necrotic stomatitis is sufficient. In more severe cases, general therapy is necessary. Local treatment with mild course of vensan's ulcerative necrotic stomatitis is sufficient. In more severe cases, general therapy is necessary. For antimicrobial action, metronidzol (trichopol, flagyl, CLion) is prescribed for 7-10 days 2 times a day in 0.25 g. Metronidazole is also used for local treatment (liquid water slurry is applied to ulcerative surfaces after removal of necrotic tissue). In severe cases, broad-spectrum antibiotics are sometimes used (Bicillin-3, 300,000 units once every 3 days, or 600,000 units once every 6 days; yerithromycin, oletetrin, oxytetracycline in daily dose 800 000—1 000 000 5-10 eating in the day). yerithromycin, oletetrin, oxytetracycline in daily dose

800 000— 1 000 000 5-10 eating in the day). At any weight level, it is recommended to prescribe B vitamins, ascorutin (0.1 g orally 2 times a day, for 10-14 days).

With proper treatment, the improvement in the condition of patients with wensan's ulcerative necrotic stomatitis occurs after 24-48 hours, the pain decreases or disappears, patients can eat, sleep. With proper treatment, the improvement in the condition of patients with wensan's ulcerative necrotic stomatitis occurs after 24-48 hours, the pain decreases or disappears, patients can eat, sleep. Swelling and oper treatment, the improvement in the condition of patients with wensan's ulcerative necrotic stomatitis occurs after 24-48 hours, the pain decreases or disappears, patients can eat, sleep. Swelling and hyperemia of the oral mucosa decreases, and epithelization of the wound begins, which is completed up to 3-6 days with a mild degree of the disease and a satisfactory condition of the oral cavity. In an unsanitized oral cavity, epithelialization of ulcerated surfaces proceeds more slowly. After the general condition of the patient has improved and acute inflammation has disappeared, it is necessary to thoroughly sanitize the oral cavity with the removal of tartar, tooth roots, treat caries teeth, periodont diseases.

With timely and correct treatment of the consequences, the prognosis is favorable. In the acute process, epithelization of ulcerative surfaces occurs after 3-6 days, in the chronic process — a little slower. With timely and correct treatment of the consequences, the prognosis is favorable. In the acute process, epithelization of ulcerative surfaces occurs after 3-6 days, in the chronic process — a little slower. When there is timely and correct treatment of the consequences, the prognosis is favorable. In the acute process, epithelization of ulcerative surfaces occurs after 3-6 days, in the chronic process — a little slower. When there are many traumatic factors in the unsanitary oral cavity and timely or improper treatment, there may be a lack (retardation) or deformation of the gums, resorption of the bone tissue of the alveolar tumor. These changes contribute to the further development of periodontitis. Patients suffering from wensan stomatitis are actively monitored for a year. The first examination is carried out after 1-2 months, the next after 6 months.

Prevention. It consists in maintaining oral hygiene, regular sanitation, full and timely treatment of infectious and other diseases that lead to a decrease in immunity.

Syphilis

Syphilis (syphilis, lues) is a chronic infectious disease, the causative agent of which is a whitish treponema. Syphilis (syphilis, lues) is a chronic infectious disease, the causative agent of which is a whitish treponema. The mucous membrane of the mouth is the plaque. Syphilis (syphilis, lues) is a chronic infectious disease whose causative agent is isaxm. (syphilis, lues) is a chronic infectious disease, the causative agent of which is a whitish treponema. The mucous membrane of the mouth is the site where syphilitic rashes deposit during all periods of syphilis. There is congenital and acquired syphilis. In congenital venomous, pale treponema enters the fetus through the placenta from a sick mother. Orthic syphilis develops when it is transmitted through direct contact (primarily sexual), through the skin and mucous membranes, or through various objects contaminated with secretions containing the pathogen. If the sterilization regimen is violated, the source of infection can be.

Due to the wavy course of syphilis, four periods are distinguished: incubation, primary, secondary, and tertiary, characterized by a change in the periods of active manifestations and latent stages of the disease, with various clinical and morphological signs occurring at different stages of the disease.

The incubation period of syphilis lasts an average of 3-4 weeks.

Primary Syphilis - Bleeding begins at the entry point of the treponema. Syphilis lasts from 6-7 weeks to several months. In the first 3 weeks, it is seronegative, then seropositive, i.e., the serological reaction is positive. When pale treponema enters the oral mucosa, primary syphiloma develops, and a dense, painful ulcer (hard chancre) develops. The hard chancre is found on the red border of the lips and very rarely on the oral mucosa. Solid chancre - consists mainly of lymphoid and plasma cells, 2-3 cm in diameter, dense, infiltrated, with a limited base. A flesh-red erosion develops in the central part of the infiltrate, which hardens after a few weeks. 1-2 weeks after the formation of the ulcer, painless, dense, elastic, mobile lymph nodes appear.

Often, one hard chancre is found in the mouth (95%), rarely it is common. Primary syphilis is accompanied by weakness, fever, headache, and fatigue.

Comparative diagnosis. Herpes simplex, chronic recurrent aphthous stomatitis, chancre-shaped pyoderma, with general sore throat, cancerous tumors, miliary-ulcerative tuberculosis, traumatic erosion, lupus erythematosus, trophic ulcer

Simple herpes. Before the appearance of erosion, the eruption of vesicles is observed, which quickly rupture, forming an erosion, and are prone to rapid epithelialization, which is not characteristic of hard chancre. Multinucleated giant cells are detected during cytological examination.

Chronic recurrent aphthous stomatitis is the main element of aphthous stomatitis. The base of the aphtha is soft, there is no hardening, usually after healing, the aphtha does not leave a trace, but in the deep or deforming aphthous form - the ulcerative formation deepens and heals with the formation of a scar. When scraped from the surface of the aphtha, pale treponema is not detected.

Cancer wound. It is located deeper than the hard chancre, its bottom is dense and the edges are uneven, resembling cauliflower, the lower part is soft, easily bleeding. The cancer ulcer can develop after traumatic erosion, the erosive ulcerative form of leukoplakia, the erosive ulcerative, hyperkeratotic form of lichen planus. Atypical cells are found on the surface of the ulcer during cytological examination.



Figure 47. Syphilitic cut in corner of lip



Figure 48. Hard chancre at the tip of the tongue

Secondary syphilis-begins 4-10 weeks after the appearance of the wound. It begins with an injury to the skin, mucous membrane of the oral cavity and internal organs. Secondary syphilis begins 4-10 weeks after the appearance of the wound. It begins with an injury to the skin, mucous membrane of the oral cavity and internal organs. Secondary syphilis lasts an average of 3-4 years. In half of patients with symptoms of secondary poisoning, damage to the oral mucosa in the form of roseolous and papular elements is observed. Pustulous rashes on the mucous membrane of the mouth are very rare. In the secondary period of venereal poisoning, latent periods can be observed several times (3-4 or more), which is explained by the individual state of immunity inherent in the venereal. Rashes of secondary recurrent venereal are different from rashes in secondary new venereal: roseolous-the number of papular elements decreases. The Wasserman reaction and other serological reactions are positive during the secondary period of the venereal.

Roseola is a specific element of the secondary period of syphilis. The oral mucosa usually appears in the palate arcs, soft palate, tonsils. Roseola is a specific element of the secondary period of syphilis. The oral mucosa usually appears in the palate arcs, soft palate, tonsils. The roseola is a specific element of the secondary period of

syphilis. The oral mucosa usually appears in the palate arcs, soft palate, tonsils. The roseola has the property of joining, as a result of which it occupies several areas. Damaged Sox have a border, tishi will be red or copper-red. Unlike catarrhal angina, syphilis angina does not have a subjective feeling of pain, accompanied by an increase in body temperature.

Papulosis is a frequent manifestation of the secondary period of syphilis in the oral cavity, especially in kayta skating. apulosis is a frequent manifestation of the secondary period of syphilis in the oral cavity, especially in kayta skating. They can appear anywhere on the mucous membrane, but the most apulosis is a frequent manifestation of the secondary period of syphilis in the oral cavity, especially in kayta skating. They can appear anywhere on the mucous membrane, but the most favorite location Sox are the tonsils, the arch of the palate, the soft palate, which can merge and form a papular angina. Most often, papules are formed on the tongue, cheeks, gums, the type of papula depends on the duration of its existence. At first, when a papule appears, it wears a stable-hyperemic area on the mucous membrane (up to peas in size), and a small infiltration appears under it. t first, when a papule appears, it wears a stable-hyperemic area on the mucous membrane (up to peas in size), and a small infiltration appears under it. The papule margin is distinct, with t first, when a papule appears, it wears a stable-hyperemic area on the mucous membrane (up to peas in size), and a small infiltration appears under it. The papule margin is distinct, with a soft consistency and is purplish. When the surface of the papule is scratched (high-matsification (glare) of the epithelium is replaced by Red erosion. Okish treponema is always found on the surface of erosion. In areas that cause damage to the oral mucosa, papules can rise significantly and merge in the form of hard plaque. When eating spicy food, the pain actually disappears or is very insignificant, when the papules are located in the corner of the mouth, pain occurs due to constant damage to the infiltrated skin and the mucous membrane of the corner of the mouth. hen eating spicy food, the pain actually disappears or is very insignificant, when the papules are located in the corner of the mouth. hen eating spicy food, the pain actually disappears or is very insignificant, when the papules are

located when eating spicy food, the pain actually disappears or is very insignificant, when the papules are located in the corner of the mouth, pain occurs due to constant damage to the infiltrated skin and the mucous membrane of the corner of the mouth. Poison writhing bleeds, becomes covered with a thick, and spreads over the surrounding skin, taking on the appearance of a kandidom. When papules are disturbed in the oral cavity, they are prone to hypertrophy and develop into broad warts. This is possible when the papules are located along the gburm of the transition, along the adhesive of the teeth on the cheeks, along the lateral parts of the tongue. In the course of poor quality, it soon decomposes and turns into deep painful wounds, purulent necrotic tissues, and large swollen infiltrates are observed, which are often accompanied by acute inflammation. In the course of poor quality, it soon decomposes and turns into deep painful wounds, purulent necrotic tissues, and large.

Comparative diagnosis-roseolous rashes differ from catarrhal sore throat, medicinal rashes. omparative diagnosis-roseolous rashes differ from catarrhal sore throat, medicinal rashes. This omparative diagnosis-roseolous rashes differ from catarrhal sore throat, medicinal rashes. This is an increase iomparative diagnosis-roseolous rashes differ from catarrhal sore throat, medicinal rashes. This is an increase in body temperature, pain, edema of both tonsils anrs.



Image 49. Secondary damage

Diagnosis of the secondary period of syphilis is established on the basis of clinical symptoms, laboratory tests (detection of pale Treponema in damage to elements, positive Wasserman reactions, sedimentation and RIBT

The third period of syphilis-syphilis is not observed in all patients, even those who have not been treated. The disease begins 4-6 years after its onset. The third period of syphilis is not observed in all patients, even those who have not been treated. The disease begins 4-6 years after its onset. This period can extend for decades and is characterized by the third period of syphilis is not observed in all patients, even those who have not been treated. The disease begins 4-6 years after its onset. This period can extend for decades and is characterized by the formation of inflammatory infiltrates (gum and Hawthorn), is prone to decay and often causes significant irreversible changes in organs and tissues. The number of leakage treponemes in the body is significantly reduced, the Wasserman reaction can be positive in 60-80% of patients.

Gummosis Syphilis is a manifestation of the third stage of Syphilis. Gummosis Syphilis is a manifestation of the third stage of Syphilis. Gumma can be located anywhere on the mucous membrane of the oral cavity, but is often formed on Gummosis Syphilis is a manifestation of the third stage of Syphilis. Gumma can be located anywhere on the mucous membrane of the oral cavity, but is often formed on the soft palate, haummosis Syphilis is a manifestation of the third stage of Syphilis. Gumma can be located anywhere on the mucous membrane of the oral cavity, but is often formed on the soft palate, hard palate, tongue. Gumma can be one or two. The disease begins with the appearance of a painless node (a foci of syphilitic necrotic inflammation), which gradually grows (the mucous membrane above it becomes stagnant-red). Then it opens, from where the gummosis core is torn off, and a gummosis granular wound is formed, even with dense edges. It is characterized by the formation of a star-shaped scar. The procedure can take 3-4 months and practically does not produce subjective signs in the patient. It is characterized by the formation of a star-shaped scar. The procedure can take 3-4 months and practically does not produce subjective signs in the patient. One of the

most severe forms of manifestat is characterized by the formation of a star-shaped scar. The procedure can take 3-4 months and practically does not produce subjective signs in the patient. One of the most severe forms of manifestation of tertiary syphilis in the oral head is diffuse interstitial syphilitic glossitis, in which there is a strong infiltration of the tongue by plasma cells. In this case, the tongue becomes denser, its mucous membrane thickens. Over time, the Infiltrate changes with scar tissue, which leads to deformation of the tongue, painful cracks and trophic ulcers form on its surface. Over time, the Infiltrate changes with scar tissue, which leads to deformation of the tongue, painful cracks and trophic ulcers form on its surface. Gummosis against tver time, the Infiltrate changes with scar tissue, which leads to deformation of the tongue, painful cracks and trophic ulcers form on its surface. Gummosis against the background of interstitial glossitis, gummas may appear on aloxia. Gummosis injury to the hard palate can initially manifest itself only in the form of a runny nose with purulent or bloody discharge, and then a puncture of the palate. Food enters the nasal cavity due to a violation of the hard and soft palate, perforation of the palate. A similar process can occur in the nose, causing the bridge of the nose to suddenly be pulled back (saddle shape).

Diagnosis and comparative diagnosis . Solid chancre is more difficult to diagnose when its atypical form is in the form of a fracture or in the form of cracks. iagnosis and comparative diagnosis . Solid chancre is more difficult to diagnose when its atypical form is in the form of a fracture or in the form of cracks. When browsing, it is necessary to stretch the corner of the mouth or the fold Diagnosis and comparative diagnosis . Solid chancre is more difficult to diagnose when its atypical form is in the form of a fracture or in the form of cracks. When browsing, it is necessary to stretch the corner of the mouth or the fold of the skin, which is examined for an oval-shaped dense border lesion groove and base. When the hard chancre settles in mindaline, it is diagnosed from angina. In this case, it is necessary to pay attention to the nature of painless unilateral damage and significant hardness in the poison. Hard chancre on the red border of the lip is diagnosed by erosion on ordinary herpes. That

is, herpes has a soft swelling, and the edges and base are hypirimized, with severe pain and uneven fever. Often the hard chancre was observed in the oral cavity when it was damaged by injury. hat is, herpes has a soft swelling, and the edges and base are hypirimized, with severe pain and uneven fever. Often the hard chancre was observed in the oral cavity what is, herpes has a soft swelling, and the edges and base are hypirimized, with severe pain and uneven fever. Often the hard chancre was observed in the oral cavity when it was damaged by injury.

The rump is stiff and less painful. With a cancer wound, significant difficulties may arise in a comparative diagnosis. The wound surface for cancer has an uneven texture. he rump is stiff and less painful. With a cancer wound, significant difficulties may arise in a comparative diagnosis. The wound surface for cancer has an uneven texture. The final diagnosis is confirmed by cytological examination by findin The rump is stiff and less painful. With a cancer wound, significant difficulties may arise in a comparative diagnosis. The wound surface for cancer has an uneven texture. The final diagnosis is confirmed by cytological examination by finding atypical epithelial cells. Gumma's wound can sometimes be mistaken for a hard chancre. Primary syphilis is largely absent from regionar lymphadenitis haracterous Gumma. The comparative diagnosis of cancer ulcers and severe chancre can be a challenge.

In comparative diagnosis, it is necessary to pay attention to the fact that the deep, uneven edges of the nisabatan are firmly embedded in the wound syphiloma in the cancer wound.

The bottom of the wound is uneven, easily bleeding, and has a ghastly prickly appearance. iagnosis, it is necessary to pay attention to the fact that the deep, uneven edges of the nisabatan are firmly embedded in the wound syphiloma in the cancer wound.

In diagnostics, the data of cytological (atypical cells are detected in cancer) and bacterioscopic studies (detection of leakage Treponema in the composition of ulcers) are decisive. n diagnostics, the data of cytological (atypical cells are detected in cancer) and bacterioscopic studies (detection of leakage Treponema in the

composition of ulcers) are decisive. For all clinical manifestations of syx, bilateral regional lymphadenitis is present, characterized by palpation of dense, moderately enlarged, motile, painless lymph nodes (sometimes slightly painful).

Diagnosis. Based on blood tests for syphilis. They are divided into two groups – triponemic and triponemic. For mass examinations, triponemless analyzes are used. diagnosis. Based on blood tests for syphilis.



Photo 50. Tertiary syphilis

The treatment of syphilis should be complex and individual, the basis of treatment is antibiotics, the treatment of people with syphilis is carried out in dermatovenerology hospitals and dispensaries. Local treatment of syphilitic wound in the oral cavity is carried out with 8% biokhinox suspension, 7% bismoverol suspension, miarsenol solution, 10% novarsenol suspension in glycerin, 10% osarsol suspension in Peach oil and symptomatic agents.

Congenital syphilis

Congenital syphilis differs in many respects from the classic disease. Congenital syphilis is divided into early (from birth to 5 years old) and late (from five years old) stages. differs in many respects from the classic disease. Congenital syphilis is divided into early (from birth to 5 years old) aongenital syphilis differs in many respects from the classic disease. Congenital syphilis is divided into early (from birth to 5 years old) and late (from five years old) stages. Congenital syphilis is transmitted from the mother to the fetus through the placenta. A patient with aongenital syphilis differs in many respects from the classic disease. Congenital syphilis is divided into

early (from birth to 5 years old) and late (from five years old) stages. Congenital syphilis is transmitted from the mother to the fetus through the placenta. A patient with a poison does not develop true immunity. It has been found that patients with and treated with syphilis can become infected with it again (reinfection), which is the development of primary syphiloma and a more characteristic feature of syphilis. The initial stage of poisoning is characterized by various clinical signs. Basically, clinical signs appear two months after the birth of a child. The manifestations of congenital syphilis affect the skin and mucous membranes. The manifestations of congenital syphilis affect the skin and mucous membranes. Syphilitic papules are more common in breast-aged infants in congenital Zahn and are the manifestations of congenital syphilis affect the skin and mucous membranes. Syphilitic papules are more common in breast-aged infants in congenital Zahn and are the manifestations of congenital syphilis affect the skin and mucous membranes. Syphilitic papules are more common in breast-aged infants in congenital Zahn and are similar to the secondary period of Zahn, but differ in their tendency to merge. Papules are more localized in the skin around the mouth and lip, diffuse mouth circumference infiltration is observed-i.e. caused by syphilitic papular exposure. After the papules disappear, scars remain around the mouth in congenital syphilis. In the place of deep papules on the lips and at the corner of the mouth, scars of an uneven, deep, rounded or oval shape are formed. In the place of deep papules on the lips and at the corner of the mouth, scars of.

A characteristic feature of late congenital syphilis (over 5 years of age) is gumma. The gumma affects the hard and soft palate, and interstitial syphilitic glossitis is observed. Dystrophy of the teeth is characteristic of congenital late syphilis.

The triad symptom is characteristic of congenital syphilis:

-parenchymatous keratitis, congenital deafness, Hutchinson's teeth.

Hutchinson's teeth are a developmental anomaly of teeth in which the upper central incisors have a screwdriver or barrel-shaped crown (larger than the edge of the neck) and a crescent-shaped depression (groove) on the incisal edge. Sometimes crescent-shaped teeth are not covered with enamel.

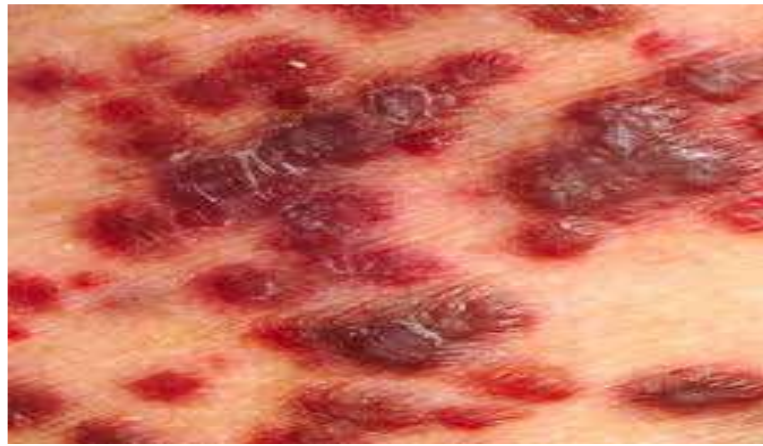


Figure 51. Syphilis

Clinical symptoms provide sufficient information for diagnosis, the doctor confirms the diagnosis according to the results of the study: a microscopic examination of a rash, blisters or nasal mucosa and a blood test for antibodies, a picture of diagnostic ausules, serodiagnostics are prescribed. nical symptoms provide sufficient information for diagnosis, the doctor confirms the diagnosis according to the results of the study: a microscopic examination of a rash, blisters or nasal mucosa alinical symptoms provide sufficient information for diagnosis, the doctor confirms the diagnosis according to the results of the study: a microscopic examination of a rash, blisters or nasal mucosa and a blood test for antibodies, a picture of diagnostic ausules, serodiagnostics are prescribed.

Treatment of congenital venereal disease. The main method of treating syphilis is antibiotic therapy, most often antibacterial substances of the penicillin line are used, cephalosporins can be used. Neuromuscular therapy is carried out in conjunction with antibiotic pyrotherapy.

Complications of congenital syphilis: damage to the central nervous system, deformities of the skull, endolumbar involves the administration of antibiotics, and pyrogenal, prodigiosan is used. omplications of congenital syphilis: damage to the central nervous system, deformities of the skull, endolumbar involves the administration of antibiotics, and pyrogenal, prodigiosan is used. Complications include physical and mental weakness.

Prophylaxis (prevention) of congenital syphilis. The main way to prevent this form of poisoning is through the mandatory examination of women during early pregnancy.rophylaxis (prevention) of congenital syphilis. The main way to prevent this form of poisoning is through the mandatory examination of women during early pregnancy. If this pathology is detected in the early stages of pregnancy, it is possible to carry.

Gonorrhea

Gonorrhea is a disease in which an infectious disease is called by gonococci. This disease is sexually transmitted and is characterized by damage to the mucous membrane of the urinary organs.Gonorrhea is a disease in which an infectious disease is called by gonococci. This disease is sexually transmitted and is characterized by damage to the mucous membranGonorrhea is a disease Gonorrhea is a disease in which an infectious disease is called by gonococci. This disease is sexually transmitted and is characterized by damage to the mucous membrane of the urinary organs.Belonging to sexually transmitted diseases, the route of transmission is damage to the oral mucosa in men who are homosexual and in people who have orogenital contact.

Transmission routes:

- 1.Direct and frequent exogenous injection of gonococci into the oral cavity by autoinoculation or infection;
2. Lymph and hematogen.

The clinical-incubation period is from 1 day to 1 month, or more.he clinical-incubation period is from 1 day to 1 month, or more. 3-4 days later, through intercellular spaces, it reaches the oral cavity to the intercostal subepithelial connective tissue, causing an inflammatory process with the formation of purulent exudate.

Gonococcal stomatitis. On the mucous membrane of the oral cavity, the oral process usually proceeds in an asymptomatic way. In rare cases, there is a sore throat, body temperature rises.onococcal stomatitis. On the mucous membrane of the oral cavity, the oral process usually proceeds in an asymptomatic way. In rare

cases, there is a sore throat, gonococcal stomatitis. On the mucous membrane of the oral cavity, the oral process usually proceeds in an asymptomatic way. In rare cases, there is a sore throat, body temperature rises. The first symptoms of gonococcal stomatitis include hyperemia, edema, the appearance of small erosions in the oral cavity, and sticky mucous - purulent detachment. If left untreated, a large amount of erosion and scarring can occur on the mucous membrane of the cheeks, tongue and teeth. The wound is usually small in size, superficial, uneven, with distorted edges, less painful, yellow-gray Carache, gonococci are found in the detachment. Tongue damage is almost always ulcerative-membranous in nature. The ventral surface is first damaged; then all sides of the tongue can be damaged. Tongue damage is almost always ulcerative-membranous in nature. The ventral surface is first damaged; then all sides of the tongue can be damaged. The mucous membrane turns red and swells, exudate appears, when the lesion increases, a yellowish-purulent discharge appears in the affected areas, when examined, gonococci are found. Any external impact (maturation, minor damage) leads to cracks and erosion, which easily turn into wounds, bleed and become covered with yellowish-gray Carache. The presence of a large number of erosions on the dorsal surface of the tongue is similar to acute allergic glossitis.



Figure 52. Gonadal stomatitis

Differential diagnosis is carried out with diphtheria, drug-induced allergic stomatitis, multiform exudative erythema, ulcerative and fungal stomatitis.

Diagnosis: Diagnosis is confirmed by the presence of gonococcus on exudate microscopy.

Treatment. Treatment of gonorrheal stomatitis is carried out similarly to the treatment of purulent-necrotic lesions of the oral mucosa. Local etiotropic therapy is carried out with antibiotics and other antimicrobial agents (oleandomycin, gentamicin). Before recovery, it is necessary to take measures to protect the patient's family members from infection.

Tuberculosis

Sil-sil Mycobacterium (Cox's stick 1888.) is characterized by a chronic high-load disease that is transmitted from Tomini. This pathology belongs to a chronic disease. **il-sil Mycobacterium** (Cox's stick 1888.) is characterized by a chronic high-load disease that is transmitted from Tomini. This pathology **il-sil Mycobacterium** (Cox's stick 1888.) is characterized by a chronic high-load disease that is transmitted from Tomini. This pathology belongs to a chronic disease. As a rule, the inflammation characteristic of tuberculosis is inappropriate for the oral mucosa and as a secondary sign around the lips. The mucous membrane of the oral cavity is not inferior to the most favorable environment for the development and reproduction of mycobacteria that cause tuberculosis. As a rule, mycobacteria enter this environment and die. However, in the presence of favorable conditions (microtrauma, mechanical damage to the oral cavity, opening the sockets for infection), bacteria penetrate inside and lead to the formation of a tuberculosis wound. **owever**, in the presence of favorable conditions (microtrauma, mechanical damage to the oral cavity, opening the sockets for infection), bacteria penetrate inside and lead to the format **owever**, in the presence of favorable conditions (microtrauma, mechanical damage to the oral cavity, opening the sockets for infection), bacteria penetrate inside and lead to the formation of a tuberculosis wound. Tuberculosis is transmitted by airborne droplets. The incubation period lasts from eight to thirty days, and after that the formation of a clearly painful uneven, blurred wound begins. After a few days, it gradually begins

to grow. At the same time, the adjacent lymph nodes swell. Tuberculosis of the oral cavity occurs mainly in immunocompromised patients and is a disease that develops. The disease is caused by Cox's wand. Tuberculosis of the oral cavity occurs mainly in immunocompromised patients and is a disease that develops. The disease is caused by Cox's wand. Inflammatory foci located in the oral cavity of patients are in most cases secondary. They develop as a result of the spread of infection from the primary focus and spread through the circulatory or lymphatic system. If the patient has pulmonary tuberculosis, the infection spreads to the oral cavity due to the penetration of mycobacteria in the sputum.

The routes of infection spread:

- Exogenous, endogenous (through the lymphatic system, through the person, through the lymphatic system)
- Factors that are important in the pathogenesis of tuberculosis
- General condition of the organism Exogenous, endogenous through the lymphatic system, through the person, through the lymphatic system)
- Factors that are important Exogenous, endogenous (through the lymphatic system, through the person, through the lymphatic system)
- Factors that are important in the pathogenesis of tuberculosis
- General condition of the organism
- Dietary habits
- Home - household conditions
- Changes in the nervous and endocrine systems

There are two types of tuberculosis:

- Primary (very rare in the oral cavity)
- Secondary (silicosis of the oral cavity,

Sil runner. the most common form is secondary tuberculosis of the facial jaw socket
runner. the most common form is secondary tuberculosis of the facial jaw socket.

The main Oral mucosa primary primary damage element is a lupoma(til runner. the most common form is secondary tuberculosis of the facial jaw socket. The main Oral mucosa primary primary damage element is a lupoma(tuberculum) of red or yellow — red color with a soft consistency, 1-3 mm in diameter. They settle on the basis of groups: new ones are formed along the periphery, and in the center, the cottage cheese tends to crumble and merge with neighboring dzmbochala. At the same time, soft, uneven, cut edges, covered with a bright red or yellow-red color, papillomatous raspberry-like wounds form growths covered with yellowish caraches, bleeding easily.

Clinically, it passes through infiltrative, tuberculous, ulcerative, and scarring stages. linically, it passes through infiltrative, tuberculous, ulcerative, and scarring stages. Tuberculosis runner lesions are mainly on the skin of the face (in the form of a "butterfly"), the upper lip, less often — on the gums and alveolar tumor of the upper jaw in the frontal area, the upper lip and lunges, the hard and soft palate are located, the edges linically, it passes through infiltrative, tuberculous, ulcerative, and scarring stages. Tuberculosis runner lesions are mainly on the skin of the face (in the form of a "butterfly"), the upper lip, less often — on the gums and alveolar tumor of the upper jaw in the frontal area, the upper lip and lunges, the hard and soft palate are located, the edges are with an irregular wound cut, and the lower part is filled with bleeding granulations. Sometimes the process is observed only on the red border of the lips. At the same time, the lips become swollen and swollen, the volume becomes dense, cracks appear on them, covered with bloody-purulent crusts.t the same time, the lips become swollen and swollen, the volume becomes dense, cracks appear on them, covered with bloody-purulent crusts.

The diagnosis of clinical diagnosis is provided by the presence of the disease "apple jelly" symptoms (with diascopia) and is carried out on the basis of the (Pospelova)fracture.

Histologically, a lump of connective tissue is detected, a tubercle consisting of epitheloid cells surrounded by a plasma, lymphocyte axis, consisting of large Pyrogov—Langhans cells is detected.istologically, a lump of connective tissue is

detected, a tubercle consisting of epithelioid cells surrounded by a plasma, lymphocyte axis, consisting of large Pyrogov—Langhans cells is detected. Caseose necrosis is not observed or poorly expressed. Elastic and collagen fibers in a special infiltration zone are damaged. There are many dilated blood vessels in and around the Infiltrate, including those newly formed.

Comparative diagnosis. Sil jogurugi is different from drumsticks found in ternary Venom. The Sycamore shells are larger than the silt rush, they are denser. Comparative diagnosis. Sil jogurugi is different from drumsticks found in ternary Venom. The Sycamore shells are larger than the silt rush, they are denser. The edges of the wounds in the poison are comparative diagnosis. Sil jogurugi is different from drumsticks found in ternary Venom. The Sycamore shells are larger than the silt rush, they are denser. The edges of the wounds in the poison are smooth, dense, and in the case of tuberculosis, they have a soft, indentation. Unlike tuberculosis, syphilitic rashes do not occur again and again in scars. There is no probing or apple jelly symptom in the wound.

Miliary tuberculosis is a form of tuberculosis that affects the respiratory system and is found in patients with advanced bacillary tuberculosis. Miliary tuberculosis is a form of tuberculosis that affects the respiratory system and is found in patients with advanced bacillary tuberculosis. On the mucous membrane of the oral cavity (as well as around the skin, buttocks, anus) tuberculosis is a form of tuberculosis that affects the respiratory system and is found in patients with advanced bacillary tuberculosis. On the mucous membrane of the oral cavity (as well as around the skin, buttocks, anus of the lower extremities), small, ulcerative, very painful nodules appear, which combine to form large ulcerative foci up to 2.5 cm in diameter. The wounds are shallow, irregular in shape, with deep, jagged, soft edges. A slow inflammatory focus is observed around them. The lower part of the wound is uneven, usually bulging, gray-yellow-red, covered with papillary growths, sometimes with spot bleeding and small purulent plaque. The lower part of the wound is uneven, usually bulging, gray-yellow-red, covered with papillary growths, sometimes with spot bleeding and small purulent plaque. The lower part of the wound is uneven, usually bulging,

gray-yellow-red, covered with papillary growths, sometimes with spot bleeding and small pur.

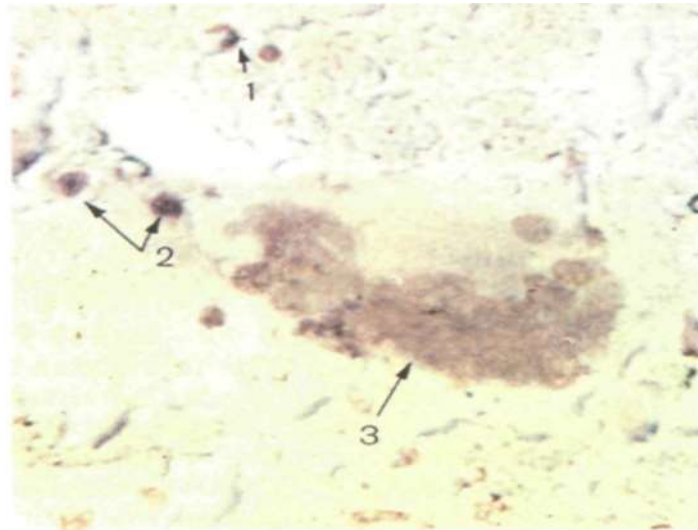


Figure 53. Scraping from a tuberculous wound
 — neutrophil leukocytes in the stage of deep Decay; 2—lymphocytes; 3-giant
Pyrogov-Langhans cell. figure 53.

Comparative diagnosis. Unlike Miliar ulcerative tuberculosis, painless, it is performed with a dental floss wound with a craterlike shape with a dense roll-shaped edge. Comparative diagnosis. Unlike Miliar ulcerative tuberculosis, painless, it is performed with a dental floss wound with a craterlike shape with a dense roll-shaped edge. Tuberculosis wounds should also be distinguished from ulcerative necrotic Vensan stomatitis, traumatic, trophic and cancer wounds. The results of Cytological and bacterioscopic examination are decisive in the diagnosis.

Treatment. Treatment of tuberculosis of the oral mucosa is carried out according to generally accepted methods in tuberculosis dispensaries. The prognosis for most patients is currently good. Treatment of tuberculosis of the oral mucosa is carried out according to generally accepted methods in tuberculosis dispensaries. The prognosis for most patient treatment. Treatment of tuberculosis of the oral mucosa is carried out according to generally accepted methods in tuberculosis dispensaries. The prognosis for most patients is currently good. Dentists play an important role in the early detection of patients with tuberculosis of the oral mucosa and in referring

them to tuberculosis dispensaries for treatment. This is because silicosis and miliary tuberculosis can only be observed in the oral cavity for a long time, so patients usually first consult a dentist.

Scrofuloderma. Scrofuloderma of the oral mucosa is very rare, mainly observed in children. Scrofuloderma of the oral mucosa is very rare, mainly observed in children. This is manifested by the formation of dense nodes the size of nuts or more, which protrude from the subcutaneous tissue. Scrofuloderma of the oral mucosa is very rare, mainly observed in children. This is manifested by the formation of dense nodes the size of nuts or more, which protrude from the subcutaneous tissue. Gradually they soften, the skin and mucous membrane above them acquire a purple color. Soon the nodes open, forming a series of flowing paths and wounds. The edges of the wounds are cut, sluggish granulations appear on their lower part, which, in turn, form jagged scars.

Comparative diagnosis. Cancer is done with ulcers, tertiary syphilis, actinomycoses.

Treatment:

* general

- dispatch to silga karshi dispensary

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- activazide adults 1.0-1.5 gr for 6-7 months

- children 0.03-0.04 gr per 1 kg of body weight every day for 6-7 months

- Vitamin D2 100 thousand Me for 6-7 months a day

- Streptomycin 0.5-1 gr per day for 6-7 months

* Vitamins C, V

* common ultra violet light

- Unsalted parkhez

Maxillary treatment:

* antiseptic treatment of wounds with 0.02% chlorhexidine solution

- Phtivazide, streptomycin appliques

* Proteolytic enzymes

- ogiz gap dating (during remission)

Candidiasis

Candidiasis is a disease caused by fungi that belong to the genus *Candida* - often caused by *Candida albicans*, sometimes by *Candida tropicalis* or other species. Candidiasis is a disease caused by fungi that belong to the genus *Candida* - often caused by *Candida albicans*, sometimes by *Candida tropicalis* or other species. Different types of yeast-like fungi Candidiasis is a disease caused by fungi that belong to the genus *Candida* - often caused by *Candida albicans*, sometimes by *Candida tropicalis* or other species. Different types of yeast-like fungi live on the mucous membrane of the mouth. Pathological changes are often caused by *Candida albicans*, *Candida tropicalis*, *Candida krusei*, *Candida guilliermondi*. The location of candidiasis in the oral cavity is diverse: different areas of the mucous membrane, carious cavities, root canals. *Candida albicans* is a representative of the resident microflora of the human oral cavity, in small quantities and inactive in 50-70% of rare individuals. It is also present on the surface of healthy skin. *Candida albicans* is a representative of the resident microflora of the human oral cavity, in small quantities and inactive in 50-70% of rare individuals. It is also present on the surface of healthy skin. The fungus favors an "acidic" environment (pH 5.8—6.5) and produces many enzymes that break down proteins, lipids and carbohydrates.

Etiology and pathogenesis. The manifestation of pathogenic properties of fungi of the genus *Candida* largely depends on the state of macroorganism. Etiology and pathogenesis. The manifestation of pathogenic properties of fungi of the genus *Candida* largely depends on the state of macroorganism. The results of recent studies

have shown that the main role in the development of candidiasis is played by a weakening of the immune system. Candidiasis usually occurs against the background of more or less pronounced immunodeficiency or an imbalance of the immune system. Severe diseases can contribute to the development of candidiasis: malignant tumors, HIV infection, tuberculosis, diseases of the endocrine system (diabetes mellitus, hypothyroidism, hypoparathyroidism, hypo - and hyper-function of the adrenal glands). Every disease can contribute to the development of candidiasis: malignant tumors, HIV infection, tuberculosis, diseases of the endocrine system (diabetes mellitus, hypothyroidism, hypoparathyroidism, hypo - and hyper-function of the adrenal glands). Diseases of the gastrointestinal tract, especially gastric juice and low acidity of stomach, often cause the development of candidiasis of the oral mucosa. In diabetes, impaired carbohydrate metabolism is a favorable background for the development of candidiasis. Therefore, the candidiasis of Oral mucosa often becomes the first clinical sign in all cases of chronic candidiasis, especially in relapses it is necessary to determine whether there is glucose in the blood, in order to determine whether there is diabetes mellitus or not. Therefore, the candidiasis of Oral mucosa often becomes the first clinical sign in all cases of chronic candidiasis, especially in relapses it is necessary to determine whether there is glucose in the blood, in order to determine whether there is diabetes mellitus or not. The development of candidiasis in the oral cavity is facilitated by long-term treatment with corticosteroid drugs, which reduces the body's immune system and increases the virulence of yeast-like fungi.

Due to the widespread use of antibiotics, the number of candidiasis in the oral cavity in recent years has increased significantly. Due to the widespread use of antibiotics, the number of candidiasis in the oral cavity in recent years has increased significantly. Due to the widespread use of antibiotics, the number of candidiasis in the oral cavity in recent years has increased significantly. Due to the widespread use of antibiotics, the number of candidiasis in the oral cavity in recent years has increased significantly.

significantly. With long-term use of antibiotics, the content of the resident microflora of the oral cavity is disrupted-dysbiosis is formed. The virulence of opportunistic *Candida* fungi that cause oral mucosal disease increases dramatically (superinfection, autoinfection). A similar effect is achieved through the long-term use of various antimicrobial drugs (trichopoly, chlorhexidine, sanguiridin, etc. Along with dysbiosis in the oral cavity, antibiotics also cause dysbiosis in the intestine, which leads to hypo - and vitamin deficiency of the yasa B, C, PP groups, which in turn negatively affects the functional state of the oral mucosa. long with dysbiosis in the oral cavity, antibiotics also cause dysbiosis in the intestine, which leads to hypo - and long with dysbiosis in the oral cavity, antibiotics also cause dysbiosis in the intestine, which leads to hypo - and vitamin deficiency of the yasa B, C, PP groups, which in turn negatively affects the functional state of the oral mucosa. In the development of candidiasis, great importance is attached to the condition of the oral mucosa and its immunity. The appearance of candidiasis is caused by acute and chronic injuries in the oral cavity (sharp edges of teeth, poor dentures, etc.). The allergenic effect of acrylic plastic dentures is observed during prolonged contact with the oral cavity. he allergenic effect of acrylic plastic dentures is observed during prolonged contact with the oral cavity. Chronic trauma allergenic effect of acrylic plastic dentures is observed during prolonged contact with the oral cavity. Chronic trauma reduces the resistance of the oral cavity, allowing *Candida* fungi to easily infiltrate and cause disease. In addition, *Candida* fungus grows well on the surface of dentures made of acrylic plastics, supporting chronic inflammation of the mucous membrane under the denture. Oral mucosal candidiasis often affects infants and the elderly, especially those weakened by chronic, severe diseases.

N. D. According to Sheklakov's classification, the following are available:

Mucous membranes, superficial candidiasis of the skin and nails, chronic generalivovannian (granulomatous) candidiasis of children, visceral (systemic) candidiasis. ucous membranes, superficial candidiasis of the skin and nails, chronic generalivovannian (granulomatous) candidiasis of children, ucous membranes, superficial candidiasis of the skin and nails, chronic generalivovannian

(granulomatous) candidiasis of children, visceral (systemic) candidiasis. More superficial forms of candidiasis occur. Dentists treat patients with candidiasis of the oral mucosa belonging to this group. Candidiasis of Oral mucosa can occur separately or with damage to other mucous membranes and skin. Sometimes, negative factors, first of all, in the presence of immunodeficiency, as well as timely and insufficient treatment, can be generalized by mucosal candidiasis with damage to internal organs, the consequences of which are very serious.

Clinical picture clinical picture. The clinical manifestations of candidiasis of the oral mucosa are diverse and depend on age, the state of the immune system, the presence of concomitant diseases, taking medications (antibiotics, corticosteroids) clinical picture. The clinical manifestations of candidiasis of the oral mucosa are diverse and depend on age, clinical picture. The clinical manifestations of candidiasis of the oral mucosa are diverse and depend on age, clinical picture. The clinical manifestations of candidiasis of the oral mucosa are diverse and depend on age, the state of the immune system, the presence of concomitant diseases, taking medications (antibiotics, corticosteroids) and other factors. According to the clinical course, there are acute and chronic forms of candidiasis of the oral mucosa. Acute candidiasis can occur in the form of acute pseudomembranous candidiasis and acute atrophic candidiasis. Chronic candidiasis also exists in two clinical forms: chronic hyperplastic and chronic atrophic.

Acute pseudomembranous candidiasis. One of the most common forms of candidiasis of Oral mucosa. In infants, lilies are more common and are relatively mild. Acute pseudomembranous candidiasis. One of the most common forms of candidiasis of Oral mucosa. In infants, lilies are more common and are relatively mild. Acute pseudomembranous candidiasis in adults is often accompanied by any common somatic serious diseases: diabetes mellitus, blood diseases, hypovitaminosis, poorly compressive tumors, etc.

Most often, the back mucous membrane of the tongue, lunge, palate and lips is affected. The mucous membrane of the mouth is clearly hypermured and dry. Most often, the back mucous membrane of the tongue, lunge, palate and lips is affected.

The mucous membrane often, the back mucous membrane of the tongue, lunge, palate and lips is affected. The mucous membrane of the mouth is clearly hypermured and dry. Against the background of hyperemia, there is aost often, the back mucous membrane of the tongue, lunge, palate and lips is affected. The mucous membrane of the mouth is clearly hypermured and dry.

Acute pseudomembranous glossitis should be distinguished from descvumative glossitis, in which the epithelial areas of descvumation occur at the back of the tongue, which are surrounded by the edge of the epithelium, which constantly migrates along the back of the tongue. cute pseudomembranous glossitis should be distinguished from descvumative glossitis, in which the epithelial areas of descvumation occur at the back of the tongue, which are surrounded by the edge of the epithelium, which constantly migrates along the back of the tongue. Acute candidiasis stomatitis should be differentiated between leukoplakia and kyat, in which, due to hyperkeratosis, whitening films and nodules form on the surface of the mucous membrane, and therefore cannot be removed. Candidiasis should also be distinguished from mild leukoplakia or white sponge nevus, in which the location of the jaroxates is mainly along the line of closure of the teeth and on the mucous membrane of the lips. andidiasis should also be distinguished from mild leukoplakia or white sponge nevus, in which the location of the jaroxates is mainly along the line of closure of the teeth andidiasis should also be distinguished from mild leukoplakia or white sponge nevus, in which the location of the jaroxates is mainly along the line of closure of the teeth and on the mucous membrane of the lips. With mild leukoplakia on the affected area, the color of the mucous membrane is white-gray, its surface is rough, uneven, with several small superficial erosions (friction). The final diagnosis is made on the basis of bacteriological examination data.

Acute atrophic candidiasis (candidosis acuta atrophica) is characterized by significant pain, Burns and dryness in the oral cavity. The mucous membrane is fiery red, dry. cute atrophic candidiasis (candidosis acuta atrophica) is characterized by significant pain, Burns and dryness in the oral cavity. The mucous membrane is fiery red, dry. When located on the tongue, its back becomes Crimson, dry, shiny, and the

filamentous suckers atrophy. Caraches will not no or will be preserved in deep folds, the exfoliated epithelial conglomerate is a large number of fungi of the genus Candida (mycelia, pseudomycelia) with difficulty removed acute atrophic candidiasis should be distinguished from an allergic reaction to the plastic of removable dentures. Caraches will not no or will be preserved in deep folds, the exfoliated epithelial conglomerate is a large number of fungi of the genus Candida (mycelia, pseudomycelia) with difficulty removed acute atrophic candidiasis should be distinguished from an allergic reaction to the plastic of removable dentures. In this case, clinical observation of the dynamics of changes in Oral mucosa after removal of the prosthesis and bacteriological examination plays an important role. The general condition of patients with acute candidiasis is satisfactory: body temperature does not change, lymph nodes are not palpable.

Chronic hyperplastic candidiasis (candidosis chronica atrophica) is characterized by the presence of a thick plate of dressing in the form of nodules or plaques that tightly adhere to an uneven surface on the hyperemic mucous membrane of the oral cavity. Chronic hyperplastic candidiasis (candidosis chronica atrophica) is characterized by the presence of a thick plate of dressing in the form of nodules or plaques that tightly adhere to an uneven surface on the hyperemic mucous membrane of the oral cavity. Carache is usually spread on the back of the tongue, on the palate. When placed on the tongue, the area characteristic of diamond-shaped glossitis is more affected. Chronic hyperplastic candidiasis suckers on the palate have the form of hyperplasia. In cases of prolonged persistent disease, carash is impregnated with fibrin, yellowish-gray films are formed and firmly settle on the lower mucous membrane. In cases of prolonged persistent disease, carash is impregnated with fibrin, yellowish-gray films are formed and firmly settle on the lower mucous membrane. In cases of prolonged persistent disease, carash is impregnated with fibrin, yellowish-gray films are

formed and firmly settle on the lower mucous membrane. When scratched with a spatula, karash is moved with difficulty, after which hyperemic bleeding is observed, the erosive surface is opened. Patients complain of pain — when there is dryness, burns and erosion in the oral cavity. This form of candidiasis should be distinguished from leukoplakia and QYaT.

Chronic atrophic candidiasis (*candidosis chronica atrophica*) is also manifested by dryness in the oral cavity, a burning sensation and pain when wearing a removable prosthesis. Chronic atrophic candidiasis (*candidosis chronica atrophica*) is also manifested by dryness in the oral cavity, a burning sensation and pain when wearing a removable prosthesis. The area of the mucous membrane corresponding to the boundaries of the prosthetic arch is hyperemic, swollen, painful in people who use removable dentures for a long time, chronic atrophic candidiasis is often characterized by damage to the oral mucosa. Under prostheses (hyperemia, yerosia, papillomatosis) mycotic (yeast) flexion and candidiasis in combination with atrophic glossitis, in which the posterior part of the tongue becomes red, dry, shiny, filamentous suckers atrophy. Under prostheses (hyperemia, yerosia, papillomatosis) mycotic (yeast) flexion and candidiasis in combination with atrophic glossitis, in which the posterior part of the tongue becomes red, dry, shiny, filamentous suckers atrophy. White-gray Carache is present in small quantities only in deep folds and lateral surfaces of the tongue. Karashis move with difficulty. Under the microscope, spores and mycelium of a fungus of the *Candida* type are found in it. This triad (inflammation of the palate, tongue and corners of the mouth) is so characteristic of atrophic candidiasis stomatitis that it is not difficult to diagnose.

A comparative diagnosis is made with QYaT; allergic stomatitis caused by the action of acrylic plastics, various forms of medicinal stomatitis, syphilis are dressed with papules. comparative diagnosis is made with QYaT; allergic stomatitis caused by the action of acrylic plastics, various forms of medicinal stomatitis, syphilis are dressed with papules. Mycotic (yeast) writhing is mainly observed in elderly people with a low bite height due to improper prosthetics, clear decay of hard tooth tissue or adentia presence of deep folds in the corners of the mouth and constant maturation

of these skin areas with saliva create favorable conditions for the formation of candida writhing. Patients complain of a burning sensation and pain in the corners of the mouth. Patients complain of a burning sensation and pain in the corners of the mouth. The disease is characterized by the appearance of gray, colorless gills, thin crusts or caraches in the corners of the mouth, which patients complain of a burning sensation and pain in the corners of the mouth. The disease is characterized by the appearance of gray, colorless gills, thin crusts or carates patients complain of a burning sensation and pain in the corners of the mouth. The disease is characterized by the appearance of gray, colorless gills, thin crusts or caraches in the corners of the mouth, which easily migrate, dry and slightly wet erosion or cracks appear after removal. Along with damage to the corners of the mouth, the process can go to the mucous membrane of the red hoshia of the lips, as a result of which candidiasis halite develops. It is characterized by the presence of hyperemia, swelling, Gray fluff and small transverse cracks. Pain occurs when the red border of the lips is exposed to the sun. Pain occurs when the red border of the lips is exposed to the sun. Mycotic writhing should be distinguished from streptococcal writhing, which is characterized by exudation, hyperemia spreading beyond the skin fold. Pain occurs when the red of the lips is exposed to the sun. Mycotic writhing should be distinguished from streptococcal writhing, which is characterized by exudation, hyperemia spreading beyond the skin fold. Erosion in the form of a crack is covered with a honey-yellow crust. A comparative diagnosis should also be carried out with a hard chancre, with a syphilitic papule. The final diagnosis is made on the basis of the results of a microscopic examination of scraping from the site of the injury, as well as the reaction of Wasserman. Candidiasis is also characterized by a deficiency of vitamin B2 and vitamin B2.

Diagnosis. The diagnosis of candidiasis is based on the typical complaints of patients, their clinical appearance and laboratory data (microscopic examination of scraping from the surface of the oral mucosa). Diagnosis. The diagnosis of candidiasis is based on the typical complaints of patients, their clinical appearance and laboratory data (microscopic examination of scraping from the surface of the oral

mucoagnosis. The diagnosis of candidiasis is based on the typical complaints of patients, their clinical appearance and laboratory data (microscopic examination of scraping from the surface of the oral mucosa). A clinical blood test and a study of serum glucose are also required. Skin and nails are examined. According to the instructions, consultations are made with a mycologist, endocrinologist, gynecologist. Candidiasis is diagnosed based on the identification of a fungus of the genus *Candida*, when it is scraped from the surface of the affected mucosa of the mouth. Microscopic examination of scraps from the Oral mucosa and from the surface of removable dentures is carried out. A sample from the material for the study should be done on an empty stomach or 4-5 hours after eating or rinsing before brushing your teeth and mouth.



Figure 54. Candidiasis. Acute from the mucous membrane of the mouth candidiasis. 1-spores; 2-pseudomycelia.F

Pseudomycelia filaments are formed by stretching cells and placing them in long chains, called pseudomycelia. Yeast-like mushrooms have almost no true mycelium. pseudomycelia filaments are formed by stretching cells and placing them in long chains, called pseudomycelia. pseudomycelia filaments are formed by stretching cells and placing them in long chains, called pseudomycelia. Yeast-like mushrooms have almost no true mycelium. The acute course of the disease is accompanied by a predominance of cellular forms, rounded,

partial budding. In chronic course, mainly pseudomycelia strands and chains of rounded elongated bud cells are identified. Microscopic examination of the material should be repeated after the end of the course of treatment and the disappearance of clinical signs of the disease. Identification of yeast-like fungi from the obtained materials is carried out based on the morphological characteristics of bacterial cells and the appearance of the grown colonies. Identification of yeast-like fungi from the obtained materials is carried out based on the morphological characteristics of bacterial cells and the appearance of the grown colonies. Solid and liquid nutrients with carbohydrates are used to obtain Candida fungi. In some cases, serological studies are carried out to make a diagnosis.

Treatment. Treatment of patients with candidiasis includes exposure to the pathogen and treatment of concomitant diseases, increasing specific and non-specific protective factors, sanitation of the oral cavity, rational treatment. Treatment of patients with candidiasis includes exposure to the pathogen and treatment of concomitant diseases, increasing specific and non-specific protective factors, sanitation of the oral cavity, rational nutrition. Patients with persistent chronic forms of candidiasis should be examined. Treatment of general and visceral forms of candidiasis is carried out by mycologists. An important condition for the successful treatment of candidiasis is a complete examination of the patient and the treatment of related diseases, especially gastrointestinal pathology, diabetes mellitus, leukemia.

General treatment **General treatment.** To affect the causative agent of the disease, it is necessary to prescribe anti-fungal drugs such as Nystatin or levorin L 6-8 times a day after meals, the daily dose should be at least 3,000,000 units. To affect the causative agent of the disease, it is necessary to prescribe anti-fungal drugs such as Nystatin or levorin L 6-8 times a day after meals, the daily dose should be at least 3,000,000 units. The drugs are taken for 10 days. It is recommended to crush the tablets and place them under the tongue and suck them, as they are poorly absorbed in the

gastrointestinal tract. Levorin is best prescribed in the form of transbuccal (cheek) tablets. Each tablet contains 500,000 units of levorin. From the absorption of decamine, which is prescribed in the form of caramel, a good zambrugal effect is obtained: 1-2 caramel 6-8 times a day (every 3-4 hours).rom the absorption of decamine, which is prescribed in the form of caramel, a good zambrugal effect is obtained: 1-2 caramel 6-8 times a day (every 3-4 hours). One or two capsules are placed under the tongue or behind the lunge and held the absorption of decamine, which is prescribed in the form of caramel, a good zambrugal effect is obtained: 1-2 caramel 6-8 times a day (every 3-4 hours). One or two capsules are placed under the tongue or behind the lunge and held until complete resorption without making swallowing movements as much as possible so that the drug comes into contact with the oral mucosa as long as possible. One caramel contains 0.00015 g of decamine. Amphoglucamine is administered orally 2 times a day 200,000-500,000 units after meals. Amphotericin B is used to treat chronic persistent forms of candidiasis of the Oral mucosa, which is prescribed at a dose of 1 unit per 250 kg of body weight and in the form of an ointment.in B is used to treat chronic persistent forms of candidiasis of the Oral mucosa, which is prescribed at a dose of 1 unit per 250 kg of body wmphtericin B is used to treat chronic pert forms of candidiasis of the Oral mucosa, which is prescribed at a dose of 1 unit per 250 kg of body weight and in the form of an ointment. Duflikan has a more pronounced antifungal effect. It is prescribed in capsules of 50-100 mg (depending on the severity of the disease) once a day. To reduce dry mouth and the effect on the fungal flora, a 2-3% solution of potassium iodide, 1 tablespoon, is prescribed 2-3 times a day after meals.

In addition, iodine exhibits fungistatic properties during its release through the skin, oral mucosa and mucous glands. In addition, iodine stimulates the saliva well.n addition, iodine exhibits fungistatic properties during its release through the skin, oral mucosa and mucous glands. In addition, iodine stimulates the saliva well. Patients with candidiasis need high-quality nutrition with a decrease in the amount of easily digestible carbohydrates. Inside, vitamins of Group V (V " V2, V6), RR, S are prescribed.

- **Local treatment.** Includes the use of anti-zambrug drugs. Local treatment. Includes the use of anti-zambrug drugs. For the application and lubrication of Oral mucosa, a solution of 0.5% decamine ointment, amphotericin V ointment (30,000 U Local treatment. Includes the use of anti-zambrug drugs. For the application and lubrication of Oral mucosa, a solution of 0.5% decamine ointment, amphotericin V ointment (30,000 U / g), 1% ointment and 1% c Local treatment. Includes the use of anti-zambrug drugs. For the application and lubrication of Oral mucosa, a solution of 0.5% decamine ointment, amphotericin V ointment (30,000 U / g), 1% ointment and 1% clotrimazole (kanesten) is used. To treat the mucous membrane of the mouth and the red border of the lips, solutions of aniline dyes are used, first of all purple (1-2% solution of Gentian Violet, 2% methylene blue, fucorcin solution). An effective means of alkalizing the environment in the oral cavity, which has a detrimental effect on the fungal flora. For this purpose, rinsing is used with a solution of 2-5% borax (sodium tetroborate), a solution of 2% sodium bicarbonate, a solution of 2% boric acid. or this purpose, rinsing is used with a solution of 2-5% borax (sodium tetroborate), a solution of 2% or this purpose, rinsing is used with a solution of 2-5% borax (sodium tetroborate), a solution of 2% sodium bicarbonate, a solution of 2% boric acid. At the same time, at least 1 glass is washed off at a time, repeating the procedure 5-6 times a day. For application and lubrication, 20% borax solution in glycerin, Lugol solution in glycerin, etc. used. With yeast infection and cheilitis, Nystatin ointment (1 pc per 100,000 g of base), 5% levorin ointment, 0.5% decamine ointment have a good effect. For topical treatment, it is better to prescribe several different medications and change them throughout the day or every day. For topical treatment, it is better to prescribe several different medications aor topical treatment, it is better to prescribe several different medicatioor topical treatment, it is better to prescribe several different medications and change them throughout the day or every day.

Careful oral sanitation is important in treatment, excluding any mucosal damage. Oral sanitation can begin 2-3 days after the start of antifungal treatment. In acute

and chronic candidiasis, prostheses must be carefully treated with the same means (except for dyes) that are used to treat Oral mucosa.

Prevention. It consists primarily in the correct and regular care of the oral cavity and dentures. It consists primarily in the correct and regular care of the oral cavity and dentures. With long-term treatment with antimicrobial agents, antibiotics, corticosteroids, Nystatin or levorin per 1,500,000 units per day, prevention. It consists primarily in the correct and regular care of the oral cavity and dentures.

Allergic Diseases

Currently, allergic diseases are widespread, and their number and, crucially, the severity of their course are constantly increasing, making this a social problem. The reasons for such widespread prevalence of allergic diseases are diverse. First of all, the contamination of the environment with industrial waste, exhaust gases, the use of pesticides, herbicides, etc., in agriculture plays a significant role. The rapid development of the chemical industry and the appearance of various synthetic materials, dyes, washing powders, cosmetics, and other substances in daily life and production also contribute to the spread of allergic diseases.

The widespread and often uncontrolled use of medicinal products also leads to an increase in the number of allergic reactions. Hypersensitivity to medicinal substances often arises due to the irrational simultaneous use of several drugs (polypragmasy). At the same time, common causes of allergic reactions include irrational and uncontrolled self-medication, especially with antibiotics, lack of sufficient knowledge among doctors regarding the pharmacokinetics of the prescribed drug, etc.

The influence of climatic factors (insolation, humidity), heredity, general somatic pathology, and the nature of nutrition also play a role in the occurrence of allergic diseases.

Allergy is the increased and altered sensitivity of the body to drugs compared to normal individuals. The state of the nervous system, as well as the endocrine system

and pathology of the gastrointestinal tract, are considered important in the development of allergy.

Allergy can be caused by various substances—from simple chemical compounds (iodine, bromine) to the most complex (proteins, polysaccharides, and their compounds)—which, when introduced into the body, induce an immune reaction of the humoral or cellular type. Substances that cause an allergic reaction are called allergens. The number of allergens in nature is vast, and they are diverse in composition and properties.

Some of them enter the body from outside, called exoallergens, while others are formed in the body and represent the body's own, but altered proteins (endoallergens or autoallergens). Exoallergens can be of non-infectious (pollen, house dust, animal hair, drugs, food products, detergent powders, etc.) and infectious (bacteria, viruses, fungi, and their waste products) origin. Exoallergens enter the body through various routes: the respiratory tract, the digestive tract, the skin, and mucous membranes, damaging various organs and systems.

Endoallergens are formed in the body from its own proteins under the influence of various damaging factors, which can be bacterial antigens and their toxins, viruses, thermal effects (burns, cooling), ionizing radiation, etc.

Allergens can be complete antigens or incomplete haptens. Haptens can combine with the body's macromolecules, which induce antigen production and lead to an allergic reaction. The specificity of the immunological reaction is directed against the hapten, not against its carrier; the formation of antigenic complexes with body molecules, where antigens are only in the complexes, not in the components.

Due to the abundance of allergens found in nature and formed in the body, the manifestations of allergic reactions are also diverse. However, allergic reactions, despite their different clinical pictures, have common pathogenetic mechanisms.

Stages of Allergic Reactions

There are three stages of allergic reactions:

1. Immunological
2. Pathochemical (Biochemical)

3. Pathophysiological (or the stage of functional and structural disturbances)

The Immunological Stage begins with the contact of the allergen with the body, resulting in the sensitization of the latter, i.e., the formation of antibodies or sensitized lymphocytes capable of interacting with this allergen. If the allergen is removed from the body before antibodies are formed, painful phenomena do not occur. The first introduction of the allergen into the body has a sensitizing effect. Upon repeated exposure to the allergen in an already sensitized body, an allergen-antibody complex or an allergen-sensitized lymphocyte is formed.

From this moment, the Pathochemical Stage of the allergic reaction begins, which is characterized by the release of biologically active substances (mediators of allergy): histamine, serotonin, bradykinin, etc.

The Pathophysiological Stage of the allergic reaction, or the stage of clinical manifestation of the damage, is the result of the action of the isolated biologically active substances on tissues, organs, and the body as a whole. This stage is characterized by disturbances in blood circulation, spasm of the smooth muscles of the bronchi and intestines, changes in the composition of blood serum, disturbances in its coagulation, cytolysis of cells, etc.

Types of Allergic Reactions (Gell and Coombs Classification)

According to the mechanism of development, there are four types of allergic reactions:

1. Immediate-type reaction (Reagin type)
2. Cytotoxic type
3. Tissue damage by immune complexes (Arthus type)
4. Delayed-type reaction (Cellular hypersensitivity)

Each of these types has a specific immune mechanism and its own set of specific mediators that determine the specific features of the clinical picture of the disease.

1. Type 1 Allergic Reaction (Immediate/Anaphylactic/Atopic)

Also called the reagin, anaphylactic, or atopic reaction type. It develops primarily with the formation of antibodies called reagins, belonging to the IgE and IgG4 classes. Reagins attach to mast cells and basophil leukocytes. When reagins combine

with the corresponding allergen, mediators are released from these cells: histamine, heparin, serotonin, platelet-activating factor (PAF), prostaglandins, leukotrienes, etc., which determine the clinical picture of the immediate allergic reaction. The clinical manifestations of the reaction appear 15-20 minutes after contact with the specific allergen; hence the name "immediate-type reaction."

2. Type 2 Allergic Reaction (Cytotoxic Type)

This type is characterized by the formation of antibodies against tissue cells and is mainly represented by IgG and IgM. This type of reaction is caused only by antibodies capable of activating the complement system. The antibodies combine with the body's altered cells, leading to the activation of complement, which causes damage and even destruction of the cells. As a result of the cytotoxic type of allergic reaction, cell destruction, followed by phagocytosis and the removal of destroyed cells and tissues, occurs. Drug allergies characterized by leukopenia, thrombocytopenia, and hemolytic anemia belong to the cytotoxic type of reactions.

3. Type 3 Allergic Reaction (Immune Complex/Arthus Type)

This type of reaction arises as a result of the formation of circulating immune complexes containing IgG and IgM antibodies. Antibodies of this class are called precipitins because they form a precipitate when combined with the corresponding antigen. Allergens in this type of reaction can be bacterial or food-related.

This type of reaction is leading in serum sickness, allergic alveolitis, some cases of drug and food allergies, and a number of auto-allergic diseases (systemic lupus erythematosus, rheumatoid arthritis, etc.).

4. Type 4 Allergic Reaction (Delayed-Type Hypersensitivity - DTH/Cellular)

In this type of reaction, the role of antibodies is performed by sensitized T-lymphocytes that have receptors on their membranes capable of specifically interacting with the antigen. When such a lymphocyte combines with an allergen that may be dissolved or located in cells, mediators of cellular immunity—lymphokines—are released. There are more than 30 lymphokines that exert their effect in various combinations and concentrations depending on the properties of the allergen, the genotype of the lymphocyte, and other conditions. Lymphokines cause

the accumulation of macrophages and other lymphocytes, resulting in inflammation. One of the main tasks of the mediators is to involve them in the process of destroying the antigen (microorganisms or foreign cells) to which the lymphocytes are sensitive. If a foreign tissue graft acts as antigenic material stimulating delayed-type hypersensitivity, it is destroyed and rejected. The delayed reaction usually develops 24-48 hours after contact with the allergen in a sensitized body.

The cellular type of reaction underlies the immunity to viral and some bacterial infections (tuberculosis, syphilis, leprosy, brucellosis, tularemia), certain forms of infectious-allergic bronchial asthma, rhinitis, and the development of transplantation and anti-tumor immunity.

The type of development of the allergic reaction is determined by the nature and properties of the antigens, as well as the state of the body's reactivity. Specific
Diagnosis of Allergic Diseases

Specific diagnosis of allergic diseases consists of taking an allergological history, performing diagnostic tests, and conducting laboratory tests.

Allergological History

When collecting an allergological history, attention should be paid to identifying the entire complex of domestic and industrial contacts with various substances that may act as an allergen. History taking allows us to determine the presence of an allergic predisposition (hereditary or acquired), as well as possible exo- and endogenous factors (climatic, endocrine, mental, etc.) affecting the course of the disease. It is necessary to determine how the patient reacted to vaccines, serums, and medications, as well as to ascertain their living and working conditions. Establishing professional contacts with various substances is very important. It is known that contact with simple chemical substances often causes delayed-type allergic reactions (contact dermatitis). Complex organic substances can cause immediate allergic reactions with the development of diseases such as Quincke's edema, urticaria, allergic rhinitis, bronchial asthma, and others.

A carefully collected history indicates the possible type of allergic reaction and the likely allergen. The specific allergen that caused the development of the disease is identified using special diagnostic tests and laboratory examinations.

Allergic Diagnostic Tests

Allergic diagnostic tests are a method of determining the specific sensitivity of the body. These tests are performed outside the exacerbation phase of the disease, in the period when the body's sensitivity to the allergen is reduced, typically 2-3 weeks after a severe acute allergic reaction.

There are skin tests and provocation tests.

Skin tests are based on introducing the allergen through the skin and evaluating the nature of the resulting inflammatory reaction to determine the specific sensitivity of the body. The following methods of conducting skin tests exist: application (patch), scarification (scratch), and intradermal (intracutaneous).

The choice of skin testing method is determined by the nature of the disease, the type of allergic reaction, and the group membership of the tested allergen. For example, application tests are most convenient for diagnosing drug allergy. Sensitivity to allergens of bacterial and fungal origin is determined by intradermal tests. Provocation tests are carried out in cases where the allergological data does not match the results of skin tests. Provocation tests are based on reproducing the allergic reaction by introducing the allergen into the organ or tissue where the damage is leading in the clinical picture of the disease. Nasal, conjunctival, and inhalation provocation tests are distinguished. Provocation tests also include cold and hot tests, which are used for cold and heat urticaria.

Laboratory Diagnostic Methods

Specific diagnosis of allergic reactions is also carried out using laboratory research methods, such as:

- Basophil Leukocyte Degranulation Reaction (Shelley Test)
- Leukocyte Blast Transformation Reaction
- Neutrophil Damage Reaction
- Leukocytolysis Reaction, and others.

The advantage of in vitro diagnostic methods for allergic reactions is the complete absence of the risk of anaphylactic shock.

Anaphylactic Shock

Anaphylactic shock does not have specific manifestations in the oral cavity but is the most serious prognostic allergic disease found in dental practice and often leads to death.

Etiology. In dental practice, many materials and drugs are used: anesthetics, analgesics, antiseptics, antibiotics, vitamins, acrylic plastics, amalgam, pastes for filling root canals (based on formalin, eugenol), etc. Most of these substances have distinct antigenic properties, especially acrylic plastics, mercury, and Novocaine (Procaine). Anaphylactic shock most often develops as a result of parenteral, especially intravenous, drug administration. However, there are known cases of anaphylactic shock with oral and local use of medicinal substances (application to the oral mucosa, periodontal dressing, injection of the preparation into the socket of an extracted tooth, etc.). Under certain conditions, related to the individual reactivity of the body, as well as the type and method of use of these substances, they can act as an allergen and cause an allergic reaction.

Pathogenesis. Anaphylactic shock is an immediate-type allergic reaction based on the formation of reagin antibodies. Their subsequent contact with the antigen (allergen) leads to the formation of biologically active substances (histamine, leukotrienes, prostaglandins, etc.), which form the clinical picture of anaphylactic shock caused by the following mechanisms:

Clinical picture. Clinical manifestations of anaphylactic shock are diverse and may have several clinical variants. Clinical picture. Clinical manifestations of anaphylactic shock are diverse and may have several clinical variants.

A hemodynamic variant dominated by symptoms of a clinical picture. Clinical manifestations of anaphylactic shock are diverse and may have several clinical variants.

A hemodynamic variant dominated by symptoms of acute cardiovascular failure: weak rapid pulse, skin hyperemia, pale sweating, profuse sweating, elevated blood pressure. The patient is pale and, in severe cases, loses consciousness.

Anaphylactic shock can occur with a predominance of central nervous system disorders. Patients become anxious, fearful, tremulous, symptoms of brain tumors, restless. Anaphylactic shock can occur with a predominance of central nervous system disorders. Patients become anxious, fearful, tremulous, symptoms of brain tumors, restless. In the clinical picture of anaphylactic shock, respiratory diseases (bronchospasm, laryngeal edema, lung symptoms) or the gastrointestinal tract (nausea, vomiting, diarrhea, pain in the stomach and intestines) may prevail.

From the moment the Antigen is administered to the appearance of clinical signs, the time for the development of anaphylactic shock ranges from a few minutes to half an hour. The shorter the latent period of anaphylactic shock, the more severe it is. From the moment the Antigen is administered to the appearance of clinical signs, the time for the development of anaphylactic shock ranges from a few minutes to half an hour. The shorter the latent period of anaphylactic shock, the more severe it is. And if the patient is not provided with timely assistance, a fatal outcome may occur. In patients with hypersensitivity, neither the dosage nor the method of using the drug have a decisive effect on the severity of anaphylactic shock. There are three severity levels of anaphylactic shock: mild, moderate, and severe.

Typical manifestations of anaphylactic shock observed in many patients are characterized by the following clinical signs: a state of discomfort, vague painful sensations of fear of death "patients complain of itching of the face, scalp and sudden weakness, headache, dizziness, a feeling of blood flow to the head, face, tongue, chest tightness. Typical manifestations of anaphylactic shock observed in many patients are characterized by the following clinical signs: a state of discomfort, vague painful sensations of fear of death "patients complain of itching of the face, scalp

and sudden weakness, headache, dizziness, a feeling of blood flow to the head, face, tongue, chest tightness. They complain of heart pain, difficulty breathing, and sometimes abdominal pain. In severe anaphylactic shock, the patient does not have time to complain and immediately loses consciousness.

The objective symptoms of anaphylactic shock are hyperemia of the skin of the face and body, associated with pallor and cyanosis, swelling of the eyelids, lip red hoshia and oral mucosa. The objective symptoms of anaphylactic shock are hyperemia of the skin of the face and body, associated with pallor and cyanosis, swelling of the eyelids, lip red hoshia and oral mucosa. A detailed clinical picture of a severe form of anaphylactic shock is characterized by serious disorders of vital organs and systems. Cardiovascular and hemodynamic disorders develop: strong sweating, weakening of cardiac activity are observed. Pulse, tachycardia, blood pressure drops rapidly, and in severe cases, diastolic pressure is not detected. Cardiovascular failure often leads to a fatal outcome. Cardiovascular failure often leads to a fatal outcome.

Usually, at the same time as the formation of cardiovascular failure, shortness of breath, wheezing with wheezing and froth appear from the mouth. Later, pulmonary edema develops, which indicates a severe course of anaphylactic shock. Cardiovascular failure often leads to a fatal outcome.

Usually, at the same time as the formation of cardiovascular failure, shortness of breath, wheezing with wheezing and froth appear from the mouth. Later, pulmonary edema develops, which indicates a severe course of anaphylactic shock. Gastrointestinal diseases are often observed. Spastic pain in the abdomen, vomiting and often diarrhea with a mixture of blood are observed. Gastrointestinal diseases are often observed. Spastic pain in the abdomen, vomiting and often diarrhea with a mixture of blood are observed. Spasms of smooth intestinal diseases are often observed. Spastic pain in the abdomen, vomiting and often diarrhea with a mixture of blood are observed. Spasms of smooth muscles and other organs occur. This is accompanied by neuropsychiatric disorders in the form of intense arousal, followed by complete apathy, headaches, vision,

rejuvenation and imbalance. A coma condition develops, sometimes urine emptying and diarrhea are observed. Causes of death are usually asphyxia due to vascular insufficiency or bronchospasm or laryngeal edema. An increase in body temperature is not characteristic; in most cases, it even decreases. An increase in body temperature is not characteristic; in most cases, it even decreases.

The outcome of anaphylactic shock depends not only on the severity of the clinical picture, but in many ways on the timely and full value of the treatment carried out. An increase in body temperature is not characteristic; in most cases, it even decreases.

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The outcome of anaphylactic shock depends not only on the severity of the clinical picture, but in many ways on the timely and full value of the treatment carried out. In the diagnosis of anaphylactic shock, the degree of vigilance of the doctor in this regard is of great importance. Usually the diagnosis is not a hassle, since it is very easy to establish a connection between the reaction of the body and exposure to the allergen.

Differential diagnosis. Anaphylactic shock is distinguished from the following acute heart failure, risk of myocardial infarction, epilepsy (sudorgi)

Treatment. The fight against anaphylactic shock should begin immediately after the appearance of the first signs of anaphylaxis, and first of all should be aimed at stopping the subsequent intake of the allergen into the body or reducing its absorption (if the drug has already been introduced). The fight against anaphylactic shock should begin immediately after the appearance of the first signs of anaphylaxis, and first of all should be aimed at stopping the subsequent intake of the allergen into the body or reducing its absorption (if the drug has already been introduced). To do this, adrenaline is applied above the injection site (if possible) or a 0.3—0.5 ml 0.1% epinephrine solution is made to the injection site. These measures help reduce the absorption of the allergen. The patient is given a horizontal position on the back with

his head slightly lowered, the lower jaw is pushed forward to prevent asphyxia due to tongue involvement or aspiration of vomiting, removable dentures are removed from the mouth. The patient is given a horizontal position on the back with his head slightly lowered, the lower jaw is pushed forward. The patient is given a horizontal position on the back with his head slightly lowered, the lower jaw is pushed forward to prevent asphyxia due to tongue involvement or aspiration of vomiting, removable dentures are removed from the mouth. They relieve the neck, chest, and abdomen from tightness. Oxygen therapy is delivered through a mask or nasal catheter. In the absence of spontaneous breathing, artificial ventilation of the lungs should first be started "from mouth to mouth" and then artificial ventilation using the apparatus. To increase blood pressure, sympathomimetics is used: 0.5 ml of 0.1% epinephrine solution or 0.3—1.0 ml of 1% mesaton solution or intravenous (drip) 2-4 ml of 0.2% norepinephrine solution is diluted in 1 liter of 5% glucose solution or isotonic sodium chloride solution is injected subcutaneously or intramuscularly. To increase blood pressure, sympathomimetics is used: 0.5 ml of 0.1% epinephrine solution or 0.3—1.0 ml of 1% mesaton solution or intravenous (drip) 2-4 ml of 0.2% norepinephrine solution is diluted in 1 liter of 5% glucose solution or isotonic sodium chloride solution is injected subcutaneously or intramuscularly. For faster absorption, it is better to divide the drugs into different parts of the body (for example, adrenaline is administered fractionally 0.5 ml every 15-30 minutes until the patient comes out of shock). In severe cases, intravenous administration of 0.1-0.2 ml of 0.1% epinephrine solution is indicated for 3-5 minutes. Long-term treatment is carried out if there is no effect. In severe cases, intravenous administration of 0.1-0.2 ml of 0.1% epinephrine solution is indicated for 3-5 minutes. Long-term treatment is carried out if there is no effect. Add 1 ml of 0.1% adrenaline solution to 250.0 ml of 5% glucose solution. Infusion begins at a rate of 50-60 drops per minute. Antihistamines are used after blood pressure has returned to normal. Intramuscular injections of 1% diphenhydramine solution, 2.5%

diprazine solution, 2% suprastin yeritma, 2 ml tavegil solution, or other antihistamines are administered. In moderate to severe forms of anaphylactic shock, water-soluble glucocorticosteroid drugs are used, which have a pronounced desensitizing and anti-inflammatory effect. In moderate to severe forms of anaphylactic shock, water-soluble glucocorticosteroid drugs are used, which have a pronounced desensitizing and anti-inflammatory effect. They are injected intravenously (reactive or drip) in a 5% glucose solution or isotonic sodium chloride solution. Most often, 50-150 mg hydrocortisone gemisuccinate is used, in severe cases the dose is increased to 300 mg, 60-120 mg prednisone gemisuccinate.

To relieve bronchospasm, a 2.4% solution of eufillin is used, diluted intravenously in 5-10 ml of 10 ml of isotonic sodium chloride solution or in 10 ml of 10% or 40% glucose solution.

In the presence of neuroleptics and tranquilizers (seduxen, relanium, yelenium, droperidol, etc.) in the exacerbation of the patient's sudorgias and excitability.) are used. In the presence of neuroleptics and tranquilizers (seduxen, relanium, yelenium, droperidol, etc.) in the exacerbation of the patient's sudorgias and excitability.) are used. If anaphylactic shock has developed from penicillin, 1,000,000 units of penicillinase should be injected intramuscularly once in 2 ml of isotonic sodium chloride solution. Due to the rapid development of anaphylactic shock, the time for emergency medical care is limited to minutes, so the doctor must have a set of tools prepared at his disposal to provide emergency care for such patients. Due to the rapid development of anaphylactic shock, the time for emergency medical care is limited to minutes, so the doctor must have a set of tools prepared at his disposal to provide emergency care for such patients. Due to the rapid development of anaphylactic shock, the time for emergency medical care is limited to minutes, so the doctor must have a set of tools prepared at his disposal to provide emergency care for such patients. This includes:

Sympathomimetics: ampoules with epinephrine solutions (0.1%),

norepinephrine (0.2%), mesaton (1%) < BR > ;

Antihistamines: ampoules with solutions of suprastin (2%), diphenhydramine (1%), tavegil (0.001 g 2 ml;

Corticosteroids: ampoules with prednisolone solutions

hyemisuccinate (25 mg in ampoules), prednisolone tablets

5 mg, hydrocortisone hemisuccinate (25 mg in ampoules), prednisolone tablets

5 mg, hydrocortisone hemisuccinate

yemisuccinate (25 mg in ampoules), prednisolone tablets

5 mg, hydrocortisone hemisuccinate

25 and 25 mg in ampoules), prednisolone tablets

5 mg, hydrocortisone hemisuccinate

25 and 100 mg yemisuccinate (25 mg in ampoules), prednisolone tablets

5 mg, hydrocortisone hemisuccinate

25 and 100 mg yemisuccinate (25 mg in ampoules), prednisolone tablets

5 mg, hydrocortisone hemisuccinate

25 and 100 mg ampoules, hydrocortisone Solu-Cortef

for intravenous injection in 300 mg vials;

bronchodilators: ampoules with yephedrine injections

hydrochloride (5%), eufillin (2.4% - 10.0 ml;

anticonvulsants: calcium pantothenate ampoules

(2 ml by 20%);

10% glucose injection in 5 ml ampoules,

0.9% Sodium Chloride peanuts in ampoules of 5-10 ml and

In 400 ml vials, hemodesis in 200 and

400 ml vials;

1 mln in ampoules. dona penicillinase;

Prevention of anaphylactic shock consists in careful collection of Anamnesis. Before the introduction of the drug, it is necessary to determine whether its use or drugs of the corresponding group are accompanied by any reactions. revention of

anaphylactic shock consists in careful collection of Anamnesis. Before the introduction of the drug, it is necessary to determine whether its.

Quincke's Edema

Angioneurotic Quincke's edema (oedema angioneuroticum Quincke) is a disease characterized by a limited deep swelling of the skin and subcutaneous tissue or mucous membranes that develops acutely, sometimes disappearing on its own and recurring frequently. It was first described by the German therapist Quinke (1862). The development of Quinke angioedema is based on a rapid type of allergic reaction. Under the influence of biologically active substances (histamine, serotonin, heparin, etc.), microvascular permeability increases and edema appears in tissues. Quincke's edema is often combined with krapivnitsa, since both of these diseases have a common pathogenesis.

The cause of quincke's edema can affect various food and drug allergens (sulfanilamides, antibiotics, acetylsalicylic acid, bromides, etc.) the cause of quincke's edema can affect various food and drug allergens (sulfanilamides, antibiotics, acetylsalicylic acid, bromides, etc.), is an increase in sensitivity to cosmetics, odors, and cold. A number of authors attach special importance to heredity in the pathogenesis of edema, the activity of the vegetative nervous system, foci of chronic infection, diseases of the gastrointestinal tract.

Clinical picture. The disease begins suddenly. In a few minutes, sometimes more slowly, a clearly limited swelling appears on different parts of the face, on the oral mucosa or other parts of the body. Clinical picture. The disease begins suddenly. In a few minutes, sometimes more slowly, a clearly limited swelling appears on different parts of the face, on the oral mucosa or other parts of the body. At the clinical picture. The disease begins suddenly. In a few minutes, sometimes more slowly, a clearly limited swelling appears on different parts of the face, on the oral mucosa or other parts of the body. At the same time, the color of the mucous membrane of the skin or mouth does not change. In the area of edema, tissue tension is noted, there is no

deep dressing under pressure on it, palpation is painless. Most often, Quincke's edema settles on the lower lip, eyelids, tongue, larynx. In the case of swelling of the tongue, it increases significantly and practically does not fit into the mouth. Advanced swelling of the tongue and larynx is considered the most dangerous, since it can lead to the rapid development of asphyxia. In these areas, the process is developing very quickly. The patient has difficulty breathing, develops aphonia, cyanosis of the tongue. Swelling of the tongue and larynx is considered



Figure 55. Quincke's Edema (Angioedema)

If Quincke's edema reaches the brain and meninges, corresponding neurological disorders appear (epileptiform seizures, aphasia, hemiplegia, etc.). Quincke's edema can last for several hours or days. It then disappears without a trace, but may periodically recur later. The edema is rarely accompanied by pain; patients often complain of a feeling of tension in the tissues.

Differential Diagnosis (Quincke's Edema)

Quincke's edema in the lip area must be differentiated from:

- Melkersson-Rosenthal Syndrome
- Erysipelas
- Lymphostasis and collateral edema in periostitis

In Melkersson-Rosenthal Syndrome, the lip edema is chronic and less clearly defined than Quincke's edema, and is simultaneously accompanied by fissured tongue and inflammation (neuritis) of the facial nerve.

In erysipeloid inflammation, unlike Quincke's edema, there is fiery redness (hyperemia) in the affected area.

Quincke's edema in the tongue area must be differentiated from various types of macroglossia. The diagnosis is primarily based on anamnesis data and the characteristic acute manifestation of Quincke's edema.

Treatment (Quincke's Edema)

The main goal in treating Quincke's edema is the immediate elimination of contact with the allergen (where possible).

- Antihistamines (Diphenhydramine, Suprastin, Tavegyl, Pipolfen, etc.) are used intramuscularly or, in mild cases, orally in tablets 2-3 times a day.
- Ascorutin is also prescribed, as it reduces vascular permeability.
- In case of laryngeal edema, 25 mg of Prednisolone hemisuccinate is additionally injected intramuscularly. If indicated, the patient is hospitalized under the supervision of an ENT specialist or surgeon due to the potential need for tracheotomy.
- In severe cases of Quincke's edema with a drop in blood pressure. Epinephrine solution is injected subcutaneously.

Prognosis: The prognosis is favorable when the edema is localized on external parts of the body, but serious when the edema involves the larynx and meninges.

Prevention (Quincke's Edema)

The prevention of recurrent Quincke's edema is achieved by avoiding contact with the allergen that caused it.

Drug Allergy (*Allergia Medicamentosa*)

The problem of complications of Pharmacotherapy is especially relevant at the moment. This is due, on the one hand, to a significant increase in the arsenal of synthesized drugs, which are allergens for the human body. The problem of complications of Pharmacotherapy is especially relevant at the moment. This is due, on the one hand, to a significant increase in the arsenal of synthesized drugs, which are allergens for the human body. On the other hand, exposure to negative

environmental factors leads to sensitivity. The result of these processes does not arise from the properties of drugs, but from the peculiarities of their interaction with the human body, the state of its reactivity, the presence of common somatic diseases.

Allergic reactions account for 6-25% of complications from drug treatment. Allergic reactions account for 6-25% of complications from drug treatment. Drug allergies (allergia medicamentosa) can be caused by any drug, but the most common cause of allergic reactions are antibiotics (penicillin and its derivatives, tetracycline, streptomycin), sulfanillergic reactions account for 6-25% of complications from drug treatment. Drug allergies (allergia medicamentosa) can be caused by any drug, but the most common cause of allergic reactions are antibiotics (penicillin and its derivatives, tetracycline, streptomycin), sulfanilamide drugs, analgesics, novocaine, iodine, bromides, tranquilizers, etc. The speed and development of an allergic reaction is often determined by the method of use of the drug. It is known that with the local use of medicinal substances (in the form of application to the mucous membrane of the mouth or skin), the highest sensitivity appears. It is known that with the local use of medicinal substances (in the form of application to the mucous membrane of the mouth or skin), the highest sensitivity appears. The frequency of allergic reactions with intramuscular administration of drugs is lower than that of administration. It is known that with the local use of medicinal substances (in the form of application to the mucous membrane of the mouth or skin), the highest sensitivity appears. The frequency of allergic reactions with intramuscular administration of drugs is lower than that of administration—that is, with regard to applicative anesthesia. In addition, sensitization has been proven to develop more often with a higher dose of the drug than with a lower dose. This is especially evident in the use of medicinal substances with mahaly, when the concentration of a drug substance is more important than its absolute amount.

The pathogenesis of drug allergies can include any type of allergic reaction, or often a combination of them, which can be associated with the body's individual reactivity, the presence of common somatic diseases, the nature of the allergen drug, the method of its use, etc., therefore, the division of allergic reactions in the clinic

into immediate and delayed types is partially conditional. The pathogenesis of drug allergies can include any type of allergic reaction, or often a combination of them, which can be associated with the body's individual reactivity, the presence of common somatic diseases, the nature of the allergen drug, the method of its use, etc., therefore, the division of allergic reactions in the clinic into immediate and delayed types is partially conditional. There are two types of hypersensitivity that arise from the action of several determinant groups of one or different medicinal products at the same time. The clinical manifestations of drug allergy are associated with the predominance of hypersensitivity of any type at the General stage of the disease or at a certain stage of it. The clinical manifestations of drug allergy are associated with the predominance of hypersensitivity of any type at the General stage of the disease or at a certain stage of it. For example, the clinical manifestations of drug allergy are associated with the predominance of hypersensitivity of any type at the General stage of the disease or at a certain stage of it. For example, drug allergies can manifest themselves in the form of anaphylactic shock, Quincke's edema, which are mainly rapid type reactions, where humoral antibodies play a key role. However, often allergic reactions with medicinal allergens occur in a delayed manner. Clinical manifestations of delayed drug allergy are very diverse, ranging from lesions located on the skin and oral mucosa to lesions of various organs and systems (gastrointestinal tract, respiratory organs, kidneys, etc.). Clinical manifestations of delayed drug allergy are very diverse, ranging from lesions located on the skin and oral mucosa to lesions of various organs and systems (gastrointestinal tract, respiratory organs, kidneys, etc.). Allergic drugs of the oral mucosa are common, since the oral cavity is the site of the patient's body's first contact with drugs. The clinical manifestations of drug allergies in the oral cavity are diverse. Depending on the location of pathological changes in Oral mucosa, medicinal stomatitis, halitosis or glossitis are distinguished. Catarrhal and catarrhal hemorrhagic stomatitis (halitosis, glossitis). Depending on the location of

pathological changes in Oral mucosa, medicinal stomatitis, halite or glossitis are distinguished.

Catarrhal and catarrhal hemorrhagic stomatitis (halite, glossitis). This is the mildest form of drug allergy. Patients usually complain of itching, burns, impaired taste sensitivity, dryness and pain when eating hot and spicy food.

Clinical picture. When examining the oral cavity, diffuse or limited hyperemia, swelling of the mucous membrane (traces of teeth on the lateral surfaces of the tongue and cheeks), violation of the integrity of the epithelium is not observed. Clinical picture. When examining the oral cavity, diffuse or limited hyperemia, swelling of the mucous membrane (traces of teeth on the lateral surfaces of the tongue and cheeks), violation of the integrity of the epithelium is not observed. Sometimes, with more pronounced changes, punctate or larger bleeding is noted, along with hyperemia. The characteristic appearance is represented by medicinal glossitis: the back of the tongue turns bright red, sometimes the phenomena of the desquamation of the epithelium and atrophy of the filamentous suckers (lacquered tongue) are expressed.

Comparative diagnosis. What specific clinical signs of catarrhal stomatitis are caused by the medicinal result are absent and hypovitaminosis C, Group B, mucosal-like changes in the mouth should be distinguished; diseases of the gastrointestinal tract; infectious; fungal lesions.

Treatment. It consists in canceling the medication or replacing it with others; antihistamines, calcium preparations are prescribed. Treatment. It consists in canceling the medication or replacing it with others; antihistamines, calcium preparations are prescribed. In the presence of hemorrhagic jaroxates, vitamins C and r do not require special local treatment of the oral mucosa, except for the use of pain relievers according to the instructions.

(applications, oral baths with 2% trimekin solution, 1-2% pyromecaine solution, anesthesia with glycerin, etc.). It is recommended to drink non-irritating food and plenty of fluids.

Erosive stomatitis (halite, glossitis). This is a more severe form of allergic reaction to drugs. It is accompanied by significant pain, which increases in nutrition and speech. Erosive stomatitis (halite, glossitis). This is a more severe form of allergic reaction to drugs. It is accompanied by significant pain, which increases in nutrition and speech. Against the background of the hyperemic and swollen mucous membrane of the mouth, blisters o Erosive stomatitis (halite, glossitis). This is a more severe form of allergic reaction to drugs. It is accompanied by significant pain, which increases in nutrition and speech. Against the background of the hyperemic and swollen mucous membrane of the mouth, blisters of various sizes appear on the palate, gums, lips lungs and tongue with a transparent composition, after rupturing the buttocks, erosion is formed and covered with fibrinous abrasions. Often a single erosion merges to form wide erosive surfaces. Milk suckers are hyperemic, swollen, and bleed easily. Hyposalivation, unpleasant sensations in the throat, and tickling may occur. posalivation, unpleasant sensations in the throat, and tickling may occur. The general condition of the patient can worsen: weakness, decreased appetite, an increase in body temperature yposalivation, unpleasant sensations in the throat, and tickling may occur. The general condition of the patient can worsen: weakness, decreased appetite, an increase in body temperature to 37.5—38 C. The lymph nodes under the jaw can become enlarged and painful. The severity of the course of erosive drug stomatitis depends on the spread of pathological changes in the oral mucosa; this can be exacerbated by an unsatisfactory state of oral hygiene, the presence of caries teeth, periodontic diseases. In addition, the presence of chronic foci of infection also aggravates the severity of erosive stomatitis during the action of the drug.

Comparative diagnosisn addition, the presence of chronic foci of infection also aggravates the severity of erosive stomatitis during the action of the drug.

Comparative diagnosis. Erosive drug stomatitis is differentiated: acute herpetic stomatitis; multiform exudative erythema; and pemphigus.

In its comparative diagnosis, it is important to determine whether changes in Oral mucosa from Anamnesis are associated with taking the drug and the rapid elimination of clinical signs of jarokhat after its removal. In its comparative diagnosis, it is important to determine whether changes in Oral mucosa from Anamnesis are associated with taking the drug and the rapid elimination of clinical signs of jarokhat after its removal. Herpetic stomatitis differs from erosive medicinal stomatitis in the comb-like contour of erosions, their typical location and the presence of huge herpes cells when scraped off the surface of erosion.

Treatment. It consists in the cancellation of the drug that caused the harm and the appointment of antihistamines. Treatment. It consists in the cancellation of the drug that caused the harm and the appointment of antihistamines. In severe treatment. It consists in the cancellation of the drug that caused the harm and the appointment of antihistamines. In severe erosive drug stomatitis, corticosteroid drugs are used (prednisone 15-30 mg, dexamethasone 10-14 mg for 2-3 days). Treatment. It consists in the cancellation of the drug that caused the harm and the appointment of antihistamines. In severe erosive drug stomatitis, corticosteroid drugs are used (prednisone 15-30 mg, dexamethasone 10-14 mg for 2-3 days). Local anesthesia, antiseptic treatment of the oral cavity, application to the area of erosion of proteolytic enzymes and agents that stimulate epithelization (vitamins A, ye macular solutions, namatak, actovegin ointment, solkoseril, etc.).

Ulcerative necrotic stomatitis or gingivitis. It is the most severe manifestation of allergic stomatitis caused by the use of drugs. It rarely occurs only on the oral mucosa. Ulcerative necrotic stomatitis or gingivitis. It is the most severe manifestation of allergic stomatitis caused by the use of drugs. It rarely occurs only on the oral mucosa. Usually, ulcerative necrotic stomatitis or gingivitis. It is the most severe manifestation of allergic stomatitis caused by the use of drugs. It rarely occurs only on the oral mucosa. Usually, ulcerative necrotic drug stomatitis develops against the background of severe general allergic reactions with damage to the skin, mucous

membranes and often internal organs. Ulcerative necrotic drug stomatitis occurs with a violation of the general condition of the body (weakness, fever). The disease begins sharply. Patients complain of general malaise, headache, worsening breathing, hypersalivation.

Eating and speaking become difficult due to severe pain. When examining the oral cavity, acute hyperemia and swelling of the mucous membrane are observed, against which there will be foci of yellowish-gray necrosis. The affected areas can cover a small area in the oral cavity, but sometimes affect almost the entire mucous membrane. The lymph nodes under the jaw become enlarged and painful. Ulcerative necrotic drug stomatitis, as well as catarrhal and erosive stomatitis, do not have specific signs of damage in the oral cavity, therefore, in its diagnosis and treatment, it is important to collect a complete Anamnesis to identify the allergen that caused the disease (drug). Ulcerative necrotic drug stomatitis, as well as catarrhal and erosive stomatitis, do not have specific signs of damage in the oral cavity, therefore, in its diagnosis and treatment, it is important to collect a complete Anamnesis to identify the allergen that caused the disease (drug).

Comparative diagnosis it is necessary to distinguish between ulcerative necrotic drug stomatitis: ulcerative necrotic stomatitis of Vinsan, ulcerative damage to the oral mucosa, treatment of trophic ulcers in blood diseases (leukemia, agranulocytosis), cardiovascular diseases.

Treatment. It consists in stopping taking the drug antihistamines are prescribed, parenteral corticosteroid drugs (prednisone per day up to 40-60 mg) are used in severe patients. Treatment. It consists in stopping taking the drug antihistamines are prescribed, parenteral corticosteroid drugs (prednisone per day up to 40-60 mg) are used in severe patients. Symptoms of pronounced intoxication, intravenous infusion of 30% sodium thiosulfate sodium solution is carried out from 5-10 ml per day. Treatment in the hospital polyglucine and others are administered intravenously.

Foods with sensitizing effects (coffee, eggs, chocolate, caviar, etc.), it is also foods with sensitizing effects (coffee, eggs, chocolate, caviar, etc.), it is also parched from foods that irritate the oral mucosa (spicy, hot, spicy).

Contact allergic stomatitis (stomatitis contactilis allergic). Contact allergic stomatitis is a manifestation of a delayed allergic reaction. In dental practice, there are enough materials and drugs that cause contact sensitivity. Contact allergic stomatitis (stomatitis contactilis allergic). Contact allergic stomatitis is a manifestation of a delayed allergic reaction. In dental practice, there are enough materials and drugs that cause contact sensitivity. Contact allergic reactions can develop in fillings made of silver amalgam, prostheses made of dissimilar and homogeneous metals. Of the metals that make up dental prostheses, nickel and its compounds with cobalt, chromium and palladium often cause allergic reactions. Allergies to silver and gold are often combined with allergies to nickel, chromium, and cobalt. A large proportion of contact stomatitis is observed in patients using prostheses made of acrylic plastic. Allergies to silver and gold are often combined with allergies to nickel, chromium, and cobalt. A large proportion of contact stomatitis is observed in patients using prostheses made of acrylic plastic. Plastic components (monomer, various dyes, hydroquinone, benzoyl peroxide) can be used as allergens. Contact allergies can develop on the red border of the lips from cosmetics (lipstick, cream); in the oral cavity — from toothpastes, elixirs.

Clinical picture. Clinically, contact allergy usually manifests itself after 5-7 days, in some cases a few months after the first contact with the allergen, which is determined by the state of reactivity of the patient's body, susceptibility to allergic reactions, the nature of clinical picture. Clinically, contact allergy usually manifests itself after 5-7 days, in some cases a few months after the first clinical picture. Clinically, contact allergy usually manifests itself after 5-7 days, in some

cases a few months after the first contact with the allergen, which is determined by the state of reactivity of the patient's body, susceptibility to allergic reactions, the nature of the allergen, etc. At the place of contact with the Allergen, swelling of the mucous membrane develops, erythema, hemorrhagic phenomena may appear. Some patients may experience small blisters at hyperemic Oral mucosa, which quickly crack and form erosion. At the same time, patients complain of itching, burning, Oral mucosa dryness, violation of taste sensations until complete loss. Allergies can occur on the lips due to the use of cosmetics. At the same time, patients complain of itching, burning, Oral mucosa dryness, violation of taste sensations until complete loss. Allergies can occur on the lips due to the use of cosmetics. In this case, not only the red border of the lips is affected, but also the skin around the mouth, where erythema, swelling, sometimes vesicle or lichenoid rashes appear

Diagnosis. When diagnosing a contact allergic reaction of Oral mucosa, attention is paid primarily to the effect and characteristic clinical picture of jaroxate on sensitivity. iagnosis. When diagnosing a contact allergic reaction of Oral mucosa, attention is paid primarily to the effect and characteristic clinical picture of jaroxate on sensitivity. Diagnosis, elimination test results, skin tests and laboratory tests (leukopenic and thrombocytopenic tests, reaction of specific agglomeration of leukocytes, etc.) important.

Treatment. Etiotropic treatment of patients with contact allergic stomatitis is associated with the elimination of the allergen. Treatment of contact stomatitis from prostheses consists in the production of a new prosthesis from another material. reatment. Etiotropic treatment of patients with contact allergic stomatitis is associated with the elimination of the allergen. Treatment of contact stomatitis from prostheses consists in the production of a new prosthesis from another material. treatment. Etiotropic treatment of patients with contact allergic stomatitis is associated with the elimination of the allergen. Treatment of contact stomatitis from prostheses consists in the production of a new prosthesis from another material. conta

Oral changes in systemic diseases

Epidermal toxic necrolysis (epidermalis toxica necrolysis, syndrome Layella). This disease is one of the most severe forms of moderately toxic-allergic reaction. pidermal toxic necrolysis (epidermalis toxica necrolysis, syndrome

Layella). This disease is one of the most severe forms of moderately toxic-allergic reaction. It is characterized by serious damage to the skin and oral mucosa against the background of a significant deterioration in the general condition. Leila disease occurs more often after taking medications (sulfonamides, bromine salts, iodine, antibiotics, nonnarcotic analgesics), tetanus anotoxin, etc. For this reason, the disease is considered a syndrome of a toxic-allergic nature and is a hyperergic reaction of the body against the background of previous sensitivity. For this reason, the disease is considered a syndrome of a toxic-allergic nature and is a hyperergic reaction of the body against the background of previous sensitivity. In some cases, epidermal necrolysis is the result of poor-quality foods or toxic-allergic effects of staphylococcal infection (mainly in children).

Clinical picture. Epidermal toxic neurolysis begins sharply, with an increase in body temperature to 38-41, the patient develops with a sharp deterioration in axvoli. Clinical picture. Epidermal toxic neurolysis begins sharply, with an increase in body temperature to 38-41, the patient develops with a sharp deterioration in axvoli. Then large erythema (palm-sized) appear on the skin. Hyperemic spots of different sizes also appear on the mucous membrane of the oral cavity, which are mainly located on the tongue, gums and lips. Sometimes the jarokhat is scattered. After 2-3 days, blisters form in the center of erythema, the epidermis and epithelium migrate, which is similar to the clinical picture of 2-degree burns. Painful wide, bleeding erosions form on the mucous membrane of the mouth and skin. Nikolsky's fracture is positive. During cytological examination, acantholytic cells are not detected. Painful wide, bleeding erosions form on the mucous membrane of the mouth and skin. Nikolsky's fracture is positive. During cytological examination, acantholytic cells are not detected. The

general condition of patients is very severe: high body temperature, drowsiness, headache, symptoms of dehydration

observed. According to clinical manifestations, Leila syndrome simultaneously resembles a severe form of KShEE and pemphigus. observed. According to clinical manifestations, Leila syndrome simultaneously resembles a severe form of KShEE and pemphigus. The development of the disease is based on necrosis of the surface layers of the epidermis and epithelium, swelling of the Horn layer, v observed. According to clinical manifestations, Leila syndrome simultaneously resembles a severe form of KShEE and pemphigus. The development of the disease is based on necrosis of the surface layers of the epidermis and epithelium, swelling of the Horn layer, violation of intercellular connections with the formation of internal and subepithelial located vesicles.

Comparative diagnosis. The disease differs from the following: multiform exudative erythema; and pemphigus.

Treatment. Stop taking the drug that caused the disease. Desensitizing drugs (diphenhydramine, suprastin, tavegil, etc.reatment. Stop taking the drug that caused the disease. Desensitizing drugs (diphenhydramine, suprastin, tavegil, etc.), corticosteroids, detoxifying therapy (intravenous 30% sodium thiosulfate solution or 10% calcium chloride solution for a course of 10 ml,10 injections), water in the body, electrolytes and other drugs that maintain protein balance, vitamins C and R are taken. With the exception of allergenic products (coffee, chocolate, eggs, caviar, smoked meats, etc.), it is necessary to carefully care for the patient, taking high-calorie foods.).ith the exception of allergenic products (coffee, chocolate, eggs, caviar, smoked meats, etc.), the exception of allergenic products (coffee, chocolate, eggs, caviar, smoked meats, etc.), it is necessary to carefully care for the patient, taking higith the exception of allergenic products (coffee, chocolate, eggs, caviar, smoked meats, etc.), it is necessary to carefully care for the patient, taking high-calorie foods.). If epidermal necrolysis has a " nutritional " etiology, a milk-vegetable diet should be followed.Topical treatment includes anesthetic, antiseptic obrobotka, removal of necrotic tissue with proteolytic enzymes, and the use of

keratoplastics. The end. This depends on the time of initiation of treatment, and in severe cases, it can be fatal.

Prevention. Prevention of the development of Layla syndrome consists in the careful collection of Anamnesis, especially about sulfonamides and antibiotics. Prevention of the development of Layla syndrome consists in the careful collection of Anamne

Erythema Multiforme Exudativum

Multi-shaped exudative erythema is an inflammatory disease of the mucous membranes and skin, characterized by a pronounced rash of polymorphisms (blisters, spots, blisters) of its pathological elements. Multi-shaped exudative erythema is an inflammatory disease of the mucous membranes and skin, characterized by a pronounced rash of polymorphisms (blisters, spots, blisters) of its pathological elements. Multi-shaped exudative erythema is an inflammatory disease of the mucous membranes and skin, characterized by a pronounced rash of polymorphisms (blisters, spots, blisters) of its pathological elements. It can have a special effect on the mucous membrane of the mouth or skin, but their combined damage is often observed. Multiform exudative erythema is characterized by acute onset and lasts for years. Recurrence of the disease differs mainly in the autumn-spring period. Most often, young people (20-40 years old) get sick, most often men get sick.

Etiology and pathogenesis. They have not been fully identified. According to the etiological principle, two curinations of multiform exudative erythema are distinguished. Etiology and pathogenesis. They have not been fully identified. According to the etiological principle, two curinations of multiform exudative erythema are distinguished. Chin or idiopathic, infeogy and pathogenesis. They have not been fully identified. According to the etiology and pathogenesis. They have not been fully identified. According to the etiological principle, two curinations of multiform exudative erythema are distinguished. Chin or idiopathic, infectious and allergic in nature. In most patients with this form of the disease, bacterial allergens are administered through skin tests (Streptococcus, Staphylococcus, ye. coli)

sensitivity is determined. The source of sensitivity is chronic infection foci. A decrease in the reactivity of the body due to hypovitaminosis, hypothermia, viral infections, stress leads to an increase in multi-form exudative erythema. The toxic-allergic or symptomatic form has a clinical picture similar to Real infectious-allergic multi-form exudative erythema, but in essence the hyperergic reaction of the body to drugs (antibiotics, salicylates, amidopyrin, etc.) is characterized.

Clinical picture. The disease begins suddenly. Tremors, Tremors, weakness appear, body temperature rises (in severe cases 38 or higher).linical picture. The disease begins suddenly. Tremors, Tremors, weakness appear, body temperature riselinical picture. The disease begins suddenly. Tremors, Tremors, weakness appear, body temperature rises (in severe cases 38 or higher). Patients complain of inical picture. The disease begins suddenly. Tremors, Tremors, weakness appear, body temperature rises (in severe cases 38 or higher). Patients complain of headaches, pain throughout the body, pain in muscles and joints, and sore throat. After 1-2 days, purple-red spots appear on the skin of the hands, wrists, calves, sometimes on the face and neck, rising slightly above the surrounding skin. Their central part is slightly depressed and takes on a purple hue, while the peripheral part retains a pinkish-red color (cockscomb). Later, the central part will have a pydo of subepidermal serous or hemorrhagic fluid-filled vesicles. Skin elements are sometimes accompanied by itching and aching, or may even go away painlessly., the central part will have a pydo of subepidermal serous or hemorrhagic fluid-filled vesicles. Skin elater, the central part will have a pydo of subepidermal serous or hemorrhagic fluid-filled vesicles. Skin elements are sometimes accompanied by itching and aching, or may even go away painlessly. The mucous membranes of the lips, cheeks, floor of the mouth, tongue, and soft palate are most often affected. The first manifestations of multi-shaped exudative erythema in the oral cavity are diffuse or restricted erythema and swelling of the mucous membrane, against which subepithelial vesicles of various sizes appear. Damage to the oral mucosa is accompanied by severe pain, even in a calm state. In the movement of the tongue and lips, the pain increases sharply, which makes it difficult to eat.amage to the oral mucosa is accompanied by

severe pain, even damage to the oral mucosa is accompanied by severe pain, even in a calm state. In the movement of the tongue and lips, the pain increases sharply, which makes it difficult to eat. Patients become hungry, which further worsens their condition. The blisters burst very quickly forming painful erosion on the oral mucosa and are covered with fibrinous crusts. Erosion on the red chaff of the lips is covered with bloody crusts, which makes it difficult to eat and open the mouth. In the first days after the bubbles burst, on the edge of the erosions, you can see the remains of a gray white bubble cap, when stretched, it is impossible to separate the epithelium (a negative symptom of Nikolsky). In the first days after the bubbles burst, on the edge of the erosions, you can see the remains of a gray white bubble cap, when in the first days after the bubbles burst, on the edge of the erosions, you can see the remains of a gray white bubble cap, when stretched, it is impossible to separate the epithelium (a negative symptom of Nikolsky). An unsatisfactory condition of oral hygiene, the presence of caries teeth aggravates the course of multi-form exudative erythema. Infection of erosive surfaces with oral microflora occurs. In some cases, the course of multi-form exudative erythema is complicated by the addition of fusospirochetosis. Erosions on the oral mucosa are covered with a thick layer of yellowish-gray plaque, plaque appears on the teeth and tongue, and there is a bad smell, hypersalivation is observed. Erosions on the oral mucosa are covered with a thick layer of yellowish-gray plaque, plaque appears on the teeth and tongue, erosions on the oral mucosa are covered with a thick layer of yellowish-gray plaque, plaque appears on the teeth and tongue, and there is a bad smell, hypersalivation is observed. Local lymph nodes are enlarged and painful. The recurrence period lasts 2-4 weeks. Epithelialization occurs after 7-12 days. After the disease is cured, no traces remain. During an exacerbation of multiform exudative erythema, the peripheral blood corresponds to an acute inflammatory process. The severity of clinical picture of multi-form exudative erythema is mainly associated with damage to the mucous membrane of the oral cavity. The severity of clinical picture of multi-form exudative erythema is mainly associated with damage to the mucous membrane of the oral cavity. The severity of clinical picture of multi-form exudative erythema is mainly

associated with damage to the mucous membrane of the oral cavity. The severe form is characterized by the severity of clinical picture of multi-form exudative erythema is mainly associated with damage to the mucous membrane of the oral cavity. The severe form is characterized by a pronounced hyperergic reaction of the body, as well as general damage to the mucous membrane of the mouth and skin. If multi-form exudative erythema is mild, the general condition of patients does not change significantly, individual pathological elements are detected in the oral mucosa. However, with an increase in the duration of the disease, its severity is exacerbated by single elements of pathology. However, the severity of the disease worsens with increasing duration. Recurrent multiform exudative erythema of an infectious allergic nature is characterized by a long recurrence. Recurrence is mainly observed in the autumn-spring months, the severity of the disease worsens with increasing duration. Recurrent multiform exudative erythema of an infectious allergic nature is characterized by a long recurrence. Recurrence is mainly observed in the autumn-spring months. 1-20 times a year. Multiform exudative erythema can last for years.

Stevens is a severe form of Johnsons syndrome (acute mucosal, cutaneous eye syndrome, ectodermosis erosiva plurioficialis) accompanied by characteristic symptoms of multiform exudative erythema. Stevens is a severe form of Johnsons syndrome (acute mucosal, cutaneous eye syndrome, ectodermosis erosiva plurioficialis) accompanied by characteristic symptoms of multiform exudative erythema. The disease begins suddenly, with high fever. Stevens is a severe form of Johnsons syndrome (acute mucosal, cutaneous eye syndrome, ectodermosis erosiva plurioficialis) accompanied by characteristic symptoms of multiform exudative erythema. The disease begins suddenly, with high fever and joint pain. Against the background of a severe general condition, blisters appear on the lips, lower mucous membrane, tongue, soft palate, back wall of the pharynx, arch of the palate, larynx and skin, after the blisters burst, bleeding erosions are formed. Combining, they constantly bleed becomes a sharply painful surface. Part of the erosion was covered with fibrinous crusts. It also affects the mucous membrane of the eye (bilateral

conjunctivitis and keratitis), nose (rhinitis, nosebleeds) and genitals. Part of the erosion was covered with fibrinous Carache. It also affects the mucous membrane of the eye (bilateral conjunctivitis and keratitis), nose (rhinitis, nosebleeds) and genitals. Due to general damage to the oral cavity and lips, patients cannot speak, eat, which leads to their fatigue. This disease can end in death.

Comparative diagnosis. Acantholytic pemphigus; non-Acantholytic pemphigus; acute herpetic stomatitis; secondary syphilis.

Unlike pemphigus, the pathological element in the multi-shaped exudative erythema is characterized by acute course with pronounced inflammatory phenomena, Nikolsky's fracture negative, erosion, and the absence of acantholytic cell vesicles in the exudate. Unlike pemphigus, the pathological element in the multi-shaped exudative erythema is characterized by acute course with pronounced inflammatory phenomena, Nikolsky's fracture negative, erosion, and the absence of acantholytic cell vesicles in the exudate.

Acute herpetic stomatitis is characterized by large erosions that do not have a multi-shaped exudative erythema polycyclic contour, the absence of pathological elements in the oral mucosa characteristic of herpetic stomatitis, and the absence of multicellular cells when scraped from erosion.

The mild course of multiform exudative erythema may be similar to the eroded papules in secondary syphilis, on the basis of which there is always infiltration. The mild course of multiform exudative erythema may be similar to the eroded papules in secondary syphilis, on the basis of which there is always infiltration. Hyperemia around the syphilitic papules, including the mild course of multiform exudative erythema may be similar to the eroded papules in secondary syphilis, on the basis of which there is always infiltration. Hyperemia around the syphilitic papules, including erosions, has the appearance of being sharply delimited from the healthy mucosa, while with multi-shaped exudative erythema there is extensive, diffuse hyperemia. The pain of syphilitic papules is somewhat expressed, pale treponemes

are found when scraping from their surface, serological reactions to syphilis are positive. The toxic-allergic form is diagnosed based on Anamnesis data on Drug Administration, as well as results from immunological studies (Shelley basophilic degranulation test, lymphocyte blasting transfusion test, cytopathological test) and discontinuation of the process after removal of the allergen drug. The toxic-allergic form is diagnosed based on Anamnesis data on Drug Administration, as well as results from immunological studies (Shelley basophilic degranulation test, lymphocyte blasting transfusion test, cytopathological test) and discontinuation of the process after removal of the allergen drug. In the case of a skin rash, it is not difficult to diagnose.

Treatment. Symptomatic treatment in the acute period of the disease is aimed at reducing intoxication of the body, desensitization, elimination of inflammation and acceleration of epithelization of the affected oral mucosa

General treatment is carried out using desensitizing drugs: diphenhydramine, suprastin, tavegil, fencarol, Claritin, etc. General treatment is carried out using desensitizing drugs: diphenhydramine, suprastin, tavegil, fencarol, Claritin, etc. Salicylates (acetylsalicylic acid) General treatment is carried out using desensitizing drugs: diphenhydramine, suprastin, tavegil, fencarol, Claritin, etc. Salicylates (acetylsalicylic acid, sodium salicylate), calcium gluconate, calcium glycerophosphate, etc. used for anti-inflammatory treatment. For the same purpose, internal injections of sodium thiosulfate are prescribed (10 ml of 30% solution per day, for a course of 8-10 injections). Be sure to prescribe vitamins of Group B (B1, B2, B6), ascorutin. The use of eturridine lactate (from 0.05 g three times a day for 10-20 days) in combination with levamisole (150 mg per day, 2 days a week in a row, with five-day breaks for 2 months) quickly relieves the exacerbation of multi-form exudative erythema. Severe course of multi-form exudative erythema patients should be treated in a General Hospital, where they are prescribed complex treatment: detoxification; desensitization; the use of anti-inflammatory drugs is recommended. Severe course of

multi-form exudative erythema patients should be treated in a General Hospital, where they are prescribed complex treatment: detoxification; desensitization; the use of anti-inflammatory drugs is recommended. For this, corticosteroid drugs are used — prednisone, dexamethasone and B. (20-30 mg per day, depending on the severity of the procedure). The indicated dose of the drug is taken for 5-7 days, then every 2-3 days the dose of Prednisolone is reduced to 5 mg until the drug is completely discontinued. The initial dose of dexamethasone is 3-5 mg. In Stevens-Johnson syndrome, corticosteroids are prescribed in high doses (60-80 mg prednisone per day). The initial dose of dexamethasone is 3-5 mg. Local treatment is aimed at eliminating inflammation, swelling and accelerating the epithelization of the affected oral mucosa. Local treatment is aimed at eliminating inflammation, swelling and accelerating the epithelization of the affected oral mucosa. Before treating the oral still focal treatment is aimed at eliminating inflammation, swelling and accelerating the epithelization of the affected oral mucosa. Before treating the oral still floor, it must be anesthetized with a solution of 1-2% trimecaine, a solution of 1-2% pyromecaine, a solution of 1-2% lidocaine. To reduce pain, oral baths with a 1-2% trimecaine solution are prescribed before meals. Antiseptic treatment of the oral mucosa is carried out with a solution of 0.25% — 0.5% hydrogen peroxide, a solution of 0.25% chloramine, a solution of 0.02% furacilin, a solution of 0.5% ethonium, etc. In the presence of necrotic crust on the erosion surface, it is recommended to apply proteolytic enzymes (trypsin, chymotrypsin, lysoamidase), after which keratoplastic agents (carotolin, and an oily solution of namatak oil vitamins, etc. the presence of necrotic crust on the erosion surface, it is recommended to apply proteolytic enzymes (trypsin, chymotrypsin, lysoamidase), after which keratoplastic agents (carotolin, and an oily solution of namatak oil vitamins, etc.) is used in the presence of necrotic crust on the erosion surface, it is recommended to apply proteolytic enzymes (trypsin, chymotrypsin,

lysoamidase), after which keratoplastic agents (carotolin, and an oily solution of namatak oil vitamins, etc.) is used to accelerate the epithelization of the damaged mucous membrane. During the period of recurrence of the disease, treatment of the mucous membrane of the oral cavity should be carried out daily, and in hospital treatment-2-3 Matras per day. Skin injuries, as a rule, do not require special treatment. In the case of itching and aching in the area of erythema on the skin, it is recommended to lubricate them with Castellani liquid or 2% salicylic alcohol. In the case of itching and aching in the area of erythema on the skin, it is recommended to lubricate them with Castellani liquid or 2% salicylic alcohol. In the case of itching and aching in the area of erythema on the skin, it is recommended to lubricate them with Castellani liquid or 2% salicylic alcohol. In the case of eye damage, 0.5% hydrocortisone eye ointment, 0.1% dexamethasone solution are used. An indispensable condition for the successful treatment of patients with multi-shaped exudative erythema is the detection of foci of chronic infection and its elimination. During the remission period of the disease, patients must undergo a full examination and rehabilitation. In the presence of microbial sensitization, specific hyposensitization therapy is performed with the identified allergens. In the presence of microbial sensitization, specific hyposensitization therapy is performed with the identified allergens.

Chronic Recurrent Aphthous Stomatitis (CRAS)

Chronic Recurrent Aphthous Stomatitis (*Stomatitis aphtosa recidiva*) is a chronic inflammatory disease of the oral mucosa, characterized by the recurrent eruption of aphthous ulcers and a prolonged course during the relapse period. CRAS is one of the most common diseases of the oral mucosa. The prevalence of CRAS, according to various authors, ranges from in different age groups of the population. In the last years, there has been a tendency to increase the number of patients with CRAS and its severe form.

Etiology and Pathogenesis. Most researchers studying the etiology and pathogenesis of CRAS believe that the immune system plays a leading role in the pathogenesis of this disease. CRAS is a disease characterized by a decrease in immunological reactivity and a disruption of non-specific protection. The causes include foci of chronic infection in the body (tonsillitis, chronic pharyngitis, gastrointestinal tract diseases, etc.), as well as the effect of a number of negative factors (chronic stressful situations, frequent changes in climatic zones, occupational hazards, etc.). Disturbances in the state of immunity and non-specific protection have been found in patients with CRAS: depression of the T-immune system is established, expressed by a decrease in the number and functional activity of T-lymphocytes. A disruption of the T-lymphocyte subpopulation is determined, with a significant decrease in the number of T-helpers and an increase in T-suppressors. Changes in the B-immune system are noted, manifested by circulating immune complexes against the background of an increased number of B-lymphocytes, an increase in Immunoglobulin G (IgG) level, and a decrease in Immunoglobulin M (IgM) content. In CRAS, indicators of non-specific humoral and cellular protection change (decrease in Lysozyme concentration and increase in Beta-lysins in blood serum, decrease in complement fractions and increase in fractions). Against the background of weakened phagocytic activity of leukocytes to most studied microbial allergens, its intensification is noted for *S. salivarius* and *C. albicans*. Patients with CRAS exhibit a disruption of local protective factors in the oral cavity, characterized by a decrease in Lysozyme concentration, an increase in Beta-lysins, and a decrease in the content of secretory Immunoglobulin A (sIgA) in the oral fluid. As a result, the protection of the oral mucosa from the effects of microorganisms is disrupted, and the number and species composition of the resident microflora change. Consequently, the number of microbial associations in the oral cavity increases, and their virulence increases.

The microbial association of the oral mucosa in patients with CRAS is mainly represented by coccal flora, in which an important place is occupied by coagulase-negative staphylococci and anaerobic cocci (peptococci, peptostreptococci), and the

number of anaerobes increases. With an increase in the virulence of microorganisms, the bacterial sensitivity of the body of patients with CRAS increases – a chain of immunological reactions of immediate and delayed types is activated, leading to frequent relapses of the disease. Immunological reactions play an important role in the pathogenesis of CRAS, interacting according to the following principle. On the surface of the oral mucosa of patients with CRAS, a large number of streptococci (52.9%) are present, among which *Streptococcus mutans*, *sanquis*, *salivarius*, *mitis*, have antigenic similarity with the cells of the oral mucosa. The disruption of target cell recognition by T-lymphocytes in CRAS is partially genetically determined, and at the same time, a diverse antigenic spectrum is present on the surface of the oral mucosa. As a result, the mechanism of antibody-dependent cytotoxicity is activated, which, according to some authors, is the cause of this disease. This mechanism can explain the formation of aphthae as a result of the manifestation of the Arthus phenomenon, as well as the significance of gastrointestinal diseases and chronic foci of infection, which are accompanied by a disruption of the balance between the macroorganism and the bacterial flora.

Clinical Manifestations. There are two clinical forms of recurrent aphthous stomatitis of the oral mucosa: mild and severe (recurrent deep scarring aphthae), which have specific clinical features. The complex of symptoms that determine the severity of the course of CRAS includes the frequency of disease recurrence and, accordingly, the duration of its remission, the nature, number, and duration of resolution of pathological elements on the oral mucosa. CRAS can be a manifestation of Behçet's disease (general aphthosis), in which the genital and intestinal areas are affected. In addition to recurrent aphthous eruptions on the oral mucosa, eye damage and sometimes pyoderma appear.

Recurrent Aphthous Stomatitis (Mild Form). The appearance of typical aphthae on the oral mucosa is characteristic. The process of aphthae formation begins with the appearance of a small spot with a diameter of up to which is hyperemic, sharply delimited, round or oval shape, painful, and slightly elevated above the surrounding mucosa after a few hours. After a few more hours, the element erodes and is covered

with a fibrinous gray-white, firmly seated plaque. This focus of fibrinous-necrotic plaque is surrounded by a thin hyperemic border. The aphtha is very painful on palpation and soft to the touch. Infiltration forms at the base of the aphtha, due to which the aphtha is slightly elevated above the surrounding tissues; on its surface, necrotic masses form a gray fibrinous film. The aphtha is sharply delimited, surrounded by a distinct hyperemic, slightly swollen border. It is sharply painful and is often accompanied by lymphadenitis.

After 2-4 days, the necrotic masses detach, and after another 2-3 days, the aphtha usually resolves; a slight hyperemia remains for some time. Often, a few days before the aphtha appears, patients feel a sensation of burning or pain.

In the mild form of CRAS, one or two aphthae appear simultaneously, rarely more. A specific feature of the disease is the recurrent nature of the rashes. The frequency of aphthae appearance in CRAS varies from a few days to several months. The rashes are often localized on the mucous membranes of the cheeks, lips, tip and lateral surfaces of the tongue, but they can appear on any part of the oral mucosa.

Chronic Recurrent Aphthous Stomatitis (Cont.)

Chronic Recurrent Aphthous Stomatitis (CRAS) - Mild Form

The mild form of recurrent aphthous stomatitis, as a rule, often goes unnoticed by the patient because its initial clinical signs are very mildly expressed.

In 50% of patients with this form of CRAS, recurrences occur 1-2 times a year, so they rarely consult a doctor. The remaining half of patients with mild recurrent stomatitis seek medical help more often because they experience 5-6 exacerbations a year, and the course of the disease becomes more severe.

Factors that provoke recurrence include:

- Trauma to the oral mucosa
- Stress
- Fatigue
- Viral infection
- Premenstrual period, etc.

In some cases, patients cannot associate the onset of a relapse with any specific factor. Clinical observations show that during the first three years of its existence, CRAS is predominantly in the mild form. There are sometimes cases of a rapid transition of the disease to the severe form. This is mainly characteristic of young people (17-20 years old). In most cases, the duration of CRAS for more than three years is accompanied by a worsening of its clinical course and the transition of the mild form to the severe one. Factors accelerating the transition of the mild form of CRAS to the severe one include: occupational hazards, frequent changes in climatic zones, chronic diseases (tonsillitis, gastrointestinal tract diseases – chronic gastritis, colitis, stomach and duodenal ulcers), and in some cases, young age (up to 25 years).

Histological Examination (Mild Aphtha)

Histological examination of a typical aphtha reveals deep fibrinous-necrotic inflammation of the mucous membrane. The process begins with changes in the lamina propria (connective tissue layer of the mucosa) and the submucosa. As a result of vascular dilation, small perivascular infiltration, edema of the stratum spinosum of the epithelium, followed by spongiosis and micro-cavity formation occur. Alterative changes lead to necrosis of the epithelium and erosion of the mucous membrane. The epithelial defect is filled with fibrin that is firmly attached to the underlying tissues.

Differential Diagnosis (Mild Aphtha)

Aphthae have similarities in appearance with:

- Traumatic erosion
- Herpetic erosion
- Syphilitic papules, on the surface of which, some time after their appearance, a necrotic gray-white plaque forms.
- Herpetic erosion differs from aphtha by its polycyclic contours, less pronounced pain, and a diffuse inflammatory reaction in the surrounding area; herpetic erosion is preceded by the appearance of grouped vesicles.

- Syphilitic papules are characterized by minimal pain, the presence of infiltration at the base, the stable nature of the inflammatory ring around the periphery, and the presence of pale treponemes in the scraping from the erosion focus.

Chronic Recurrent Aphthous Stomatitis - Severe Form (Recurrent Deep Scarring Aphthae / Sutton's Aphthae) The severe form of recurrent aphthous stomatitis can have the following clinical variations:

1. The single lesion in the oral mucosa is an aphtha, with an epithelialization period of 14-20 days. The course of the disease is characterized by the occurrence of monthly relapses.
2. Deep, crater-like, sharply painful ulcers appear, with a long epithelialization period (25-35 days). Exacerbations occur 5-6 times a year.
3. Aphthae and ulcers are found simultaneously in the oral mucosa. Their epithelialization period is 25-35 days.

The severe form of CRAS worsens 5-6 times a year or monthly. The course of the disease is chronic. In a number of patients, aphthae appear paroxysmally for several weeks, succeeding one another or appearing in large numbers simultaneously. In other patients, a single aphtha develops at different times. The course of the disease in the same patient can change over time.

In patients with severe CRAS, the general condition worsens: irritability, poor sleep, loss of appetite are noted (in 70% of patients). In 22% of patients, a neurotic state is formed due to constant pain in the oral cavity. Local lymphadenitis is often present.

The influence of seasonal factors on the exacerbation of severe CRAS is minimal. As a rule, exacerbations of the severe form of CRAS occur monthly, and the disease becomes permanent, with the severity of its clinical course worsening as the frequency of recurrence increases. The exacerbation of the disease usually begins with the appearance of a localized painful induration of the mucous membrane, in which a superficial, fibrinous plaque initially forms, and then a deep, crater-like ulcer with slight hyperemia around it. The ulcer, as a rule, enlarges. Sometimes a

superficial aphtha forms at the beginning, at the base of which an infiltrate forms after 6-7 days, and the aphtha itself turns into a deep ulcer.

Ulcers in the severe form of CRAS epithelialize very slowly (up to 1.5–2 months). After healing, coarse scars of connective tissue remain, which leads to deformation of the oral mucosa. If such ulcers are located at the corners of the mouth, deformations may appear, sometimes leading to microstomia (reduced mouth opening). The duration of aphtha scarring varies from 2 weeks to 2 months. The rashes are often localized on the lateral surfaces of the tongue, the mucous membrane of the lips and cheeks, and are accompanied by intense pain.

Diagnosis (Severe Aphtha). Histologically, with deep recurrent aphthae, a complete destruction of the epithelium and basement membrane is determined, as well as a focus of necrosis with scarring in the lamina propria and submucosa. Salivary glands with periglandular infiltration are often found in the affected areas, which led Sutton to call the disease recurrent necrotic periadenitis of the mucous membrane. However, A.L. Mashkilleyson observed deep scarring aphthae without phenomena of periadenitis.

Differential Diagnosis (Severe Aphtha)

The severe form of recurrent aphthous stomatitis is differentiated from:

- Traumatic erosion
- Traumatic ulcer
- Recurrent Herpes
- Behçet's disease
- Vincent's ulcerative-necrotic stomatitis
- Ulcers in specific infections (syphilis, tuberculosis)
- Cancerous ulcer
- In Behçet's disease, combined aphthous-ulcerative lesions of the oral, ocular, and genital mucous membranes are observed.
- In Vincent's ulcerative-necrotic stomatitis, an abundance of fusobacteria and spirochetes is found in the ulcer.

- The edges of a cancerous ulcer are dense, slightly painful, and often a history of chronic trauma is present. Atypical cells are detected in the cytological examination.



Figure 56. Chronic Recurrent Aphthous Stomatitis (CRAS) - Treatment and Behçet's Syndrome

The treatment of Chronic Recurrent Aphthous Stomatitis (CRAS) is effective only with a thorough clinical and immunological examination of the patient, which allows for the selection of an appropriate individualized complex pathogenetic treatment based on the data obtained. Before starting treatment, the patient should be examined by a therapist, otorhinolaryngologist, and other specialists to identify concomitant diseases, primarily those of the gastrointestinal tract, foci of chronic infection, and the state of the immune system. Particular attention should be paid to the detection and treatment of dental and periodontal diseases. All these measures allow for the individualization of the complex pathogenetic treatment process for patients with CRAS. The complex pathogenetic treatment of patients with CRAS includes the use of immunocorrective agents and metabolic corrective drugs with mandatory rehabilitation of chronic infection foci.

Patients are prescribed two complexes of drugs with metabolic action:

1. First Metabolic Complex (For 10 days)

Aimed at improving energy processes in lymphocytes:

- Calcium Pantothenate (2 ml of 20% solution IM or 0.1 g orally 4 times a day)
- Riboflavin Mononucleotide (1 ml of 1% solution IM)
- Lipamide (0.025 g 3 times a day after meals)

- Cocarboxylase (0.05 gIM)
- Potassium Orotate (0.5 g 3 times a day 1 hour before meals)

2. Second Metabolic Complex (For the next 10 days)

Prescribed after the first complex:

- Vitamin B12 (1 ml of 0.01% solution IM)
- Folic Acid (5 mg 3 times a day)
- Pyridoxal Phosphate (0.02 g 3 times a day after meals)
- Methylmethionine Sulfonium Chloride (0.1 g 3 times a day after meals)
- Calcium Pangamate (0.05 g 3-4 times a day)
- Potassium Orotate (0.5 g 3 times a day 1 hour before meals)

The sequence of administration is determined by cytochemical analysis but, as a rule, the first complex optimizes cell energy, and the second requires energy readiness of the tissues to utilize it. To achieve a state of stable clinical remission of CRAS, characterized by the normalization of the cytochemical status of blood lymphocytes, 4-6 courses of metabolic therapy should be conducted with an interval of 4 months, regardless of the features of the clinical course of the disease.

In some cases, the set of therapeutic measures includes sedatives (Valerian root, "minor" tranquilizers). Particular attention should be paid to measures to eliminate foci of chronic infection in the body of patients with CRAS (treatment of chronic diseases of the ENT organs, gastrointestinal tract, etc.), which should be carried out simultaneously with metabolic correction. The presence of chronic septic foci in patients with CRAS causes constant bacterial sensitization, including of lymphocytes, which leads to frequent exacerbations of the disease.

Immunocorrection

- Thymogen is used, which has a regulatory effect on the reactions of cellular and humoral immunity, as well as on non-specific resistance factors of the body. Thymogen is administered intramuscularly at 100 µg per day for 10 days. Immunogram monitoring should be performed before and after treatment.
- Levamisole (Decaris) is also used to treat CRAS. The drug is prescribed 2 days a week (consecutively or with an interval of 3-4 days, 150 mg once or 50 mg 3

times a day). Treatment is carried out for 1.5-2 months under the control of the clinical formula of peripheral blood and the patient's general condition.

Metabolic Stimulation. To normalize the cellular metabolism of lymphocytes, patients with recurrent aphthous stomatitis are prescribed metabolic drugs that stimulate metabolic processes at the mitochondrial level. The choice of drugs and the duration of metabolic therapy are determined by the cytochemical parameters of the enzyme status of blood lymphocytes (activity of mitochondrial Succinate Dehydrogenase, alpha-Glycerophosphate Dehydrogenase).

Physical Therapy

Modern methods of physical influence on the body (transdermal electrical stimulation, laser therapy for reflexogenic zones, aeroion massage on the affected oral mucosa) are effectively used to stimulate specific and non-specific defense factors in patients with CRAS.

Summary of Complex Therapy

A complex of therapeutic measures, including immunocorrective, metabolic, and reflexotherapy, helps to quickly eliminate the exacerbation of the disease, significantly prolong its remission periods, eliminate cell and tissue hypoxia, and normalize immunological parameters in patients with recurrent aphthous stomatitis. A positive therapeutic effect can also be obtained by applying each of the aforementioned treatment methods individually, but its effectiveness is less pronounced. Therefore, the combined use of these methods for the treatment of CRAS is recommended.

Patients are prohibited from eating spicy, sour foods, alcoholic beverages, and smoking.

Local Treatment

Local treatment is reduced to rehabilitation of the oral cavity, elimination of traumatic factors and foci of chronic infection.

- During the period of exacerbation, pain relievers are used, as aphthae and especially ulcers are painful. For anesthesia, anesthetics are used (1-2% Lidocaine solution, 1-2% Trimecaine solution, 1-2% Pyromecaine solution, or 5% Pyromecaine

ointment). A 5 or 10% suspension of the anesthetic in glycerin or liquid oils (peach, apricot, sunflower) is also used for anesthesia.

- Fibrinous and necrotic plaque is removed from the surface of the aphtha and ulcer using proteolytic enzymes (Trypsin, Chymotrypsin, Chemopsin, Lysoamidase).
- Antiseptic treatment is carried out with antiseptic solutions (1% Ethacridine Lactate solution, 0.02–0.06% Chlorhexidine solution, 0.02% Furacillin solution, etc.).
- To stimulate the epithelialization of the affected oral mucosa in CRAS, it is recommended to prescribe oil solutions of Vitamins A, E, Carotolin, 5% Linethol ointment, Solcoseryl salve and gel, 5% ointment and 20% Actovegin gel, etc.

Prognosis: The prognosis for recurrent aphthous stomatitis is favorable, especially with early diagnosis and treatment of its mild form.

Prevention. Prevention consists mainly of the timely detection and elimination of foci of chronic infection, including in the oral cavity, early diagnosis and treatment of chronic diseases of the gastrointestinal tract, nervous, and endocrine systems. Systematic oral care and regular sanitation are important. Strict adherence to the regime of work and rest, active physical education, hardening, and rational, balanced nutrition are necessary.

Behçet's Syndrome

The disease was first identified as a syndrome by the Turkish dermatologist Behçet and subsequently named "Behçet's Syndrome."

The described syndrome includes three main symptoms:

1. Recurrent aphthae on the mucous membranes of the mouth and external genitalia.
2. Eye damage (often both eyes), manifested as hypopyon-iritis, sometimes leading to blindness. Patients with Behçet's syndrome often exhibit skin manifestations (erythema nodosum rash, pyoderma, vasculitis), as well as damage to the joints and nervous system. Touraine (1941) described the disease—large aphthosis—which is clinically characterized by recurrent eruptions of aphthae on the mucous membranes of the mouth, external genitalia, and intestines.

Etiology and Pathogenesis.The causes of Behçet's syndrome are not established. Most authors believe that autoimmune processes are at the center of the recurrent aphthous lesions of the mucous membranes of the mouth, genitals, eyes, and in some cases, the intestines. There are also opinions about a genetic predisposition to the occurrence of Behçet's symptom.

Clinical Manifestations.The disease is characterized by a chronic course with remissions lasting from a few days to several years. People of both sexes suffer from this disease equally often, mainly at the age of 20-30 years.



Figure 57. Behçet's Syndrome and Exogenous Intoxications

Behçet's Syndrome

The image description highlights typical lesions: an ulcer with hyperemic infiltrated edges, covered with a gray-yellow plaque, and a hyperemic border around an aphtha on the lower lip mucosa. Aphthae in the oral mucosa are often found as the first manifestation of Behçet's syndrome, which, as a rule, is indistinguishable from the typical aphthae that occur with recurrent aphthous stomatitis (CRAS). In some patients, deep, long-lasting, non-healing aphthae form, which, after epithelialization, leave coarse connective tissue scars, deforming the mucous membrane. The aphthae are sharply painful, and their number varies.

Genital Lesions. Painful aphthous ulcers appear in the external genital area (on the mucosa, skin of the labia, scrotum, penis) with dense infiltration beneath the rash. The elements are slightly elevated above the surrounding skin, and the bottom of the ulcers is covered with a gray-yellow plaque.

Eye Damage. According to reports, 70-85% of patients with Behçet's syndrome have eye damage. In a number of patients, this occurs before the appearance of aphthous rashes in the oral mucosa and is the first sign of the disease. Initially, painful photophobia is noted, followed by rapidly developing hypopyon-iritis, cyclitis, hemorrhages in the vitreous body and fundus. This leads to a temporary, and later permanent, decrease in vision. The process often ends in blindness.

Skin and Systemic Manifestations

Rashes such as recurrent erythema nodosum, erythema multiforme exudativum, and pyoderma elements often appear on the skin in Behçet's syndrome.

Pathergy of the skin is observed in Behçet's syndrome in areas of mild trauma. For example, various pustular elements appear at the sites of biopsy and subcutaneous injections. This is highly characteristic of Behçet's syndrome, so it is believed that a diagnosis can be established based on the presence of only two symptoms—aphthae on the oral mucosa and rash on the genitalia—in combination with pathergy. The process is often complicated by rheumatoid damage to soft tissues and joints, hemorrhages (due to increased fragility of blood vessels), recurrent thrombosis, and thrombophlebitis. Aphthous rashes can spread to the mucous membrane of the pharynx, trachea, larynx, esophagus, and intestines. Sometimes, swelling of the salivary and lacrimal glands, as well as various changes in the internal organs and nervous system, are present. Fatal outcomes are observed. Behçet's syndrome is characterized by a chronic recurrent course and an increase in all clinical manifestations.

Diagnosis (Histology) Histologically, during the formation of the aphtha element, infiltration of lymphocytes and monocytes into the epithelium is determined. After epithelial damage, erosion occurs, at the bottom and sides of which an infiltrate consisting of neutrophils, monocytes, and lymphocytes appears. Sometimes vascular changes are detected in the form of obliterating endarteritis. In long-lasting elements, vasculitis is determined, but such changes are less common in new rashes.

Treatment. The treatment of Behçet's syndrome is a difficult task.

- The use of antihistamines and detoxifying agents is recommended.
- Considering the activation of chemotaxis, the use of the cytostatic Colchicine is suggested.
- The use of corticosteroid preparations is often effective, for example, Dexamethasone prescribed at 3-6 mg per day, followed by transition to maintenance doses.
- In recent years, there have been reports of the beneficial effect of Levamisole (Decaris) (150 mg 2 times a week) in patients with severe aphthosis. To prolong the remission period, patients took the drug for six months or more.

Sjögren's Syndrome

Sjögren's Syndrome (*Syndromum Sjögren*) is a systemic disease described by Sjögren in 1933. Infectious and allergic genesis, the leading importance of endocrine disorders, and hereditary predisposition associated with impaired vitamin A metabolism are assumed. At the same time, the detection of hypergammaglobulinemia and the presence of antinuclear factors and pronounced autoantibodies in the blood serum of patients with Sjögren's syndrome provide grounds for a number of authors to confirm the autoimmune nature of the disease.

Clinical Manifestations. For a long time, the initial changes may manifest as a decrease in the amount of saliva and an increase in its viscosity. Subsequently, changes in the mucous membrane are observed: atrophy of the papillae of the tongue appears, the tongue becomes hyperemic, and a sensation of burning occurs. By this

time, atrophic changes in the mucous membrane of the upper respiratory tract (dry tracheobronchitis, rhinitis) may appear. The sense of smell may decrease.

Complaints depend on the severity of the process. With increasing hyposalivation, patients complain of dry mouth and pain during eating, as well as the appearance of cracks at the corners of the mouth (angular cheilitis). The eyes become dry, accompanied by conjunctivitis, pain, photophobia, and lack of tears. In the later stage, in addition to dryness of the mucous membranes of the mouth, eyes, and nose, atrophic changes are observed on the genital mucosa, dryness and scaling of the skin, dryness and brittleness of the hair, and a predisposition to dermatosis. Changes in the type of chronic arthritis may be present (in about 1/3 of patients). In some patients, pneumosclerosis, neuralgia of the peripheral and trigeminal nerve, and other changes appear.

Upon examination, along with reduced salivation and the changes described above in the oral mucosa, a tumor-like enlargement of one or both parotid and other salivary glands is detected. Sometimes stones form in the parotid gland.

Diagnosis and Histology

- Histological examination of the oral mucosa shows focal lymphocytic infiltration, and a significant decrease in the secretory activity of the salivary glands, including the submucosal ones. Histological changes in the salivary glands are characterized by infiltration of lymphocytes, plasma cells, and an increase in connective tissue.
- The diagnosis of Sjögren's syndrome does not cause particular difficulties due to the typical clinical picture. Sjögren's syndrome must be differentiated from xerostomia (dry mouth of other causes).

Treatment is comprehensive, carried out by a dentist, ophthalmologist, and therapist.

- Saliva-stimulating drugs, Vitamins A, E, B12, and ascorbic acid are prescribed.
- Corticosteroid preparations and Levamisole are used.

Changes in the Oral Mucosa in Exogenous Intoxication

Damage to the oral mucosa can be caused by negative factors of a person's professional activity. The effect of relatively low but long-term concentrations of

harmful chemicals can lead to the development of chronic intoxication, often with specific symptoms in the oral mucosa.

The property of heavy metals (such as Lead, Mercury, Bismuth, etc.) to accumulate in the body with the formation of depots in bones, parenchymal organs, subcutaneous tissues, etc., is very important in the development of chronic intoxication. Those working with such metals may eventually develop chronic intoxication, which manifests with a clinical picture characteristic of each metal in the oral cavity. Changes in the oral cavity can also occur as a result of the long-term use of drugs used to treat chronic diseases: Bismuth (Biyokinol, Pentabismol, Vismuverol), Mercury, Gold (Chrysanola, Auranofin). The severity of the toxic effect of the medicinal substance is determined by its dose and concentration in the body. The salts of heavy metals are partially excreted by the salivary glands, which is why signs of intoxication are often manifested in the oral cavity.

Bismuth Intoxication

Develops mainly in people who have used bismuth preparations for long-term treatment (Syphilis, non-syphilitic lesions of the CNS).

Clinical Manifestations

- Manifested by general disorders (nephropathy, gastrointestinal tract, nervous system damage: weakness, apathy) and damage to the oral mucosa.
- The mild degree of intoxication is accompanied by catarrhal inflammation of the oral mucosa.
- In severe cases, erosive-ulcerative gingivostomatitis develops with hypersalivation and fetid breath.
- Long-term use of bismuth preparations results in the accumulation of bismuth particles in the gingival mucosa, leading to a characteristic blue-gray border at the gingival margin (Bismuth line).
- In some cases, bismuth intoxication is accompanied by the formation of dark, almost black pigmentation in other parts of the oral mucosa (tongue, cheeks, lips, palate, tonsils). Sometimes the pigmentation resembles black circles around the openings

of the excretory ducts of the salivary glands. The bismuth line is the result of the formation of Bismuth Sulfide upon excretion with saliva.

- With severe intoxication, a deep ulcer surrounded by a blue-gray border appears. In the presence of ulcerative gingivostomatitis with painful sensations, tartar, and carious teeth, ulcerative-necrotic gingivostomatitis rapidly develops in an uncleansed oral cavity.

Treatment

- First of all, the intake of bismuth preparations must be stopped.
- A complex of active detoxification and symptomatic general therapy is carried out.
- Locally, the affected oral mucosa is treated with proteolytic enzymes, antiseptics, and epithelialization-stimulating agents.

Lead Intoxication. Develops as a result of exposure to harmful professional factors among workers in metallurgy, battery production, chemistry, mining, and others. Intoxication is caused by the inhalation of lead vapors and dust, which accumulate in the body, mainly in bone tissue.

Clinical Manifestations

- The initial symptom is a pale gray color (lead pallor) caused by lead damage to blood vessel walls and their spasm.
- One of the clinical manifestations of severe lead intoxication is "lead colic." Peripheral paralysis and blindness associated with damage to the central nervous system may develop.
- Weakness, apathy, digestive disorders, and kidney damage (nephropathy) are observed. Anemia, thrombocytopenia, and monocytosis are detected in the blood.
- Characteristic symptoms in the oral cavity are a sweet metallic taste and fetid breath-"lead breath"-as well as the appearance of the "lead line" (halo saturninus), which is the first sign of saturnism in the oral cavity.
- The border is located along the gingival margins around the necks of the frontal teeth, mainly on the vestibular surface, and has a gray-black color. Upon careful examination, it can be seen that it is not uniform, like the bismuth line, but consists of fine grains.

- Lead pigmentation in the form of black and blue spots may simultaneously appear on the gums, cheeks, tongue, lips, and palate.
- In severe cases, ulcerative stomatitis may develop.
- Damage to the oral mucosa is associated with skin damage in some cases. Erythematous or bullous dermatitis appears.

Treatment

- Contact with lead must be stopped.
- General detoxification, oxygen, and symptomatic therapy are carried out according to indications.
- Vitamin preparations (C, B1, B2, B6, B12), as well as food rich in calcium and phosphorus, are prescribed.
- Local treatment includes the removal of dental deposits, necrotic areas of the gums using proteolytic enzymes, antiseptics, and keratoplastic preparations (oil solutions of Vitamins A, E, rosehip oil, Solcoseryl, etc.).

Prevention. Prevention of lead poisoning mainly involves adherence to the Maximum Permissible Concentration (MPC) of lead in the air of workplaces. The use of respirators, washing with 1% acetic acid solution, irrigation of the oral cavity with oxygen-saturated solutions, and vitamins using special devices are necessary to prevent the toxic effects of lead on the tissues and organs of the oral cavity.

Mercury Intoxication. Can occur as a result of exposure to harmful professional factors in workers in the mercury industry. In this case, mercury and its compounds enter the human body mainly through the respiratory tract in the form of vapor and dust, and partially through the gastrointestinal tract and skin.

Chronic mercury intoxication can also occur with the long-term use of mercury-containing preparations (Mercuzal, Novurit, Sublimate, Calomel, mercury-containing ointments). When mercury-containing preparations are used topically, mercury interacts with tissue proteins to form albuminates. In this case, the astringent or irritating effect of the drugs is manifested. The solubility and

penetration depth of mercury compounds increase in the presence of salts of sodium chloride and other halogens.

Clinical Manifestations

- In case of poisoning with mercury vapors, signs of damage to the respiratory tract (rhinitis, tracheobronchitis, toxic pneumonia) are most clearly manifested. At the same time, the gastrointestinal tract and central nervous system are affected. Drowsiness appears, alternating with periods of excitation, and hand tremors ("mercury tremor") appear.
- With oral and local application of mercury preparations, the gastrointestinal tract is more affected (severe abdominal pain, vomiting, diarrhea with blood).
- Clinical manifestations of mercury intoxication in the oral cavity begin with catarrhal gum disease. Mercury gingivitis develops, characterized by swelling, pain, bleeding of the gums, and the appearance of an ulcerative-necrotic process.
- A characteristic black border appears at the gingival margin as a result of the reaction of mercury with hydrogen sulfide to form Mercury Sulfide.
- In severe cases, the process can spread to the mucous membrane of the cheeks, tongue, and tonsils, where foci of necrosis surrounded by a hyperemic border form.
- There is a fetid odor from the mouth, and salivation increases. Due to the swelling of the mucous membrane of the tongue and cheeks, tooth marks are clearly visible on them. Local lymph nodes are enlarged, mobile, and painful.
- Patients complain of a metallic taste in the mouth, pain in the gums, headache, general weakness, and an increase in body temperature. Speech and swallowing are difficult, and chewing is painful.

Treatment. First of all, the further intake of mercury into the body must be stopped, and medical measures aimed at its elimination must be carried out. For this purpose, methods of active detoxification of the body are used (gastric lavage, forced diuresis, hemodialysis). 5% solution of Unithiol, 30% solution of Sodium Thiosulfate, vitamins (B6, B12), etc., are administered.

- Local treatment includes antiseptic treatment of the oral cavity and removal of dental deposits. In the presence of ulcerative processes, proteolytic enzymes and keratoplastics are used.

Prevention. Prevention of mercury poisoning consists in strict adherence to the rules for handling and storing mercury in production and especially in everyday life. Strong ventilation is required in rooms where mercury is handled. Sanitary and hygienic control of the indoor air environment for the content of mercury and its compounds is necessary. Sanitary and educational work is a very important factor in the prevention of mercury poisoning.

Gold Preparations. These drugs (Chrysanola, Auranofin) are used in the treatment of rheumatoid arthritis and lupus erythematosus as a means of stimulating cellular immunity and inhibiting humoral immunity. Gold preparations mainly accumulate in the lungs and kidneys. They are excreted very well with urine. Overdose of gold preparations can lead to nephropathies, dermatitis, stomatitis, and anemia.

Changes in the oral mucosa during an overdose of gold preparations are characterized by hypersalivation and the appearance of erythematous swelling (often on the tongue). Dense white, pearl-like plaques develop, which resemble the clinical picture of Lichen Planus.

Changes in the Oral Mucosa in Hypovitaminosis

Vitamins are biologically active organic substances that, along with proteins, lipids, and carbohydrates, are necessary for the normal functioning of the human body. Vitamins are biological catalysts that interact with enzymes and hormones. They are part of many enzymes as cofactors or participate in their synthesis. The absence or deficiency of any vitamin leads to a disruption in the formation of enzymes and hormones that regulate metabolism in the body.

The oral mucosa is highly sensitive to vitamin deficiency. Vitamin deficiency reduces the resistance of the oral mucosa to damage and microorganisms, and the processes of its regeneration slow down. The main source of vitamins is food. Furthermore, it is important to note that not only a low absolute quantity of vitamins but also a disruption of the balance between the qualitative composition of the food

and the quantitative content of vitamins in it can be significant. For instance, the need for Vitamin B1 increases when consuming carbohydrate-rich foods, and the absorption of Vitamin B2, Nicotinic Acid, and Ascorbic Acid decreases with protein deficiency, and so on.

Hypovitaminosis can be:

- Primary (exogenous): Due to a low vitamin content in the diet, as well as monotonous, unbalanced, or insufficient nutrition.
- Secondary (endogenous): Occurs in diseases of the gastrointestinal tract, when the absorption of nutrients, including vitamins, is reduced; or with impaired utilization of vitamins after absorption, such as in liver diseases, endocrine diseases, etc.

It is necessary to specifically mention conditions caused by an increased demand for vitamins by the body. This can occur under the influence of low or high ambient temperatures, physical exertion, neuropsychic stress, oxygen deficiency, and so on. The need for ascorbic acid, pyridoxine, folic acid, calciferol, and tocopherol increases during pregnancy and lactation.

Most vitamins are closely related in metabolic processes. It is believed that a deficiency of only one type of vitamin almost never occurs. In this regard, it is important to emphasize the possibility of synergism and antagonism in vitamin action. With increased intake of ascorbic acid (Vitamin C), the need for Vitamin B1 decreases, and with B1 deficiency, the level of Vitamins C and B2 in the tissues decreases.

Retinol Deficiency (Vitamin A). One of the important functions of Vitamin A is the regulation of differentiation and keratinization processes of epithelial cells, including those of the oral mucosa (OM). The participation of Vitamin A in normalizing metabolic processes in epithelial tissues is considered its main physiological function in the body. Furthermore, retinol has the ability to restore the structural integrity not only of the epithelium but also of the connective tissue, leading to the thickening of collagen and elastic fibers.

Clinical Manifestations. Vitamin A deficiency leads to significant epithelial changes—atrophy and inflammation—as a result of which the protective properties of the skin and OM are significantly reduced. Hypovitaminosis A is characterized by dryness of the oral and skin mucosa, increased keratinization processes (hyperkeratosis), and significant dysfunction of the sebaceous glands. Hyposalivation is observed. The inflammation on the mucous membrane can be complicated by erosion and ulceration.

Histology. Histologically, in hypovitaminosis A, epithelial metaplasia is detected, with the replacement of some cell rows of the stratum spinosum with keratinizing cells.

Treatment

- Retinol is prescribed at 50,000-100,000 IU per day. Large doses of Vitamin A should be used under medical supervision, as hypervitaminosis can develop, accompanied by drowsiness, weakness, headache, facial hyperemia, followed by skin peeling. Vitamin A is prescribed in the form of a concentrate or tablets.
- In dental practice, Vitamin A is widely used as a means of accelerating epithelialization of erosions and ulcers on the oral mucosa; for the treatment of hyperkeratosis (leukoplakia, lichen planus, etc.); and to reduce dryness of the oral mucosa.
- Vitamin A is prescribed orally after meals in the form of tablets of 3300 IU or drops, 1-2 times a day, with a course of treatment for 1.5-2 months. Oil solutions of Vitamin A are also used for local treatment of erosions and ulcers of the oral mucosa to accelerate their epithelialization.

Thiamine Deficiency (Vitamin B1)

Vitamin B1 plays a particularly important role in carbohydrate metabolism by carrying out the phosphorylation reaction. Thiamine deficiency leads to incomplete combustion of carbohydrates and an increase in pyruvic and lactic acids in the body, which negatively affects the functioning of the nervous and cardiovascular systems. Vitamin B1 is also involved in protein metabolism. Its derivative, Thiamine

Diphosphate, is part of the enzyme cocarboxylase, which catalyzes the cleavage of CO₂ from keto acids.

Clinical Manifestations.In humans with B1 deficiency, the disease Beriberi develops, characterized by disorders of the cardiovascular and nervous systems.

- In the state of hypovitaminosis, various functional disorders of the nervous system can occur: paresthesia, neurotic states, poor sleep, anxiety, weakness, weight loss.
- Allergic reactions of the oral mucosa may occur.

Treatment

- Vitamin B1 has a beneficial effect in the treatment of neuritis and trigeminal neuralgia, herpes zoster, paresthesia of the oral mucosa, drug-induced and fungal stomatitis.
- For medicinal purposes, Vitamin B1 is prescribed orally (after meals) or parenterally. For patients with gastrointestinal diseases, it is recommended to administer Vitamin B1 intramuscularly or intravenously.
- Thiamine chloride preparations are used for treatment. Therapeutic daily doses for adults are prescribed at 0.012 g 1 to 3 (up to 5) times a day. The course of treatment is usually 30 days.
- In cases of malabsorption in the intestines, Thiamine Chloride (0.025-0.05 g) or Thiamine Bromide (0.03-0.06 g) is injected intramuscularly.
- Cocarboxylase (the coenzyme of Vitamin B1) is also used. The course of treatment consists of 10-15 injections.

Note: Simultaneous parenteral administration of Vitamin B1 solution with Pyridoxine (B6) and Cyanocobalamin (B12) is not recommended. Cyanocobalamin enhances the allergenic effect of thiamine, and pyridoxine makes it difficult for Vitamin B1 to transform into the active (phosphorylated) form. Vitamin B1 and Nicotinic Acid, which destroys it, should also not be mixed in the same syringe.

Riboflavin Deficiency (Vitamin B2). Riboflavin is part of the prosthetic groups of many enzymes (flavins) that participate in oxidation-reduction processes. Riboflavin is involved in carbohydrate, lipid, and protein metabolism.

Clinical Manifestations. Riboflavin deficiency manifests as a triad: dermatitis, cheilitis, glossitis.

- Dermatitis develops in the area of the nasolabial folds, wings of the nose, and eyelids in the form of redness, scaling, cracks, and scabs at the corners of the mouth (angular cheilitis), which, after removal, leave an erosion.
- Scaling is detected on the red border of the lips. Furthermore, numerous painful, sometimes bleeding, cracks appear on it.
- Changes on the tongue begin with hyperemia, then its papillae gradually atrophy until they completely disappear. As a result, the dorsum of the tongue becomes bright red, smooth, shiny, and dry (sometimes called a "magenta" tongue).
- Changes in the lips and mouth are accompanied by burning and pain, especially when speaking and eating.

For diagnostic clarification, biochemical blood and urine tests are used. Riboflavin has a selective effect on the skin of the face and the mucous membranes of the mouth and eyes. In riboflavin deficiency, these areas are affected first. Therefore, Vitamin B2 should be included in the complex treatment of cheilitis, glossitis, and angular stomatitis.

- Riboflavin is prescribed orally at 0.01 g 3 times a day for 1 to 1.5 months.

Nicotinic Acid Deficiency (Vitamin PP). Nicotinic acid deficiency in humans leads to Pellagra. Nicotinic acid is a specific anti-pellagic agent, which is why it is called "Vitamin PP" (Pellagra Preventive). Nicotinic acid participates in the regulation of metabolism, primarily carbohydrate metabolism. Hypo- and avitaminosis PP mainly develop with its insufficient intake from food. At the same time, the deficiency of Tryptophan in food, as well as alcoholism, plays a certain role.

Clinical Manifestations. With hypovitaminosis PP:

- Appetite worsens, nausea and diarrhea appear.

- The tongue becomes enlarged, edematous, and its papillae are initially hypertrophied. The dorsum of the tongue is usually covered with a thick layer of plaque separated by tubercles. Then the plaque gradually exfoliates, and the tongue in these places becomes bright red, shiny, and sharply painful.
- Hyperemia of the oral mucosa and erythema on the skin develop.
- At the same time, patients complain of weakness, apathy, rapid fatigue, and memory impairment.

With Pellagra, typical changes are observed in the form of a triad:

1. Dementia (Disorders of the nervous system, expressed by mental illness in the form of depression).
 2. Diarrhea (Characterized by persistent, debilitating diarrhea).
 3. Dermatitis (The skin becomes pigmented, especially in areas exposed to sunlight (back of the hands, neck, face), slightly hyperemic, dry, scaly, and thin).
- The papillae of the tongue completely atrophy, the tongue becomes lacquered, shiny, bright red, and painful.

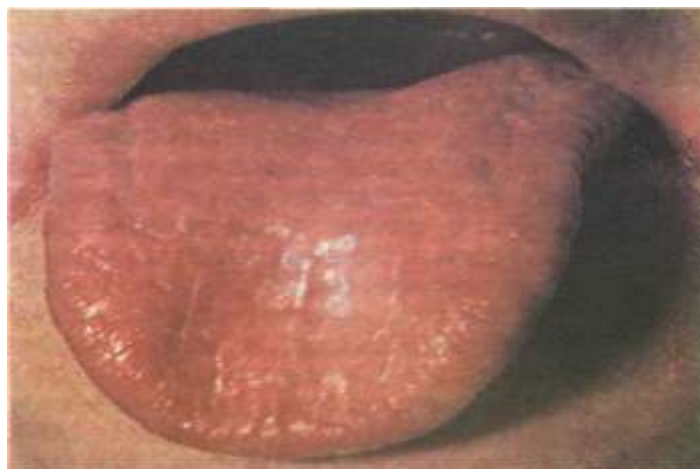


Figure 58. Albatta, mana gipovitaminozlar (B2, C, B6, B12, E) va ayrim tizimli kasalliklar (asosan OIT) fonida og‘iz bo‘shlig‘idagi o‘zgarishlarga oid ma'lumotlarning inglizcha tarjimasini.

English Translation: Hypovitaminosis and Changes in the Oral Mucosa in Systemic Diseases Riboflavin Deficiency (Vitamin B2)The tongue is hyperemic, smooth, shiny, with atrophy of the papillae.

The oral mucosa (OM) is edematous, hyperemic, hypertrophied, and vesicles may appear on it, which turn into painful erosions. Patients note dry mouth and taste disturbances.

It is now established that insufficient intake of Vitamins B1, B2, and B6 also plays a role in the onset of pellagra. Vitamin PP deficiency is confirmed by the results of laboratory tests. Treatment for Pellagra (Vitamin PP Deficiency)

Treatment consists of administering Nicotinic Acid orally (after meals) at 0.1 g 2-3 times a day or parenterally for 15-20 days. Thiamine (B1), Riboflavin (B2), and Pyridoxine (B6), as well as general tonic substances, are administered concurrently.

Ascorbic Acid Deficiency (Vitamin C). The absence or low intake of ascorbic acid in food leads to the development of Scurvy (also known as *scorbutus* or Möller-Barlow disease). While avitaminosis is rare in Russia, cases of hypovitaminosis are observed.⁰

Vitamin C deficiency disrupts the oxidative-reductive enzyme system, causes significant changes in carbohydrate and protein metabolism, reduces the body's activity (decreased activity of phagocytic cells and antibody formation), and impairs collagen synthesis and maturation of connective tissue, which increases the permeability of blood vessels and tissues. The combination of Ascorbic Acid deficiency and Rutin (Vitamin P) deficiency particularly sharply increases permeability.

Clinical Manifestations

Changes resulting from Vitamin C deficiency consist of the manifestation of hemorrhagic syndrome and complications arising from secondary infection.

- Hypovitaminosis C, and especially avitaminosis, is accompanied by restlessness, weakness, fatigue, decreased appetite, weight loss, and pain in the calf muscles.
- The skin darkens due to melanin accumulation, becoming dry and easily fractured.
- Hemorrhagic syndrome appears due to a sharp increase in the permeability of capillaries and small vessels: petechial hemorrhages are observed in various areas of the oral mucosa, subcutaneous tissue, and joints.

- Oral Mucosa Changes: Vitamin C deficiency always manifests with changes in the oral cavity. Extensive hemorrhages appear in the damaged areas of the mucosa, along with petechiae.
- At the beginning of the disease, swelling of the gingival papillae occurs; they turn blue-red, gradually increase in size, covering the crowns of the teeth, and bleed profusely.
- Mobility and loss of teeth are noted.
- Subsequently, an ulcerative-necrotic process develops in the areas of bleeding, which is associated with the addition of secondary infection. Necrosis is often localized on the tonsils and gingival margins.

Differential Diagnosis

- In the initial stage, the enlargement of the gingival papillae in hypovitaminosis C must be differentiated from hypertrophic gingivitis.
- If punctate hemorrhages are present on the oral mucosa, blood diseases that may have a similar appearance should be excluded.
- Ulcerative-necrotic changes are differentiated from Vincent's ulcerative-necrotic stomatitis and changes in the oral mucosa in leukemia.
- The history, the presence of other signs of scurvy, and laboratory test results are crucial for diagnosis. Hypo- or avitaminosis is confirmed by detecting reduced levels of Ascorbic Acid in the blood.

Treatment

- Ascorbic Acid is prescribed at 0.1 g 4-5 times a day (after meals) or Sodium Ascorbate parenterally with 1-3 ml of a 5% solution.
- Concurrently, taking Rutin (50-100 mg 2-3 times a day) is recommended.
- In cases of pronounced bleeding, Calcium Chloride and Vitamin K are also prescribed.

Pyridoxine Deficiency (Vitamin B6). Pyridoxine is actively involved in the **metabolism of amino acids** (tryptophan, methionine, cysteine, serine, glutamic acid, etc.). Vitamin B₆ participates in lipid metabolism processes, particularly normalizing it in atherosclerosis.

Clinical Manifestations. Vitamin B6 deficiency impairs the function of the central and peripheral nervous systems. Lethargy, drowsiness, increased excitability, peripheral neuritis, neurodermatitis, stomatalgia, glossitis, glossalgia, eczematous cheilitis, and seborrheic dermatitis develop.

Treatment

- In dental practice, Pyridoxine is used in the complex treatment of eczematous and allergic cheilitis, herpes zoster
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and lupus erythematosus**.
- Pyridoxine is prescribed orally (after meals) in tablets of 0.002 g, 0.005 g, and 0.01g 1-2 times a day.
- Parenterally (subcutaneously, intramuscularly, or intravenously), adults are administered 0.05-0.1 g per day (in 1-2 doses). The course of treatment is 1 month.

Cyanocobalamin Deficiency (Vitamin B12)

Cyanocobalamin is essential for normal hematopoiesis and the maturation of red blood cells. It is a catalyst in the metabolic reaction of synthesizing nucleic acids and nucleoproteins, which are necessary for normal hematopoiesis. The effect of Vitamin B12 on hematopoiesis occurs in the presence of Folic Acid, which is converted into its active form under its influence and promotes normal hematopoiesis. Vitamin B12 and Folic Acid participate in hemoglobin synthesis.

Clinical Manifestations

Vitamin B12 deficiency leads to the development of Addison-Biermer pernicious megaloblastic anemia. In addition to impaired hematopoiesis, hypovitaminosis B12 is characterized by damage to peripheral nerves (neuritis, neuralgia, paresthesia of the tongue and oral mucosa).

- The initial signs of B12 deficiency anemia can be damage to the tongue: burning, tingling, paresthesia of the tongue, hyperemia of the tip and lateral surfaces of the tongue against the background of atrophy of the papillae—the "polished" tongue (Hunter's glossitis or Möller-Hunter glossitis).

Treatment.For the treatment of Addison-Biermer anemia, Vitamin B12 is injected intramuscularly or subcutaneously at 100-200 μ g every 2 days.Folic Acid is prescribed concurrently, which enhances its effect.Cyanocobalamin is also used in the treatment of candidiasis

Tocopherol Deficiency (Vitamin E) Tocopherol is a natural antioxidant. It regulates the process of lipid peroxidation and, consequently, the products of oxidation in organs and tissues. It participates in cellular metabolism processes, as well as in the synthesis of heme and proteins. Vitamin E normalizes the permeability and strength of the blood vessel wall, primarily the gingival capillaries. Vitamin E deficiency is associated with increased permeability and fragility of capillaries, degenerative changes in nerve cells, skeletal and heart muscles, and fatty infiltration of the liver.

In dentistry, Vitamin E is used to treat periodontitis. The preparation is used intramuscularly, as well as in the form of periodontal dressings.

In the treatment of oral mucosa diseases, Vitamin E is used to accelerate regeneration.

Tocopherol Acetate combined with Vitamin A is used for application to the area of erosions and ulcers.When injected intramuscularly, Tocopherol Acetate is prescribed at 50-100 mg per day for 20-40 days. For this purpose, 5%, 10%, and 30% solutions of Tocopherol Acetate in oil are used. The oral doses are the same. Capsules of 0.1 ml and 0.2 ml of 50% solution correspond to 0.05 g and 0.1 g of Tocopherol Acetate, respectively.

Changes in the Oral Mucosa in Some Systemic Diseases

The oral cavity is closely connected with various internal organs and systems. Modern clinical and experimental research methods allow establishing close anatomical and physiological connections with various body systems: the receptors

of the oral mucosa have extensive connections with the central nervous system, as well as the gastrointestinal tract (GIT) and the endocrine system. This has led researchers to conclude that the majority of oral mucosa diseases stem from impaired functions of certain systems, primarily the GIT.

The first signs of some systemic diseases often appear on the oral mucosa, leading patients to initially seek help from a dentist. Furthermore, changes in the oral mucosa often do not have specific manifestations characteristic of a particular somatic disease, which makes it difficult to correctly recognize them. Therefore, the involvement of other specialist doctors is necessary for a correct diagnosis in some cases. The oral mucosa is involved in the pathological process in various somatic diseases to varying degrees. This section focuses only on diseases accompanied by explicit changes in the oral mucosa, the knowledge of which is necessary for early diagnosis of the underlying disease and the selection of correct treatment tactics for its manifestations in the oral cavity.

Changes in the Oral Mucosa in Gastrointestinal Tract Diseases.

GIT diseases are often accompanied by changes in the oral cavity. This is primarily due to the morphofunctional unity of the oral mucosa and the entire digestive tract. Studies by many authors have shown that various changes are detected in the oral mucosa in chronic gastritis, gastric and duodenal ulcers, chronic colitis, and enterocolitis, the severity of which depends on the form, severity, and duration of the underlying disease.

Treatment of the underlying disease leads to a decrease or disappearance of pathological changes in the oral cavity.

A connection has been established between impaired acid-forming function of the stomach and pathological changes in the oral mucosa. Gastric secretion, as shown by S. P. Kolomiets' studies (1970), is closely related to salivary gland secretion. Exacerbation of peptic ulcer disease is accompanied by a decrease in the reactivity of the oral mucosa. V. V. Khazanova's studies indicate that recurrent aphthous stomatitis occurs against the background of intestinal dysbiosis. Many authors have described changes in patients with recurrent aphthous stomatitis that are similar to

lesions in the small intestine. Data obtained by V. E. Rudneva (1971) show that in patients with peptic ulcers, the concentration of histamine in the blood serum increases, while the activity of histaminase decreases, and the activity of hyaluronidase increases. One of the significant factors causing changes in the oral mucosa in GIT pathology is vitamin deficiency, especially of the B group.

Thus, based on data obtained by many authors, all the above-mentioned factors in the pathogenesis of GIT diseases (increased vascular and tissue permeability, hypovitaminosis, intestinal dysbiosis, immune system imbalance, etc.) are understood to be related to changes in the oral cavity.

Changes in the Tongue

The state of the tongue is the best studied in GIT diseases. The appearance of the tongue, according to many authors, can have significant diagnostic value and indicate existing pathology of the digestive tract.

Tongue Coating (*Oblazhennost*). A coated tongue is detected in many cases. The plaque mainly consists of keratinized epithelial cells, bacteria, fungi, and food debris. The increase in plaque is associated with various causes. The amount of plaque on the tongue increases mainly with a decrease in self-cleaning during chewing. The composition and consistency of the food consumed, as well as adherence to individual hygiene rules and other factors, are important. The amount of plaque on the tongue changes during the day: there is more in the morning than during the day and evening, as the amount of plaque decreases after eating.

Disruption of the process of normal (physiological) keratinization and shedding of the epithelium also determines the amount and density of the plaque. Thus, with atrophy of the filiform papillae of the tongue, the plaque may be minimal or absent. With hypertrophy of these papillae, there is a thick layer of plaque on the tongue surface, which is difficult to remove, consisting mainly of fused keratinized filiform papillae. Tongue coating is one of the characteristic signs of various diseases of the GIT. With the exacerbation of gastritis, peptic ulcers, enterocolitis, and colitis, the amount of plaque increases, covering the entire dorsum of the tongue, predominantly located in its posterior parts. Plaque formation, as a rule, is not

accompanied by subjective sensations in patients. Only when a thick, dense layer of plaque is present on the tongue may there be a slight feeling of discomfort and a slight decrease in taste sensitivity. The plaque on the tongue usually has a grayish-white color, but it can have various colors (yellow, brown). The color of the plaque is mainly caused by food additives, medications, or the exacerbation of GIT diseases: peptic ulcer, chronic hepatitis, cholecystitis (yellow, brown).

Tongue Edema

Edema (swelling) of the tongue is an important sign of GIT diseases. As a rule, tongue edema is detected by the doctor during examination, as it does not cause painful sensations, except in cases of significant swelling when the tongue is bitten during eating or talking. Edema is detected upon examination by clear tooth marks on its lateral surfaces, as well as an increase in its size. The MacClure-Aldrich test is used as an objective method for detecting edema. Blister tests can also be used to detect latent edema, allowing for the diagnosis of early (preclinical) changes. V. A. Epishev (1970) established a violation of the hydrophilicity of the oral mucosa in chronic gastritis. A decrease in the resorption time of the blister sample was found in anacid gastritis, and an increase in hyperacid gastritis. It should be borne in mind that tongue edema is a constant condition associated with chronic intestinal diseases (enterocolitis, colitis), in which impaired water metabolism is detected.

Changes in Tongue Papillae

Changes in the papillae of the tongue are often found in patients with GIT pathology. The pathogenesis of these changes is mainly due to trophic disorders, as well as disrupted vitamin balance due to insufficient absorption and synthesis of Vitamins B1, B2, B6. Depending on the development and color of the papillae, some authors distinguish hyperplastic and atrophic glossitis.

Hyperplastic glossitis is more often observed in patients with gastritis with increased acidity, with exacerbation of peptic ulcer disease. It is characterized by hypertrophy of the filiform papillae, the presence of dense plaque, and an increase in tongue size due to pronounced edema.

Atrophic glossitis occurs in patients with gastritis with secretory insufficiency, hepatitis, gastroenteritis, and colitis. In this form of glossitis, atrophy and smoothing of the tongue papillae, as well as the absence of filiform papillae, are observed. Sometimes the atrophy of the papillae is sharply pronounced. The tongue can be hyperemic (erythematous) or pale pink. In some cases, the tongue has a "lacquered" appearance with bright red spots and stripes, similar to Möller's glossitis. Atrophy of the tongue papillae can cause a burning sensation, pain, and itching when eating spicy or sour food.

Desquamation of the Tongue Epithelium

Desquamation of the tongue epithelium is often observed in GIT diseases and can be developed to varying degrees. Desquamation is more common in patients with chronic gastritis with secretory insufficiency, chronic colitis, and liver diseases.

It is characterized by the appearance of foci of desquamation of the epithelium of the filiform papillae on the dorsum of the tongue.

A combination of desquamative glossitis with atrophy and smoothing of the tongue papillae is often observed in patients with secretory insufficiency and infectious liver lesions. These changes, as a rule, do not cause painful sensations, and patients often do not suspect their existence, complaining only occasionally of burning and pain when consuming irritating food. It is characterized by the appearance of foci of desquamation during the exacerbation of chronic GIT diseases and their disappearance during remission.

Changes in the Oral Mucosa in Gastrointestinal Tract Diseases (Cont.)

Paresthesia of the tongue often accompanies diseases of the digestive system. Burning, tingling, and crawling sensations on the tongue are observed. These sensations often accompany desquamative glossitis, but they can also manifest without visible changes on the tongue.

Disturbance of Taste Sensitivity

Taste sensitivity of the tongue is determined by the functional mobility of its receptors. The number of functioning receptors is known to depend on age and the functional state of the digestive tract. Normally, the maximum activity of the taste

buds is observed on an empty stomach. After eating, their level of mobility decreases. The reaction of the taste receptors of the tongue occurs in response to afferent impulses coming from the receptors of the gastric mucosa. In peptic ulcer disease and stomach tumors, the reflex connection between the tongue and stomach receptors is disrupted due to impaired secretory and motor functions. This manifests as various disturbances in the functional mobility of the taste buds (increased activity and lack of demobilization after eating, etc.). Disturbance of taste sensitivity can also occur with changes in the papillary apparatus of the tongue (thickly coated tongue, atrophy, or desquamation phenomena), which is associated with damage to the taste buds.

Erosive and Ulcerative Lesions of the Oral Mucosa

There are numerous reports of the presence of erosions and ulcers on the oral mucosa in peptic ulcer disease, liver diseases, colitis, enterocolitis, and others. The connection of recurrent aphthous stomatitis with gastrointestinal pathology is known. Approximately 50% of patients with GIT diseases (chronic gastritis, gastric and duodenal ulcers) suffer from recurrent aphthous stomatitis of varying severity.

Color Changes and Inflammation of the Oral Mucosa

The color of the oral mucosa can change against the background of gastrointestinal pathology. During the exacerbation of peptic ulcer disease, enterocolitis, and colitis, catarrhal gingivitis, glossitis, or stomatitis often develops, the severity of which depends on the duration and frequency of exacerbations of the underlying disease. The oral mucosa in the affected area is characterized by hyperemia with the appearance of cyanosis due to the chronic course. In such cases, patients complain of burning in the mouth, changes in the color of the mucosa, and sometimes pain when consuming irritating food. Catarrhal gingivitis and stomatitis appear during the exacerbation of chronic colitis. During the period of remission of GIT diseases, the signs of catarrhal gingivitis or stomatitis ease or completely disappear. Note: It should be remembered that such changes in the oral mucosa can also occur in other diseases and conditions of the body (infectious, including fungal, allergic,

cardiovascular, and hypovitaminosis diseases). In this regard, a complete examination of the patient is necessary for successful diagnosis and selection of treatment methods.

Salivary Gland Disturbances

These can manifest as hyper- or hyposalivation. Studies by a number of authors have shown that patients with gastric and duodenal ulcers have morphological and functional changes in the minor salivary glands. In the initial stage (up to one year) of peptic ulcer disease, as well as during its exacerbation, an increase in salivation is observed, with subsequent development of hyposalivation. Patients begin to complain of dry mouth. Clinically, salivary disturbances are often combined with other changes in the oral cavity previously described as characteristic of gastrointestinal pathology.

Changes in the Oral Mucosa in Cardiovascular Diseases

Changes in the oral cavity in case of cardiovascular system disorders are associated with the degree of circulatory disturbance and damage to the vascular wall. Compensated forms of cardiovascular insufficiency are, as a rule, not accompanied by significant changes in the oral cavity. Edema and Cyanosis of the Oral Mucosa and Lips These are usually observed in decompensated forms of cardiovascular insufficiency: hypertension, rheumatic heart disease, or other diseases. Tooth marks appear on the lateral surfaces of the tongue and cheeks. This condition is often combined with cyanosis of the red border of the lips. Tongue edema can be significantly pronounced, causing it to enlarge and speech to become difficult. In myocardial infarction, tongue edema may be combined with a change in its color, the appearance of cracks, erosions, and ulcers. The development of these changes is determined by the severity of the underlying disease. In a large-focal infarction, the damage to the oral mucosa is more pronounced. Improvement in the patient's condition after general treatment leads to an improvement in the situation in the oral cavity—tongue edema decreases, and epithelialization of erosions and ulcers occurs. Despite the significant changes in the mucosa, most patients do not experience subjective sensations, but the doctor, if edema is detected, must establish

the cause of its occurrence. Differential diagnosis should be carried out with similar changes in the oral mucosa in gastrointestinal pathology, infectious, and other diseases. Bullous and Hemorrhagic Syndrome in the Oral Cavity

This is characterized by the appearance of dense vesicles of various sizes with transparent or hemorrhagic contents. The authors explain the mechanism of vesicles in patients with cardiovascular pathology by the rupture of small vessels of the oral mucosa due to increased permeability and fragility of the vascular wall. At the same time, the weakening of the connection between the epithelium and the connective tissue layer of the oral mucosa was undoubtedly associated with the destruction of the basement membrane. T. I. Lemetskaya found a decrease in the resistance of the oral mucosa capillaries to vacuum (Kulazhenko test) in patients with pulmonary vascular syndrome. The bullous and hemorrhagic syndrome is more common in women aged 40-75 years. The vesicles appear suddenly, often during meals. The appearance of the vesicles is usually associated with an increase in blood pressure, which patients often do not suspect. Localization: On the mucous membrane of the soft palate, lateral surfaces of the tongue, and cheeks. The vesicles can persist unchanged for several hours to several days. Sometimes they disappear without opening, but more often they open with the formation of erosions that epithelialize within 3-7 days, depending on the size. Smears from the surface of erosions reveal peripheral background elements, but no acantholytic cells are found. Nikolsky's sign is negative.

Differential Diagnosis

The bullous and hemorrhagic syndrome is differentiated from:

- Pemphigus
- Angiomas
- Erythema multiforme exudativum

Treatment. First of all, it is aimed at treating the background cardiovascular diseases, without which the recurrence of vesicles cannot be prevented. Drugs that normalize the state of the vascular wall (Ascorutin, Complamin) are prescribed. Local treatment consists of sanitation of the oral cavity, including rational prosthetic rehabilitation.

The oral mucosa is treated with antiseptics (solution of chlorhexidine, chloramine, etc.). Keratoplastic agents are used (Vitamins A and E, rosehip oil, sea buckthorn oil, Actovegin ointment, Solcoseryl, etc.).

In cases of rapid spontaneous epithelialization of erosions, special local treatment is sometimes not required 2-3 days after their appearance. **Ulcerative-Necrotic Lesions of the Oral Mucosa with the Formation of Trophic Ulcers**

Against the background of a deterioration in the patient's general condition (weakness, shortness of breath, edema of the extremities), pain appears in the oral cavity, and eating becomes difficult. Single, rarely multiple, ulcers appear on the oral mucosa. The appearance of trophic ulcers is provoked by the effect of sharp edges of teeth, poor-quality prostheses, or other traumatic factors. Ulcers often form on the lateral surfaces of the tongue, the mucous membrane of the cheeks, the floor of the mouth, the palate, etc. The edges of the ulcers are uneven, and the bottom is covered with a grayish-white necrotic plaque. If the necrotic tissues do not slough off for a long time, they acquire a dark color. A characteristic feature of a trophic ulcer is the absence of a pronounced inflammatory reaction in the surrounding tissues. The necrotic process can spread to adjacent areas of the face and nasopharynx. Breathing becomes difficult, and saliva becomes viscous. Cases of ulcerative-necrotic lesions of the oral mucosa as a result of circulatory disorders with necrosis and sequestration of the jaw bone tissue and the formation of defects in the cheek tissues are observed. The decay of necrotic tissues can lead to severe bleeding. The long-term existence of trophic ulcers can lead to their malignancy.

Diagnosis. Cytological examination of a scrape from the surface of a trophic ulcer shows a decrease in the number of epithelial cells, which is expressed by a decrease in cell volume; some cells show a lack of clear contours, and pyknosis. Neutrophils of varying degrees of disintegration are detected. A significant decrease in histiocytic cells is found, which characterizes the areactive nature of the inflammatory process.

Pathohistologically, extensive necrosis and chronic inflammatory process with proliferation of interstitial tissue are found in the area of trophic ulcers; sclerotic changes in blood vessels and damage to nerve fibers are observed.

Differential Diagnosis. A trophic ulcer is differentiated from

- Traumatic ulcer
- Cancerous ulcer
- Tuberculous ulcer
- Ulcerative-necrotic stomatitis of Vincent
- Necrotic lesions of the OM in blood diseases

Treatment. This is carried out in contact with the treating physician.

General treatment should be aimed at eliminating cardiovascular insufficiency. In some cases, it is advisable to carry out general treatment in a hospital setting.

Symptomatic therapy is carried out locally according to indications. First of all, all local traumatic factors are eliminated. Necrotic tissues are removed using proteolytic enzymes. The OM is treated with antiseptics and drugs that stimulate epithelialization (Vitamins A and E, sea buckthorn oil, rosehip oil, propolis, Sigeroll, etc.). A soft, high-calorie, vitamin-rich diet is prescribed.

Changes in the Oral Mucosa in Endocrine Diseases

The endocrine system is the regulator of metabolic processes in the human body. It ensures the consistency of the functions of individual organs and systems, and also controls the balance of a number of body functions (blood glucose and calcium levels, temperature, etc.). Changes in the oral mucosa and the red border of the lips are observed in some endocrine diseases. Clinical manifestations in the oral cavity can lead to the need to study certain functions of the endocrine system. For example,

the progressive development of periodontal diseases may be associated with diabetes mellitus or other diseases of the endocrine system. Changes in the oral cavity are often detected in diabetes mellitus, disorders of the gonads, thyroid and parathyroid glands, pituitary gland, and adrenal cortex. Therefore, the timely and correct assessment of the local manifestations of these diseases in the oral cavity by a dentist helps in their early diagnosis.

Diabetes Mellitus

This is characterized by a disruption of carbohydrate metabolism, against the background of which changes in the oral cavity occur. Their course depends on the severity and duration of diabetes. Oral manifestations are often the first signs of the disease. Xerostomia is a dryness in the oral cavity resulting from decreased salivation, the development of which varies among all patients. Dryness is often combined with increased thirst and appetite, burning and itching in the area of the tongue, lips, and gums; sometimes accompanied by itching and burning in the genitals or other parts of the body. On examination, the oral mucosa is dry, shiny, edematous, and hyperemic, especially on the tongue. Paresthesia may appear simultaneously.

Differential Diagnosis: Xerostomia in diabetes mellitus is differentiated from salivary disturbances in neurological disorders, Sjögren's syndrome, sialoadenitis and sialodochitis, and Mikulicz's disease. Catarrhal, Erosive, and Ulcerative Stomatitis, Glossitis. Inflammation of the oral mucosa or its individual parts in diabetes occurs as a result of decreased resistance due to disruption of redox processes, dysbiosis in the oral cavity, local immune imbalance, and other reasons. All this leads to a change in the reactivity of the oral mucosa to local stimuli, a decrease in resistance to infection; there is a tendency to inflammation, and the processes of healing erosions and ulcers are slowed down. Even a slight effect of a traumatic factor leads to inflammation of the oral mucosa and, in some cases, to the disruption of its integrity with the formation of cracks, erosions, and ulcers. Patients complain of pain that appears when eating hot, sharp, or dry food.

Erosions and ulcers are characterized by a prolonged course due to a decrease in the regenerative properties of the mucous membrane. Periodontal Tissue Involvement. Periodontal tissues are also frequently affected in diabetes mellitus. Initially, catarrhal gingivitis develops with pronounced edema and bleeding of the gums, which subsequently rapidly progresses to periodontitis, characterized by a progressive course with pronounced inflammatory and destructive changes in all periodontal tissues. The more severe the clinical picture of diabetes and the longer its duration, the more severe the damage to the periodontal tissues. Destructive changes in the periodontal tissue are especially characteristic of young patients with diabetes.

Fungal Stomatitis (Candidiasis), Mycotic Cheilitis

Candidiasis

Shutterstock is a very common form of oral disease in diabetes mellitus. Dysbiosis in the oral cavity and disruption of the acid-base balance with the accumulation of unoxidized metabolic products (lactic and pyruvic acids) in the tissues create favorable conditions for its development in diabetic patients. Candidiasis of the oral mucosa in diabetes is characterized by a persistent and stubborn course. Mycotic cheilitis is frequent, in which cracks covered with small grayish-white crusts form at the corners of the mouth (angular cheilitis).

Diagnosis of Candidiasis: For the diagnosis of candidiasis, the general signs of diabetes, laboratory data (increased glucose levels in the blood and urine), as well as the results of bacterioscopic examination of scrapes from the oral mucosa surface are important. Antifungal Treatment is carried out in consultation with the endocrinologist treating the diabetes mellitus. Paresthesia, Taste Disturbances

In diabetic patients, paresthesia sometimes appears, along with dryness and burning of the oral mucosa. In the decompensated form of diabetes, some patients show a decrease in sensitivity to sweet, salty, sour, and in some cases, bitter tastes. Sometimes disorders of the nervous system (neuritis, neuralgia of the branches of the trigeminal nerve) are observed.

Diagnosis: Anamnesis data, including lifestyle, the description of changes in the oral cavity, and the results of laboratory tests (blood and urine tests) are important in the diagnosis.

Treatment is carried out jointly with an endocrinologist. Along with the treatment of diabetes mellitus, sanitation of the oral cavity and symptomatic local therapy are performed.

Itsenko-Cushing's Disease

This disease develops as a result of hyperfunction of the adrenal cortex due to damage to the hypothalamic-pituitary system. The increased production of steroid hormones by the adrenal cortex leads to osteoporosis of bone tissue, including the jaws, and predisposes to periodontal diseases.

Some patients develop macrocheilitis (swelling of the lips).

Candidiasis

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is frequently observed.

Trophic ulcers may appear on the mucous membrane of the tongue, cheeks, and other parts of the oral cavity.

Treatment:

The underlying disease is managed by an endocrinologist.

Local treatment includes sanitation of the oral cavity.

Symptomatic therapy is carried out according to indications.

Addison's Disease

This disease is caused by adrenal insufficiency (hypofunction). One of the initial signs of the disease may be pigmentation of the skin and mucous membranes as a result of excessive deposition of melanin in the connective tissue and epithelial cells. Pigmentation on the skin appears in sun-exposed areas (face, back of hands). As the disease progresses, the skin acquires a light brown or bronze color. In the oral mucosa, small spots or streaks (from one to several square millimeters) of a gray-black, dark brown, or blue-gray color appear on the cheeks, along the edges of the

tongue, on the palate, and gums, without signs of inflammation. At the same time, patients often do not have subjective symptoms related to these changes.

The patient's general condition may deteriorate, characterized by asthenia, gastrointestinal disorders (nausea, vomiting, diarrhea, abdominal pain), headache, memory loss, and weight loss.

Differential Diagnosis: Addison's disease is differentiated from: Congenital pigmentation of the oral mucosa, which occurs in some people. Poisoning with heavy metal salts.

Treatment: The underlying disease is treated by an endocrinologist. Specific local treatment is not required.

Myxedema

This is a disease caused by severe hypothyroidism (underactive thyroid gland). It mainly affects women. The main symptom of myxedema is widespread mucous edema of the skin and subcutaneous tissue. The edema results from impaired lymph drainage and the accumulation of a specific mucous substance composed of glycosaminoglycans in organs and tissues, which retains water and forms the edema. In the advanced stage of the disease, the patient's face has a characteristic appearance: puffy, pale, the lips and nose are thickened, the upper eyelids are sharply swollen, almost covering the palpebral fissure. The tongue is significantly enlarged (macroglossia), and tooth marks are visible on its lateral surfaces. Anemia, swelling, and dryness of the oral mucosa are noted. Speech is reduced and slowed down due to edema of the laryngeal and vocal cord mucosa. The general condition of the patients deteriorates, characterized by drowsiness, sluggishness, decreased memory, and hearing loss. In children with hypothyroidism, enamel hypoplasia, hyposalivation, high intensity of carious lesions, and delayed eruption of teeth are observed; cretinism develops.



Figure 59. Oral Manifestations of Systemic Diseases (Cont.)
Endocrine Diseases (Cont.)

Diffuse Toxic Goiter (Thyrotoxicosis, Basedow's Disease). This develops due to hyperplasia and hyperfunction of the thyroid gland. Patients complain of burning in the oral mucosa, decreased taste sensitivity, and deterioration of general well-being (shortness of breath, fatigue, irritability, sweating, etc.). Upon examination of the oral cavity, the mucosa is pale and edematous. Desquamative glossitis is sometimes detected. Accelerated eruption of teeth and abnormal development of enamel are characteristic.

Treatment: General treatment is carried out by an endocrinologist. Local treatment is performed if indicated.

Juvenile Gingivitis

This occurs in adolescent boys and girls and with menstrual cycle disturbances. The changes in the oral mucosa are caused by the action of gonadotropin hormones of the pituitary gland. Local factors contribute to the development of juvenile gingivitis: poor oral hygiene, malocclusion, and others. The gum mucosa is edematous, hyperemic, and easily bleeds. Hypertrophy of the gingival papillae develops, mainly in the area of the anterior teeth of the lower jaw.

Treatment: Systematic hygienic procedures, removal of dental plaque/calculus, and sanitation of the oral cavity are necessary. Orthodontic treatment is carried out according to indications.

Gingivitis of Pregnant Women (Pregnancy Gingivitis)

This develops in connection with pregnancy or its course worsens during pregnancy. It manifests as hypertrophy of the interdental papillae and sometimes the marginal gum. "False" pockets are formed, and bleeding is observed. The severity of the course can vary. Sometimes the hypertrophied papillae protrude up to the incisal edges of the teeth, which causes pain and bleeding during eating.

The symptoms of gingivitis existing before pregnancy are aggravated in most cases. After childbirth, the gums often return to their previous state (normal), but sometimes (with poor oral hygiene, dental calculus, poor-quality prosthetics, malocclusion, and other local traumatic factors) gum hypertrophy persists.

Treatment: Gingivitis of pregnant women primarily requires careful systematic oral care and the use of anti-inflammatory toothpastes ("Lesnaya," "Romashka," "Zodiac," etc.). If hypertrophic gingivitis persists after childbirth, its conventional treatment is carried out according to the generally accepted method.

Changes in the Oral Mucosa in Diffuse Connective Tissue Diseases

This includes a group of diseases characterized by diffuse inflammatory lesions, fibrinoid degeneration, and sclerosis of the connective tissue. The term "collagenoses," previously used for this group of diseases, is not used in the modern international classification of diseases because it does not reflect the essence of their pathogenesis (it was found that not only collagen but also other components of the connective tissue are damaged). The group of connective tissue diseases includes Systemic Lupus Erythematosus, Systemic Scleroderma, Dermatomyositis, Sjögren's Disease, and Rheumatoid Arthritis. Their main clinical feature is polysyndromic involvement, meaning changes occur in almost all organs and systems (skin, mucous membranes, muscles, joints, internal organs) resulting from a single pathogenesis—diffuse damage to the connective tissue. Changes in the oral mucosa in connective tissue diseases, as in other diseases of internal organs, are a local manifestation of the general pathology.

Desquamative Glossitis

Desquamative glossitis is a very common lesion of the tongue mucosa in patients with connective tissue diseases (Systemic Lupus Erythematosus, Systemic Scleroderma, Rheumatoid Arthritis). Xerostomia occurs to varying degrees in different connective tissue diseases. Saliva reduction is most pronounced in Sjögren's syndrome. In a number of patients, it reaches a significant degree, which reduces the resistance of the oral mucosa to traumatic and infectious factors and also leads to difficulties in chewing food and speaking for the patients. Atrophic Stomatitis, Glossitis, Gingivitis. Lesions of the oral mucosa are a general symptom of Scleroderma. In this disease, the oral mucosa is pale, atrophic, and thin, making its small blood vessels clearly visible. Telangiectasia (small vessel dilation) often appears. The red border of the lips also becomes atrophic and thin.

There is pronounced atrophy of the tongue papillae, giving it a varnished appearance. If the muscles of the tongue are affected, its volume may decrease. The gum mucosa is compressed, anemic, and atrophy of the marginal gingiva is frequently noted. Due to the development of atrophic processes, the resistance of the oral mucosa decreases. It is easily injured, and erosions or ulcers appear that do not epithelialize for a long time.

Generalized Skin Lesions in Scleroderma

Generalized skin lesions are characteristic of Scleroderma. Their nature is determined by the clinical form of Scleroderma. Plaque-form lesions on the skin resemble bluish-pink spots or streaks slightly elevated above the skin surface. Their central part gradually scleroses, acquiring an ivory color, while a purple rim remains at the edges, indicating the further progression of the process. In the superficial form of Scleroderma, the skin lesions resemble several white spots whose resolution leaves behind foci of atrophy. The skin is cold to the touch. The skin on the face is also thin and anemic, with multiple telangiectasias. Sometimes there is pain in the temporomandibular joint, which may result in limited mouth opening. Lesions of the skin and oral mucosa pass through the stages of edema, induration (hardening), and atrophy. Lesions are predominantly localized on the face (mask-like appearance) and

hands (sclerodactyly). The skin acquires a yellowish tint, thickens, and telangiectasia appears.

Treatment: Patients with connective tissue diseases are treated by rheumatologists, internists, and others. Dental care primarily consists of thorough sanitation of the oral cavity. Given the reduced resistance of the oral mucosa to microflora, it is very important to teach patients rational oral hygiene. In some cases, due to limited mouth opening, performing a number of dental manipulations may have certain specific features. The ongoing sclerotic processes in the oral mucosa must also be taken into account when administering anesthetics or other medications in these diseases. Considering the pathogenesis of connective tissue diseases, patients should be examined to identify and eliminate foci of odontogenic infection.

Changes in the Oral Mucosa in Diseases of Hematopoietic Organs

Diseases of the blood and hematopoietic organs are often accompanied by changes in the oral mucosa, which in some cases precede the clinical signs of the main disease. This forces patients to seek dental care first. The dentist is often the first to encounter such patients, so they must be well aware of the manifestations of hematopoietic system pathology in the oral cavity. This allows for the timely diagnosis and treatment of the disease. Furthermore, not knowing about a disease of the hematopoietic organs leads to incorrect diagnosis and, consequently, inappropriate treatment, which can significantly worsen the patient's condition.

The dentist must pay special attention to the psychological and deontological aspects when communicating with a hematological patient for the first time. It is important to be able to carefully and confidently explain to the patient the need for certain special examination methods or consultation with a hematologist in connection with the specificity of the oral mucosa lesions. It is very important not to frighten or cause psychological trauma to the patient during communication, as the fear of a malignant tumor (acute and chronic leukemia) is a natural reaction for most people.

The treatment of patients with diseases of the hematopoietic organs involves the use of special means and methods. As a rule, it is carried out in specialized hematology institutions. The volume of therapeutic manipulations by the dentist is determined by

the severity and stage of the clinical course of the underlying disease and is carried out in some cases by the dentist or in a specialized hematology department by the dentist. Acute Leukemia (Acute Leukaemia) This is characterized by an increase in the number of blast, "young" cells in the bone marrow, liver, lymph nodes, and other organs and tissues. Based on the morphological and cytochemical characteristics of the blast cells, several forms of acute leukemia are distinguished: myeloblastic, lymphoblastic, plasmoblastic, and others. Oral mucosa lesions are observed in 90.9% of patients with acute leukemia. This occurs mainly in young patients (under 30 years old). General symptoms (general weakness, malaise, fatigue, pallor of the skin) play an important role in the diagnosis of acute leukemia. Body temperature can be high with large fluctuations, but sometimes it can be subfebrile. The patient gives the impression of being seriously ill. Due to the sharp decrease in the body's immunity, candidiasis sometimes develops in patients with leukemia, and herpetic infection manifests itself in the oral cavity. Blast cells are present in the blood, and the number of platelets and red blood cells is often reduced. Symptoms in the oral cavity are of great diagnostic value in acute leukemia. They are very characteristic and easy to detect. Examination reveals pallor of the skin, pastiness (edema), easy vulnerability and bleeding of the oral mucosa, hemorrhages (bleeding spots) on the gums, cheeks (especially along the occlusion line), palate, and tongue are noted. Hematomas and hemorrhages can be observed on both the mucosa and the skin.

Hemorrhagic Syndrome

Gum bleeding is often the first clinical sign of acute leukemia. The gums become loose, bleed, and ulcerate. Bleeding occurs with the slightest palpation and sometimes spontaneously. Bleeding can occur not only from the gums but also from the tongue, cheeks along the occlusion line, and other parts of the oral mucosa. Sometimes extensive hemorrhages and hematomas are found on the oral mucosa.

The clinical manifestations of the hemorrhagic syndrome can be varied: from small pinpoint hemorrhages on the mucous membrane of the mouth and skin to extensive hematomas and massive bleeding.

Hemorrhagic syndrome is detected in 50-60% of patients and is based on the development of thrombocytopenia, impaired normal hematopoiesis as a result of leukemic hyperplasia, and bone marrow infiltration.

Differential Diagnosis:

The hemorrhagic syndrome in acute leukemia should be differentiated from: Hypovitaminosis C. Traumatic injuries (especially on the cheek mucosa along the occlusion line). The final diagnosis is determined based on the clinical picture of the disease, complete blood count, and bone marrow aspirate examination.



Figure 60. Acute Leukemia. Hyperplastic and Hemorrhagic Ulcerative Necrotic Gingivitis

Gingival hyperplasia is a common symptom of acute leukemia, especially in its severe course, and is highly valued by hematologists. Hyperplasia and infiltration of the gums by leukemic cells can be so significant that the crowns of the teeth are almost completely covered by loose, bleeding, sometimes ulcerated gums, which impedes the patient's eating and speaking. Characteristically, at the onset of the disease, hyperplasia develops more clearly on the inner (palatal or lingual) surface than on the buccal surface. This clinical symptom sometimes helps differentiate leukemic gingival hyperplasia from banal hypertrophic gingivitis.

Histological data suggest that gingival hyperplasia results from the infiltration of the connective tissue layer of the mucosa by myeloid cells, which leads to trophic disturbances, followed by tissue necrosis and ulceration.

Ulcerative Necrotic Lesions

Ulcerative necrotic lesions of the oral mucosa often develop in acute leukemia.

It is characteristic that the tips of the gingival papillae become necrotic. The gum in the center of the necrosis is bluish, while the entire mucosa is pale.

Necrosis spreads rapidly, and soon an ulcer appears around the tooth, covered with a dirty-gray plaque. Necrosis is often found on the tonsils, in the retromolar area, and in other parts of the oral cavity. A specific feature of the necrotic process in acute leukemia is its tendency to spread to adjacent areas of the mucosa. As a result, large ulcers with irregular contours covered with a gray necrotic plaque may appear. Reactive changes around the ulcer are absent or poorly expressed. In the presence of necrotic changes in the oral cavity, patients complain of severe pain during eating, difficulty swallowing, fetid odor from the mouth, general weakness, dizziness, and headache. At the beginning of the development of ulcerative necrotic changes, hypersalivation may appear, followed by a decrease in the amount of saliva, which is confirmed by dystrophic changes in the salivary glands.

The causes of necrotic processes in the oral mucosa in acute leukemia are not fully understood. It is believed that necrosis may arise as a result of the spread of leukemic infiltrates, in areas of extensive hemorrhages, as well as due to neurotrophic disturbances in the tissues and the violation of the body's defense forces. An important factor contributing to the appearance of necrosis is the effect of external factors, especially microorganisms. In acute leukemia, the lymph nodes are slightly enlarged (up to 0.5-1 cm), they are soft and painless. In some cases, acute leukemia may manifest with the formation of specific leukemic infiltrates on the skin and oral mucosa. The formation of infiltrates in the gums causes sharp deformation of the gingival margin. This condition is often misdiagnosed as hypertrophic gingivitis. Leukemic infiltrates can ulcerate, which often leads to bleeding.

Differential Diagnosis:

The phenomena of acute leukemia in the mucous membrane must be differentiated from:

Hypertrophic gingivitis

Vincent's ulcerative necrotic stomatitis

Hypovitaminosis

Poisoning with heavy metal salts

Clinical laboratory results are crucial in the diagnosis of leukemia.

Treatment:

The task of the dentist is the correct and timely detection of acute leukemia based on clinical signs in the oral cavity and other manifestations, and referral for peripheral blood analysis.

Treatment of acute leukemia is carried out in a specialized hematology department. Local treatment is performed in consultation with a hematologist. Oral hygiene is very important.

Dental treatment, extractions, and treatment of dental calculus are carried out in a hospital setting under the supervision of a hematologist.

In the presence of ulcerative necrotic lesions of the oral mucosa, it is treated with anesthetic antiseptic solutions (hydrogen peroxide, chloramine, ethonium, romazulan, furacilin, etc.).

Proteolytic enzymes and agents that accelerate epithelialization (rosehip oil, sea buckthorn oil, oily solution of Vitamin A, propolis preparations, etc.) are used.

If fungal or herpetic lesions of the oral mucosa are detected

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traditional antiviral and antifungal therapy is administered.

Chronic Leukemia

Based on the nature of the lesion of the hematopoietic organs, chronic leukemia is divided into myeloproliferative (myeloid leukemia) and lymphoproliferative (lymphocytic leukemia) forms.

Chronic Myeloid Leukemia

It passes through two stages: benign, which lasts for several years, and malignant (terminal), which lasts for 3-6 months. The first stage often begins without clear clinical symptoms, manifesting later with neutrophilic leukocytosis with a shift to promyelocytes and single blast cells.

In the second stage, as in the first, there are many blast cell forms (myeloblasts, hemocytoblasts) in the blood during blast cell breakdown; a rapid increase in the number of leukocytes in the blood is observed. The progression of leukemia leads to the development of anemia and thrombocytopenia accompanied by bleeding.

Chronic myeloid leukemia proceeds asymptotically for a long time and can be diagnosed accidentally during clinical laboratory examination. Laboratory examination is characterized by high leukocytosis ($50-60 \times 10^9/L$). In blood smears, various intermediate forms of the myeloid series are found: myeloblasts, promyelocytes, myelocytes. Hemorrhagic manifestations are characteristic of chronic myeloid leukemia, although their intensity is much lower than in acute leukemia. Bleeding in the gums does not occur spontaneously but is observed when injured. The oral mucosa is pale, the gums are swollen, bluish, bleed upon palpation, and inflammation is absent. The absence of inflammation and pronounced bleeding are characteristic signs of leukemia. Erosive ulcerative lesions of the oral mucosa are observed in $1/3$ of patients with myeloid leukemia (V. M. Uvarov, 1975). The appearance of severe necrotic lesions is considered an unfavorable sign indicating the exacerbation of the process. Candidiasis often develops in the severe stage of the disease.

Chronic Lymphocytic Leukemia

It usually develops in middle-aged and elderly people, characterized by a long-term benign course and a **significant increase in the number of leukocytes** in the blood (among which lymphocytes dominate). The number of leukocytes can reach $100 \times 10^9/L$. The initial period of chronic lymphocytic leukemia is often unnoticed by the patient. The only complaint may be **enlarged lymph nodes** (upon palpation they are not very dense, mobile, painless).

In chronic lymphocytic leukemia, leukemic lymphocytic infiltrates or tumor-like nodules form in the oral cavity. They are often located in the gums, cheeks, tongue, palatal arch, and tonsil mucosa. The formed infiltrates (nodules) have a soft (doughy) consistency, bluish color, are mobile, rise above the mucous membrane, and

ulceration is rare. Bleeding occurs with minor trauma. Leukemic lymphocytic infiltrates are also located in the bone marrow, lymph nodes, spleen, and liver.

Treatment: General and local treatment is carried out as in acute leukemia.

Agranulocytosis

A syndrome characterized by a decrease or complete absence of granulocytes (granular leukocytes) in the peripheral blood. According to the mechanism of occurrence, myelotoxic and immune agranulocytosis are distinguished. Myelotoxic agranulocytosis is caused by the disruption of granulocyte formation in the bone marrow under the influence of ionizing radiation, cytotoxic drugs, benzene vapors, and others. Immune agranulocytosis occurs when granulocytes in the blood are destroyed by immune complexes formed in people with increased sensitivity to certain drugs (amidopyrine, butadion, analgin, phenacetin, sulfonamides, some antibiotics, etc.). Agranulocytosis often begins with the manifestation of changes in the oral cavity, forcing patients to consult a dentist at the onset of the disease. Against the background of deterioration of the patient's general condition (fever, lethargy, headache, pain when swallowing, fetid breath), rapidly spreading ulcerative necrotic processes develop in the oral cavity. The destruction of the oral mucosa proceeds without a pronounced leukocyte reaction of the surrounding tissues and with little pain. The fetid ulcers bleed easily.

Sometimes the bone tissue of the jaw is involved in the ulcerative necrotic process. The gums, lips, tongue, cheeks, and tonsils are most often affected. When the tonsils are affected, pain occurs when swallowing. Ulcers are observed along the digestive tract in some patients. Lymph nodes are enlarged. The number of lymphocytes in the blood is significantly reduced. The number of granulocytes in the leukocyte formula is sharply reduced or they are completely absent; only lymphocytes and monocytes are found.

Diagnosis: Diagnosis is based on anamnesis, clinical presentation, and the results of peripheral blood and bone marrow aspirate examination.

Differential Diagnosis:

Agranulocytosis should be differentiated from Vincent's ulcerative necrotic stomatitis and other blood diseases.

Treatment:

General treatment is carried out under the supervision of an internist and hematologist, primarily including blood transfusions, elimination of the etiological factor, etc. Local treatment includes the use of anesthetics, the application of proteolytic enzymes to the affected areas of the oral mucosa, antiseptic treatment, and the use of epithelialization-accelerating drugs.

Thrombocytopenic Purpura

A syndrome characterized by increased bleeding due to a decrease in the number of platelets (thrombocytes) in the blood. Thrombocytopenic purpura can be an independent disease—Werlhof's disease—as well as developing as a result of other diseases (malignant neoplasms, radiation sickness, hepatitis, infectious diseases, drug intoxication, etc.)—symptomatic form. Idiopathic thrombocytopenic purpura or Werlhof's disease can occur in acute or chronic form. The main clinical symptom of the disease is hemorrhage (bleeding) on the skin and mucous membranes, as well as bleeding from the nose, gums, etc., which occurs spontaneously or under the influence of minor trauma. Hemorrhages are observed in the serous membranes, retina, and other parts of the eye. Hemorrhages in the heart and brain are life-threatening. Patients have a mild increase in body temperature and pronounced thrombocytopenia, as a result of which blood clot retraction is impaired and the bleeding time is prolonged up to 10 minutes or more (normally 3-4 minutes). Iron deficiency anemia can develop as a result of large blood loss.

Diagnosis: The diagnosis of Thrombocytopenic Purpura is based on anamnesis data (identifying contact with various chemicals, uncontrolled administration of analgesics, sleeping pills, etc.), clinical presentation, and blood analysis.

Differential Diagnosis: The disease is differentiated from:

Hemophilia

Scurvy (Hypovitaminosis C)

Hemorrhagic vasculitis

Treatment:

General treatment is carried out by a hematologist, as a rule, in a hospital setting. Dental interventions should be performed **very cautiously** after consultation with a hematologist.

Polycythemia (Erythremia or Vaquez's Disease)

A chronic disease of the hematopoietic system characterized by a persistent increase in the number of red blood cells and hemoglobin per unit of blood and an increase in circulating blood volume. The disease is associated with increased production of erythrocytes, leukocytes, and platelets in the bone marrow.

It develops slowly and has a long chronic course. It is more common in men aged 40-60 years. The color of the skin and oral mucosa changes. They acquire a dark cherry color with a bluish tint, which is associated with an increase in reduced hemoglobin in the capillaries. Gum bleeding is often observed. Bleeding may occur from the stomach, intestines, and uterus. The color of the lips and cheeks is distinctly dark cherry with a bluish tint. Kuperman's symptom is observed in the oral cavity: pronounced cyanosis of the soft palate mucosa and pallor of the hard palate. Many patients experience severe skin itching and paresthesia (abnormal sensations) of the oral cavity, which many authors believe is associated with increased blood filling of blood vessels and neurovascular disorders. The peripheral blood picture is characterized by an increase in the number of erythrocytes, hemoglobin and a decrease in ESR.

Treatment: It is carried out by a **hematologist**.

Specific local treatment is not indicated.

Stomatodynia (Glossodynia)

Stomatodynia (synonyms: glossalgia, glossodynia, paresthesia of the tongue) is a chronic painful condition characterized by persistent orofacial pain, usually not accompanied by lesions of the oral and tongue mucosa. The first scientific description of stomatodynia was given by Vaisse in 1838. However, the etiology, pathogenesis, and method of treatment of stomatodynia are still not fully understood. Due to the ambiguity of the mechanism of disease development, there is

no single concept for stomatodynia. Many terms have been proposed: Burning Tongue Syndrome, Glossalgia or Oral Dysesthesia, Glossodynia, Vegetosis of the Tongue, Sensory Neurosis of the Tongue, Glossoperosis. R. M. Basker, D. M. Main (1991) propose a new term for this suffering: "Burning Mouth Syndrome" (BMS), which more accurately reflects the leading complaint of the patients.

Etiology and Pathogenesis. Some authors suggest that increased monomers in the acrylic bases of dentures are the cause of stomatodynia, but not all studies confirm an allergic reaction to denture acrylics as a significant cause. Mechanical irritation from dentures and parafunctional habits may be more likely causes. Although it was previously believed that galvanic currents play a major role in the development of stomatodynia, recent research indicates this is not the case. Patients with stomatodynia show increased sensitivity to mercury, nickel, cobalt, gold, palladium, copper, zinc, and food additives. Hypersensitivity reactions to mercury are the most common, however, replacing amalgam fillings relieves the burning sensation in the oral cavity for less than half of the patients. The onset of stomatodynia can be observed after the use of enalapril, captopril, and lisinopril (Angiotensin-Converting Enzyme Inhibitors, ACE-I). Many chemical agents and drugs can cause pathological changes localized in the orofacial area. There is a prevalence of xerostomia (dry mouth) in approximately 20% of this pathology. Complaints of dry mouth may not always indicate hypofunction of the salivary glands. Attempts have been made to establish a link between stomatodynia and salivary gland dysfunction. There is evidence of reduced salivary gland function in stomatodynia.

Given that most patients with stomatodynia are post-menopausal women, many authors suggest that dry mouth may be a consequence of decreased functional activity of the salivary glands during menopause. However, most researchers have shown no significant decrease in salivary gland function during menopause.

The cause of xerostomia may be coexisting somatic pathology and the use of certain drugs for its treatment. In histological examination of the salivary glands, some authors report morphological changes, while others refute this data.

Clinical Picture. The pain in patients with stomatodynia is often difficult to localize and can be classified as dysesthesia. Pain can be superficial or deep, mainly on the tip and edges of the tongue, spreading to the oral floor, pharynx, and lips, and can be combined with a burning sensation in the oral cavity, dry mouth, thirst, taste disturbances, anxiety, irritability, and a decreased desire for social interaction.

The degree of pain in stomatodynia, although qualitatively different, is similar in intensity to toothache. In the case of dysesthesia and paresthesia, the burning sensation appears in the morning, increases during the day, reaches a maximum in the evening, and decreases or completely disappears while eating. A clear diurnal rhythm of pain is visible. The condition recurs for several months with periods of exacerbation. This condition sometimes appears after affective trauma, the death or cancer of a loved one, or as a result of personal and professional difficulties. Women often suffer from stomatodynia, mainly during menopause. They feel irritation in the area of the tongue and oral mucosa. Increased excitability of the tongue is added. Patients examine their tongue for a long time, trying to identify changes in it. All these symptoms are combined with short-term general excitement. Patients fear the development of cancer. Many patients with stomatodynia complain of taste disturbance — dysgeusia. Dysgeusia is detected in 68% of patients with stomatodynia, especially when consuming bitter food.

The clinical symptoms of stomatodynia disappear under the influence of hot and bitter food. This effect is associated with capsaicin present in bitter foods. Capsaicin (a preparation of capsicum for external use) shows a good effect in the treatment of orofacial pain. A correlation has been established between the level of beta-endorphin and the severity of stomatodynia in patients. Stomatodynia should be considered among other pathological manifestations of chronic stress. Many authors point to the frequent combination of stomatodynia with diseases of the gastrointestinal tract, cardiovascular pathology, osteochondrosis, endocrine and autoimmune pathology, and nasopharyngeal diseases. Others do not find a link between stomatodynia and these pathological conditions. It is assumed that hormonal changes in menopause may cause the development of oral mucosal discomfort. The

most characteristic features of stomatodynia are sleep disturbances, pronounced neurovegetative reactivity, and the presence of depression with a somatic reaction to stress. However, masked depression is also detected. Although many studies have suggested a link between depression and stomatodynia, not all definitively support this conclusion. Nerve damage or dysfunction resulting from oral or facial injuries, as well as after medical interventions, may be the source of pain in stomatodynia. Chronic pain may be associated with changes in the sympathetic nervous system, as well as impaired blood flow. Recent studies have shown that damage or inflammation of peripheral tissues or nerves can induce neuroplastic changes in central nociceptive systems, which plays an important role in stomatodynia. Given the likelihood of such changes, treatment methods for stomatodynia associated with damage to facial tissues and nerves (tooth extraction, etc.) should be avoided, as they are usually ineffective and can enhance the development of neuroplastic changes and symptoms.

Differential Diagnosis. Neuralgia and neuropathy of the lingual nerve have a clinical presentation similar to stomatodynia, in which sensory disturbances in the form of pain or hypesthesia (reduced sensation) are more clearly expressed. However, the main difference between stomatodynia and these conditions is the disappearance of all painful manifestations while eating.

Treatment. The treatment of stomatodynia requires a comprehensive application of therapeutic measures: drug therapy, physiotherapy, acupuncture, laser therapy, hyperbaric oxygenation, and psychotherapy. Drug therapy includes the use of antidepressants, benzodiazepines, antihistamines, sex hormones, vitamins, antifungals, analgesics, vasoactive drugs, and vegetotropic agents. Antidepressants have been widely used to treat chronic pain, with typical daily doses of 25-100 mg. Complications (orthostatic hypotension, arrhythmia) may occur in patients, especially those with cardiovascular pathology. Benzodiazepines are often administered to patients with chronic pain, although long-term use can stimulate or enhance depression. Phenazepam 0.25-0.5 mg 2-3 times a day, tazepam (daily dose 30-90 mg), mezepam (daily dose 10-30 mg) are often used. During periods of

exacerbation of unpleasant sensations in stomatodynia, Relanium is prescribed intramuscularly, and in severe cases of the disease, neuroleptics are used, for example, haloperidol is prescribed at 3.6-15 mg per day. The use of vitamins is necessary (parenteral administration of thiamine, riboflavin, pyridoxine, cyanocobalamin, ascorbic acid; oral administration of tocopherol acetate and retinol). The intake of vegetative-tropic drugs is recommended: cholinolytics (bellaspon, belloid, platyphylline); anticholinesterase preparations (galantamine); ganglioblockers (gangleron, benzohexonium). Antihistamines are used—pipolfen, dimedrol, suprastin are prescribed. Local anesthetics (anesthetic suspension in glycerin, trimecaine and lidocaine solutions) are prescribed for mouth baths to improve peripheral mechanisms. To improve salivary gland function and reduce xerostomia, pilocarpine (5 mg once a day sublingually), Vitamin A, and potassium iodide are used. Bromhexine is used to change the consistency of saliva. Physiotherapy procedures are widely used: massage of the shoulder and head, galvanization of the superior cervical sympathetic ganglia, endonasal electrophoresis of novocaine, etc. Many authors point to the positive effect of laser therapy, which plays the role of a biostimulator and has an analgesic effect. A treatment method for glossalgia with medical leeches is also used, which is especially effective in patients with cardiovascular diseases. According to a number of authors, a course of psychotherapeutic treatment consisting of hypnotherapy and autogenic training is effective. It is necessary to perform sanitation of the oral cavity, grinding of sharp edges of teeth, and restoration of occlusal surfaces. Despite the existence of many drugs and techniques used to treat stomatodynia, their effectiveness is low, which requires the joint efforts of doctors of different specialties—dentists, neuropathologists, and psychiatrists—to succeed in the study and treatment of this condition.

Taste Disturbances (Dysgeusia)

Taste disturbances (dysgeusia) occur with lesions of the facial nerve if the lesion is located in the facial nerve canal (Fallopian canal). This is because the chorda tympani passes next to the facial nerve in this segment, carrying taste fibers that transmit

information from the taste receptors of the anterior 2/3 of the tongue to the cortex of the cerebral hemispheres. Accordingly, in this case, a unilateral loss of taste sensitivity in the anterior 2/3 of the tongue is detected. Outside the facial nerve canal, the taste fibers are located as part of the lingual nerve. Therefore, if it is affected, taste sensitivity in the anterior 2/3 of the tongue is lost, but in combination with other symptoms. It should be noted that unilateral loss of taste sensitivity in the posterior 1/3 of the tongue is associated with damage to the glossopharyngeal nerve. Complete bilateral, and rarely unilateral, loss of taste sensitivity occurs with organic lesions of the CNS (brain tumors, neurosyphilis, etc.). Taste disturbances can manifest as its complete loss (ageusia), reduction (hypogeusia), or distortion (dysgeusia). This applies to all types of taste sensitivity (sweet, salty, sour, bitter) or only some. Taste buds are mainly located in the oral mucosa of the tongue, so the etiological factors affecting them and leading to taste disturbances are diverse: viral infections, allergic glossitis, radiation injury, trauma, avitaminoses A and B, Sjögren's syndrome, diseases of the gastrointestinal tract, and others. At the same time, desquamation or temporary loss of epithelium is enhanced on the tongue, due to which the taste buds are exposed and are available for excessive excitability. In such cases, taste sensitivity is restored with the regeneration of the papillae. With severe hyperkeratosis (thickening) and extensive lesions of the tongue in leukoplakia, taste is often reduced, which is associated with the difficult penetration of gustatory substances to the taste buds. A sensation of sourness may occur in patients with galvanic phenomena. Gingivitis, periodontitis, chronic tonsillitis, and periodontitis can be a source of unpleasant taste in the oral cavity. A sensation of bitterness or sourness may arise with hepatitis, cholecystitis, or gastritis.

Treatment. Treatment is often carried out by a neuropathologist in accordance with the principles of treating the corresponding diseases and taking into account their etiology.

Salivary Secretion Disturbances

Pathology of the **innervation of the salivary glands** and **lesions of the salivary glands themselves** and the oral mucosa often lead to **salivary secretion disturbances**.

There are two types of salivary secretion disturbances:

1. **Hypersalivation (Sialorrhea)**
2. **Hyposalivation (Xerostomia)**, with its extreme degree, which is discussed next in the text.

Hypersalivation (Sialorrhea)

Hypersalivation (Sialorrhea) is the excessive secretion of saliva.

Etiological factors causing hypersalivation include acute inflammatory processes of the oral mucosa which, due to the severe irritation of the mucosa, are accompanied by a reflex increase in salivary secretion—a positive protective reaction of the body. In some cases, hypersalivation occurs as a result of the direct excitation of the secretory cells of the salivary glands when substances like mercury and iodine enter the body. Prolonged hypersalivation is observed in some organic diseases of the Central Nervous System (CNS), such as Parkinsonism, bulbar and pseudobulbar paralysis, and in some cases, after a stroke. Impaired swallowing in these and other conditions exacerbates hypersalivation (the patient can't clear the saliva). The cause of hypersalivation can also be helminthic invasion (worms). In neurosis, when patients complain of excessive saliva, it may be false hypersalivation, which is not confirmed by objective examination. Patients with hypersalivation should be examined by a neurologist and internist (therapist), in addition to a dentist, according to indications.

Treatment: Treatment in all cases of hypersalivation should be aimed at eliminating the underlying disease that caused the condition. A temporary reduction in saliva can be achieved with atropine preparations.

Hyposalivation (Xerostomia)

Hyposalivation is the reduced secretion of saliva. The causes of hyposalivation are diverse: Vitamin A, B, E deficiencies, hypsiderosis (iron deficiency); diabetes mellitus, systemic diseases of connective tissue, radiation damage, bilateral chronic parotitis, and reduced salivation after surgical removal of large salivary glands. Dryness of the mouth (xerostomia) is a manifestation in patients with Sjögren's syndrome. Hyposalivation is also associated with thyrotoxicosis, menopause, and sympathetic nervous system hyperactivity in neurotic conditions. Temporary xerostomia can occur during acute infectious diseases, botulism, and after taking certain medications (e.g., atropine groups). Mouth dryness in people who are forced to breathe through the mouth due to impaired nasal breathing (polyps, deviated nasal septum) is caused by increased evaporation of fluid from the oral cavity (false hyposalivation). Dry mouth may also be the result of sleeping with the mouth open only at night, which is more common in old age. It should be noted that salivation is reduced in old and senile age, which is why factors that reduce it are more effective in these age groups. In hyposalivation and especially xerostomia, patients complain of dry mouth, difficulty eating and speaking, pain with sour and hard food, burning sensation, and a feeling of roughness of the mucosa. Reduced salivation is often combined with dryness of other mucous membranes. Upon objective examination, the mucosa may be slightly moist or dry and matte. Saliva is scant, may be frothy, or completely absent. The active growth of microflora (due to the elimination of the protective effect of saliva) leads to inflammation of the mucosa, and the appearance of erosions, ulcers, and injuries during eating. In patients with long-term hyposalivation, the amount of plaque increases, and multiple dental caries develop.

Treatment: This consists of eliminating the cause of hyposalivation. Sanitation of the oral cavity is mandatory, including prosthetics. Locally, the use of anti-inflammatory drugs and products that stimulate the mucosa is recommended (glycerin, lysozyme, peach, sunflower oil, etc.). As a rule, vitamins are prescribed (A, B1, B6, B12, C, E); according to indications—sex hormones; iodine preparations are prescribed

internally. Galvanization of the area of the large salivary glands gives a good effect. Anticholinesterase preparations are successfully used as symptomatic agents, particularly a 0.5% galantamine solution, 1.0 ml subcutaneously, or by electrophoresis, or orally daily for 1 month.

A 1% solution of pilocarpine hydrochloride is prescribed, 4 drops 1-2 times a day.

Changes in the Oral Mucosa in Dermatoses

Almost all dermatoses have manifestations on the oral mucosa and the red border of the lip (Pemphigus Erythematosus, Lichen Planus, Pemphigoid, Lupus Erythematosus, Dermatitis Herpetiformis, Pigmented Papillary Dystrophy, etc.). This section provides information on the dermatoses most commonly encountered in dental practice.

Lichen Planus

Lichen Ruber Planus (LRP) is a chronic inflammatory disease of the skin and mucous membranes manifested by the formation of **keratinized papules**. This disease is more common in **women aged 40-60 years**. Changes in the **oral mucosa** and the **red border of the lips** are frequently noted along with skin lesions in LRP. According to Abramova's data, simultaneous lesions of the skin and oral mucosa are noted in **25%** of patients, and lesions of the skin without oral involvement make up **75%** of patients. Lichen planus is also localized on other mucous membranes: **genitalia, anus, conjunctiva, esophagus, stomach, urinary tract**.

Etiology. The etiology is not fully established. Neurogenic, viral, and toxic-allergic theories exist regarding the origin of this disease. Lichen planus, as a rule, occurs against the background of chronic diseases of the gastrointestinal tract, hypertension, diabetes mellitus, neuroses, immune system imbalance, etc. A significant increase in the permeability of blood vessel walls and a decrease in immunological reactivity have been found in many patients.

Trauma has some importance in the development of LRP on the oral mucosa (sharp edges of teeth, poor-quality restorations, galvanic phenomena, allergic reactions to denture plastic, etc.).

Clinical Picture The main morphological element of the lesion is a keratinized papule of a round or polygonal shape with a diameter of up to 2mm. On the skin, the papules are usually flat, waxy in appearance, and have a pink or bluish-red color. On the oral mucosa, due to the keratinization of the epithelium and constant maceration, they acquire a white-gray color, standing out against the background of the normal or hyperemic mucosa. A characteristic feature of LRP is the confluence of papules into a pattern resembling a spider web, snowflakes, dendritic branches, sometimes rings, or lines (Wickham's striae). The papules are slightly raised above the surface of the mucosa, giving it a roughness. On the dorsum and lateral surfaces of the tongue, the papules merge, forming hyperkeratotic plaques of various sizes that often resemble leukoplakia; the papillae in this area are flattened



Figure 61. Typical Form of Lichen Planus (LRP)

Clinical Presentation in the Oral Cavity

The typical form of Lichen Ruber Planus (LRP) is characterized by the formation of multiple papules on the oral mucosa that merge into patterns. On the buccal mucosa, papules form patterns. In smokers, the papules are more distinct and larger, and they may be covered with patches resembling leukoplakia. On the red border of the lips, papules may merge to form a whitish band and sometimes take on a star shape. LRP on the red border and mucosa of the lips often leads to the appearance of glandular cheilitis. Typical locations for LRP on the oral mucosa are: The area where the large molars meet on the buccal mucosa. The vestibular folds (mucobuccal folds). The lateral and dorsal surfaces of the tongue, and the transition area to the lower surface

near the large molars. The lips, gums, palate, and floor of the mouth are less commonly affected. Forms of Oral Lichen Planus

Due to the variety of clinical manifestations of LRP in the oral cavity, the following forms are distinguished:

1. Typical (Simple) form
2. Exudative-Hyperemic form
3. Erosive-Ulcerative form
4. Bullous form
5. Hyperkeratotic form

Typical Form

The Typical form is encountered more frequently than the others. It is characterized by white-pearly papules located singly or grouped in patterns of rings, lines, or streaks (Wickham's striae) on the unchanged oral mucosa. With this typical manifestation of LRP, subjective symptoms are minimal and may manifest as a burning sensation, roughness, or dryness of the oral mucosa. Often, the disease is asymptomatic and can only be detected during a dental examination.



Figure 62. Lichen Planus (LRP) - Forms (Cont.)

2. Exudative-Hyperemic Form

This form is less common. The papules are located on a hyperemic (reddened) and edematous (swollen) oral mucosa. This form is accompanied by a more pronounced sensation of pain and a burning sensation, which intensifies when consuming spicy

food or talking. Against the background of the inflamed, hyperemic mucosa, the patterns of the papules may lose the clarity of their contours or even partially disappear. However, during the process of inverse development, when the swelling and hyperemia of the mucosa decrease, the papule patterns reappear.

3. Erosive-Ulcerative Form

This is considered the most severe type of all forms. It may occur as a complication of the typical and exudative-hyperemic forms due to the effect of various traumatic factors (sharp edges of teeth, dentures, galvanic phenomena, etc.) on the affected mucosa, resulting in erosions and sometimes ulcers.

In this form, there are erosions, and sometimes ulcers, on the hyperemic and swollen oral mucosa, around which the papules characteristic of LRP are present in a pattern against a background of clear inflammation. The irregularly shaped erosions or ulcers are covered with a fibrinous plaque that bleeds easily when removed. They can be single, small, and cause little pain, but they can also be accompanied by severe pain. Such erosions and ulcers persist for a long time, sometimes for months or even years, without epithelization. Often, under the influence of treatment, they are partially or completely epithelized, but they soon recur in the same or another area of the mucosa, sometimes immediately after stopping treatment. In some cases, areas of mucosal atrophy appear in place of long-existing erosions and ulcers. In certain cases, a long-term erosion or ulcer can transform into a malignant process.

4. Bullous Form This form is rarely encountered. It is characterized by the appearance of vesicles (bullae) with a diameter of 1 mm to 10 mm, along with the typical arrangement of white-pearly papules. The vesicles may contain serous (clear) or hemorrhagic (bloody) content, and they open very quickly. Their duration of existence is maintained from a few hours up to 2 days. The erosions formed in place of the vesicles are epithelized very quickly, which differentiates the bullous form of LRP from the erosive-ulcerative form.

The duration of the bullous form can vary, with vesicles sometimes appearing over many months. The vesicles can appear simultaneously with the papules on the oral

mucosa or later join them. Sometimes they appear before the papules, which makes the diagnosis of LRP difficult.



Figure 63. Lichen Planus (LRP) - Forms and Pathology (Cont.)

5. Hyperkeratotic Form

This form is rarely observed. It is characterized by the presence of various shapes and contours of hyperkeratotic plaques that are raised above the surface of the mucosa with sharp borders. Papular eruptions characteristic of Lichen Planus are present around the foci of hyperkeratosis. Most often, this form is localized on the buccal mucosa and the dorsum of the tongue.

Transformation and Course of LRP The described forms of LRP can transform into one another. For example, the Typical form can progress to the Exudative-Hyperemic and Erosive-Ulcerative forms as a result of exacerbation. The process of transformation is determined by the influence of general (systemic diseases) and local factors. Local factors that contribute to the transition from the Typical form to a more severe form include: sharp edges of teeth and dentures, dissimilar metals (galvanism), dental anomalies and deformities, untreated periodontitis, and dental calculus. LRP is a chronic disease characterized by a persistent, long-term course. LRP can last for decades with periods of remission, the duration of which is affected by severe systemic diseases and the presence of local traumatic factors in the oral cavity. Oral LRP can transform into a malignant process. This is more often observed in elderly people who have suffered from the Erosive-Ulcerative or

Hyperkeratotic forms for a long time. Signs of malignant transformation include: the formation of an induration (seal) at the base of the lesion, increased branching (verrucous) processes, and the appearance of vegetation (outgrowths) on the surface of long-standing, unhealed erosions and ulcers.

Pathological Anatomy of Lichen Planus. Pathohistological changes are characterized by hyper- and parakeratosis, as well as acanthosis in the epithelium. In some cases, the formation of a granular layer is detected. Edema occurs in the stroma, along with a diffuse inflammatory infiltrate (mainly from lymphocytes and plasmacytes), whose cells penetrate the epithelium through the basal membrane (exocytosis), as a result of which the membrane becomes poorly defined. The infiltrate almost never penetrates into the deep layers and the proper lamina of the mucosa; it closely approaches the epithelium, seemingly lifting it up from below. Depending on the type of lesion, certain differences are observed: In Erosive and Ulcerative lesions, in addition to hyper- and parakeratosis, ulceration and destruction of the epithelium and hemorrhages in the mucosa are noted. The infiltrate is detected directly beneath the basal membrane. Bullous lesions develop sub-epithelially as a result of extensive edema in the connective tissue. The vesicles are located sub-epithelially, and beneath them is a large round-cell infiltration in the proper lamina of the mucosa.

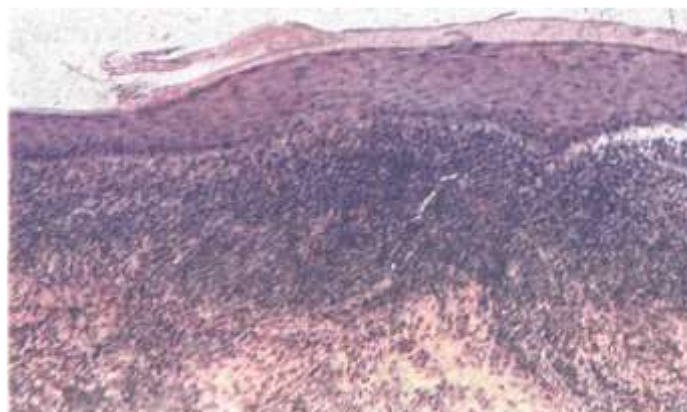


Figure 64. Lichen Planus (LRP) - Diagnosis and Treatment

Histopathology of LRP Forms

Typical Form: Pathohistologically, there is hyperkeratosis and parakeratosis in the epithelium. Erosive-Ulcerative and Bullous Forms: The epithelial changes characteristic of LRP are found in the areas bordering the defect (erosion/ulcer).

Diagnosis. Usually, the diagnosis of LRP is not difficult, especially when there are lesion elements on the skin. Correct diagnosis is more challenging with isolated lesions of the oral mucosa.

Differential Diagnosis

Lichen Planus Erythematosus must be differentiated from the following conditions:

Candidiasis

Lupus Erythematosus (LE)

Papular Syphilis

Allergic Stomatitis

Chronic Trauma-Induced Lesions

Bowen's Disease

Erosive Leukoplakia

Differentiation Points:

Erosive Leukoplakia: Unlike LRP, the area of keratinization has the appearance of a hard gray-white plaque slightly elevated above the mucosal surface, and no inflammation or papules are observed around it. Unlike LRP, leukoplakia is mainly located in the anterior parts of the oral cavity, often in the areas of the commissures of the mouth and buccal mucosa.

- **Candidiasis:** DDx is based on clinical differences. In candidiasis, papules are absent, and the plaques partially or completely scrape off, which is not possible with LRP plaques. A definite diagnosis is established based on bacterioscopic examination.
- **Lupus Erythematosus (LE):** Lesions on the oral mucosa are mainly located on the lips, palate, and less often on the cheeks. They are hyperemic, infiltrated, and

hyperkeratosis is present only in the center of the inflammation, where atrophy is observed—a feature not seen in LRP.

- **Syphilitic Papules:** These are significantly larger, round or oval-shaped. Their surface is covered with a gray-white plaque that scrapes off easily. Pale treponema are observed from the surface of open erosions.
- **Allergic Stomatitis:** LRP is differentiated from this based on anamnesis data, the disappearance of the lesion after eliminating the allergen, and the results of allergological tests.

Chronic Trauma-Induced Lesions: Oral mucosal lesions in LRP should be differentiated from similar changes under the influence of local traumatic factors, where the damaged area corresponds to the irritating effect, and the focus of inflammation disappears after the irritant is eliminated.

The Bullous and Erosive-Ulcerative forms of LRP must also be differentiated from other diseases that present with vesicles and erosions, such as Erythema Multiforme Exudativum, Acantholytic and Non-Acantholytic Pemphigus, Herpes Simplex, and the Erosive-Ulcerative form of Chronic Lupus Erythematosus.

The Hyperkeratotic form must be differentiated from Verrucous Leukoplakia and Cancer.

Treatment. Treatment for LRP is often complex and requires addressing underlying systemic issues. Systemic Examination and Treatment: LRP patients should undergo a complete examination to identify underlying systemic diseases.

Particular attention is paid to the state of the gastrointestinal tract and nervous system. Blood pressure and blood glucose levels are determined. If pathology is detected, the patient must be treated by the relevant specialists. Oral Sanitation and Traumatic Factor Elimination: All LRP patients must undergo complete oral sanitation, which begins with the elimination of traumatic factors by the dentist. Sharp edges of teeth and dentures must be smoothed. If dissimilar metals are present in the oral cavity, they, or at least one of the dissimilar metals, must be removed. Removable plate dentures should be made of colorless plastic with an internal elastic layer. It is advisable to replace amalgam fillings with cement. It is

recommended to stop smoking, avoid irritating food, and adhere to rational oral hygiene.

Corticosteroid Use Precautions:

When treating with corticosteroids, their possible side effects must be considered. During treatment, blood tests (complete clinical and glucose), blood pressure measurement, and prescription of potassium, calcium, multivitamins, and a special diet are necessary. If contraindications to corticosteroids exist for the Erosive-Ulcerative form, hyposensitization therapy with repeated use of Histoglobulin is recommended (several courses with a two-month break; 2 ml subcutaneously 2 times a week, 8-10 injections).

Malignant Transformation: It is crucial to remember the risk of malignant transformation of long-standing, unhealed erosions, ulcers, and foci of hyperkeratosis. In such cases, they should be removed followed by histological examination.

Cornuopia (Pemphigus)

Puffs containing acantholytic cells are characteristic. For this reason, acantholysis is called purulent. The blisters observed in Cornuopia (Pemphigus) Puffs containing acantholytic cells are characteristic. For this reason, acantholysis is called purulent. The blisters observed in this disease are a degenerative process as the acantholysis process increases. Acantholytic cells occur in epithelial tissue. This process leads to the formation of pores and skin crusting.

There are 4 clinical forms of pemphigus: vulgar; vegetative; leafy; seborrheic (erythematous).

Most often, the mucous membrane of the mouth is damaged by vulgar pemphigus (in 75% of patients), so this is of great interest to dentists. Most often, the mucous membrane of the mouth is damaged by vulgar pemphigus (in 75% of patients), so this is of great interest to dentists. Most often, the mucous membrane of the mouth is damaged by vulgar pemphigus (in 75% of patients), so this is of great interest to dentists. Vulgar pemphigus, the mucous membrane of the mouth is damaged by vulgar pemphigus (in 75% of patients), so this is of great interest to dentists. Vulgar pemphigus almost

always begins with damage to the mucous membrane of the mouth or larynx, and then spreads to the skin. Even if the disease begins with the excitation property of the skin, damage to the OBSHQ is almost always detected. Vulgar pemphigus (pemphigus vulgaris). Vulgar or common pemphigus is much more common than other forms. Men and women over 35 years old suffer from this disease, children get very little. Etiology. Unknown. There are viral and autoimmune theories of this disease.

Clinical picture. Damage to the mucous membrane of the mouth and lips with pemphigus is characterized by the formation of vesicles with serous or hemorrhagic content. Clinical picture. Damage to the mucous membrane of the mouth and lips with pemphigus is characterized by the formation of vesicles with serous or hemorrhagic content. Due to clinical picture. Damage to the mucous membrane of the mouth and lips with pemphigus is characterized by the formation of vesicles with serous or hemorrhagic content. Due to constant renewal in the oral cavity, the vesicles open very quickly, so it is very rare to see them in the oral cavity. Remnants of the former Bubble are more common. Instead of bubbles, round or oval erosions are formed, which do not heal for a long time. Erosions of a bright red color are observed against the background of an unchanged or slightly inflamed mucous membrane. The dimensions of erosions in the trunk can be different-from small friction to large surfaces of a stagnant red color will be characteristic. The dimensions of erosions in the trunk can be different-from small friction to large surfaces of a stagnant red color will be characteristic. The dimensions of erosions in the trunk can be different-from small friction to large surfaces of a stagnant red color will be characteristic. As a rule, carashalr is not observed on the surface of erosion, or easily removable fibrinous carash can be thin. Most often, erosion settles on the mucous membrane of lungs (especially in the retromolar sac), on the lower surface of the tongue, on the palate and on the lower part of the oral cavity. New erosions appear if left untreated, which combine with each other to form wide erosive surfaces that do not end. The pain is

very intense, and increases when eating and talking. Erosion is rapidly damaged, especially in the sanitized oral cavity. The pain is very intense, and increases when eating and talking.

Erosion is rapidly damaged, especially in the sanitized oral cavity. The addition of the pain is very intense, and increases when eating and talking. Erosion is rapidly damaged, especially in the sanitized oral cavity. The addition of cocci, zambucal flora and fusospirochetosis aggravates the patient's condition, with specific oral odors. Hypersalivation is observed. Bubbles and erosions covered with a hemorrhagic shell can also appear on the red border of the lips and at the corners of the mouth. Sometimes wheezing occurs, which indicates damage to the larynx.

Blisters on pemphigus skin appear mainly in areas of friction with clothing, pressure, maturation (abdomen, back, trough folds, etc.). Blisters on pemphigus skin appear mainly in areas of friction with clothing, pressure, maturation (abdomen, back, trough folds, etc.). Erosion on the skin formed after the bubbles. Open is much more painful. Nikolsky's fracture in pemphigus is usually positive. It has three manifestations: if the cap of the bladder or the upper layer of the epithelium at the edge of erosion is held and pulled with tweezers, the epithelial film is separated to the unchanged healthy mucous membrane and skin. The thin film of the epithelium is very fragile and easily decomposes with tweezers; Rubbing the unchanged mucous membrane or skin between the affected areas leads to the rapid formation of blisters or erosions; if you rub the mucous membranes away from the affected area, there will also be a cleansing of the upper layers of the epithelium from the lower layers. The second and especially third manifestations of the Nikolsky symptom indicate an increase in the intensity of acantholysis. The second and especially third manifestations of the Nikolsky symptom indicate an increase in the intensity of acantholysis. In addition

to the skin and mucous membrane of the mouth with pemphigus, other mucous membranes. The second and especially third manifestations of the Nikolsky symptom indicate an increase in the intensity of acantholysis. In addition to the skin and mucous membrane of the mouth with pemphigus, other mucous membranes (intestines, stomach, esophagus, pharynx), as well as internal organs and the central nervous system, can be affected by pemphigus. Pemphigus is characterized by a turbulent course, with the recurrence alternating with periods of remission, which rarely occur spontaneously, often under the influence of treatment. If the process begins on the mucous membrane of the mouth, appropriate treatment measures are not taken after 1-6 months, sometimes it spreads to the skin of the face and legs. If the process begins on the mucous membrane of the mouth, appropriate treatment measures are not taken after 1-6 months, sometimes it spreads to the skin of the face and legs.

Diagnosis. It is made based on clinical manifestations, positive Nikolsky's symptom, cytological examination results, and direct immunofluorescence reaction.

Cytological examination. For the diagnosis of pemphigus, a method of cytological examination of fingerprints or scraping from the bottom of erosion is mandatory. The presence of acantholytic cells in them serves to confirm the diagnosis of acantholytic pemphigus. Cytological examination. For the diagnosis of pemphigus, a method of cytological examination of fingerprints or scraping from the bottom of erosion is mandatory. The presence of acantholytic cells in them serves to confirm the diagnosis of acantholytic pemphigus. Acantholytic cells or Tsanka cells indicate a change in the thoracic layer. They have rounded outlines and are smaller than the normal cells of the thorny layer. The nucleus is large compared to the whole cell, with a diameter of $\frac{1}{3}$ — $\frac{1}{2}$ and more than the diameter of the cell, which is dyed dark blue, often with 1 to 6 and lighter nucleoli. The nucleus is large compared to the whole cell, with a diameter of $\frac{1}{3}$ — $\frac{1}{2}$ and more than the diameter of the cell, which is dyed dark blue, often with 1 to 6 and lighter nucleoli. The cythe nucleus is large compared to the whole cell, with a diameter of $\frac{1}{3}$ — $\frac{1}{2}$ and more than the diameter of the cell, which is dyed dark blue, often with

1 to 6 and lighter nucleoli. The cytoplasm of cells has a different color: light blue around the nucleus and dark blue along the periphery. Acantholytic cells are characterized by polymorphism in color, cell size, and nucleus size. The cotalization of the disease increases the number of acantholytic and multinucleated cells. They merge into a continuous conglomerate of polymorphic cells. During remission and treatment with corticosteroids, the number of acantholytic cells decreases.

The cytological appearance in Vegetative pemphigus is no different from that of vulgar.uring remission and treatment with corticosteroids, the number of acantholytic cells decreases.

The cytological appearance in Vegetative pemphigus is no different from that of vulgar. In seborrheic pemphigus, multicellular cells are usually undetectable, acantholytic cells are found in small amounts, which are monomorph-xysobed.

Pathogistological examination. The main morphological changes in acantholytic pemphigus have been shown to be acantholysis and edema, resulting in epithelial intra-vesicles.athogistological examination. The main morphological changes in acantholytic pemphigus have been shown to be acantholysis and edema, resulting in epithelial intra-vesicles. The bonds are broken betweenathogistological examination. The main morphological changes in acantholytic pemphigus have been shown to be acantholysis and edema, resulting in epithelial intra-vesicles. The bonds are broken between the cells of the thoracic layer-the phenomenon of acantholysis, as a result of which intercellular bridges dissolve, cracks form between the cells, and then bubbles appear. The lower part of such bubbles, as well as the later erosion surface, is mostly covered with acantholytic cells.

Comparative diagnosisomparative diagnosis. Acantholytic (ischemic) pemphigus should be distinguished from other bullous lesions of the oral mucosa: multi-form exudative erythema; pemphigoid; drug allergy; QYAT form of bullous; with herp

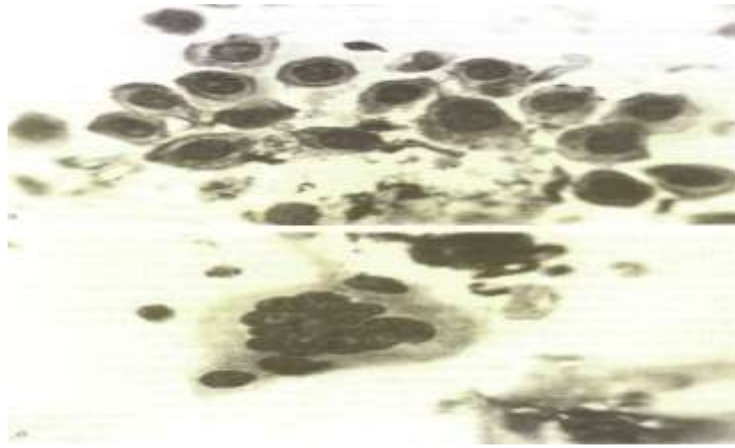


Figure 65. Pemphigus: Differential Diagnosis and Treatment

Differential Diagnosis (Cont.)

Acantholytic pemphigus is distinguished from other diseases with blister formation mainly by the location of the blisters relative to the epithelium. Acantholytic pemphigus is distinguished from other diseases with blister formation mainly by the location of the blisters relative to the epithelium. Thus, KShEE blisters are located under the epithelium, Nikolsky's test is negative. In addition, KShEE is characterized by acute onset, seasonality of reactions, pronounced inflammation of the oral mucosa and a short duration of course.

In bullous pemphigoid, the vesicles are located under the epithelium, their cap is thick, so their migration is longer. Bullous pemphigus is more common in people over 60 years of age. In bullous pemphigoid, the vesicles are located under the epithelium, their cap is thick, so their migration is longer. Bullous pemphigus is more common in people over 60 years of age. In bullous pemphigoid, the vesicles are located under the epithelium, their cap is thick, so their migration is longer. Bullous pemphigus is more common in people over 60 years of age. Nikolsky's fracture is negative, acantholytic cells are not detected. Acantholytic pemphigus is diagnosed by Anamnesis as distinct from drug-induced allergic stomatitis. Stomatitis is quickly eliminated after stopping taking the Allergen drug. Medicinal stomatitis blisters are located under the epithelium, Nikolsky's fracture is negative, there are no acantholytic cells.

In the form of kyat bullous, the blisters are epithelial under, there is no acantholysis. Around the blisters or in other parts of the oral mucosa, there will be many papules characteristic of a red flat surface. In the form of kyat bullous, the blisters are epithelial under, there is no acantholysis. Around the blisters or in other parts of the oral mucosa, there will be many papules characteristic of a red flat surface. During herpetic dermatitis is characterized by the subepithelial arrangement of blisters that initially appear on the skin. Nikolsky's synapse is negative, without acantholytic cells. It is characterized by a significant amount of eosinophils in the vesicles, and eosinophilia is also recorded in the blood. Patients with during herpetic dermatitis experience increased iodine sensitivity. In some cases, with pemphigoid, QYaT and other cystic diseases, when there is a lower epithelia location of vesicles or yerosias around them, the entire layer of epithelium located in the area of inflammation can be easily cleaned. In some cases, with pemphigoid, QYaT and other cystic diseases, when there is a lower epithelia location of vesicles or yerosias around them, the entire layer of epithelium located in the area of inflammation can be easily cleaned. At the same time, the bubble cap is thick, difficult to tear. This symptom is called a false Nikolsky symptom or a symptom of peripocal subepithelial detachment

Treatment. Currently, corticosteroid and cytostatic drugs are used to treat pemphigus. Treatment. Currently, corticosteroid and cytostatic drugs are used to treat pemphigus. Prednisolone, methylprednisolone (metipred, urbazone) treatment. Currently, corticosteroid and cytostatic drugs are used to treat pemphigus. **Prednisolone, treatment.** Currently, corticosteroid and cytostatic drugs are used to treat pemphigus. Prednisolone, methylprednisolone (metipred, urbazone), dexamethasone (dexazone), triamcinolone (polcor-tolone, kenacort) are prescribed. In many patients, treatment with corticosteroids leads to the loss of clinical signs of the disease. However, in the case of discontinuation of corticosteroids, relapse of the disease, so treatment of patients with pemphigus, even if the clinical manifestations

of the disease are incomplete, should be permanent. Prednisolone is prescribed in high doses of 50-80 mg per day, triamcinolone 40-80 mg, and dexamethasone 8-10 mg, depending on the patient's condition. Prednisolone is prescribed in high doses of 50-80 mg per day, triamcinolone 40-80 mg, and dexamethasone 8-10 mg, depending on the patient's condition. Such high, so-called shock doses are taken by patients until new blisters appear and almost complete epithelization of erosions is stopped, after which the daily dose of the drug is gradually reduced. The dose of prednisolone is first reduced by 5 mg every 5 days, then these periods are extended to 7-10 days. Reducing the Daily Dose is carried out to a minimum, the so-called individual daily dose, which is applied continuously. 10-15 mg is usually recommended for prednisolone, and 0.5—1 mg for dexamethasone. Reducing the Daily Dose is carried out to a minimum, the so-called individual daily dose, which is applied continuously. 10-15 mg is usually recommended for prednisolone, and 0.5—1 mg for dexamethasone. Reducing the Daily Dose is carried out to a minimum, the so-called individual daily dose, which is applied continuously. 10-15 mg is usually recommended for prednisolone, and 0.5—1 mg for dexamethasone. Long-term use of corticosteroid drugs causes various side effects in patients (increased blood pressure, increased glucose in the urine, osteoporosis of bone tissue, provoking thrombosis, etc.). In this regard, corticosteroid treatment is recommended to limit the amount of table salt and water in food to reduce complications. The diet should mainly contain protein with a restriction of fats and carbohydrates. In this regard, corticosteroid treatment is recommended to limit the amount of table salt and water in food to reduce complications. The diet should mainly contain protein with a restriction of fats and carbohydrates. In this regard, corticosteroid treatment is recommended to limit the amount of table salt and water in food to reduce complications. The diet should mainly contain protein with a restriction of fats and carbohydrates. Inside, potassium preparations (potassium chloride, asparkam), ascorbic acid, B vitamins, calcium preparations, calcitonin are prescribed. Cytostatics, mainly methotrexate,

are also used to treat pemphigus. It is prescribed for 35-40 mg once a week at the same time as corticosteroids.

Local treatment is mainly aimed at preventing secondary infection of erosion and ulceration. Local treatment is mainly aimed at preventing secondary infection of erosion and ulceration. This involves the use of painkillers in the form of oral baths; antiseptic drugs in non-damaging concentrations; application of the oral mucosa or corticosteroid ointment is applied. After each meal and before applying ointments containing corticosteroids, warm solutions of potassium permanganate, 0.25% chloramine, 0.02% chlorhexidine solution, etc. are washed off. Careful sanitation of the oral cavity is important for the rapid epithelization of erosions in the mucous membrane. Careful sanitation of the oral cavity is important for the rapid epithelization of erosions in the mucous membrane. When the skin of the lips is damaged, careful sanitation of the oral cavity is important for the rapid epithelization of erosions in the mucous membrane. Careful sanitation of the oral cavity is important for the rapid epithelization of erosions in the mucous membrane.

Pemphigoid

In the pemphigoid case, the blisters are dressing and do not elect acantholytic cells in the liquid contained. Nikolsky's synapse is negative.

In the pemphigoid case, the blisters are dressing and do not elect acantholytic cells in the liquid contained. Nikolsky's synapse is negative.

- In the pemphigoid case, the blisters are dressing and do not elect acantholytic cells in the liquid Pemphigoid

- In the pemphigoid case, the blisters are dressing and do not elect acantholytic cells in the liquid Pemphigoid

- In the pemphigoid case, the blisters are dressing and do not elect acantholytic cells in the liquid contained. Nikolsky's synapse is negative. This group of diseases includes diseases with a view to pemphigus diseases:

- Lever's bullous pemphigoid or private nonacantholytic form (N. D. According to Sheklakov).;

- scarred pemphigoid or mucous synechial bullous dermatitis, pemphigus of the eye;
- a good quality non-antimicrobial blister (B. M. According to Pashkov).

Etiology. Not fully determined. Etiology. Not fully determined. All types of pemphigoid are characterized by the absence of acantholysis in epithelial cells, subepithelial arrangement of vesicles, pronounced inflammation, absence of acantholytic cells, antiolgy. Not fully determined. All types of pemphigoid are characterized by the absence of acantholysis in epithelial cells, subepithelial arrangement of vesicles, pronounced inflammation, absence of acanthotiology. Not fully determined. All types of pemphigoid are characterized by the absence of acantholysis in epithelial cells, subepithelial arrangement of vesicles, pronounced inflammation, absence of acantholytic cells, and negative Nikolsky's fracture. Pemphigoid is safe, the general condition of patients is satisfactory, the consequences for life are good.

Bullous pemphigoid (pemphigoid bullosa). The disease usually occurs in people over 50 years of age. The skin and mucous membranes of the mouth, nose and genitals are damaged. In about 10% of cases, the disease is caused by damage to the oral mucosa.

Clinical picture. It is characterized by the formation of subepithelial vesicles in the mouth. Clinical picture. It is characterized by the formation of subepithelial vesicles in the mouth. Against the background of swollen and defl. Clinical picture. It is characterized by the formation of subepithelial vesicles in the mouth. Against the background of swollen and defl. Clinical picture. It is characterized by the formation of subepithelial vesicles in the mouth. Against the background of swollen and deformed oral mucosa, bubbles of serous or hemorrhagic content with a diameter of 5-20 mm appear. They last for several hours, sometimes days, then erosion is formed in their place, which is covered with fibrinous Carache. Unlike acantholytic pemphigus, erosion is epithelized after 10-15 days without scarring and atrophy, but can reappear after a certain period of time. The favorite location of bubbles in non-antholytic pemphigus is the border of the hard and soft palate, lunge. The disease lasts for months and years and can go away on its own.

Sometimes the pathological process can settle in the gums. At the same time, the mucous membrane on the vestibular surface on the banks of the gums is hyperemic, swollen and bleeding is observed. Sometimes the pathological process can settle in the gums. At the same time, the mucous membrane on the vestibular surface on the banks of the gums is hyperemic, swollen and sometimes the pathological process can settle in the gums. At the same time, the mucous membrane on the vestibular surface on the banks of the gums is hyperemic, swollen and bleeding is observed. In this case, Nikolsky's fracture will be positive, but acantholytic cells will not be detected. The course of the disease is constant and prolonged.

Pathological anatomy. Histologically, subepithelial vesicles are detected. Swelling and inflammatory infiltration are noted on the private plate of the mucous membrane.

Comparative dental. Bullous pemphigoid should first be distinguished: Acantholytic pemphigus, with during herpetic-shaped dermatitis.

Treatment comparative dental. Bullous pemphigoid should first be distinguished: Acantholytic pemphigus, with during herpeo comparative dental. Bullous pemphigoid should first be distinguished: Acantholytic pemphigus, with during herpetic-shaped dermatitis.

Treatment. The most effective treatment for non-antholytic pemphigus is corticosteroid drugs (prednisone 20-30 mg / day). In some cases, a good effect is achieved through combined treatment — prednisone, broad-spectrum antibiotics, anti-malaria drugs and multivitamins.

Eye pemphigus (pemphigus cicatri-cans) or mucous synechial atrophic bullous dermatitis. e pemphigus (pemphigus cicatri-cans) or mucous synechial atrophic bullous dermatitis. For Scar pemphigoid, instead ye pemphigus (pemphigus cicatri-cans) or mucous synechial atrophic bullous dermatitis. For Scar pemphigoid, instead of previous blisters, the appearance of scars, adhesions and atrophy is characteristic. Affects the mucous membranes, mainly the conjunctiva of the eyelids, sometimes on the skin. As a result, there may be a narrowing of the eyeball, disarticulation of the eyeball, and blindness.

Treatment. This should be done in conjunction with an ophthalmologist, effective treatment is prescribed corticosteroid drugs (prednisone 20-30 mg per day).reatment. This should be done in conjunction with an ophthalmologist, effective treatment is prescribed corticosteroid drugs (prednisone 20-30 mg per day). Ieatment. This should be done in conjunction with an ophthalmologist, effective treatment is prescribed corticosteroid drugs (prednisone 20-30 mg per day). In some cases, a good effect is achieved through combined treatment — prednisone, broad-spectrum antibiotics, anti-malaria drugs and multivitamins.

The end. satisfactory, but sometimes the development of blindness and scarring of the mucous membranes can significantly reduce the working capacity of patients.

Non-antigenic pemphigus of the oral mucosa only. In 1959, B.onon-antigenic pon-antigenic pemphigus of the oral mucosa only. In 1959, B. M. Pashkov and N. D. Described by Sheklakov.



Figure 68. Pemphigoid. Pathogistologically, subepithelial location of the bladder, swelling and inflammatory infiltration

The clinical course is almost identical to that of non-antithetic pemphigus.he clinical course is almost identical to that of non-antithetic pemphigus. Vesicles in this disease are smaller (3-10 mm in diameter), located subepitelically, without acantholysis events; they are observed in some patients almostthe clinical course is almost identical to that of non-antithetic pemphigus. Vesicles in this disease are smaller (3-10 mm in diameter), located subepitelically, without acantholysis events; they are observed in some patients almost continuously, in others with remissions from several days to several weeks. Subjective complaints of patients are reduced

by mild burns and hardening of the mucous membrane. After the bubbles burst, erosion quickly epithelializes, how hyech leaves no trace and atrophy.

Treatment. Antihistamines, small doses of corticosteroid drugs (15-20 mg prednisone) are carried out in combination with antimalarial drugs. Vitamins C and R are prescribed.

Local treatment mainly aimed at preventing erosion and secondary infection of the wound, it involves the use of pain relievers in the form of oral baths; antiseptic drugs in non-damaging concentrations; application of the oral mucosa or corticosteroid ointment is applied. Local treatment mainly aimed at preventing erosion and secondary infection of the wound, it involves the use of pain relievers in the form of oral baths; antiseptic drugs in non-damaging concentrations; application of the oral mucosa or corticosteroid ointment is applied. After each meal and before applying ointments containing corticosteroids, warm solutions of potassium permanganate, 0.25% chloramine, 0.02% chlorhexidine solution, etc. are washed off. Careful sanitation of the oral cavity is important for the rapid epithelization of erosions in the mucous membrane. Careful sanitation of the oral cavity is important for the rapid epithelization of erosions in the mucous membrane.

Lupus Erythematosus

Erythritis connective tissue disease is characterized and is accompanied by autoimmune changes. In this case, the antigen accumulates between the epidermis and the derma, a cluster of antitelo and complement. Erythritis connective tissue disease is characterized and is accompanied by autoimmune changes. In this case, the antigen accumulates between the epidermis and the derma, a cluster of antitelo and complement.

Erythritis connective tissue disease is characterized and is accompanied by autoimmune changes. In this case, the antigen accumulates between the epidermis and the derma, a cluster of antitelo and complement. The disease often begins between the ages of 20 and 40, with women getting sick more often than men. There are two forms of rubella (rubella): chronic (discoid) — a relatively good clinical form of rubella and acute (systemic) — severe form.

Both forms of red runny nose can affect the red Hesia of the lips and the mucous membrane of the mouth.

Etiology and pathogenesis. The causes of lupus have not been determined.oth forms of red runny nose can affect the red Hesia of the lips and the mucous membrane of the mouth.

Etiology and pathogenesis. The causes of lupus have not been determined. According to modernred runny nose can affect the red Hesia of the lips and the mucous.



Figure 69. Epilepsy. The cortex on the red border of the lip.

Chronic lupus

Clinical pictureclinical picture. Chronic lupus usually begins with erythematous spotting on the skin of the face (often in the form of a butterfly on the nose, forehead, cheeks), ear Supras, scalp, red Hesia of the lips and other open pcal picture. Chronic lupus usually begins with erythematous spotting on the skin of the face (often in the form of a butterfly on the nose, forehead, cheeks), ear Supras, scalp, red Hesia of the lips and other open parts of the body. The oral mucosa is rarely affected. Hyperkeratosis soon develops on the surface of the erythema. Atrophy forms in the center of the lesion, gradually covering the entire furnace, and erythema and hyperkeratosis are maintained. Atrophy forms in the center of the lesion, gradually covering the entire furnace, and erythema and hyperkeratosis are maintained. In

addition to these three main clinical signs of lupus, there are additional signs: infiltration, teleangiectasia and pigmentation. The signs of each of these signs may differ.

Damage to the oral mucosa and the red border of the lips is accompanied by burning and pain, which intensifies when eating and speaking. Damage to the oral mucosa and the red border of the lips is accompanied by burning and pain, which intensifies when eating and speaking. There are 4 clinical differences in red runny nose in the red Hoshia of the lips: typical; clinically clear without atrophy; erosive and ulcerative; deep In the typical form of red runny nose, infiltrated Oval foci are formed on the red Hoshia of the lips, or the process can diffusely cover the entire lip red border. In the typical form of red runny nose, infiltrated Oval foci are formed on the red Hoshia of the lips, or the process can diffusely cover the entire lip red border. In the typical form of red runny nose, infiltrated Oval foci are formed on the red Hoshia of the lips, or the process can diffusely cover the entire lip red border. The affected areas are purple-red, characterized by permanently enlarged veins and pronounced infiltration foci. Their surface is covered with tightly wrapped white-gray scales, which, when scraped, cause bleeding and noticeable pain. In the center of the jarokhat, atrophy of the red hoshia of the lips and skin is noted. On the peripheral surface of the jarokhat, an epithelium of a different shape, white in color, is observed. On the peripheral surface of the jarokhat, an epithelium of a different shape, white in color, is observed. With a pathologically typical form, parakeratosis, hyperkeratosis, acanthosis in the peripheral surface of the jarokhat, an epithelium of a different shape, white in color, is observed.



Photo 70. Chronic red run. On the skin of the cheeks, nose, against the background of erythematous spots, there are wide sores covered with crusts.

The lack of development of atrophy in the clinic in lupus is characterized by the appearance of diffuse dimmed hyperemia with hyper - and para-keratotic scales on the red border of the lips, which are more easily excreted than in the typical form. The lack of development of atrophy in the clinic in lupus is characterized by the appearance of diffuse dimmed hyperemia with hyper - and para-keratotic scales on the red border of the lips, which are more easily excreted than in the typical form. This form of hyperkeratosis is less pronounced than the typical form of lupus erythematosus.

In the red hoshia of the lips, the erosive and ulcerative shape of the Red runner is manifested by pronounced inflammation, foci of bright red color, cracks covered with swollen, erosive and bloody purulent crusts. In the red hoshia of the lips, the erosive and ulcerative shape of the Red runner is manifested by pronounced inflammation, foci of bright red color, cracks covered with swollen, erosive and bloody purulent crusts. In the red hoshia of the lips, the erosive and ulcerative shape of the Red runner is manifested by pronounced inflammation, foci of bright red color, cracks covered with swollen, erosive and bloody purulent crusts.

bloody purulent crusts. Patients will be worried about severe burns, pain and itching that will increase during meals. After the jarokhat furnace is finished, atrophic scars remain in its place. In the deep form of the Kaposi—Irganga of the Red runner, lesions on the lips are rare. The nodular-shaped lesion protrudes from the surface of the lip red border, on the surface of the erythema and hyperkeratosis.

Lupus erythematosus of the lips is often complicated by secondary glandular cheilitis. Mucous membrane lupus erythematosus affects the lips less frequently than the red erythematosus of the lips is often complicated by secondary glandular cheilitis. Mucous membrane lupus erythematosus affects the lips less frequently than the red erythematosus of the lips is often complicated by secondary glandular cheilitis. Mucous membrane lupus erythematosus affects the lips less frequently than the red border of the lips. The process is localized on the mucous membrane of the lips, on the cheeks along the line of closure of the teeth, less often on the tongue, palate, and other places. The following forms of lupus erythematosus are distinguished on the oral mucosa: typical; exudative-hyperemic; erosive-ulcerative.

The typical form is characterized by foci of hyperemia, dimmed by infiltration and hyperkeratosis. There is atrophy in the center of the pharynx, hyperkeratosis in the periphery, one joined to one along the white lines. The typical form is characterized by foci of hyperemia, dimmed by infiltration and hyperkeratosis. There is atrophy in the center of the pharynx, hyperkeratosis in the periphery, one joined to one along the white lines. The typical form is characterized by foci of hyperemia, dimmed by infiltration and hyperkeratosis. There is atrophy in the center of the pharynx, hyperkeratosis in the periphery, one joined to one along the white lines. Due to strong inflammation, hyperkeratosis and atrophy in the exudative hyperemic form do not develop clearly. In the presence of a traumatic factor, the exudative-hyperemic form quickly develops into an erosive-ulcerative form, in which painful erosion or sores appear in the center of the jarokhat. Against the background of erythema, erosion or radial divergent white stripes are visible around the wound. Hyperkeratosis multiplies along the periphery of the pharynx, and sometimes a

hyperkeratotic border is formed, consisting of tightly adjacent lines and dots. Hyperkeratosis multiplies along the periphery of the pharynx, and sometimes a hyperkeratotic border is formed, consisting of tightly adjacent lines and dots. After the red runny hyperkeratosis multiplies along the periphery of the pharynx, and sometimes a hyperkeratotic border is formed, consisting of tightly adjacent lines and dots. After the red runny nose injury is treated, scars and atrophy usually remain. Chronic red jogging is long with exacerbation in the spring and summer period (years -decades). The erosive and ulcerative form of chronic lupus, which is located in the red hyacinth of the lips, can go into a dangerous process.

Pathological anatomy. The histological appearance of the lesion in chronic discoid lupus erythematosus of the oral mucosa and lips is characterized by the presence of hyperkeratosis, acanthosis, and parakeratosis. The histological appearance of the lesion in chronic discoid lupus erythematosus of the oral mucosa and lips is characterized by the presence of hyperkeratosis, acanthosis, and parakeratosis. The histological appearance of the lesion in chronic discoid lupus erythematosus of the oral mucosa and lips is characterized by the presence of hyperkeratosis, acanthosis, and parakeratosis alternating with atrophy. The basal layer of the epithelium is characterized by vacuolar degeneration of the cells and indistinctness of the basement membrane due to the infiltration of infiltrating cells from the stroma into the epithelium. The lamina propria of the mucous membrane contains massive lymphoid-plasma cell infiltration, capillary dilatation, and stasis. The loss of collagen fibers is particularly important under the epithelium and around small blood vessels. In the erosive and ulcerative form, epithelial defects, edema, and inflammation are significantly pronounced.

Diagnosis. If the skin has red runny sores at the same time, this will not cause difficulties. If the skin has red runny sores at the same time, this will not cause difficulties. Damage to the oral mucosa or red hoshia of the lips can cause difficulties in diagnosis, therefore, additional research methods (histological, fluorescent diagnostics and direct RIF) are used along with clinical examination. In

wood Rays, hyperkeratosis areas on the red hyacinth of the lips give a snow-blue or snow-white shine, on the mucous membrane of the mouth — a white or pale white shine in the form of stripes and dots. yperkeratosis areas on the red hyacinth of the lips give a snow-blue or snown wood Rays, hyperkeratosis areas on the red hyacinth of the lips give a snow-blue or snow-white shine, on the mucous membrane of the mouth — a white or pale white shine in the form of stripes and dots.

Differential diagnosis. Chronic lupus erythematosus should be differentiated from SLE and leukoplakia. When the process is located on the red border of the lips, the process differs from actinic halite and abrasive precancerous Manganotti halite.

Treatment. Treatment for patients with chronic lupus begins with a thorough examination to identify and eliminate foci of chronic infection, as well as to determine the systemic nature of jaroxate.reatment. Treatment for patients with chronic lupus begins with a thorough examination to identifreatment. Treatment for patients with chronic lupus begins with a thorough examination to identify and eliminate foci of chronic infection, as well areatment. Treatment for patients with chronic lupus begins with a thorough examination to identify and eliminate foci of chronic infection, as well as to determine the systemic nature of jaroxate. Treatment of medicinal substances is carried out using Quinoline-type drugs (plaquenil, delagil). Small doses of corticosteroid drugs are prescribed at the same time: prednisolone (u— 15 mg), triamcinolone (8-12 mg) and dexamethasone (1.5—2.0 mg). A complex of vitamins is widely used: B2, B12, nicotinic and ascorbic acids are prescribed. Intravenous administration of hinghamin solutions is applied 1-2 days after the acute inflammatory process has subsided. Corticosteroid ointments are used for topical treatment ("Flucinar"," Sinas", prednisolone, etc. Intravenous administration of hinghamin solutions is applied 1-2 days after the acute inflammatory process has subsided.

A sharp red runner. It is a severe systemic disease. sharp red runner. It is a severe systemic disease. It is characterized by high temperature, joint pain, damage to internal organs (polycerositis, yendocarditis, glomerulonephritis, polyart sharp red runner. It is a severe systemic disease. It is characterized by high temperature, joint

pain, and sharp red runner. It is a severe systemic disease. It is characterized by high temperature, joint pain, damage to internal organs (polycytemia, endocarditis, glomerulonephritis, polyarthritis, damage to the gastrointestinal tract, etc.). In the blood-increased leukopenia, anemia, SOE. The disease can occur in acute and acute subsurface forms. In acute lupus, changes in the mucous membrane are detected in almost 2/3 of patients. Hyperemic and swollen spots appear on the mucous membrane of the palate, tongue, gums, sometimes with a pronounced hemorrhagic character; bubbles of various sizes with a hemorrhagic composition, which turn into erosion, covered with fibrinous Carache. There are spots of hyperemia on the skin, sometimes swelling and blisters appear. Bubbles of various sizes with a hemorrhagic composition, which turn into erosion, covered with fibrinous Carache. There are spots of hyperemia on the skin, sometimes swelling and blisters appear. The course of this form of lupus is characterized by the development of various organs and tissues with a gradual involvement in the process.

Diagnosis. They are put on the basis of detectable skin lesions and the condition of internal organs, as well as the presence of "red runner cells" in blood and bone marrow punctures. Diagnosis. They are put on the basis of detectable skin lesions and the condition of internal organs, as well as the presence of "red runner cells" in blood and bone marrow. Diagnosis. They are put on the basis of detectable skin lesions and the condition of internal organs, as well as the presence of "red runner cells" in blood and bone marrow punctures. Many patients with lupus tend to have secondary immunodeficiency.

Treatment. Treatment of patients with acute lupus should be started in the hospital as early as possible. The course of treatment should be long and continuous. In the active period, glucocorticoid drugs are prescribed in large doses.

Herpetic-shaped dermatitis of Dühring

Dühring's herpetiform dermatitis (dermatitis herpetiformis Dühring) is a very rare disease. Dühring's herpetiform dermatitis (dermatitis herpetiformis Dühring) is a very rare disease. According to Pashkov, this disease affyuring's herpetiform dermatitis

(dermatitis herpetiformis Duhring) is a very rare disease. According to Pashkov, this disease affects only 10% of patients with oral mucosa.

Etiology and pathogenesis. Not fully accurate. Patients with herpes-shaped dermatitis of Duhring have increased sensitivity to iodine preparations. When a solution of 3-5% potassium iodide is given inside, the usual rashes for this disease appear.

Clinical picture. The oral mucosacininin, which is secreted in the Gill floor, is not recorded, but changes occur in the oral cavity along with normal skin jaroxates. linical picture. The oral mucosacininin, which is secreted in the Gill floor, is not recorded, but changes occur in the oral cavity along with normal skin jaroxates. The clinical picture of During in herlinical picture. The oral mucosacininin, which is secreted in the Gill floor, is not recorded, but changes occur in the oral cavity along with normal skin jaroxates. The clinical picture of During in herpetic-shaped dermatitis is characterized by polymorphism of rashes. On the hyperemied and swollen mucous membrane of the mouth, papules, blisters and blisters appear. Sometimes only hyperemia occurs in basal groups, subepitelially, with transparent contents and thick-coated vesicles. It makes them look like herpes, so the disease got its name. After 3-4 days, the bubbles open and form a bright red erosion with a combed outline, the pain is poorly expressed. Nikolsky's fracture is negative, there are no acantholytic cells. fter 3-4 days, the bubbles open and form a bright red erosion with a combed outline, the pain is poorly expressed. Nikolsky's fracture is negative, there are no acantholytic cells. Erosion in the oral cavity open and form a bright red erosion with a combed outline, the pain is poorly expressed. Nikolsky's fracture is negative, there are no acantholytic cells. Erosion in the oral cavity occurs for 2-3 weeks, what trace remains of hych after epithelization. The disease is characterized by a cyclical course. The duration of remission periods can vary significantly.

In Dyuring's herpetiform dermatitis, eosinophilia is detected in the peripheral blood and in the contents of the vesicles.

Diagnosis of Dyuring dermatitis the diagnosis is usually based on clinical manifestations (cluster location of OBSHQ vesicles, negative Nikolsky symptom,

absence of acantholytic cells, cyclic retardation of the disease, typical polymorphic rashes on the skin), as well as eosinophilia. Diagnosis of Dyuring dermatitis the diagnosis is usually based on clinical manifestations (cluster locatio Diagnosis of Dyuring dermatitis the diagnosis is usually based on clinical manifestations (cluster location of OBShQ vesicles, negative Nikolsky symptom, absence of acantholytic cells, cyclic retardation of the disease, typical polymorphic rashes on the skin), as well as eosinophilia. based on the increased content of peripheral blood, vesicles and iodine sensitivity.

Comparative diagnosis. The disease is distinct from: Haqiqi acantholytic pemphigus, nonacontolytic pemphigus, multiform exudative erythema.

Treatment. Sulfones, especially dimosiphone, are used for treatment during dermatitis.reatment. Sulfones, especially dimosiphone, are used for treatment during dermatitis. In the case of severe, persistent disease, these drugs are combined with corticosteroids in moderate daily doses (20-30 mreatment. Sulfones, especially dimosiphone, are used for treatment.

Anomalies and independent diseases of the tongue

This section provides information about the anomalies and diseases of the language, which are usually called independent glossitis – it develops only in the language, without affecting other parts of the **OBShQ**.nomalies and independent diseases of the tongue.This section provides information about the anomalies and diseese.



Image 71. Twisted tongue

Twisted tongue

This is an innate anomaly of the shape and size of the tongue. Twisted tongue (lingua plicata) is equally common in children and adults, but children have less deep folds. This is an innate anomaly of the shape and size of the tongue. Twisted tongue (lingua plicata) is equally common in children and adults, but children have less deep folds. This is an innate anomaly of the shape and size of the tongue. Twisted tongue (lingua plicata) is equally common in children and adults, but children have less deep folds. The pleated tongue is characterized by the presence of deep furrows (folds) on its surface, which are located mainly symmetrically in longitudinal and transverse directions. The deepest longitudinal folds are usually located along the midline of the tongue. It originates at the tip of the tongue and often goes to the level of location of the orofacial suckers. Transverse folds are deeper, usually directed to the main and deeper longitudinal folds, which combine them into a single system and create an image of the division of the tongue into many parts. Transverse folds are deeper, usually directed to the main and deeper longitudinal folds, which combine them into a single system and create an image of the division of the tongue. Transverse folds are deeper, usually directed to the main and deeper longitudinal folds, which combine them into a single system and create an image of the division of the tongue into many parts. The lower and side surfaces of the folds are covered with suckers characteristic of the normal mucous membrane of the tongue. Suckers are well expressed, sometimes hypertrophied. The folds are clearly visible when the tongue is stretched. In most cases, this is accompanied by an average increase in the volume of the tongue — macroglossia, in addition, its thickening is often noted. As a developmental anomaly, the folding and enlargement of the tongue can only cover the anterior third of the tongue. Curly tongue can be one of the symptoms of Melkersson-Rosenthal syndrome. As a developmental anomaly, the folding and enlargement of the tongue can only cover the anterior third of the tongue. Curly tongue can be one of the symptoms of Melkersson-Rosenthal syndrome. In addition, as a developmental anomaly, the folding and enlargement of the tongue can only cover the anterior third of the tongue. Curly tongue can be one of the symptoms of

Melkerson-Rosenthal syndrome. In addition, the pleated tongue is compared with desquamative glossitis in 30-50% of cases. In older people, a slight crease of the tongue and langes may occur due to a decrease in mucous membrane turgor. Complaints with a wrinkled tongue are usually not, but they find an unusual appearance of the tongue, and patients see a doctor as cracks in the folds. With an uncannulated oral cavity, saliva depletion, food residues, exfoliated epithelial cells, and microflora accumulate in the folds, which can lead to inflammation, burns, and pain complaints. With an uncannulated oral cavity, saliva depletion, food residues, exfoliated epithelial cells, and microflora accumulate in the folds, which can lead to inflammation, burns, and pain complaints. The presence of an uncannulated oral cavity, saliva depletion,

Black-haired ("hairy") tongue

Black-haired tongue (lingua villosa nigra) is a disease characterized by branching and hyperplasia of the filamentous suckers of the tongue of varying degrees. Black-haired ("hairy") tongue

Black-haired tongue (lingua villosa nigra) is a disease characterized by branching and hyperplasia of the filamentous suckers of the tongue of varying degrees. This is a very rare disease, mainly affecting men, often in middle and old age.

Etiology. Unknown. This disease is often found in people who smoke and abuse alcohol.

Clinical presentation. There may be no complaints. Clinical presentation. There may be no complaints. Often, patients are concerned about the unusual appearance of the tongue, sometimes a feeling of a foreign body on it, a gag reflex when talking, itching in the palate, and decreased taste sensitivity. At the back of the tongue, in front of the grooved sucker, the filamentous sucker extends, thickens, and becomes a light brown to black color. The filamentous sucker sometimes reaches 2 cm in

length and is up to 2 mm in diameter, externally resembling sucker hair. The nipples are thicker at the base but less pigmented than at the tip.

A characteristic feature of the black-haired ("hairy") tongue is that the color of the tongue is determined by the activity of chromatogenic bacteria. characteristic feature of the black-haired ("hairy") tongue is that the color of the tongue is determined by the activity of ch characteristic feature of the black-haired ("hairy") tongue is that the color of the tongue is determined by the activity of chromatogenic bacteria. Most often, hyperplastic suckers are slightly brown to black in color, but sometimes they can be straw yellow, brown, dark gray. The modified part usually has an oval or triangular shape and is located along the middle line. The front of the tongue and its lateral surfaces are not involved in the process and have a normal appearance. This jarochate can last indefinitely without treatment, sometimes it can go away on its own, and after a while it will go back again.his jarochate can last indefinitely without treatment, sometimes it can go away on its own, and after a while it will go back again. During bacterioscopic examination, an increase in the number of leptotrichia is often detected.

Differential diagnosis. Black hairy tongue must be differentiated from clinically similar conditions, such as tongue suckers and false black tongue, which have a distinct pigmentationifferential diagnosis. Black hairy tongue must be differentiated from clinically similar conditions, such as tongue suckers and false black tongue, which have a distinct pigmentation of the tongue. The cause of such pigmentation can be poor oral hygiendifferential diagnosis. Black hairy tongue must be differentiated from clinically similar conditions, such as tongue suckers and false black tongue, which have a distinct pigmentation of the tongue. The cause of such pigmentation can be poor oral hygiene, exacerbation of diseases of the gastrointestinal tract, the use of drugs (washing with solutions of potassium permanganate, chloramine, chlorhyexidine, taking antibiotics, metronidazole, etc.). Differential diagnosis facilitates the identification of the history. When stained with drugs or other exogenous dyes, pronounced hyperplasia of the filamentous sucker disappears; in addition, after stopping the drug or other substance that causes the entire body part

of the tongue to stain, the color will normalize within a few days. When stained with drugs or other exogenous dyes, pronounced hyperplasia of the filamentous sucker.



Figure 72. Black-haired "hairy" tongue. On the back of the tongue there is a triangular area on the elongated filamentous suckers of black color

Treatment. The oral cavity is sanitized (oral hygiene, removal of local traumatic factors). Treatment. The oral cavity is sanitized (oral hygiene, removal of local traumatic factors). For local treatment, it is recommended to use keratolytic agents - lubricate the back of the tongue. The oral cavity is sanitized (oral hygiene, removal of local traumatic factors). For treatment. The oral cavity is sanitized (oral hygiene, removal of local traumatic factors). For local treatment, it is recommended to use keratolytic agents - lubricate the back of the tongue with 3-5% resorcinol solution, 5-10% salicylic alcohol solution (2-3 times a day). For the same purpose, injections of a 0.25% calcium chloride solution are used under jaroxate. 0.5-1 ml 0.25% calcium chloride is administered with 0.5 ml 1-2% novocaine twice a week under jaroxate (for a course of 3-5 injections). Smoking and the abuse of irritating foods are prohibited.

Desquamative glossitis

Most often, desquamative glossitis (glossitis desquamativa, geographical tongue, exfoliative or migratory glossitis) occurs in diseases of the gastrointestinal tract, vegetative-endocrine diseases, rheumatic desquamative glossitis. The disease occurs equally often in different age groups.

Clinical picture. The process begins with the appearance of a white-gray area of epithelial opacity with a diameter of several millimeters. The process

begins with the appearance of a white-gray area of epithelial opacity with a diameter of several millimeters. It then swells and its thread-like suckers move, revealing a bright pink or red circular area against a slightly raised area of fuzzy epithelium. The desquamation area grows rapidly, maintaining smooth rounded contours, but the intensity of desquamation decreases. The epithelial desquamation part can be of different shapes and sizes and represents reddish spots. Sometimes the areas of desquamation have a ring or semicircular shape. In the area of desquamation, a mushroom-shaped papule is clearly visible, which has the appearance of a bright red spot. Sometimes the areas of desquamation have a ring or semicircular shape. In the area of desquamation, a mushroom-shaped papule is clearly visible, which has the appearance of a bright red spot. When the desquamation furnace reaches a significant volume, its boundaries become blurred on the surrounding mucous membrane, and after desquamation in its center, the normal branching of the filamentous suckers begins, in the places of branching, on the contrary, desquamation occurs. Desquamation foci can be single, but often multiple, and are layered on top of each other as a result of constant changes in branching and desquamation processes. Desquamation foci can be single, but often multiple, and are layered on top of each other as a result of constant changes in branching and desquamation processes. Against the background of old kilns, new kilns are formed, as a result of which desquamation foci can be single, but often multiple, and are layered on top of each other as a result of constant changes in branching and desquamation processes. Against the background of old kilns, new kilns are formed, as a result of which The Shape of the desquamation sites and the color of the tongue are constantly changing, which gives the surface of the tongue a geographical map-like appearance. This was the reason for the names "geographical language", "migratory glossitis". The rapid change in the contours of the desquamation foci is characteristic, the picture also changes when checked the next day. Desquamation foci are located on the back and side surfaces of the tongue, usually not spreading to the lower surface. Desquamation foci are located on the back

and side surfaces of the tongue, usually not spreading to the lower surface. In many patients, especially in children, changes in squamation foci are located on the back and side surfaces of the tongue, usually not spreading to the lower surface. In many patients, especially in children, changes in language occur. Squamation foci are located on the back and side surfaces of the tongue, usually not spreading to the lower surface. In many patients, especially in children, changes in language occur without what subjective sensations they are and are accidentally detected during oral examination. Only some patients complain of burns, itching, paresthesia, pain from irritating food. Patients are also concerned about the strange appearance of the tongue; carcinophobia may develop. Emotional stress conditions contribute to a more severe course of the process. Desquamative glossitis, which occurs against the background of gastrointestinal pathology and other systemic diseases, can deteriorate from time to time, which is often associated with an exacerbation of somatic diseases. Desquamative glossitis, which occurs against the background of gastrointestinal pathology and other systemic diseases, can deteriorate from time to time, which is often associated with an exacerbation of somatic diseases. Exacerbation of desquamative glossitis, which occurs against the background of gastrointestinal pathology and other systemic diseases, can deteriorate from time to time, which is often associated with an exacerbation of somatic diseases. Exacerbation of desquamative glossitis is accompanied by an increase in the intensity of desquamation of the epithelium of the mucous membrane of the tongue. Desquamative glossitis is comparable to a curled tongue in about 50% of cases. The disease lasts indefinitely, without worrying patients, sometimes disappears for a long time, then reappears in those or other places. There are cases where desquamation occurs mainly in one place.



Image 73. Desquamative glossitis.

Diagnosis Diagnosing the disease is not difficult, as its clinical signs are very characteristic. It is necessary to distinguish between desquamative glossitis: leukoplakia, plaque formation in secondary syphilis; hypovitaminosis B₂, B₆, B₁₂; allergic stomatitis. Diagnosing the disease is not difficult, as its clinical signs are very characteristic. It is necessary to distinguish between desquamative glossitis: leukoplakia, plaque formation in secondary syphilis; hypovitaminosis B₂, B₆, B₁₂; allergic stomatitis. Histological changes are characterized by thinning of the epithelium and flattening of the filamentous suckers at the site of desquamation, parakeratosis in the epithelium of the areas around the leukoplakia, and moderate hyperkeratosis. There is mild swelling and inflammatory infiltration in the mucous layer.

Treatment. If there are no complaints or unpleasant sensations, treatment is not performed. In case of burning sensation, pain, sanitation of the oral cavity, elimination of various excitation-specific substances and reasonable oral hygiene are recommended. Hygienic recommendations are especially relevant in the combination of desquamative glossitis with a pleated tongue, in which the anatomical features of the structure create favorable conditions for increased

microflora in the folds, which can cause pain-causing inflammation. Mouth baths with a solution of burning sensation, pain, mild antiseptic obrobotka, irrigation and citral (25-30 drops of 1% citral injection per half a glass of water), the use of 5-10% anesthetic suspension in a solution of vitamin ye oil, carotolin, etc. outh baths with a solution of burning sensation, pain, mild antiseptic obrobotka, irrigation and citral (25outh baths with a solution of burning sensation, pain, mild antiseptic obrobotka, irrigation and citral (25-30 drops of 1% citral injection per half a glass of water), the use of 5-10% anesthetic suspension in a solution of vitamin ye oil, carotolin, etc. Good results are achieved with calcium pantothenate treatment (0.1-0.2 g 3 times a day orally for a month). In some patients, the tongue nerve area is positively affected by the use of Novocaine blockades. (For 10 injection courses). In severe pain, it is recommended to prescribe local pain relievers. This treatment is symptomatic, aimed at eliminating or reducing pain, and reducing the frequency of relapses. Patients often develophis treatment is symptomatic, aimed at eliminating or reducing pain, and reducing the frequency of relapses. Patients often develop cancerophobia.



Figure 74. Rhombic-shaped glossitis.

On the back of the tongue there is a pink, oval-shaped lumpy formation protruding from the surface of the mucous membrane.

Rhomboid glossitis

Rhombic glossitis (glossitis mediana rhombica) - chronic inflammatory disease is characterized

Etiology and pathogenesis. Not fully defined. Currently, most research on glossitis (glossitis mediana rhombica) - chronic inflammatory disease is characterized by its rhombic-shaped or oval-shaped appearance, when held, solid, filamentous suckers are located along the anterior midline. Clinically, rhombic-shaped glossitis is manifested by its rhombic-shaped or oval-shaped appearance, when held, solid, filamentous suckers are located along the anterior midline. In different patients, the size of these wounds ranges from 1.5 to 5 cm in length and 0.5 to 2.5 cm in width. The shape resembles a rhombus, which is why it is called "rhomboid glossitis." Jarochat foci are 2-3 located along the midline of the tongue in one rare case. Suckers disappear on the surface of the tongue.

Clinical picture. Clinically, rhombic-shaped glossitis is manifested by its rhombic-shaped or oval-shaped appearance, when held, solid, filamentous suckers are located along the anterior midline. In different patients, the size of these wounds ranges from 1.5 to 5 cm in length and 0.5 to 2.5 cm in width. The shape resembles a rhombus, which is why it is called "rhomboid glossitis." Jarochat foci are 2-3 located along the midline of the tongue in one rare case. Suckers disappear on the surface of the tongue.

Rhomboid glossitis is a chronic disease that lasts for years and usually does not progress. Rhomboid glossitis is a chronic disease that lasts for years and usually does not progress. The variety of clinical course of rhombic glossitis has allowed us to distinguish three forms of the disease: flat (or smooth), hailstone (or uneven) and papillomatosis (or hyperplastic). With a flat shape, the surface of the tongue in the jarokhat area is smooth, does not rise above the surrounding mucous membrane, has a pink or red color, sometimes (mainly in older people) cyanotic appearance suckers do not occur in the affected Sox during palpation, it can be determined that the jarokhat area is slightly resilient, painless under the jaw lymph nodes

The rhombus-shaped, round or oval jarochat area in the form of a drum consists of bindings of different sizes, separated from each other by clearly defined folds.

rhombus-shaped, round or oval jarochat area in the form of a drum consists of bindings of different sizes, separated from each other by clearly dehe rhombus-shaped, round or oval jarochat area in the form of a drum consists of bindings of different sizes, separated from each other by clearly defined folds. The rhombus-shaped, round or oval jarochat area in the form of a drum consists of bindings of different sizes, separated from each other by clearly defined folds. This arrangement of the folds resembles a granite pavement. The surface of the bumps and folds is red with a cyanotic tint, there will be no suckers. The most obvious changes are observed in the sucker-like form of rhombic glossitis. Jaroxate grows like a swollen tumor that rises above the surface of the tongue. The furnace often grows to 3-5 cm in diameter. During palpation, stiffness is not detected or determined by name. With tuberculous and papillomatous forms, the surface of the center of the lesion may become branched, then it becomes milky white. With tuberculous and papillomatous forms, the surface of the center of the lesion may become branched, then it becomes milky white. The Jaroxat area does not increase during the life of the p tuberculous and papillomatous forms, the surface of the center of the lesion may become branched, then it becomes milky white. The Jaroxat area does not increase during the life of the patient, he constantly retains its shape and size. All three clinical forms of Rhombus glossitis can occur without subjective signs. Many patients are unaware that they have these changes unless they are noticed - complaints of burning, thickening of the back of the tongue, and sometimes pain in advanced cases of inflammation. The prognosis for rhomboid glossitis is poor. The clinical and morphological picture has remained unchanged for many years. The prognosis for rhomboid glossitis is poor. The clinical and morphological picture has remained unchanged for many years. Only in unfavorable conditions (constant chronic irritation, he prognosis for rhomboid glossitis is poor. The clinical and morphological picture has rerognosis for rhomboid glossitis is poor. The clinical and morphological picture has remained unchanged for many years.

Differential diagnosis. The flat form is distinguished from: desquamative glossitis; and beriberi; papillomatous and papillomatous: tumors; candidiasis is compared with.

Treatment. The oral cavity is sanitized to eliminate local traumatic factors and smoking cessation is recommended. The oral cavity is sanitized to eliminate local traumatic factors and smoking treatment. The oral cavity is sanitized to eliminate local traumatic factors and smoking cessation is recommended. When a fungus of the Candida treatment. The oral cavity is sanitized to eliminate local traumatic factors and smoking cessation is recommended. When a fungus of the Candida genus is detected,

Cheilitis

Cheilitis is a benign inflammatory disease of the lips. There are two groups of cheilitis: **Proper Cheilitis** and **Symptomatic Cheilitis**. The **Proper Cheilitis** group unites various etiologically independent lip diseases. These include exfoliative cheilitis, glandular cheilitis, meteorological cheilitis, and actinic cheilitis. The second group, **Symptomatic Cheilitis**, includes lesions of the lips that are symptoms of diseases of the oral mucosa, skin diseases, and general somatic diseases. The symptomatic cheilitis group includes atopic cheilitis, eczematous cheilitis, allergic contact cheilitis, and macrocheilitis in Melkersson-Rosenthal syndrome. Exfoliative Cheilitis

Exfoliative cheilitis (*cheilitis exfoliativa*) is a chronic disease that affects only the vermilion border (red margin) of the lips. This disease was first described by Stelwagon in 1900 under the name "persistent desquamation of the lips." Later,

Mikulicz and Kümmel proposed the name "exfoliative cheilitis." Exfoliative cheilitis occurs more often in women aged 20 to 40 years.

Etiology and Pathogenesis: Not fully studied. Many researchers believe that neurogenic factors lie at the basis of the disease. Various variants of psycho-emotional disorders have been identified in patients with exfoliative cheilitis. There is evidence of a link between thyroid diseases and exfoliative cheilitis. The role of genetic predisposition in the occurrence of exfoliative cheilitis has been noted. Immunological factors play a significant role in the pathogenesis of the disease.

Clinical Picture: There are two clinical forms of exfoliative cheilitis: **Dry** and **Exudative**. Pathological changes are located on the vermilion border of both lips or one of them and affect only part of the vermilion border from the Klein line (wet-dry line) to the center. The affected area stretches like a ribbon from one corner of the mouth to the other. The part of the vermilion border adjacent to the skin, as well as the corners of the mouth, are not affected. The process never spreads to the skin and mucous membrane of the mouth.

Dry Form: Characterized by the appearance of persistent hyperemia on the surface of the vermilion border, upon which dry, transparent, mica-like scales of a gray or gray-brown color form in the affected area. The lips are dry and peeling. The scales are removed very easily, and a persistent red surface is revealed underneath them, but erosion does not form. After 5-7 days, scales resembling mica form again. The course of the disease is long-term. The dry form of exfoliative cheilitis can transform into the exudative form.

Exudative Form: Characterized by severe pain, swelling of the lip, and hyperemia. In the Klein zone, a large number of scabs and crusts of a gray-yellow or yellow-brown color form, covering the affected area from corner to corner of the mouth. Sometimes the crusts reach significant sizes and hang from the lip in the form of an apron, causing damage to the entire vermilion border. However, the line of the vermilion border adjacent to the skin, as well as the corners of the mouth, are not affected. After removing the crusts, a bright hyperemic surface of the lip without erosion is revealed, which is a characteristic feature of exfoliative cheilitis. Patients

with the exudative form of exfoliative cheilitis are bothered by a burning sensation and pain in the lips, especially when closing them, which makes eating and speaking difficult; therefore, these patients' mouths are often slightly open. The reason for the appearance of distinct exudative phenomena is considered to be a sharp increase in capillary permeability. Under the influence of treatment, the exudative form of exfoliative cheilitis can transform into the dry form.

Pathohistology: Acanthosis, para- and hyperkeratosis are noted. This is characterized by the presence of a large number of cells in the spinous layer of the epithelium with reduced metabolic activity, unlike other actively working cells. There is loosening of the epithelial layer and the appearance of wide gaps in it due to intracellular lysis, which contributes to increased exudation.

Differential Diagnosis: The dry form of exfoliative cheilitis must be differentiated from: Meteorological cheilitis, Atopic cheilitis, and Allergic cheilitis.

Meteorological cheilitis: The entire surface of the vermilion border is damaged as a result of increased inflammation. Furthermore, the course of meteorological cheilitis is determined to be directly dependent on the influence of various meteorological factors.

Atopic cheilitis: Affects the part of the vermilion border adjacent to the skin and the corners of the mouth. It does not affect the red lip area adjacent to the mucous membrane. The alternation of periods of lichenification and relapse with remissions in atopic cheilitis often has a seasonal character.

Allergic contact cheilitis: Unlike the dry form of exfoliative cheilitis, it is characterized by the presence of erythema in the area of contact with the allergen, as well as the rapid disappearance of inflammatory conditions after the allergen is eliminated.

The exudative form of exfoliative cheilitis must be differentiated from: The exudative form of actinic cheilitis, eczematous cheilitis, and the erosive-ulcerative form of lupus erythematosus.

Exudative form of actinic cheilitis: A connection with the season of the year is observed; the lesion covers the entire surface of the vermilion border, and its swelling and the presence of erosion are noted.

Eczematous cheilitis: Expressed by polymorphic rashes (erosions, crusts, vesicles, cracks), frequent changes in lesion elements, and periods of relapse and remission. In eczematous cheilitis, the lesion covers the entire surface of the vermilion border and the transitional part of the face to the skin.

Erosive and ulcerative form of lupus erythematosus: Unlike the exudative form of exfoliative cheilitis, it proceeds with significantly distinct erythema, hyperkeratosis, formation of erosions and ulcers, and cicatricial atrophy.

Treatment: Treatment should be comprehensive and have differences for the dry and exudative forms of exfoliative cheilitis. Important significance is given to means affecting the psycho-emotional sphere. For this purpose, patients are prescribed tranquilizers: Phenazepam 0.0005 g 3 times a day, Sibazon (Seduxen, Relanium) 0.005 g 3 times a day, or neuroleptics: Thioridazine (Melleril, Sonapax) 0.01 g 3 times a day. Considering the predominance of depressive reactions in patients with the dry form of exfoliative cheilitis, they should be prescribed antidepressants (Amitriptyline, Melipramine 0.025 g 2 times a day) and tranquilizers: Trioxazine 0.03 g 3 times a day, Chlordiazepoxide (Elenium) 0.01 g 3 times a day. Locally, indifferent creams "Delight," "Spermaceti," and hygienic lipstick are recommended for lubricating the lips for patients with the dry form. For the treatment of patients with the exudative form of exfoliative cheilitis, the use of complex therapy, including the use of borderline radiation (Bucca radiation / Grenz rays), is effective – 2 Gy 2 times a week with a total dose of 16 to 30 grams (Gy). Before each session, crusts must be removed from the vermilion border. Patients with the exudative form of exfoliative cheilitis should undergo 6-8 courses of complex treatment with a break of 3-4 months. Reflexotherapy is successfully used to eliminate swelling, pain, and burning in the exudative form of exfoliative cheilitis.

Glandular Cheilitis

Glandular cheilitis (*cheilitis glandularis*) is a disease of the lip, mainly the lower lip, developing due to hyperplasia, hyperfunction, and sometimes heterotopia of the salivary glands in the area of the vermilion border and the transition zone (Klein zone). Primary and Secondary cheilitis are distinguished. The development of primary cheilitis is associated with a congenital anomaly of the salivary glands. Secondary cheilitis, as a rule, develops against the background of various diseases located on the lips (Lupus Erythematosus, Leukoplakia, etc.).

Etiology: In the etiology of primary glandular cheilitis, the leading role is assigned to hereditary anomalies of the lower lip with hyperplasia, heterotopia, and excessive secretion of minor salivary glands. Secondary cheilitis develops as a result of chronic inflammatory processes in the vermilion border. Consequently, in leukoplakia, lupus erythematosus, and other diseases located on the lips, inflammatory infiltration causes irritation, hyperplasia, and hyperfunction of the glandular tissue. Secretion, maceration of the lip, and an increase in infection are detected. However, these phenomena develop only in some people.

Clinical Picture: Glandular cheilitis develops mainly in people over 50 years of age. Primary cheilitis is detected 2 times more often in men than in women. The lower lip is affected twice as often as the upper lip. Glandular cheilitis has a typical clinical picture. In the area of the transition of the mucous membrane to the vermilion border and sometimes on the red border, the dilated openings of the salivary glands are visible in the form of red dots, from which droplets of saliva emerge. Sometimes leukoplakia develops in the form of rings around the mouths of the salivary glands, and in some cases, the vermilion border keratinizes for a long time. Due to frequent wetting of the lips with saliva and evaporation of the latter, dryness of the vermilion border develops, and maceration, cracks, and erosion appear. Subsequently, the mucous membrane and red border may gradually keratinize. Initially, this occurs around the mouths of the excretory ducts of the salivary glands, then diffusely. With the appearance of cracks and foci of hyperkeratosis due to constant irritation of the lip, with a long course of glandular cheilitis, it can proceed unfavorably with the

development of precancerous lesions. As a result of the penetration of pyogenic infection into the dilated ducts of the salivary glands, glandular cheilitis can turn into a purulent form. This is characterized by induration and swelling of the lip, covered with thick crusts of yellow-green or brown-black color. Purulent discharge from the dilated ducts of the salivary glands, cracks, and erosions are located around them. Sometimes the excretory ducts of the glands may be clogged with purulent exudate, which leads to the development of multiple or single abscesses.

Diagnosis: The clinical manifestations of glandular cheilitis are so characteristic that diagnosis, as a rule, does not cause difficulties. Pathohistological examination reveals hypertrophy of the salivary glands with minor inflammatory infiltration, mainly around their excretory ducts. In some patients, acanthosis and parakeratosis are observed in the epithelium.

Treatment: To treat primary glandular cheilitis, anti-inflammatory ointments (prednisolone, hydrocortisone, naphthalene, etc.) are prescribed; electrocoagulation or excision of hypertrophied salivary glands is used. Treatment of secondary glandular cheilitis primarily ensures the treatment of the underlying disease that caused it, and anti-inflammatory treatment is carried out locally.

Outcome: The prognosis for glandular cheilitis is satisfactory, but in the absence of timely and effective treatment, precancerous diseases may develop against its background.

Actinic and Meteorological Cheilitis

Actinic and Meteorological Cheilitis (*cheilitis actinica*, *cheilitis meteorologica*) are inflammatory diseases of the lips caused by various meteorological factors (solar radiation, high or low humidity, wind, dust in the air, cold). Unlike meteorological cheilitis, the leading factor in **actinic cheilitis** is the presence of high sensitivity to ultraviolet rays. These diseases occur more often in men aged 20 to 60 years.

Actinic Cheilitis (*Cheilitis actinica*). The main cause of actinic cheilitis is the development of a delayed reaction to ultraviolet rays.

Clinical Picture There are dry and exudative forms of actinic cheilitis. Dry Form: Manifests as dryness of the lower lip; the upper lip and facial skin are rarely affected.

The lesion covers the entire surface of the vermilion border; it is bright red and covered with small, dry, silver-white scales. Later, abrasions and erosions may appear. In some patients, hyperkeratosis and sometimes verrucous (wart-like) growths appear on the vermilion border. With a long-term course of actinic cheilitis and the addition of irritating factors (smoking, humidity, insolation), it can transform into a malignant process. **Exudative Form:** Characterized by bright red erythema, small vesicles (blisters), wet erosions, and the appearance of crusts on the swollen, hyperemic surface of the lower vermilion border. Patients are concerned about itching, burning, and pain in the lips. The course of actinic cheilitis is seasonal: exacerbation of the disease is observed in spring and summer, and remission in autumn and winter.

Differential Diagnosis. Dry form: Must be distinguished from the dry form of exfoliative cheilitis and lupus erythematosus. Exudative form: Differentiated from allergic contact cheilitis and atopic cheilitis.

Treatment First of all, it is necessary to avoid the causative factors as much as possible; if it is associated with a long stay outdoors, it may be necessary to change professions. Nicotinic acid and B-group vitamins (B2, B6, B12) are prescribed. In some cases, they are combined with antimalarial drugs (Delagil 0.25 g, 2 times a day for 2-3 weeks) and small doses of corticosteroids (e.g., Prednisolone 10 mg per day). Corticosteroid ointments are used locally (0.5% Prednisolone, "Flucinar", "Locacorten", "Lorinden", etc.).

Prognosis Satisfactory, but with a prolonged course of the dry form, the development of a malignancy process is possible.

Meteorological Cheilitis (*Cheilitis meteorologica*)

This is an inflammatory disease of the lips, the cause of which is various meteorological factors (increased or decreased humidity, solar radiation, dust, wind, cold, etc.). The duration of exposure to these factors and the constitutional characteristics of the skin play an important role in the development of this disease. Meteorological cheilitis often occurs in people with light and delicate skin or in

people with skin diseases accompanied by increased dryness (seborrhea, seborrheic eczema, diffuse neurodermatitis, etc.).

Clinical Picture In meteorological cheilitis, the vermilion border is affected; usually, the lower lip is affected along its entire length. The vermilion border is slightly hyperemic (red), infiltrated, dry, and often covered with small scales. Patients are concerned about dry lips, a sensation of tightness, and peeling. Long-term exposure to meteorological factors contributes to the appearance of erosions or cracks.

Histology Epithelial hyperplasia, mild keratinization, and stromal infiltration are detected.

Differential Diagnosis Meteorological cheilitis is differentiated from:

The dry form of actinic cheilitis.

Allergic contact cheilitis.

The dry form of exfoliative cheilitis.

Treatment (*Note: The source text repeats the treatment protocol for Actinic Cheilitis here*) First of all, avoid the causative factors as much as possible; if associated with long outdoor exposure, a change of profession may be necessary. Nicotinic acid and B-group vitamins (B2, B6, B12) are prescribed. In some cases, they are combined with antimalarial drugs (Delagil 0.25 g, 2 times a day for 2-3 weeks) and small doses of corticosteroids (e.g., Prednisolone 10 mg per day). Corticosteroid ointments are used locally (0.5% Prednisolone, "Flucinar", "Locacorten", "Lorinden", etc.).

Prognosis Satisfactory, but with a prolonged course, in rare cases, there is a possibility of transition to an obligate precancerous state (Manganotti's abrasive precancerous cheilitis, circumscribed hyperkeratosis).

Allergic Contact Cheilitis

Allergic Contact Cheilitis (*Cheilitis allergica contactilis*) develops as a result of sensitization of the vermilion border upon contact with various substances. The development of this disease is based on a delayed allergic reaction to various chemicals. Allergic contact cheilitis occurs mainly in women over 20 years of age.

Etiology and Pathogenesis Allergic contact cheilitis is the clinical manifestation of a delayed allergic reaction to substances that come into contact with the vermilion border. Often, the cause of allergic contact cheilitis is lipstick, toothpaste, and denture plastics. Cheilitis can also develop upon contact with metal objects (wind instruments, pens, pencils, etc.).

Clinical Picture The process usually develops on the vermilion border, sometimes spreading slightly to the skin. Combined lesions of the vermilion border and the oral mucosa are less common. Erythema (redness) of the vermilion border develops at the site of contact with the allergen. With a sharp increase in inflammatory phenomena, small vesicles appear on the hyperemic red border, which quickly burst and form erosions and cracks. Patients complain of severe itching, burning, and swelling of the lips. Allergic contact cheilitis can occur without a significant inflammatory reaction. In such cases, only limited erythema and slight peeling are present on the vermilion border. With a long course of the disease, the vermilion border becomes dry, and small grooves and cracks appear on it.

Differential Diagnosis The disease must be distinguished from:

Exfoliative cheilitis.

The dry form of actinic cheilitis.

Atopic cheilitis.



Figure 75. Allergic Contact Cheilitis (Continued)

Treatment Treatment of allergic contact cheilitis primarily consists of identifying and eliminating the factors that caused the disease. For local treatment, corticosteroid-containing ointments (0.5% prednisone, "Flucinar", "Lorinden") are

used; they are applied to the surface of the vermilion border 5-6 times a day. Desensitizing drugs (Suprastin, Tavegyl, Phencarol, etc.) are prescribed for oral administration.

Atopic Cheilitis

Atopic Cheilitis (*cheilitis atypicalis*) is one of the signs of atopic dermatitis or neurodermatitis, meaning it belongs to the group of symptomatic cheilitis. The disease occurs more often in children and adolescents of both sexes aged 7 to 17 years.

Etiology Genetic factors that predispose to a condition called atopic allergy play an important role in the etiology of atopic cheilitis. Allergens can be drugs, food products, floral and household dust, microorganisms, cosmetics, and others.

Clinical Picture In atopic cheilitis, the vermilion border of the lips is affected with the involvement of adjacent skin areas in the pathological process, and the lesion is most severe in the area of the corners of the mouth.

Infiltration appears on the vermilion border, and it peels with small scales. Infiltration and dryness of the lips eventually lead to the formation of cracks (fissures) and grooves (lichenification). The process never spreads to the oral mucosa. Changes in the lips are often combined with dryness and peeling of the facial skin. Patients with atopic cheilitis complain of itching, burning, and peeling of the vermilion border. Atopic cheilitis lasts for a long time; exacerbation of the disease occurs mainly in the autumn-winter period, while remission usually occurs in the summer.

Differential Diagnosis When conducting a differential diagnosis for atopic cheilitis, it is necessary to take into account skin lesions, which are present in many cases. Usually, rashes are found on the facial skin and the skin of the elbow folds (antecubital fossa).



Figure 76. Atopic Cheilitis (Continued)

Atopic Cheilitis: The vermilion border of the lips has infiltrated small **nodules/bumps** and **fissures (cracks)**.

Differential Diagnosis

Atopic cheilitis must be differentiated from clinically similar conditions:

Actinic cheilitis

Allergic cheilitis

Contact allergic cheilitis

Streptococcal angular cheilitis (perleche)

Treatment. Desensitization therapy plays an important role in the treatment of atopic cheilitis.

Oral Medications: Antihistamines (Suprastin, Diazolin, Claritin, Phencarol) are prescribed for oral consumption.

Systemic Corticosteroids: In cases of a prolonged, persistent course of the disease, corticosteroids may be prescribed orally for 2-3 weeks (Prednisone 10-15 mg per day for children aged 8-14, 15-20 mg per day for adults, or Dexamethasone).

IV Therapy: Intravenous infusions of a 30% sodium thiosulfate solution are also used (5-10 ml per day, for a course of up to 10 injections).

Histaglobulin: In some patients, the use of **Histaglobulin**, administered in courses of 6-8 injections, yields good results; the preparation is administered subcutaneously in doses ranging from 0.2 ml to 1 ml, twice a week.

Vitamins: B vitamins (B1, B6, B12) are also prescribed for oral consumption.

Local Treatment: A topical **corticosteroid ointment** (0.5% Prednisolone, Lorinden, Flucinar, etc.) should be applied to the affected surface 4-5 times a day.

Radiation Therapy: Bucca borderline rays (Grenz rays) are used when conservative treatment shows minimal effect and the disease has a persistent course.

Diet: It is necessary to strictly limit the consumption of food substances that can lead to sensitization: **caviar, chocolate, coffee, strawberries, citrus fruits**, etc., as well as spicy, salty, pungent food, and alcohol; carbohydrate consumption must also be sharply restricted.

Eczematous Cheilitis

Eczematous Cheilitis (*cheilitis eczematosa*) belongs to the group of symptomatic lip diseases, as it manifests as a symptom of a **generalized eczematous process** based on inflammation of the superficial layers of the skin of a **neuroallergic nature**. In this case, the allergens can be various factors: microorganisms, food substances, medicines, materials for prosthetic manufacture, metals such as nickel and chromium, amalgam, filling materials, and others.

Clinical Picture

Eczematous cheilitis, like any eczema, can proceed acutely, subacutely, or chronically. Damage to the vermilion border may occur either simultaneously with or separately from damage to the facial skin.

Acute Stage: Characterized by **polymorphism** (the presence of multiple types of lesions): **vesicles (blisters), scabs (crusts), and scales**. The process is accompanied by significant **swelling** of the lips. At this stage, patients complain of **itching, burning, swelling, and hyperemia** on the lips. The spread of the process to the skin adjacent to the vermilion border is highly characteristic.

Chronic Stage: Characterized by a decrease in acute inflammatory phenomena (swelling, hyperemia). The vermilion border and the affected areas of the skin around the mouth thicken due to inflammatory infiltration; nodules and crusts appear. Vesicle formation stops, and scaling/peeling occurs. In this state, the process can last for a long time.

All the described clinical signs of eczematous cheilitis develop on previously unchanged lips. Sometimes, prolonged microbial angular cheilitis (perleche) or cracks precede the onset of the eczematous process. In such cases, the developing eczema is usually regarded as microbial sensitization of the vermilion border or skin. The clinical picture of microbial eczema on the vermilion border is absolutely identical to the described clinical picture of eczematous cheilitis.

Differential Diagnosis

Eczematous cheilitis is differentiated from:

Atopic cheilitis

Allergic contact cheilitis

The exudative form of actinic cheilitis

Treatment. Desensitizing and soothing drugs are prescribed. Local treatment consists of prescribing ointments containing corticosteroids; corticosteroid aerosols are also used. For microbial eczema, combined corticosteroid and antimicrobial agents are prescribed

Macrocheilitis

Macrocheilitis is the leading symptom of a disease first described in 1901 by G. I. Rossolimo; subsequently, the Swedish physician Melkersson described the disease characterized by unilateral facial nerve paralysis and lip swelling. Later, in Rosenthal's description of the disease in 1931, the symptom of a fissured (scrotal) tongue was added to these two signs. Since then, the condition manifesting with this triad of symptoms has been called Melkersson-Rosenthal Syndrome.

Etiology. In the etiology of this disease, a number of authors assign an important role to the constitutional and hereditary characteristics of the person. Others consider Melkersson-Rosenthal Syndrome to be a form of angioedema (Quincke's edema). The infectious-allergic theory of the disease's origin also has its proponents.

Clinical Picture. Melkersson-Rosenthal Syndrome is characterized by three main symptoms: Macrocheilitis, Fissured Tongue, and Facial Nerve Paralysis. The disease is more common in women. The disease usually begins suddenly. As a rule,

swelling of one or both lips is the first symptom. Sometimes, the first sign may be facial pain of the neuralgic type, which often precedes facial nerve paralysis. **Macrocheilitis (Lip Swelling):** Swelling of one or both lips appears within a few hours. Patients note itching of the lip, its enlargement, and sometimes the appearance of swelling in other parts of the face. The lip shows shapeless swelling; it is glossy, the color is usually unchanged, though in some cases, it acquires a bluish tint. Sometimes the swelling is so great that the lips become 3-4 times thicker than their normal size. The thickening is often uneven, with one side more swollen. Upon palpation, the lip tissue feels uniformly soft or dense elastic (firm). The indentation after palpation disappears. This swelling can be persistent, fluctuating, or disappear spontaneously only to recur and become permanent. Swelling is often located on the lips, cheeks, eyelids, and other parts of the face. **Facial Nerve Paralysis (FNP):** This is the second symptom. It involves unilateral vasomotor rhinopathy, unpleasant sensations in the mouth, and changes in salivation. Due to the loss of tone in the affected half of the face, the corner of the mouth drops, and the palpebral fissure (eye opening) widens. **Fissured Tongue (Plicated Tongue):** This is the third symptom, observed in 2/3 of patients. It is considered an anomaly of development rather than a primary disease symptom. There is clinically significant swelling of the tongue, leading to its uneven enlargement.



Figure 77. Macrocheilitis in Melkersson-Rosenthal Syndrome (Continued)

Macrocheilitis in Melkersson-Rosenthal Syndrome: The lower lip is swollen, enlarged, and its color is often unchanged. In patients with Melkersson-Rosenthal

Syndrome, a combination of all three symptoms is not always present; sometimes the disease may clinically manifest only as **macrocheilitis**.

DiagnosisIf the patient has all three symptoms of the disease (macrocheilitis, fissured tongue, facial paralysis), diagnosing Melkersson-Rosenthal Syndrome is not difficult. Difficulties arise only when a single symptom—macrocheilitis—is present. In this case, differential diagnosis is performed with:

Quincke's Edema (Angioedema)

Erysipelas

Lymphangioma

Hemangioma

Treatment.Treatment is a very difficult task. Every patient must be thoroughly examined to identify the pathological change that contributes to the onset of the disease. Particular attention is paid to identifying foci of chronic infection, including in the maxillofacial region.

Systemic Therapy: Treatment of patients with Melkersson-Rosenthal Syndrome involves prescribing corticosteroids (Prednisolone 20-30 mg per day or Dexamethasone 2-3 mg) in combination with broad-spectrum antibiotics (Oletetrin 750,000–1,000,000 units per day) and synthetic antimalarial drugs (Delagil 0.25 g twice a day, for a course of 20-25 g). Antihistamines, as well as Vitamins C and B, are also used. The combination of drugs used for treatment is determined by the patient's general condition, age, clinical symptoms, and the stage of the disease. The indicated course of treatment is carried out over 30–40 days.**Hyposensitization:** If the patient has microbial allergy, specific hyposensitization therapy is conducted.**Chronic Forms:** During remission periods, persistent, chronic forms of the disease are treated with pyrogenic drugs (Pyrogenal, Prodigiosan, etc.).**Local Therapy:** Electrophoresis with Heparin and Dimexide is used to treat macrocheilitis.**Surgery:** Surgical methods are used if conservative treatment is ineffective. For cosmetic purposes, a portion of the lip tissue is excised. However, surgical treatment does not prevent the recurrence of the disease.**Prognosis:** The best results in the treatment of Melkersson-Rosenthal Syndrome are achieved in the initial stages of

the disease. It is recommended to repeat the indicated courses of treatment after 2–3 months.

Precancerous Diseases of the Oral Mucosa and Vermilion Border

Timely diagnosis of precancerous diseases of the oral mucosa and the vermillion border of the lips is the key link in cancer prevention. Cancer of the vermillion border and oral mucosa is quite common, accounting for approximately 5% of all malignant tumors. The lower vermillion border (lateral part) is most often affected; in the oral cavity, the tongue (lateral surface) and the floor of the mouth are most affected. Men over 40 years old predominate among patients with cancer of the vermillion border and oral mucosa. In many patients, cancer is preceded by certain diseases of the oral mucosa and vermillion border, which are called precancerous (pre-malignant) conditions. Their appearance is primarily caused by trauma, especially chronic trauma, including smoking and chewing tobacco, betel, *nas* (a form of chewing tobacco), and alcohol consumption. Trauma is regarded as an external factor in carcinogenesis. Cancer is often preceded by proliferative processes, benign tumors, and chronic inflammatory diseases accompanied by erosion and ulcers. The precancerous disease exists for a long time (from several months to decades), after which it may turn into cancer (but not necessarily). Timely detection and treatment of precancerous diseases eliminates the risk of cancer or allows for timely, more effective treatment. Depending on the frequency of malignant transformation, precancerous diseases of the oral mucosa and vermillion border are distinguished as obligate and facultative. Obligate precancerous diseases have a high risk of transition to a malignant process; if not treated in time, they usually turn into cancer. Many of them form carcinoma in situ (cancer in place) from the beginning of their development. Facultative precancerous disease does not always lead to the development of cancer. The classification of precancerous diseases of the oral mucosa and vermillion border proposed by A. L. Mashkilleyson in 1970 and subsequently confirmed (in 1976) is generally accepted.

Clinical Picture. In the oral mucosa, usually a single lesion is found, less often two or more. The disease manifests as a spot-nodular lesion. Initially, a bright-colored

spot with a smooth or velvety surface is formed due to the growth of papillae in the mucosa. With the long-term course of the disease, slight atrophy of the mucous membrane occurs, as a result of which the lesion slightly sinks relative to the surrounding tissue, and foci of bleeding erosions appear. The central part of the lesion resembles a small, hyperemic bump, similar to leukoplakia. When several foci merge, an irregularly shaped plaque is formed. The lesion diameter can range from 1–2 mm to 5–6 cm; its contours are clear, but uneven, and no induration (hardness) is detected at the base. In the oral mucosa, Bowen's disease is located on the soft palate, uvula, retromolar area, and tongue. When pathological elements are located on the tongue, the papillae disappear. Regional lymph nodes are usually not detectable. Subjective symptoms are typically absent, but pain may be expressed in the presence of erosion. Not all cases of Bowen's disease have a typical clinical picture. The disease may be characterized only by a small amount of hyperemia or resemble leukoplakia without significant inflammation. Sometimes a distinct gray-white pattern appears, reminiscent of Lichen Planus. The disease has a **long course**; in some cases, invasive growth occurs quickly, and trauma accelerates the process. In other patients, the disease remains in the **carcinoma in situ** stage for years.

Bowen's disease

Bowen's disease (morbus Bowen) was first diagnosed by Bowen in 1912.

Clinical picture. More single, less often two or more jarochat foci are found on the mucous membrane of the mouth. The disease is manifested by blotchy nodular discharge. linical picture. More single, less often two or more jarochat foci are found on the mucous membrane of the mouth. The disease linical picture. More single, less often two or more jarochat foci are found on the mucous membrane of the mouth. The disease is manifested by blotchy nodular discharge. Initially, a brightly colored spot with a smooth or velvety surface forms on the mucous membrane due to the growth of suckers. With a long course of the disease, mild atrophy of the mucous membrane occurs, as a result of which the wound sinks slightly compared to the surrounding tissues, and bleeding erosions appear. The central part of the jarochate furnace is similar to a hypyremia-like fine-grained bulge, similar to leukoplakia.e

central part of the jarochate furnace is similar to a hypyremia-likhe central part of the jarochate furnace is similar to a hypyremia-like fine-grained bulge, similar to leukoplakia. When sevehe central part of the jarochate furnace is similar to a hypyremia-like fine-grained bulge, similar to leukoplakia. When several kilns combine, an irregularly shaped blyashka is formhe central part of the jarochate furnace is similar to a hypyremia-like fine-grained bulge, similar to leukoplakia. When several kilns combine, an irregularly shaped blyashka is formed. Jaroxate can be from 1-2 mm to 5-6 cm in diameter, its contours are clear, uneven, at the base the hardness is not determined. The oral mucosa of Bouen's disease localizes to the soft palate, lingual, retromolar soxa, and tongue. The pathological elements were located in the language, the suckers disappeared. Maxillary lymph nodes are usually not detected. There will be no subjective signs-only erosion can represent pain.

Not all cases of Bowen's disease have a typical clinical picture. The disease can only be characterized by a small amount of hyperemia or similar to leukoplakia without significant inflammation.ot all cases of Bowen's disease have a typical clinical picture. The disease can only be characterized by a small amount of hyperemia or similar to leukoplakia without signifiot all cases of Bowen's disease have a typical clinical picture. The disease



Figure 78. Bowen's Disease

Histologically, Bouen's disease provides intra-epithelial cancer curination: polymorphism of stem layer cells, increased number of mitoses, and

disordered. histologically, Bowen's disease provides intra-epithelial cancer curination: polymorphism of stem layer cells, increased number of mitoses, and disordered. Giant cells with histologically, Bowen's disease provides intra-epithelial cancer curination: polymorphism of stem layer cells, increased number of mitoses, and disordered. Giant cells with multiple nuclei are sometimes found in the thoracic layer. mild hyper- and parakeratosis is noted, acanthosis, basal membrane and basal layer are preserved. In the upper part of the stroma there will be a small infiltrate dressing, consisting mainly of lymphocytes and plasma cells.

Comparative diagnosis is made by: with leukoplakia; red flat iron, chronic traumatic seizures.

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Pre-cancerous condition

Pre-cancerous condition of the cervix The so-called pre-cancer disease (praecancer verrucosus) manifests itself as a hemispherical nodule with an uneven surface rising above the level of the mucous membrane. The so-called pre-cancer disease (praecancer verrucosus) manifests itself as a hemispherical nodule with an uneven surface rising above the level of the mucous membrane. The so-called pre-cancer disease (praecancer verrucosus) manifests itself as a hemispherical nodule with an uneven surface rising above the level of the mucous membrane with a diameter of 4-10 mm. The Node, located on an unchanged surface, is located mainly the lower lip, palpation is painless. The color of the node can vary from the normal color of the labial red hoshia to a stagnant red color. The color of the knot can be gray-red, the surface of the knot is difficult to remove if it is covered with tightly fitting gray thickets.

Histologically, a clear-cut spread of the epithelium is observed due to the expansion of the spiny layer. istologically, a clear-cut spread of the epithelium is observed due to the expansion of the spiny layer. Acanthosis, hyperkeratosis, alternating with zones of paraceratosis, polymorphistologically, a clear-cut spread of the epithelium is observed due to the expansion of the spiny layer. Acanthosis, hyperkeratosis, alternating with zones of paraceratosis, polymorgically, a clear-cut spread of the epithelium is observed due to the expansion of the spiny layer. Acanthosis, hyperkeratosis, alternating with zones of paraceratosis, polymorphism of stem layer cells of varying degrees of weight up to acute is noted. The basal membrane is retained. The course of the so-called pre-cancer disease is very fast. The transition to an invasive form of cancer can occur 1-2 months after the onset of the disease.

Comparative diagnosis. It is necessary to distinguish between precancerous lesions of the skin: papilloma; warts, keratoacanthomas.

The diagnosis is finally justified by the results of the histological examination.

Treatment. Removal of the jarochate site, followed by histological examination.



Figure 79. Celiac cancer

Borderline cancer of the red lip hoshia hyperkeratosis

The borderline cancer of the red hokia of the lips is characterized by hyperkeratosis (hyperkeratosis precanserosacircumscripta) - a limited furnace with a diameter of 0.2 to 1 cm.he borderline cancer of the red hokia of the lips is characterized by hyperkeratosis (hyperkeratosis precanserosacircumscripta) - a limited furnace with a diametehe borderline cancer of the red hokia of the lips is characterized by

hyperkeratosis (hyperkeratosis precancerosa circumscripta) - a limited plaque with a diameter of 0.2 to 1 cm. The surface of the plaque is smooth, thin, covered with tightly fitting thickets and has a gray-white color. It can be easily removed when scraped and not eaten. In most patients, the plaque sinks slightly to the surface of the lip, but sometimes it can rise slightly higher. Stiffness is not detected during palpation.

Histological examination determines the deep penetration of the epithelium into the tissues, often with the presence of dysplasia of hyperkeratosis surfaces on the thoracic floor. Histological examination determines the deep penetration of the epithelium into the tissues, often with the presence of dysplasia of hyperkeratosis surfaces. Histological examination determines the deep penetration of the epithelium into the tissues, often with the presence of dysplasia of hyperkeratosis surfaces.

Comparative diagnosis. The disease is distinguished: leukoplakia; red runner; and with red flat iron.

Treatment comparative diagnosis. The disease is distinguished: leukoplakia; red runner; and.

Manganotti's abrasive cancerous cheilitis

Manganotti's abrasive CRAK Aldi Haliti (cheilitis abrasiva praecancerosa Manganotti) is a disease characterized by the appearance of one, rarely several, erosions in the red border of the lower lip. Manganotti's abrasive CRAK Aldi Haliti (cheilitis abrasiva praecancerosa Manganotti) is a disease characterized by the appearance of one, rarely several, erosions in the red border of the lower lip. Manganotti's abrasive CRAK Aldi Haliti (cheilitis abrasiva praecancerosa Manganotti) is a disease characterized by the appearance of one, rarely several, erosions in the red border of the lower lip. Erosion usually settles on the side of the lip, less often in the center or at the corner of the mouth. It has an oval or irregular shape, a smooth, polished surface, dark red in color. Erosion is located superficially, sometimes covered with a tightly settled bloody or serous crust, which is removed with difficulty, and a little bleeding. Erosions that are not covered with a crust are not prone to bleeding. Tissue hardening is usually not observed in and around the base of erosion. Erosions that are not covered with a crust are not prone to bleeding. Tissue hardening is usually not observed in

and around the base of erosion. Erosion in Manganottosions that are not covered with a crust are not prone to bleeding. Tissue hardening is usuarosions that are not covered with a crust are not prone to bleeding. Tissue hardening is usually not observed in and around the base of erosion. Erosion in Manganotti Halite is usually painless or mildly painful. Sometimes they appear against the background of small inflammation, characterized by instability. Erosion is a different slow and constant course, they do not respond well to drug treatment. They can go away for a long time, sometimes self-epithelialization is observed, but after a while they appear again in one place or another. The time for epithelization of erosions in Manganotti halite can vary from 3 months to 2 years. The duration of Manganotti's abrasive crayfish before it turned into cancer was individualizedhe time for epithelization of erosions in Manganotti halite can vary from 3 months to 2 years. The duration of Manganotti's abrasive crayfish before it turned into cancer was individualized. The rapid danger of the procedure can go to the process (after 4-6 months); in some patients it occurs 5-7 years after the onset of the disease. Clinical signs that indicate the possible onset of cancer transformation can be the appearance of stiffness in and around the base of erosion, the growth of suckers on its surface, bleeding after mild trauma, branching around erosion.

Histological examination is determined epithelial deffect. The epithelium at the edge of erosion is usually observed in the case of acanthosis, with broad epithelial tumors deeply embedded in the lower stroma.istological examination is determined epithelial deffect. The epithelium at the edge of erosion is usually observed in the case of acanthosis, with broad epithelial istological is determined epithelial deffect. The epithelium at the edge of erosion is usually observed in the ca



Image 81. Manganotti cheilitis on the lower lip



Figure 80. The pathogystology of Manganotti Haliti. Violation of the integrity of the epithelium, inflammatory infiltration of the mucous membrane.

Comparative diagnosis comparative diagnosis. It is necessary to distinguish the abrasive Ra Aldi halite of manganotti: the erosive form of leukoplakia; the erosive form of QYaT g; the erosive form of red runner; pemphigus, actinic halite multiform exudative erythema; herpetic erosions, lip cancer

Treatment. The treatment of Manganotti's halitis consists primarily in eliminating local traumatic factors. The oral cavity must be sanitizedreatment. The treatment of Manganotti's halitis consists primarily in eliminating local traumatic factors. The oral cavity must be sanitized. Smreatment. The treatment of Manganotti's halitis consists primarily in eliminating local traumatic factors. The oral cavity must be sanitized. Smoking, stopping the intake of harmful food together it is necessary to carry out an examination to identify and treat common somatic diseases. If the histological examination does not reveal dangerous signs of Manganotti's halite, a general and local conservative treatment can be carried out, the duration of which does not exceed 1-2 months. It is prescribed to drink Vitamin a (3.44% retinol acetate solution in oil or 5.5% retinol palmitate solution in oil) 3 drops 10 times a day, multivitamins are prescribed.t is prescribed to drink Vitamin a (3.44% retinol acetate

solution in oil or 5.5% retinol palmitate solution in oil) 3 drops 10 times a day, multivitamins are prescribed. Applying oils locally is carried out as prescribed to drink Vitamin A (3.44% retinol acetate solution in oil or 5.5% retinol palmitate solution in oil) 3 drops 10 times a day, multivitamins are prescribed.

Skin Horn

The cutaneous horn (cornu cutaneum) is a narrowed area of epithelial hyperplasia with a horny protrusion appearance with strongly pronounced hyperkeratosis. The cutaneous horn (cornu cutaneum) is a narrowed area of epithelial hyperplasia with a horny protrusion appearance with strongly pronounced hyperkeratosis. The cutaneous branch is found on the cutaneous horn (cornu cutaneum) is a narrowed area of epithelial hyperplasia with a horny protrusion appearance with strongly pronounced hyperkeratosis. The cutaneous branch is found on the red border of the lip, often on the lower lip, usually in people over 60 years of age. Often there is one skin branching, but there can be two or more. Damage to the lips or skin is clearly limited, up to 1 cm in diameter, from which a dirty gray or brownish-gray cone-shaped protrusion (Horn) emerges, up to 1 cm long, densely robust, painless. The cutaneous branch is a disease that lasts for a long time (for years). The beginning of its transition to a dangerous process is manifested by inflammation, increased branching, hardening of the base. The cutaneous branch is a disease that lasts for a long time (for years). The beginning of its transition to a dangerous process is manifested by inflammation, increased branching, hardening of the base. The final diagnosis can be made after the removal of the growth and its histological examination. From the pathological examination, the growth has a thick horny layer, which is a bounded hyperplasia of the labial red border epithelium. The epithelium at the base of the cutaneous branch is hyperkeratosis, a condition of acanthosis that is often irregular with symptoms of dysplasia and atypia.

Treatment is increased to AML in the Jarrahlik method. branched skin consists of removal i.e. healthy tissue.

Keratoakantoma

Keratoacanthoma is an epidermal benign tumor that develops rapidly and recedes spontaneously. Keratoacanthoma can be located on the red Hsia of the lips, very rarely on the tongue. Keratoacanthoma is an epidermal benign tumor that develops rapidly and recedes spontaneously. Keratoacanthoma can be located on the red Hsia of the lips, very rarely on the tongue. Keratoacanthoma is an epidermal benign tumor that develops rapidly and recedes spontaneously. Keratoacanthoma can be located on the red Hsia of the lips, very rarely on the tongue. The disease begins with the formation of a varonik node in the gray-red dense center of the lip. The tumor grows rapidly, within a month the node reaches a size of 2.5 x 1 cm². In its center, a Keratoacanthoma, filled with horny masses that easily migrate, is almost painless, mobile and does not grow into the main tissues. Keratoacanthoma has two manifestations. The most common is that it disappears on its own with the formation of an atrophic pigmented scar or it turns into cancer. The most common is that it disappears on its own with the formation of an atrophic pigmented scar or it turns into cancer. Spontaneous loss of keratoacanthoma is observed in the period from 6 weeks to 6-8 months. The disease can recur.

Histologically, the keratoacanthoma is a sharply restricted, slightly protruding, epithelial tumor that is covered with epithelium and has a crateriform recess filled with horny masses. Histologically, the keratoacanthoma is a sharply restricted, slightly protruding, epithelial tumor that is covered with epithelium and has histologically, the keratoacanthoma is a sharply restricted, slightly protruding, epithelial tumor that is covered with epithelium and has a crateriform recess filled with horny masses. An acanthosis condition is observed in the epithelium. Epithelial tumors are often visible and deeply absorbed into the connective tissue. They are polymorphic cells, sometimes reminiscent of the appearance of dyskeratosis. An inflammatory infiltrate of plasma and lymph cells is observed in the connective tissue stroma

Comparative diagnosis. Distinguish keratoacanthoma: papilloma; from Bald; disease of the so-called cancer;

Treatment. Performed in the jarrahlik method.

Prevention of cancer

In the Prevention of cancer diseases of the oral mucosa and red hoshia of the lips, the main importance is the elimination of chronic factors that damage the mucous membrane (acute edges of the teeth, protrusion, tartar deposits, destroyed tooth crowns, galvanism phenomena).n the Prevention of cancer diseases of the oral mucosa and red hoshia of the lips, the main importance is the elimination of chronic factors that damage the mucous membrane (acute edges of the teeth, protrusion, tartar deposits, destroyed tooth crowns, galvanism phenomena). stop smoking, protect against excess insolation and drying is necessary. In the Prevention of cancer diseases of the red lip hoshia, an important joint consists in the timely treatment of systemic diseases and chronic inflammatory processes of the oral mucosa - chronic lip cracks, glandular halitis, chronic herpetic lesions, etc.

In a conversation with patients, attention should be paid to the harm of taking hot, mouthwash and excessively spicy food, as well as the risk of alcohol consumption, smoking.n a conversation with patients, attention should be paid to the harm of taking hot, mouthwash and excessively spicy food, as well as the risk of alcoholn a conversation with patients, attention should be paid to the harm of taking hot, mouthwash and excessively spicy food, as well as the risk of alcohol consumption, smoking. It is necessary to pay attention to the state of oral hygiene, which is one of the joints of the Prevention of changes in the mucosa of the oral cavity. Especially it is necessary to talk to patients who have pre-cancer changes in the oral mucosa. If the necessary requirements of the doctor are not met (stop smoking, eat irritating food, regular oral care), the likelihood of cancer should be warned.

Dentists, as well as doctors of other professions, should be careful with oncological diseases when examining patients.entists, as well as doctors of other professions, should be careful with oncological diseases when examining patients. Regardless of the complaints addressed by the entists, as well as doctors of other professions,

should be careful with oncological diseases when examining patients. Regardless of the complaints addressed by the patient, it is important for the doctor to examine the cavity of the mouth and fully examine the red border of the lips. Early manifestations of precancerous diseases or signs of their transformation into cancer, as a rule, are not visible to the patient, since they proceed painlessly, therefore, their timely diagnosis is a doctor's task. The concept of "anti-oncology oboxification" refers, first of all, to the sum of specific knowledge of oncology, which allows the doctor to diagnose cancer early and on time. The concept of "anti-oncology oboxification" refers, first of all, to the sum of specific knowledge of oncology, which allows the doctor to diagnose cancer early and on time. The concept of "anti-oncology oboxification" refers, first of all, to the sum of specific knowledge of oncology, which allows the doctor to diagnose cancer early and on time. The concept of "anti-oncology oboxification" refers, first of all, to the sum of specific knowledge of oncology, which allows the doctor to diagnose cancer early and on time. The concept also includes knowledge of precancerous diseases, their diagnosis, treatment, and signs of danger. Diagnosis and malignancy, the cytological research method plays a key role in determining morphological signs, which makes it possible to make the correct diagnosis in 90-95% of cases. It is obtained by scraping or puncture for the cytological examination method. Histological examination should be carried out in patients with obligate and facultative forms of precancerosis with a high probability of malignancy. The precancerous condition of leukoplakia is solved on the basis of a complex of clinical and morphological signs. Histological examination should be carried out in patients with obligate and facultative forms of precancerosis with a high probability of malignancy. The precancerous condition of leukoplakia is solved on the basis of a complex of clinical and morphological signs. Cancer of the oral cavity and lips mucous membranes, especially patients with its mandatory forms, should be under the active supervision of a dentist or oncologist. The dentist must have a clear understanding of the organization of oncological care, the network of oncological medical institutions in

order to quickly send the patient to the address. Treatment without a diagnosis should not last more than 7 days. Treatment without a diagnosis should not last more than 7 days. It is necessary to eliminate substances that have a local excitatory property, do not use drugs that promote tumor growth. Treatment without a diagnosis should not last more than 7 days. It is necessary to eliminate substances that have a local excitatory property, do not use drugs that promote tumor growth (moxibustion, physiotherapy, etc.) it is necessary to stop. In difficult cases, the doctor is obliged to involve more experienced specialists. Treatment without a diagnosis should not last more than 7 days. It is necessary to eliminate substances that have a local excitatory property, do not use drugs that promote tumor growth (moxibustion, physiotherapy, etc.) it is necessary to stop. In difficult cases, the doctor is obliged to involve more experienced specialists in the examination of the patient. In case of the slightest suspicion of the danger of the carcinogenic process, it is necessary to immediately biopsy the area inside healthy tissues with a subsequent histological examination. Only then should the issue of further treatment tactics be resolved. Patients should be actively monitored after the treatment of cancer or pre-cancer diseases.

Стоматолог лугат - глоссарий
СТОМАТОЛОГИК ЛУҒАТ-ГЛОССАРИЙ
СТОМАТОЛОГИК ЛУҒАТ-ГЛОССАРИЙ

Глоссарий:	Глоссарий:	Glossary:
ОБШҚ – оғиз бۆшлиғи шиллик қавати	СОПР – слизистая оболочка полости	OMMD – oral mucous membrane diseases
ЎГС – ўткир герпетик стоматит	ОГС – острый герпетический стоматит	SHA – stomatitis herpetica acuta
СҚГ – сурункали қайталанувчи герпес	ХРГ - хронический рецидивирующий герпес	HCHR – herpes chronica recidivans
ЎОТ – ўраб олувчи темиртки	ОЛ – опоясывающий лишай	HS – herpes zoster
ОИТС – ортирилган иммун танқислиги синдроми	СПИД – синдром приобретенного иммунодефицита	AIDS – asquired immune deficiency syndrome
ИИБ – инсон иммун танқислик вируси	ВИЧ – вирус иммунодефицита человека	ARVI – acute respiratory viral infections
ЎРК – ўткир респиратор касалликлар	ОРЗ – острое респираторное заболевание	VANUS – vincent’s acute necrotizing ulceromembranous stomatitis
ВЯНС – венсан ярали – некротик стоматити	ЯНСВ –язвенно- некротический стоматит Венсана	AE – angioedema

КАШ – квинкенинг ангионевротик шиши	АНОК – ангио- невротический отек Квинке	ЕМЕ – erythema multiforme exudative
КШЕЕ – кўп шаклли экссудатив эритема	МЕЕ – многоформная экссудативная еритема	CHRAS – chronically recurrent aphthous stomatitis
СҚАС – сурункали қайталанувчи афтоз стоматити	ХРАС – хронический рецидивирующий афтозный стоматит	BS – Behcet's syndrome
БС – Бехчет синдроми	СБ – синдром Бехчета	SHS – Sjogren syndrome
ШС – Шегрен синдроми	СШ – синдром Шегрена	GD – gastroenteric disease
ОИТК – ошқозон – ичак тизими касалликлари	ЖКТ – желудочно- кишечный тракт	CD – cardiovascular diseases
ЮҚТК – юрак – қонтомир касалликлари	ССС – сердечно- сосудистая система	BD – blood dyscrasias
ЕК –эндокрин касалликлари	ЕЗ – эндокринные заболевания	ED – endocrinologic disorders
БТТК – бириктирувчи тўқима тарқок касалликлари	РЗСТ – распространенные заболевания соединительной ткани	EI – exogenous intoxications
ҚХҚАК – қон хосил қилувчи аъзо касалликлари	ЗККО – заболевания крови и кроветворных органов	OM – oral manifestations
АТК – асаб тизими касалликлари	ЗНС – заболевания нервной системы	NS – nervous system
ҚЯТ – қизил ясси темиртки	КПЛ – красный плоский лишай	LRP – lichen ruber planus

ҚЮ – қизил югурик	КВ – красная волчанка	LE – lupus erythematosus
БТ – бурмасимон тил	СЯ – складчатый язык	LP – lingua plicata
ДГ – дескваматик глоссит	ДГ – десквамативный глоссит	GDG – glossitis desquamativa
ҚСТ – қора сочли тил	ЧВЯ – чёрный волосатый язык	LVN – lingua villosa nigra
РГ – ромбсимон глоссит	РГ – ромбовидный глоссит	GR – glossitis rhombica
ЕксХ – эксфолиатив хейлит	ЕГ – эксфолиативный глоссит	CHE – cheilitis exfoliativa
ОГХ – оддий glandуляр хейлит	ГХ – glandулярный хейлит	CHGS – cheilitis glandularis simplex
КАХ – контактли аллергик хейлит	КАХ – контактный аллергический хейлит	CHAC – cheilitis allergic contactilis
АкХ – актиник хейлит	АХ – актинический хейлит	AkX – cheilitis aktinicalis
МХ – метеорологик хейлит	МХ – метеорологический хейлит	CHM – cheilitis meteorologica
АХ – атопик хейлит	АХ – атопический хейлит	CHA – cheilitis atypicalis
ЭХ – экзематоз хейлит	ЕХ – эксфолиативный хейлит	CHEc – cheilitis eczematosa
СОК – саратон олди касалликлари	ПЗ – предраковые заболевания	PcD – precancerous diseases
ССОК – сўгалсимон саратон олди касалликлари	БП – бородавчатый предрак	PcV – precancer verrucosus
МанХ – манганотти хейлити	ХМ – хейлит Манганотти	MB – morbus Bowen

ФСОК – факултатив саратон олди касалликлари	ФПЗ – факультативные предраковые заболевания	HPcC – hyperceratosis praecancerosa circumsripta
ОСОК – облигат саратон олди касалликлари	ОПЗ – облигатные предраковые заболевания	CHAPcM – cheilitis abrasive praecancerous Manganotti
Абсцесс – чегараланганйирингли-яллиғланиш жараён.	Абсцесс - ограниченный гнойно-воспалительный процесс.	CC – cornu cutaneum
Актиномикоз – турли тўқима структураси ва аъзоларнинг сурункали специфик яллиғланиш касаллиги бўлиб, тўлқинсимон кечиши билан ажралиб туради.	Актиномикоз - хроническое специфическое воспалительное заболевание с поражением различных тканевых структур и органов, характеризующееся волнообразным длительным течением.	Actinomycosis is a chronic specific inflammatory disease with damage to various tissue structures and organs, characterized by a wavelike long course.
Аллергик стоматит-аллергия билан чақирилган оғиз бўшлиғи шиллиқ қаватининг яллиғланиши.	Аллергический стоматит- воспаление слизистой оболочки полости рта, обусловленное аллергией.	Allergic stomatitis - inflammation of the oral mucosa due to allergies.
Аллергия - Оғиз бўшлигининг шиллиқ қавати, лабнинг қизил хошияси маҳаллий ва	Аллергия - болезненное состояние организма человека, которое проявляется в	Allergy is a painful condition of the human body, which manifests itself in intolerance to any

системали аллергия касалликларда ўзига хос шикастланади. Аллергия касалликлари негизда химоя хусусиятига эга бўлган иммун реакцияси ётади. Аллергияни физиологик механизми бўлиб, антигенга нисбатан антитаначалар пайдо бўлади. Аллергия датири ва шиллиқ қаватларда кучли зарарланиш, умумий ахвол ёмонлашиши юзага келади.	непереносимости каких-либо веществ. Физиологический механизм аллергии заключается в образовании в организме антител, что приводит к понижению или повышению его чувствительности. Аллергия проявляется сильным раздражением слизистых оболочек, кожными сыпями, общим недомоганием и т.п.	substances. The physiological mechanism of allergy is the formation of antibodies in the body, which leads to a decrease or increase in its sensitivity. Allergy is manifested by severe irritation of the mucous membranes, skin rashes, general malaise, etc.
Аллергологик синама – аллергияни аниқлаш учун бажараладиган тест.	Аллергологическая проба - тест на выявление (определение) вещества, вызывающего у человека болезненное состояние (болезненную реакцию организма).	An allergological test is a test to identify (determine) a substance that causes a person to have a painful condition (a painful reaction of the body).
Аллотрансплантация – Битта инсондан олишган тишни бошқа инсонга екиб ўтқишиш, даставал консервант сифатида	Аллотрансплантация - пересадка удаленного зуба от одного человека другому при наличии банка зубов, прошедших специальную обработку	Allotransplantation is a transplant of a removed tooth from one person to another in the presence of a tooth bank that has undergone special

банкда бўлган тишларга махсус ишлов берилади.	и хранящихся в консервантах.	treatment and is stored in preservatives.
Афта –шиллик қаватнинг диаметри 1-5 мм , оқ-саригиш караш билан қопланган ва атрофида гиперемияланган гардиши бор юмалоқ ёки овал шаклдаги юза нуқсони, яраланиши.	Афты - поверхностные болезненные изъязвления слизистой оболочки, реже кожи, округлой формы, диаметром 1-5 мм, покрытые беловато-желтым налетом и окруженные красным ободком гиперемии.	Aphthae is superficial painful ulceration of the mucous membrane, less often of skin, of a round shape, with a diameter of 1-5 mm, covered with a whitish-yellow coating and surrounded by a red rim of hyperemia.
Афтоз стоматит – оғиз бўшлиғи шиллик қаватнинг сурункали яллиғланиши, натижада вақти вақти билан қайтарилиб ва ремиссияда турувчи афталар пайдо бўлиши.	Афтозный стоматит - хроническое воспалительное заболевание слизистой оболочки полости рта, характеризующиеся возникновением афт, протекающие с периодическими обострениями и ремиссиями.	Aphthous stomatitis is a chronic inflammatory disease of the oral mucosa, characterized by the occurrence of aphthae, occurring with periodic exacerbations and remissions.
Базал (basalis) – асосида жойлашган, юнончадан «basis» - пойдевор, яъни асос.	Базальный (basalis) – лежащий в основании, обращенный к нему, находящийся у основания; в переводе с греч. «basis» - основа.	Basal (basalis) - lying at the base, facing it, located at the base; translated from Greek. "Basis" is the basis.

Биопсия (biopsia). Ташхислаш мақсадида организмдан тўқима олиб, патологияни аниқлаш учун (яллиғланиш, ўсма ва б.) уни микроскопик текшириш.	Биопсия (biopsia). Метод исследования для установления диагноза, предполагающий забор кусочка ткани организма и последующее его микроскопическое исследование на выявление патологии (воспаления, опухоли и пр.)	Biopsy (biopsia). A research method for establishing a diagnosis, involving the collection of a piece of body tissue and its subsequent microscopic examination to identify pathologies (inflammation, tumors, etc.)
Гиперсаливация – сўлак безлари секретиясини ошиши.	Гиперсаливация - увеличение секреции слюнных желез.	Hypersalivation - increased secretion of the salivary glands.
Гипосаливация - сўлак безларисекретиясини камайиши ёки йўқ бўлиши.	Гипосаливация - снижение секреторной функции больших и малых слюнных желез вплоть до ее прекращения.	Hyposalivation - a decrease in the secretory function of large and small salivary glands until its termination.
Гиперстезия – тиш тўқималарини турли механик, химиквахароратли таъсиротларга ошиши.	Гиперстезия - повышенная чувствительность тканей зуба к механическим, химическим и температурным раздражителям.	Hypersthesia is an increased sensitivity of tooth tissues to mechanical, chemical and temperature stimuli.
Глоссит – яллиғланиш оқибатида тил тўқималарининг	Глоссит - патологическое состояние тканей языка	Glossitis is a pathological condition of the tissues of the tongue of an

патологик холатга келиши, кўпинча инфекция табиатлиқақирилган бўлганда генераллашган стоматит кўринишида, ёки организмнинг бошқа касалликлари симптоми бўлиши мумкин.	воспалительного характера, представляющее собой либо генерализованный стоматит, чаще инфекционной природы, либо симптом общего заболевания организма.	inflammatory nature, which is either generalized stomatitis, often of an infectious nature, or a symptom of a general illness of the body.
Қизилча-болаларнинг ўткир юқумли касаллиги бўлиб, оғиз бўшлиғи шиллиқ қаватнинг гиперемиясига бўйин лимфа тугунларини катталаши билан характерланади.	Краснуха - острое инфекционное заболевание детского возраста, характеризующееся гиперемией слизистой оболочки полости рта, а также увеличением шейных лимфатических узлов.	Rubella is an acute childhood infectious disease characterized by hyperemia of the oral mucosa, as well as an increase in cervical lymph nodes.
Қизил ясси темирлатки – сурункали яллиғланиш-дистрофик касаллиги бўлиб, шиллиқ қават ва терида кузатилади.	Красный плоский лишай - хроническое воспалительно-дистрофическое заболевание, возникающее на коже и видимых слизистых оболочках.	Lichen planus is a chronic inflammatory-dystrophic disease that occurs on the skin and visible mucous membranes.
Ксеростомия - оғиз бўшлиғи шиллиқ қавати ва лабларнинг қуриши	Ксеростомия - сухость слизистой оболочки рта и губ вследствие снижения	Xerostomia - dryness of the mucous membrane of the mouth and lips due to

сўлак функционал пасайишидан чиқади.	безларини активности желез.	функциональной активности желез.	a decrease in the functional activity of the salivary glands.
Кератоз – ошқозон-ичак тизими касалликларида, инфекция, ўсмаларда билвосита юзага келадиган, ҳамда бевосита салбий таъсир натижасида тил, лаб ва оғиз бўшлиғи шиллик қаватини патологик мугузланиши хисобланади.	Кератоз - патологическое ороговение слизистой оболочки полости рта появляется чаще всего на языке, губах как при непосредственном воздействии неблагоприятных факторов на слизистую оболочку, так и при системных заболеваниях желудочно-кишечного тракта, инфекциях, опухолях.	Keratosis - pathological keratinization of the oral mucosa appears most often on the tongue, lips, both with direct exposure to adverse factors on the mucous membrane, and with systemic diseases of the gastrointestinal tract, infections, and tumors.	
Лейкоплакия – экзо ва эндоген сурункали яллиғланиш натижасида шиллик қават стромасининг зарарланиши.	Лейкоплакия - ороговение слизистой оболочки, сопровождающееся воспалением стромы и возникающее в ответ на хроническое как экзогенное, так и эндогенное раздражение.	Leukoplakia - keratinization of the mucous membrane, accompanied by inflammation of the stroma and arising in response to chronic both exogenous and endogenous irritation. Lymphadenitis is an inflammation of the lymph node.	

		Lymphangitis is an inflammation of the lymphatic vessels, which is usually combined with regional lymphadenitis.
Лимфаденит - лимфатиктугун яллиғланиши.	Лимфаденит - воспаление лимфатического узла.	Lymphadenitis - inflammation of the lymph node
Лимфангит - регионар лимфаденит билан бирга кечувчи лимфа томирларни яллиғланиши.	Лимфангит - воспаление лимфатических сосудов, которое обычно сочетается с регионарным лимфаденитом.	Lymphangitis is an inflammation of the lymphatic vessels, which is usually combined with regional lymphadenitis.
Микоз(кандидоз) - Candida ачитқисимон замбуруғ билан чақирилган шиллиқ қават касаллиги.	Микозы (кандидозы) - заболевания слизистой оболочки, вызванные дрожжеподобными грибами рода Candida.	Mycoses (candidiasis) - diseases of the mucous membrane caused by yeast-like fungi of the genus Candida.
Невралгия – уч шохлик нервнинг сезув толаларининг зарарланиши оқибатида биринчи ёки учинчи шохлари бўйлаб тарқалувчи қисқамуддатли (1-2 дақ) ёки доимий хуружсимон азоб берувчи оғриқлар кўпинча	Невралгия - поражение главным образом чувствительных нервов представляет собой кратковременные (1-2 мин.) или постоянные приступообразные мучительные боли в зоне иннервации первой третьей ветвей тройничного нерва.	Neuralgia - damage to mainly sensitive nerves is a short-term (1-2 min.) Or constant paroxysmal painful pain in the innervation zone of the first third branches of the trigeminal nerve. Pain syndrome is accompanied by vegetative manifestations (facial

вегетативбузилишлар (юз гиперемияси, кўз ёш ва сўлак оқиши) билан кечади.	Болевой синдром сопровождаются вегетативные проявления (гиперемия лица, слезо- и слюнотечение).	flushing, tear and salivation).
Неврит – нерв тутамида периферик нервнинг анатомик ўзгариши билан кечади, натижада функция ёқолиши симптомларини (атрофия, мушак тонуси пасайиши, сезувчанлик ва рефлексларнинг парез ва тўлиқ фалажи) ривожланиши.	Неврит - заболевание периферического нерва с выраженными анатомическими изменениями в нервных стволах. Выражены симптомы выпадения функций (атрофия, понижение мышечной силы, чувствительности и рефлексов) от парезов до полного паралича.	Neuritis is a disease of the peripheral nerve with pronounced anatomical changes in the nerve trunks. Symptoms of loss of function are expressed (atrophy, decreased muscle strength, sensitivity and reflexes) from paresis to complete paralysis. .
Петехии – оғиз бўшлиғи шиллиқ қавати ва шиллиқ ости қавати ва теридаги майда қон қуюлишлар.	Петехии - мелкие точечные кровоизлияния в подслизистый слой слизистой оболочки полости рта или кожи.	Petechiae - small point hemorrhages in the submucous layer of the oral mucosa or skin.
Пигментация – меланинпигменти оғиз бўшлиғи шиллиқ қавати ёки терида ёғилиши уларнинг ранги ўзгаришига олиб келади.	Пигментация - изменение цвета слизистой оболочки или кожи вследствие отложения пигмента меланина.	Pigmentation is a discoloration of the mucous membrane or skin due to deposition of the pigment melanin.
Рана – бу тўқималарнинг механик за	Рана - это механическое повреждение тканей с	A wound is a mechanical damage to tissues with a

парланиши натижасида тери, шиллиқ қават, кўзнинг шох қавати, склера бутунлиги бузилишиюзага келиши мумкин	нарушением целости покровов (кожа, слизистая оболочка, склера, роговица).	violation of the integrity of the integument (skin, mucous membrane, sclera, cornea).
Сўлак-тош касаллиги – сўлак безида тош хосил бўлганда ривожланади.	Слюнно-каменная болезнь - образование камней в слюнной железе.	Salivary stone disease is the formation of stones in the salivary gland.
Шилиниш – шиллиқ қаватнинг юза нуқсони бўлиб, механик таъсирот натижасида келиб чиқади.	Ссадина - поверхностный дефект слизистой оболочки, возникающий при механическом повреждении.	Abrasion is a superficial mucosal defect that occurs with mechanical damage.
Ерозия – тиш тўқималарини (эмаль, дентин) прогрессланувчи камайиши.	Ерозия - прогрессирующая убыль тканей зубов (эмали и дентина) недостаточно выявленной этиологии.	Erosion is a progressive decrease in tooth tissues (enamel and dentin) of an insufficiently identified etiology.
Етиология – касалликни ривожланиш сабаблари заболевания.	Етиология - причины возникновения заболевания	Etiology - the causes of the disease

References

1. **Kamilov H.P., Akhmedov K.R.** Diseases of the Oral Mucosa. Textbook. – Tashkent. TIGOP GATSH AUO. 2016.
2. **Kamilov H.P., Ibragimova M.H., Mirakhmedova D.U. et al.** Diseases of the Oral Mucosa. Textbook. – Tashkent. Yangi Asr Avlodi. 2005.
3. **Barer.** Diseases of the Oral Mucosa. 2015. (Russian)
4. Clinical Restorative Dentistry. (Russian)
5. **Maksimovsky Yu.M. et al.** 1.53.008 (Clinical Endodontics 1.53.008). 2016. (Russian)
6. **Rizaev J.A.** Therapeutic Dentistry. Textbook.
7. **Safarov T. Kh., Rakhmonov Kh. Sh., Safarov M. T.** Non-Carious Diseases of Dental Tissues. – Tashkent. 2011.
8. **Kamilov H. P., Yusupalikhodzhaeva S. Kh., Zoirov.** Fundamentals of Endodontics in Therapeutic Dentistry. Samarkand. 2013.
9. **Kamilov H. P., Yusupalikhodzhaeva S. Kh., Shukurova U. A.** Electronic Educational-Methodological Complex for the Faculty of Therapeutic Dentistry. Copyright Certificate (№ AGU 0231, 19.04.2013). Tashkent.
10. **Borovsky E.V. et al.** (Therapeutic Dentistry). Moscow. 2006. (Russian)
11. **Dmitrieva D.M.** (Therapeutic Dentistry). 2005. (Russian)
12. **Nikolaev A.I., Tsepov L.M.** (Practical Therapeutic Dentistry). 2006. (Russian)
13. **Beer R., Baumann M., Kim S.** (Atlas of Dentistry. Endodontology). 2006. (Russian)
14. **Balin V.N., Guk A.S.** (Symptoms and Syndromes in Dental Practice). 2000. (Russian)
15. **Banchenko G.V.** (Combined Diseases of the Oral Mucosa and Internal Organs). Moscow. 1979. (Russian)
16. **Banchenko G.V., Maksimovsky Yu.M.** (The Tongue – "Mirror" of the Organism). Moscow. Business Center "Stomatologiya". 2000. 404 p. (Russian)
17. **Borovsky E.V. and Mashkilleyson A.L.** (Diseases of the Oral Mucosa and Lips). Medpress. 2001. (Russian)

- 18.**Lukinykh L.M. (ed.).** (Diseases of the Oral Mucosa). Nizhny Novgorod. 2001.
(Russian)
- 19.**Zhumatov U.Zh., Babadzhanov S.N.** (Physical Methods for the Prevention of Dental Diseases). Tashkent. 1989. 79 p. (Russian)
- 20.**Zafarov O. O., Mirakhmedova D. U., Bektashev S. I., Zhumatov U. Zh.**(Diseases of the Oral Mucosa). Tashkent. 2001. 106 p.
- 21.**Kamilov H.P., Yuldasheva N.A., Komilova A.Z.**(Manifestation of Viral Diseases in the Oral Cavity). Study Guide. 2024.
- 22.**Kamilov H.P., Yuldasheva N.A., Komilova A.Z.**(Changes in the Oral Cavity in Metabolic and Endocrine Diseases). Study Guide. 2025.
- 23.**Kamilov H.P., Ibragimova M.H., Takhirova K.A., Kadyrbaeva A.A.** (Diseases of the Oral Mucosa). 2023.
- 24.**Kamilov H.P., Ibragimova M.H.**(Therapeutic Dentistry. Diseases of the Oral Mucosa). Tashkent. Yangi Avlod. 2005.
- 25.**Kamilov H.P., Ibragimova M.H., Komilova A.Z.**(Histological Structure of the Oral Mucosa). Tashkent. 2023.
- 26.**Barer G.M. (ed.).** Moscow Dental Institute.(Clinical Situations in Therapeutic Dentistry). Study Guide with Test Assignments. Moscow. 1999. Mashkilleyson A.J. Moscow Dental Institute. (Diseases of the Oral Mucosa). Study Guide. Moscow. 1996. **Pashkov B.M., Stoyanov B.G.**(Lesions of the Oral Mucosa and Lips in Some Dermatoses and Syphilis). Moscow. 1970. (Russian)
- 27.**Puzin M.Sh.**(Neurostomatological Diseases). WHO Expert Committee Report. Moscow. 1994. (Russian)
- 28.**Pokrovsky V.V.** (Epidemiology and Prevention of HIV Infection and AIDS). Moscow. Meditsina. 1996. 248 p. **Rabinovich I.M., Banchenko G.V.**(AIDS. Manifestations of HIV Infection in the Oral Cavity.). International Dental Site. 2003. (Russian)
- 29.**Roshchina G.I., Maksimovskaya L.I.**(Medicines in Dentistry). Moscow. 1989. 208 p. (Russian)

30. **Langlais R. P., Miller C. S.** (Atlas of Diseases of the Oral Cavity). Translated from English under the editorship of L.A. Dmitrieva. Moscow. GEOTAR-Media. 2010. (Russian Translation)
31. **Yakovleva V.I. et al.** Prevention of Dental Diseases. (Bulletin of Experiment). 2012, Vol. 154, No 8, pp. 252-255. *Burket's Oral Medicine*. 2008. (Russian/English)
32. **Brown D.** European seroprevalence of HSV-2/Serologic infections: new developments. Eur newb61. **Overall J.C.** Herpes simplex. Orn. Pediatr. Ann. 1994, vol. 33. **Gitz M.** Pseudoallergy. Vien.med/ Wolhensehr.-1996.-14. (English/German)
33. (See No. 32).
34. (Guidelines for Epidemiological Studies of Manifestations of HIV Infection Localized in the Oral Cavity). Moscow. 1995. **Rybakov A.I., Banchenko G.V.** (Diseases of the Oral Mucosa). Moscow. 1978. 232 p. (Russian)
35. **Rybakov A.I. et al.** Atlas. (Diseases of the Oral Mucosa). Tashkent. 1977. 96 p. (Russian)
36. **Rybakov A.I., Ivanov V.S.** (Clinical Therapeutic Dentistry). Moscow. 1980. (Russian)
37. **Smirnov V.A.** (Diseases of the Facial Nerves). Moscow. 1978. (Russian)
38. **Selyutina O.V.** (Towards the Diagnosis of Syphilis). Moscow. Journal Stomatologiya. 2000, No 2, 50 p. (Russian)
39. **Tretyakovich A.G., Glinnik A.V.** (Handbook of Clinical Symptoms and Syndromes in Dentistry and Maxillofacial Surgery). NGMA. 2000. (Russian)
40. **Fayzullaev T.N., Epishev V.A.** (Stomatitis and its Treatment). Tashkent. 1983. 110 p.
41. **Khantov R.M., Ignatieva G.A.** (AIDS). Moscow. Kultura. 1992. (Russian)
42. **Yuldasheva N.A., Komilova A.Z.** (Periodontitis Classification, Pathological Anatomy, Clinic, Differential Diagnosis, Types of Periodontitis and Treatment). Educational-Methodological Guide. Tashkent. 2025. (Russian)
43. **Yuldasheva N.A., Komilova A.Z., Ubaydullaeva N.I.** (Changes in the Oral Mucosa in Endocrine Diseases). Educational-Methodological Guide. Tashkent. 2025.

44. **Leus P.A. et al.** (Diseases of the Teeth and Oral Cavity). Minsk. 1998. (Russian)
45. **Borovsky E.V.** (Clinical Endodontics). 1999. (Russian)
46. **Borovsky E.V., Leontiev V.K.** (Biology of the Oral Cavity). 2001. (Russian)
47. **Khomenko L.A., Bidenko N.V.** (Practical Endodontics). 2005. (Russian)
48. **Kuzmina E.M. (ed.).** (Prevention of Dental Diseases). 1997. **Groshikov M.I.** (Non-Carious Lesions of Dental Tissues). 1985. (Russian)
49. **Banchenko G.V., Kryazheva S.S.** Atlas. (Combined Lesions of the Oral Mucosa and Skin). 1994. (Russian)
50. **Muravyannikova Zh.G.** (Diseases of the Teeth and Oral Cavity). 2006. (Russian)
51. **Kamilov H.P., Yusupalikhodzhaeva S.Kh.** (Application of Modern Pedagogical Technology in Therapeutic Dentistry). Educational-Methodological Guide. Tashkent. 2007.
52. **Kamilov H.P., Bekzhanova O.E., Shukurova U.A.** (Periodontosis, Clinical Picture, Diagnosis, and Differential Diagnosis). Educational-Methodological Guide. Tashkent. 2007. (Russian)
53. **Kamilov H.P., Bekzhanova O.E., Shukurova U.A.** (Modern Approaches to the Treatment and Prevention of Periodontosis). Educational-Methodological Guide. Tashkent. 2007. (Russian)
54. **Kuryakina N.V., Kutepova T.F.** (Diseases of the Periodontium). Moscow. 2003. (Russian)

Additional References

1. **Ream M., Bruce J., Salmel A., Trefzer C.** *Clinical Oral Medicine and Pathology*. Springer International Publishing. Textbook. 2nd edition. United States of America (USA). 2016.
2. **Laskaris G.** (Treatment of Diseases of the Oral Mucosa). Textbook. – Moscow. MIA. 2006. (Russian)
3. **Tsvetkova L.A., Arutyunov S.D. et al.** (Diseases of the Oral Mucosa). Textbook. – Moscow. MEDpress-inform. 2006. (Russian)

4. **Anisimova I.V., Nedoseko V.B., Lomiashvili L.M.**(Diseases of the Oral Mucosa and Lips. Clinic Diagnosis). Textbook. Moscow. MEDI. 2005. (Russian)
5. **Barer G.M.**(Therapeutic Dentistry. Part 3. Diseases of the Oral Mucosa). Textbook. – Moscow. GEOTAR-Media. 2005. (Electronic). (Russian)
6. **Panchishin M., Got I., Masny Z.**(Emergency Conditions in Dental Practice). Textbook. – Lviv. Gal-Dent. 2004. (Electronic). (Russian)
7. **Dmitrieva L.A.** (Therapeutic Dentistry). Textbook. – Moscow. MEDpress-inform. 2003. (Russian)
8. **Sonis S. T.**(Secrets of Dentistry). Textbook. BINOM – Nesky Dialect Publishing House. 2002. (Russian)
9. **Borovsky E.V.**(Biology of the Oral Cavity). Textbook. – Moscow. Meditsinskaya Kniga. 2001. (Electronic). (Russian)
10. **Danilevsky N.F., Leontiev V.K. et al.**(Diseases of the Oral Mucosa and Lips). Textbook. – Moscow. Stomatologiya. 2001. (Russian)
11. **Banchenko G.V., Maksimov Yu.M.**(The Tongue – "Mirror" of the Organism). Textbook. – Moscow. Business Center Stomatologiya. 2000. (Russian)
12. **Troshin V.D., Zhulev E.N.**(Pain Syndromes in Dental Practice). Publishing House of the Nizhny Novgorod State Medical Academy. 2002. (Russian)
13. **Mirziyoyev Sh. M.**(Critical analysis, strict discipline, and personal responsibility should be the daily rule of every leader's activity). O'zbekiston Publishing and Printing Creative House of the Uzbekistan Press and Information Agency. 2017.
14. **Mirziyoyev Sh. M.** (We will build our great future together with our brave and noble people). O'zbekiston Publishing and Printing Creative House of the Uzbekistan Press and Information Agency. 2017.
15. **Mirziyoyev Sh. M.**(We will build a free, prosperous, and democratic state of Uzbekistan together). O'zbekiston Publishing and Printing Creative House of the Uzbekistan Press and Information Agency. 2016.

Electronic data obtained from the website

1. [www. medlibrary. ru](http://www.medlibrary.ru)
2. [www. medline. ru](http://www.medline.ru)
3. [www. cohrane.com](http://www.cohrane.com)
4. [www. zdorove.ru](http://www.zdorove.ru)
5. [www. med.ru](http://www.med.ru)
6. [www. stom.ru](http://www.stom.ru)
7. [www. stomatologya.ru](http://www.stomatologya.ru)

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