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**PERIODONTIUM DISEASES,
ETIOPATHOGENESIS CLINIC
COMPARATIVE DIAGNOSIS,
TREATMENT AND PREVENTION**



MINISTRY OF HIGHER AND SECONDARY SPECIAL
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MINISTRY OF HEALTH REPUBLIC OF UZBEKISTAN

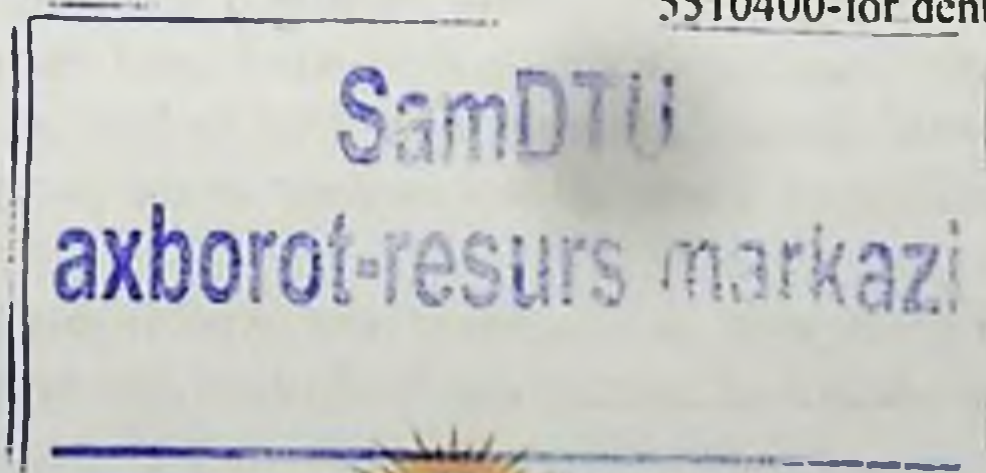
T.E.ZOYIROV B.K. RAXMONOVA A.T. ELNAZAROV



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Oral diseases are a huge medico-social problem, and the solution to this problem is the task of modern preventive dentistry. It should be noted that most dentists are currently engaged in restoration work, and the prevalence of dental caries and periodontal disease remains high. Due to lack of time, doctors do not conduct dental explanatory work, do not engage in the prevention of caries and gingivitis.

Active dental promotion, development and implementation of comprehensive preventive programs should be carried out by dentists and hygienists-nurses.

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LIST OF ABBREVIATIONS

- KTRP** -fast-progressing periodontitis in adults (KTRP)
- GBO** -is hyperbaric oxygenation(GBO)
- Lupus** -locale Juvenil parodontosis
- TYuP** -scattered Juvenil parodontosis
- AIDS** -Acquired Immunodeficiency Syndrome
- EchT** -erythrocyte deposition rate
- PMYaL** -polymorphic-nuclear leukocytes
- BJSST** -Health Organization

INTRODUCTION

Parodontology is the science of the parodont or tooth auxiliary holding apparatus. A Parodont is a tooth wrapper that holds firmly in the jaw (lot. para-around, Grec. odus-tooth) unites tissues. The following soft and hard tissues are involved in the structure of the parodont: gums, root cement, periodontal ligament, alveolar bone. The structure and function of the Parodont tissue has been sufficiently studied.

Periodont diseases are diverse. In the first place, it is manifested by the presence of various manifestations of gingivitis (inflammation of the gums with the preservation of the tooth-gum joint) and periodontitis (loss of parodont tissue as a result of inflammation) dental caraches (dental plaque).

It is known that periodont diseases, in particular gingivitis, are among the most common dental diseases to this day. Treatment measures do not always benefit, as a result of the development of the pathological process, sometimes severe complications can arise.

Today the development of Science and economics contributes to the introduction of modern and new technologies into the practice of Dentistry and thus the development of facilities in the work of dentists. But, in recent times, this trend is getting a little lame. Perhaps the reason for this is the insufficient organization of measures aimed at combating the most common among mankind – diseases such as gingivitis and caries.

It is the high prevalence of dental diseases and the high prevalence intensity that increase the relevance of the problem of mass prevention of periodont diseases.

The main etiological factors are oral microflora. In the current period of development of Dentistry, the microflora of the oral cavity is not paid enough attention not only by patients, but also by Doctor-dentists. The main cause in the development of catarrhal gingivitis is the microbial landscape of the oral cavity, which has the property of changing as a result of the influence of local and general factors. The study of the role of resident microbes in the origin of oral pathology and their participation in the formation of local immunity makes it possible to determine, correctly diagnose, prevent and timely treat the etiopathogenesis of periodont diseases, caries, diseases of the oral mucosa.

The most basic and relatively slow joints in the organization of primary preventive measures of primary dental diseases are hygienic education of the population, carrying out dental explanatory work and teaching oral hygiene.

Oral diseases are a huge medico-social problem, and the solution to this problem is the task of modern preventive dentistry. It is worth noting that now the main part of Doctor-dentists is occupied with restorative work, while the rate of spread of dental caries and periodont diseases remains high. Due to insufficient time, doctors do not carry out dental clarification work, and do not engage in the Prevention of dental caries and gingivitis.

Active dental clarification work, the creation and implementation of comprehensive preventive programs should be carried out by dentists-doctors and hygienists-nurses.

It should be noted that even the cost-effective method of increasing the dental wellness level of the population is to carry out mass preventive work using all available means and methods of dental explanatory activity.

I. GENERAL INFORMATION ABOUT PERIODONT DISEASES

The prevalence and continued growth of dental diseases are making mass prophylaxis of dental caries and periodont diseases an urgent problem. Information about the intensity of the spread of dental caries and periodont diseases is becoming the basis for the planning of preventive programs in the regions.

"Par "is the surrounding," odont "is the tooth," logos" is the science, which means Periodontology is the science that studies the tissues around the tooth. Parodont tissues include gums, alveoli, bone tissue, periodont, and dental tissue, with parodont tissues being cross-heritable (genetic), morphological, and functionally related. So, a periodont is a sum, complex of tissues in and around the tooth.

Academician N.N.Nesmeyanov (1905) was the first to propose that this tissue complex be called an amphodont member (organi). In the case of the disease, it was recommended that these tissues be taken into account by the transition of one lesion to another, or that all at once be diseased, taking them as the sum — complex of tissues. Subsequently, foreign scholars have proposed various term-terms-such as "parodensium", "parodont". In 1914-1921 Weski recommended the term "parodensium", Kantorovich (1923) "parodont".

The term Parodont is the term adopted by dentists around the world. Diseases associated with Parodont tissue include amphodontosis (I.G. Lukomsky, 1948), "parodontosis", "parodontitis", "parodontopathy", "alveolar piorreia".

In modern times, it has been accepted to refer to parodont diseases as "gingivitis", "parodontitis", "parodontosis", "idiopathic diseases", "parodontomas" each of these terms applies to its own definition. Modern epidemiological indicators are well known that the prevalence of periodont diseases among children and adults is high, as well as tartar in the etiology of the disease, poor oral hygiene, poor quality dentures and fillings, dental-jaw defects, occlusion injuries, disorders in the structure of the vestibular tissue of the oral cavity, breathing by mouth; ingestion of drugs, somatic diseases, violation of the natural immune compensatory mechanism.

In epidemiological studies of PERIODONT diseases recommended by BJSST, social factors (age, gender, social-ecological level); local causes (microbial caraches, occlusion injuries, yatrogenic; fillings, prostheses, defects of orthodontic devices); harmful habits (non-compliance with oral hygiene); systemic factors (hormonal changes in puberty, pregnancy, menopause), drugs (hydantoin, steroids, immunodepressants, peroral substances, heavy metal salts, cyclosporine) must be taken into account.

P. According to the results of research by a Leus (1990), the condition of the parodont was almost the same among adolescents of the same age in all of the settlements examined. Tooth decay and bleeding gums can be observed in children from 5 years of age. At the age of 14-17 years, the average intensity of parodont diseases was observed, and this indicator increases with age.

When Parodont diseases are generalized, chronic gingivitis occurs in 80% of cases in children, hypertrophic gingivitis – 10%, inflammatory and dystrophic changes in parodont – 10% of cases.

T. F. Vinogradova reported that marginal periodontitis in school – aged children is 8.3% in first grade, 8.5% in second grade, 8.5% in third, 9.4% in fourth, 11% in fifth, 16% in sixth, 31% in seventh and 8.9.10% respectively.

In Uzbekistan, gingivitis occurs in 6 – year – olds – 0.9%, in 7 – year – olds-5.2%, in 8-year-olds-2.8%, in 12-year-olds-11.7%, and in 15-year-olds-14% of cases (Bobojonov L.A., 1990). The prevalence of gingivitis was 12-60% when 6,300 school children aged 7-16 were examined in Moscow (Grudyanov A.I., 1985). According to the Ministry of statistics of Ukraine, 70-80% of children have gingivitis at the age of 12 (Khomenko L., 1996).

In the special literature, there are data on the presence of gingivitis in 80% -90% of people between the ages of 10 and 20, according to scientifically based evidence. And after the age of 20 (60-70%) it is noted that there are periodontitis, after the age of 40-periodontosis and other diseases.

Parodont diseases, in particular parodontitis, are particularly common in gastrointestinal diseases (T.X. Safarov, 1986), other internal

organ and system disorders also have a higher incidence of periodont disease compared to healthy people (up to 100%).

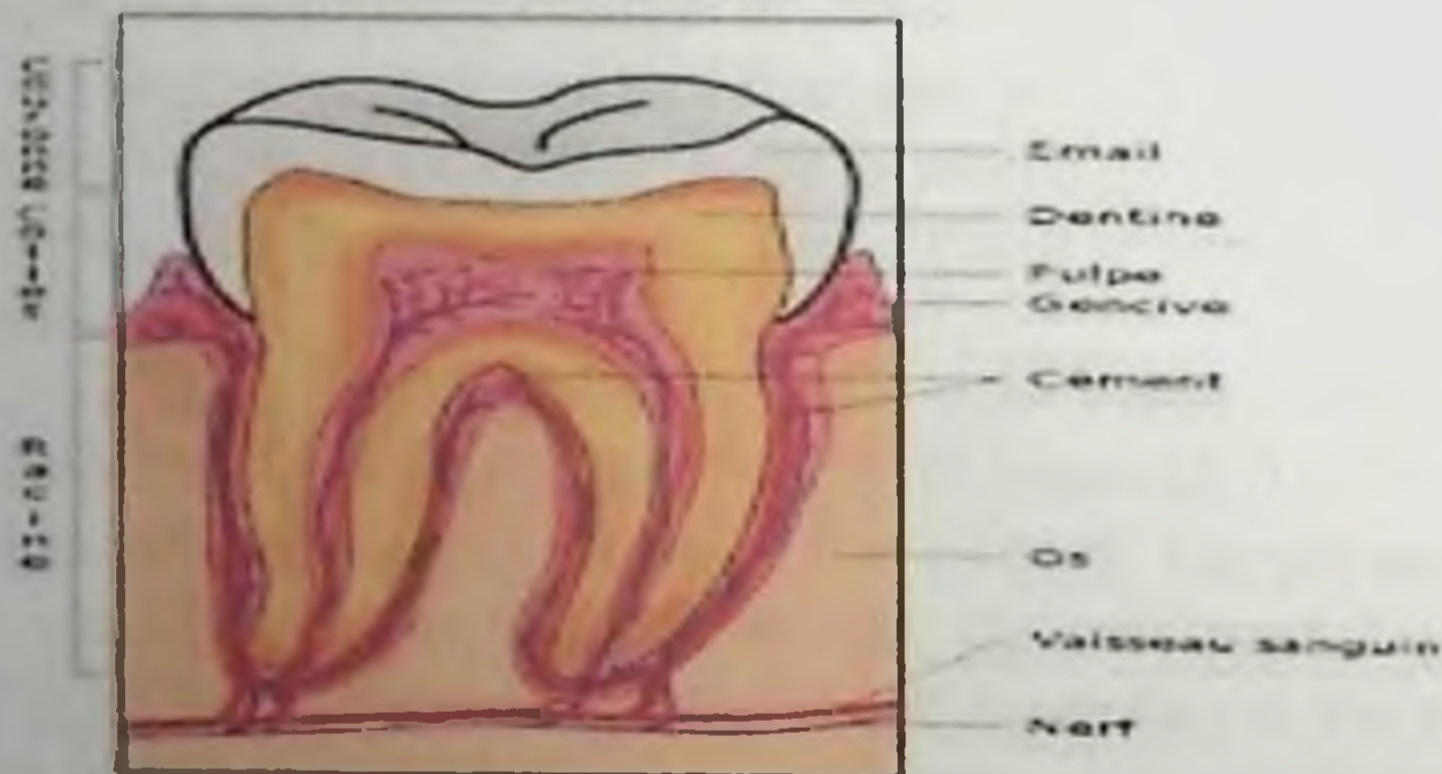
It is worth noting that epidemiological examinations of the population are carried out to assess the prevalence and intensity of oral diseases; to determine the demand for the treatment and Prevention of primary dental diseases and to determine the quality of dental care; to compare the condition of teeth and parodont in children of different regions; it makes it possible to set specific goals for dentists and secondary medical personnel, to assign specific tasks to employees of school and preschool educational institutions, to practically implement the planned program of continuous and rapid preventive measures.

II. STRUCTURE, FUNCTION OF THE PARODONT TISSUE. CLASSIFICATION OF PERIODONT DISEASES

Periodontic diseases are common among the population. According to the WHO, high rates of periodont disease are found at 35-40 years of age (65-98%) and 15-19 years of age (55-89%).

Anatomical and physiological features in the structure of the Parodont tissue are the reason for the manifestation of various pathological processes that have developed in it with different clinical manifestations. The concept of "Parodont" includes the sum of anatomical derivatives derived from the ectoderm and mesenchyma: milk, periodont, alveolar bone tissue, and dental root Cement, which are genetically and functionally one.

The gums are the mucous membranes that cover the alveolar tumor of the jawbone. In the gums, parts of the (gum-bearing) tumor, the edging gum or the gum edge (free gum), the alveolar gum (compound gum) are distinguished between the teeth.



The gum-bearing protrusions between the teeth are triangular, filling the interosseous interval between the adjacent teeth. The free gum is located combed along the edge of the alveolar tumor in the pre-neck area. It is not attached to the Supra-Bony floor and has partial mobility. The free gum is separated from the tooth by the help of a narrow slit – gum pit, the depth of which is from 0.5 to 3.0 mm, on average 1.8 mm. The bottom of the Milk pit corresponds to the area of the enamel-cement compound. The line separating the attached and free part of the gum is the gums furrow, which runs parallel to the gum edge for a distance of

0.5 to 1.5 mm, and its position almost coincides with the feather of the gum pit. The free gum sticks hard to the tooth in the neck area. It has intracellular pressure at the expense of high molecular fibrillar matter storage and is resistant to mechanical effects.

At the expense of connective tissue fibers, the attached gum grows into the hyoid membrane of the alveolar tumor of the jawbone. The surface of the attached Gum has a wavy appearance at the expense of raised parts and recesses. Such a structure of the milk ensures its high resistance to mechanical influences. The gum is covered by a multilayered flat mucosal epithelium that becomes a pit epithelium in the area of the gum pit and loses its mucosal character. The property of decay of the epithelium is considered as a protective function in relation to mechanical, thermal, chemical influences at the time of chewing motion. The multilayered flat mucosal epithelium consists of four floors: basal, thoracic, granular and mucosal floors.

The basal floor is made up of cuboid or prism cells. Compared to the mucous membrane in another area of the oral cavity, these cells have high mitotic activity and perform the function of the cambial element. The thoracic floor is made up of large cells with several layers of the wrong shape. Histochemical investigations have shown the presence of neutral glycosaminoglycans (glycogen) in this floor of the epithelium, the amount of which depends on the degree of Decay. In the course of inflammation, the mucosa of the epithelium breaks down, and the glycogen content increases sharply. The granular floor is very thin, consisting of several layers of thin cells that store n itself. The mucilaginous floor is superficial and consists of mucilaginous flat cells, while the cavity is filled with keratinous filaments instead of nuclei and organelles. Glycosaminoglycans which are contained in an adhesive substance located between the cells of the multilayered flat epithelium, perform the function of a barer, preventing microorganisms and toxins from entering the internal tissues.

Sour glycosaminoglycans – chondroitin sulfuric acid, hyaluronic acid, heparin-enter high molecular complex compounds and perform the function of feeding connective tissue, participate in the processes of growth and regeneration of tissues. Superficial keratinized layers of the epithelium have groups in addition to glycosaminoglycans in the cell cytoplasm and intercellular bridging. In gingivitis and periodontitis, groups in cells are lost due to swelling and intercellular bond elongation.

The epithelium of the gum pit forms the lateral wall of the gum tumor, growing into the gum epithelium at its apex, while the part heading towards the tooth neck borders the compound epithelium. The epithelium of the gum pit consists of basal and thoracic floors and is located between the multilayered flat and connective epithelium. The connective epithelium has few cellular folds, while the intercellular space is large, lacking mucosal and connective tumors. The connective epithelium (attachment epithelium) is a continuation of the dentary epithelium, forming a cuff around the tooth and having a strong connection to the primary cuticle. The plate of the compound epithelium will consist of 15-30 layers of cells in the area of the bottom of the gum recess, with the cells decreasing to 3-4 layers as the tooth orients towards the neck. The cells have a flat shape and are located parallel to the surface of the tooth. The connective epithelium does not contain Langerhans, which participate in immune reactions, as well as Merkel cells, which perform a receptor function.

The high mitotic huskiness of cells is manifested in their rapid renewal (every 4-10 days). The connective epithelium acts as a barre for the periodont tissues, protecting them from aggressive influences in the oral cavity, being highly permeable and providing biodegradable movement of biological substances. Toxins, enzymes, antigens (tooth Caroché metabolites) can fall from the oral cavity into the gum pocket, while a detachment of dead epithelial cells occurs from the deeper layers of the connective epithelium. Dead cells are replaced by a new one, as a result of which the epithelium is constantly renewed. In addition to the exchange of individual cells, the action of vascular cells – leukocytes with a polymorphic nucleus-is also observed in the connective epithelium. The permeability of the connective epithelium can be used in the treatment of periodontium diseases.

It turns out that antibiotics of the tetracycline group accumulate in the gum pit when peroral application, and its concentration is 2-10 times higher than in the blood serum. Opinions differ as to whether milk is attached epithelial to tooth enamel. Some authors (Baume) while epithelial cells are believed to attach using ton fibrils, others thought to be due to the adhesion and stickiness of the epithelium.

Gum honeycomb is filled by milk fluid, which is transudate and is the same in composition as blood serum. As a result of the permeability of blood vessels in the area of the epithelial compound, the fluid of the

gums originates in the gum pit. Milk fluid contains proteins similar to whey proteins: albumin, globulin, fibrin. It has also been suggested that Globulin and fibrin form a cling film, providing a strong adhesion of the epithelium of the gums to the enamel.



And from the cellular elements there are leukocytes with a polymorphic nucleus and a small amount of microorganisms. In Milk fluid, enzymes (cathepsin D, phosphatase), antibodies, AGM immunoglobulins, complement system, are an antimicrobial factor, and the pH of milk fluid can range from 6.3 to 7.9. The qualitative composition of the Milk fluid implies a high baric function for its periodontium. A small amount of fluid is released from the gum cavity into the oral cavity. Medicines sent to the Milk pit are quickly excreted, this feature of it should be taken into account in the treatment of periodontium diseases. Insulating periodontal ligaments should be used to maintain the drug for longer when instilled.

Between the connective tissue and epithelium of its plate, the mucous membrane is located the basal membrane formed from the junction of reticular fibers. Milk's own plate is made of papillary and mesh layers. The papillary is a superficial layer, formed from soft connective tissue rich in vascular and nerve fibers. The mesh floor is made up of a solid connective tissue that is located deeper and contains collagen fibers, which ensure that the fiber adheres to the alveolar tumor bone-top layer. The gum grows into its plate with collagen fibrous Tufts and forms the milk fibers of the periodontium. The personal plate of the

gums contains reticular and elastic fibers, while the floor of the glands and mucus is absent.

Between the fibrous Tufts, cellular elements are assembled. Most often, fibroblasts are found that produce collagen, in rare cases – lymphocytes, Plasmatics and fat cells can be present. Fat cells accumulate in the papillary floor around the blood vessels. In the cervical area, the gum binder consists of collagen, elastic and reticular fibers. Because of this, the milk adheres strongly to the tooth, and the chewing pressure spreads evenly. In relation to the surface of the tooth root, the fibers settle in different directions. Depending on the location and direction of the fibers, tooth-gum, tooth-peridots, tooth-alveolar and tooth-shell Tufts are distinguished.

The gums will have encapsulated and encapsulated nerve endings. Encapsulation produces Krause flasks that act as mechanoreceptors and sensory Meissner bodies. Near the enamel-cement joint is a cuff-like vascular web, connected by anastomoses to the blood vessels of the gums and periodontium. Under the gum pit, blood vessels are located not in the form of a capillary ring, but in the form of an even layer. The last parts of the blood vessels – arterioles, capillaries and venules-are located close to the surface of the epithelium of the gum pit. They are in the form of postcapillary venules and are more permeable to Capillary and arterioles. This property is important in the production of milk fluid. Lymphatic vessels of the gums-the wall will be thin, small in size and have the wrong shape. They are accompanied by veins from the periodontium towards the gums. On the personal plate of milk, especially on the papillary layer and basal membrane, sour glycosaminoglycans are spread, but in the area where blood vessels and collagen fibers are located, they are in small quantities. Fat cells of the connective tissue of the gums store biologically active substances in themselves – heparin, histamine and serotonin. They act as protection and are involved in controlling the transfer of substances to the connective tissue. In addition, fat cells are also involved in the development of glycosaminoglycans.

Periodontium.

Periodontium (periodontium) is a dense connective tissue that places the tooth root between the cement and the compact plate of the tooth cells. The area in which the periodontium is located is said to be a periodontal fissure (Rima periodontal). The change in the width of this

crack makes a person older, the functional pressure of the tooth and pathological processes are of great importance.

For its entire length, the periodontium is in constant contact with the continuous jaw bone, with the dental pulp through the apical hole, through the edge of the tooth cell - with the gums and the overlying bone fragment. Initially, the periodontium is detected in the follicular development phase of the tooth, with root development and growth, and in the development of the cortical plate of the alveolar. The formation of the periodontium ends a year after the end of the development of the root crest.

The width of the Periodontal fissure is on average 0.20-0.25 mm. E.M. According to Gofung, the width of the periodontal fissure in the lower jaw is 0.15-0.22 mm, in the upper jaw - 0.2-0.25 mm. With age, the periodontal fissure becomes larger in width and is 0.37 mm. The width of the Periodontal fissure is homogeneous in all places of the tooth: in the neck area — 0.3-0.5 mm, in the middle part of the root the fissure is 0.15-0.2 mm, going to a narrow peak and expanding the scrotum in the area of the interdental barrier peak (0.25). Thickening of periodontitis will be associated with pathological processes. In connection with a large pressure drop on the tooth, the periodontal thickens at the expense of hypertrophy. Beyond that, changes in the bone cell cause the periodontal fissure to expand. In hyper cements, the periodontal fissure narrows. As a result of inflammation, resorption of the alveolar bone wall and root cement can be observed. The formed but not ruptured tooth periodontitis is 0.05-0.1 mm thick. This indicator is considered 2 times less than a healthy functional tooth. Because the opposite tooth has reduced capacity in the absence of the opposite tooth, its thickness is reduced by 0.1-0.15 mm. In the elderly, the periodontal fissure expands at the expense of periodontium thickening. The periodontium is composed of cells, an intercellular substance consisting of a basic amorphous substance and fibers. The cells are diverse and consist of fibroblasts, plasmatic and obese cells, histiocytes, osteoblasts, cement oblasts and osteoclasts. The periodontium has clusters of epithelial cells embedded in the remains of the tooth-forming epithelium (islets of Maltase). It is glycogen in cells and is structurally reminiscent of the basal cells of the multilayered flat epithelium. Molasse epithelial cells are proliferating in nature and can cause tumors of poor quality.

The periodontium has collagen fibers, the main function of which is to ensure the uniform distribution of mechanical pressure in the alveolar bone tissue, the nerve-receptor system and the micro circular flow of the periodontal. The periodontium fibers are assembled into cohesive Tufts. Depending on the junction and direction, all collagen fiber junctions of the periodontium are divided into 5 groups:

1. Alveolar comb fibers, alveolar bone comb of the neck surface of the tooth

connects with.

2. Horizontal fibers are located below the alveolar comb fibers; their direction is horizontal, forming a right angle to the alveolar bone and root surface.

3. Oblique Tufts-mostly obliquely oriented relative to the longitudinal axis of the tooth. Their entrance to the bone hole is located above the entrance to the root cement.

4. The apical fibers are the apical part of the stem that is oriented perpendicular to the bottom of the alveolar.

5. Inter-root fibers-root bifurcation (trifurcation)in multilingual teeth

in the area of the inter-root barrier connects with the comb.

In addition to collagen fibers, periodontium also contains small amounts of reticular, elastic and oxidizing fibers.

65% of the periodontium intercellular substance is made up of glycosaminoglycans and a basic substance made up of glycopeptides. Ugel is in appearance and is made up of 70% water. With such a structure, the main substance relieves the pressure acting on the tooth. The periodontium is mainly supplied with blood by the upper and lower alveolar arteries. Blood is also supplied through the branches of the dental artery running from the periapical part of the ligament towards the gums and the branches of the superpatriots artery passing through the mucous membrane covering the alveolar tumor.

The blood vessels line parallel to the axis of the tooth root, forming a vascular tangle around the tooth. In the bulk of the capillaries, the conductivity is high, and therefore the main substance of the periodontium, which is hydrophilic, is supplied with water. The veins do not repeat the course of the arteries, leaving the periodontium and heading towards the bone barrier. The periodontium has many anastomoses between arteries and veins. The lymphatic vessel – wall of

the periodontium begins in the form of capillaries from thin, soft, fibrous connective tissue, then passes into the valve lymphatic vessels and continues along with the veins. Part of the lymph vessels diverge to the gums, while others penetrate the bone tissue.

Innervation of the periodontium is mediated by afferent and efferent fibers. Afferent fibers start from the peripheral branch of the dental nerve ahead of the entrance to the apical orifice and from the branch of the nerve entering the canal of the Intertribal and intertribal bone barrier. In the periodontium, the nerve endings are mechanoreceptors and pain receptors, often with a narrow branching appearance. Most of the nerve endings are in the apex area of the root. Non-myelinated fibers are located around the blood vessels and are involved in the regulation of local circulation.

In periodontium, neutral glycosaminoglycans are located along collagen fibrous fumes, in the endothelium of leukocytes and blood vessels, while sour glycosaminoglycans are located mainly on the wall of periodontium blood vessels, in collagen fibers along fat cells as well as the entire periodontium suture. Neutral and sour glycosaminoglycans perform a protective function, preventing connective tissue from entering infections and toxins. Sour glycosaminoglycans also perform tissue growth, regeneration and trophic functions.

Bone tissue. The alveolar growths of the upper and lower jaw are made up of external and internal cortical plates, between which is the cartilaginous bone. The space between the pore bone trabecula is filled with red bone burrow in children, and yellow bone burrow in adults. The upper jaw bone has a large scaly structure, the bone trabecula are located vertically, and the lower jaw bone is of a small cellular structure, with the trabeculae oriented horizontally. The pore bone forms Inter-Root and inter-dental barriers, in which there are vertical channels through which nerves, lymph and conic vessels pass. The tooth root is fixated on the alveoli. In it, 5 walls are distinguished: vestibular, tongue (palate), medial, distal and tubular. The size of the alveolar is smaller than the tooth length, so the edge of the alveolar does not reach the enamel-cement joint, while the apex of the root slightly touches the bottom of the alveolar at the expense of the periodontium. The bone of alveolar tumors is made up of 30-40% organic matter, the main part of which is collagen. And 60-70% of bone tissue is organic substances, the bulk of which is hydroxyapatites, which are located parallel to collagen fibers.

Normal function of bone tissue, its regeneration largely depends on the activity of osteoblast, osteoclast, osteocyte cell elements. Osteoclasts resorb bone, while osteoblasts produce collagen, whose subsequent mineralization produces bone. In the norm, this process is balanced and depends on the activity of hormones, mainly the hormone of the thyroid gland. The bone contains sour glycosaminoglycans, as in other tissues of the periodontium, mainly around osteocytes. In places where the bone is being rebuilt, their amount increases. Neutral glycosaminoglycans are around osteons. On the X – ray, the cortical plate of the bone in the form of a clear line on the edge of the alveolar, while the structure of the pore bone is squamous. The Supra-Bony veil surrounds the cortical plates of the jaw alveolar tumors. It is composed of strong connective tissue and has many blood vessels and nerve fibers. The adrenal membrane is involved in the regeneration and blood supply of bone tissue.

Cement. It is a hard tissue formed from the mesenchyme, covering the tooth root from the part bordering the enamel to its apex. Primary – cell-free and secondary-cell cement are differentiated. According to its chemical structure, cement is similar to bone tissue, it is made up of organic matter (22%), water (32%), phosphorus, calcium salts and microelements. The cell-free cement is mainly located at the neck of the stem touching the dentin. The tooth is made of collagen fibers and adhesive substance, oriented parallel to the axis. Primary cement occurs before the tooth cracks. Cellular (secondary) cement encloses the upper end of the dentin and the inter-root surface in multi-root teeth. It is made up of collagen fibers, viscous substance and cells located in its lacunae – coenocytes, which form anastomosis between themselves and with dentin tubes. Secondary cement is formed once the tooth reaches the occlusion plane. Cement is part of the tooth-holding apparatus, to which periodontium fibers come and attach, it protects the tooth from harmful effects, accumulates in the area of the root crest and acts as a corrector, preventing enamel decay and maintaining the total length of the tooth. Primary Cement has few neutral glycosaminoglycans, but secondary cement contains both neutral and sour glycosaminoglycans.

Blood supply to the periodontium. The periodontium tissue receives arterial blood from the jaw artery, a branch of the external sleeping artery. The upper jaw teeth and the tissues that surround them are supplied with blood using the upper wing-shaped and wing-palate part of the jaw artery. Duck jaw teeth and the tissues surrounding it are

supplied with blood from the lower jaw artery. There are anastomoses between the alveolar and dental arteries and the extrasolar thoracic blood vessels that go to the hyoid. In the marginal part of the periodontium near the enamel-cement joint, there is a vascular cuff formed by anastomoses with gums and periodontium blood vessels. The micro circular flow of the periodontium tissue consists of arteries, arterioles, precapillary, capillaries, postcapillaries, venules, veins and arteriole-venous anastomoses.

The structure of capillaries in the pit epithelium differs from that of the capillaries of the milk (oral) epithelium. The capillaries are arranged in a flat layer under the pitted epithelium rather than in the form of capillaries. The end of the blood vessels – the arteriole, capillary and venules – are located close to the surface of the epithelium. They are in the form of postcapillary venules and have a high permeability to arterioles and capillaries, and are also prone to thrombosis and allergic effects. Capillaries and the surrounding connective tissue serve as protection and ensure the nutrition of the periodontium tissue. The permeability and stagnation of capillaries is important in the development of a pathological process in periodontium.

The lymphatic system of the periodontium plays an important role in inflammatory pathogenesis. They consist of many vessels and are associated with microcirculation of the vascular system. Lymphatic vessels are located side by side with blood vessels and have common collators on the mucous membrane, in the connecting apparatus, in the alveolar tumor bone, in the pulp. In the gums, lymphatic vessels form deep and superficial nets. The superficial mesh has small scales, lacunar expansions, very close to the capillary. Deeply located lymphatic vessels-have large, semicircular valves. The lymphatic vessels of the periodontium surround the tooth root circularly and, together with the microcirculatory system, provide the amortizing function of the periodontium. Lymph is poured from the pulp and periodontium tissue into the lower jaw and subcutaneous lymph nodes.

The innervation of the periodontium is made from the tooth branch of the tangle of the second and third branches of the nerve. Nerve fibers divide the into two parts at the bottom of the alveoli: one is directed to the pulp, and the other – along the surface of the periodontium, parallel to the main nerve axis of the pulp. In the periodontium, many thin

parallel-located nerve fibers come out of the sheath of the main nerve fibers.

The presence of glycosaminoglycans in all tissues of the periodontium protects it in bacterial and toxic effects. The control of the permeability of the capillary-connective tissue structure depends on the enzyme system of hyaluronic acid-hyaluronidase, much more from the state. When hyaluronidase increases, depolymerization of glycosaminoglycans occurs, the bond between hyaluronic acid and protein is severed (hydrolysis), the permeability of connective tissue increases, as a result it loses its barrier property and conditions are created for microorganisms and their toxins to be able to easily penetrate.

A function of the periodontium. Morpho functional properties of the periodontium tissue are adapted to perform the following tasks: holding or shock absorber barrier, trophic, reflector and plastic functions.

The retaining (shock absorber) function is provided by the entire periodontium tissue complex, but the main role belongs to the collagen and elastic fibers in the periodontium gum, alveolar tumor bone. They hold the tooth in the alveolar moustache and resist chewing pressure. In the shock absorber, Inter-tissue slits, cellular fluid and colloid, vascular and lymphatic fluid are involved, which ensure that the force of chewing pressure is evenly distributed throughout the alveolar tumors of the jaw in the tooth row Ham. Soft connective tissue, blood vessel the web of lymph vessels, as well as tissue fluid act as a hydraulic pad.

The function of the Barrier depends on the integrity of the periodontium tissue from the morphological gyrate, which depends on the mugging of the milk epithelium, the large amount of collagen fibers, the specificity of the structure of the gum pit, the presence of the reticuloendothelial cell row, neutrophil granulocytes, fat and plasmatic cells, lymphocytes. Cellular elements maintain constant phagocytosis, the production of enzymes and antibodies. The presence of lysozyme, immunoglobulins, in the oral fluid has an important acuity in its antibacterial properties. The main substance of connective tissue are considered barrier, they are chemically caused by the entry of toxins into the cells and tissues. The protective function is achieved by resorption of bone osteoclasts and peroxidation of lysosomal enzyme activity. In mixed saliva, an increase in peroxidase activity is marked by the development of periodontitis. Milk is rich in vascular mesh, good

innervation, high permeability of its epithelium ensures its active absorption and separation functions.

Trophic function is carried out on the basis of a wide vascular network and lymphatic vessels in the ham at the end of the nerve. It depends on the state of the microcirculation process and innervation in the periodontium tissue.

The reflector function consists in the regulation of the pressure of the kettle, which is carried out on the basis of many nerve receptors. The periodontium -muscle reflex is governed by the force of contraction of the chewing muscles (chewing pressure) depending on the character of the food, the completeness of the tooth row and the condition of the periodontium.

Plastic function is manifested in the constant reconstruction of the periodontium tissue, which changes in the process of physiological and pathological reactions. Regeneration of periodontium tissue is provided by fibroblasts, osteo - and cement oblasts, obese cells. The high speed of the exchange process and the intensity of transcapillary exchange in the micro circular flow have a significant effect.

The functions of the periodontium provide a whole system of adaptation processes. Violation of any function of periodontium changes the natural physiological balance of tissues, and as a result, the ground is broken for the origin of periodontium disease.

III. ETIOLOGY AND PATHOGENESIS OF PERIODONT DISEASES, DIAGNOSIS.

The main part of Parodont diseases is the inflammatory process of the gums and tissues under it, which is called by dental Carache bacteria of a chronic nature.

Gingivitis can persist for many years without transitioning to periodontitis. Gingivitis can be treated if oral hygiene is normalized and soft and hard tooth sediments are constantly cleaned by a specialist.

Most periodontic diseases have an inflammatory nature, and in addition to some forms of dystrophy (atrophy), in adulthood (Juvenil) periodontitis (11-21 years old) also has an inflammatory character in many cases.

Etiology of periodontium diseases.

1. Local factors:

a. oral hygiene factors: - bacterial;

- number of microorganisms, virulence;

- tartar

- anatomical features

- caries

- iatrogenic factors

A. diet(nutritional properties)

B. pathophysiological factors:

- saliva (composition and properties, functional disorders)

- oral breathing

2. Time-organism reactivity

3. General (factors) resistance:

- age

- systemic diseases

- genetic changes

- alimentary factor

- effect of drugs

- psychosomatic factor

4. Occlusive surgeon

Destructive periodontium pathology has shown evidence of the development of local syndrome. The hemorrhagic siptoma of milk is caused by the aggregation of blood-shaped elements inside the vein, the development of the phenomenon and the activation of the Hageman

system factor. Periodontal disease can be considered the result of functional disorders (stresses) in patients, as well as organic changes in the vascular mesh, since the vascular wall is one of the effective managers of the processes of blood clotting and fibrinolysis.

Periodontium diseases can develop against the background of a change in the reactivity of the body as a result of the joint action of local factors (microorganisms in dental, occlusive trauma), as well as local (exogenous) and general (endogenous) factors. In the etiology of periodontium diseases, the following factors are of great importance:

- condition and composition of the exchange in the dental plaque and dental;

- oral cavity factors that enhance or attenuate the pathogenetic potential and Exchange-induced substances of microorganisms;

- pathogenic exposure in the tissues of the oral cavity are common factors that control the metabolism of reactions.

Periodontitis usually follows various degrees of gingivitis. Periodontitis can be partially cured. To date, the reasons for whether gingivitis passes into periodontitis have not been studied to the end. As it is an infectious disease, the main factors are the reproduction of pathogenic microorganisms, their toxicity, tissue entry characteristics and beyond, the individual reaction of the organism can be calculated.

Sterile oral cavity, failure to form on the surface of the tooth is an illusion, i.e. an immaturable condition. Even so, in order for the periodontium tissue to be healthy, should not be collected in large quantities on the tooth, and microorganisms with low virulence (gram-negative facultative anaerobes) should be stored in it. Another basic condition is the good functioning of the body's protective system.

As a result of the appearance of the periodontopathogenic (some gram-negative microorganisms), inflammatory and specific immune reactions occur. These reactions are the effects of protective mechanisms, which also have a destructive property (cytotoxic, immunopathological effects). The destructive effect is more pronounced when the disease persists chronically for a long time.

Bacterial inflammatory products are enzymes, antigens, toxins and also "signaling" substances that activate macrophages and T-cells. Enzymes, toxins and other metabolic products of bacteria can enter the periodontium tissue without calling the primary inflammatory process. In periodontium tissue, bacterial products such as hyaluronidase.

chondroitinsulfatase, proteolytic enzymes, cytokines, and including organic acids, ammonia, hydrogen sulfate, endotoxins (lipopolysaccharides) are ham. If bacteria enter tissues and cells directly (invasion), this is called a real infection.

Periodontitis is a multifactorial disease. In recent years, there has been a renewed view of etiology. Previously, bacteria were considered the main factor. Some pathogenic species of bacteria have been found to be associated with various periodontium diseases and their rate of development. However, the presence of pathogenic bacteria has not always led to periodontium origin and clinical development. In addition, it has been proven that the bacteria in the periodontium pocket do not always cause the origin of this pocket. On the contrary, the pocket is a suitable place for the reproduction and survival of pathogenic microorganisms.

Inflammation of the gums occurs under the influence of endotoxin, exoenzyme and antigen materials produced during the life of microorganisms in soft tooth. On the first hand, bacteria, anti-tissue properties, number and composition, as well as exchange products; on the second hand, the resistance of tissues and the whole organism determines the character and weight level of gingivitis, the origin of periodontitis and the rapid destruction of periodontal tissues. Academician Chunuk A.M. as they say - "inflammation is the reaction of living tissue in the process of evolution with respect to local mutilators, consisting of complex step-by-step changes in micro circular flow, blood system and connective tissue, aimed at isolating and losing the surgeon agent, restoring the surgeon tissue."

Hence, the appearance of inflammation is the first short reaction of tissues to intercellular damage. This reaction ends with long tissue degradation or complete recovery. Bacterial enzymes and toxins are unlikely to directly affect periodontium tissue.

It can be considered a balanced biological system, which is the result of the mutual adaptation of macro and microorganisms into the oral cavity. Normal microflora is characterized by "biological in relation to microflora, which randomly falls on the body, as well as pathogenic microflora. In addition, auto flora is also a constant stimulator of local immunity.

There are more than 200 different microorganisms in the oral cavity. Microflora contains aerobic and anaerobic bacteria, Candida and

simple microorganisms. The concentration of anaerobic and facultative is $10^7 >$ anaerobic 10^8 in 1 ml of saliva. The oral microflora is divided into permanent (resident) and random (transistor) microflora. Permanent microflora of the oral cavity includes streptococci (a and p), saprophytic, lactobacilli, anaerobic cocci (Pepto streptococci and Pepto cocci), bacteroid fuzobacteri spirochetes, etc. Gram-negative aerobic bacteria into non-permanent or random microflora, as well as *E. coli* pseudomonas proteus, include bacilla and anaerobic Clostridia.

The number and composition of microflora is relatively constant and can vary in oral hygiene, patient age, dental condition, etc. It should be borne in mind that microorganisms are not evenly distributed in the oral cavity. The greatest concentration of bacteria is found in the root of the tongue, on the surface of the gum and in dental caroches. 1 gram of dental caroches contains microorganisms equivalent to 10^{11} . Obligate anaerobes are mostly more common in the sub-mammalian facultative and microaerophilic.

In order for gingivitis to pass into periodontitis, it is necessary that there are not only specific microorganisms, but also certain reactions of the organism. Immune reactions caused by pathogens are now well studied. Immunopathological tissue damage occurs if this reaction is very strong, disproportionate to external influences. In addition to immune reactions, there are many factors that affect the origin and course of the periodontium. The main forms of gingivitis and periodontitis are caused by bacteria that adhere to the gum crust by forming dental on and below it. Tooth decay is produced by adsorption of microorganisms from the oral fluid to the gum ego. Such adsorption is achieved by the specific hellenicity of glucose, high adhesivity, water-insoluble polymers, as well as the characteristic of saliva proteins. In addition to the proliferation of microorganisms and in the cell and cells-from products refined with carbohydrates that fall into the oral cavity, soft tooth decay is formed from the synthesis of carbohydrate reserve compounds. Depending on the accumulations of the products of microorganisms, inflammation of the gums occurs. then the ratio of microorganisms forming dental changes. First of all, in these areas, colonies of Gram-positive bacteria (streptococci, actinomycetes), then with the growth of gram-negative cocci, as well as Gram-negative and Gram-negative rods and filamentous forms are found. The main triggers

of inflammation of periodontitis are Gram-negative anaerobes: fuzobacteria, spirochetes, actinomycetes, anaerobic cocci.

The main of bacteria that call for periodontium diseases acute ulcerative gingivitis - Bact. intermedius, Spirochetes

Fetal gingivitis-Bact. intermedius,

Adult periodontitis-Bact. intermedius, Backus gingival

Localized juvenile periodontitis is actinomycete capnocytophaga.

Bacterial enzymes that cause periodontium tissue degradation

Enzyme name microorganism-producer

Collagenase Prohormones gingivitis, Actinobacillus

actinomycetemcomitans.

Trypsin-like protease gingivalis,

Actinobacillus actinomycetemcomitans,

Prevotella intermedia.

Treponema denticola.

Keratinase Porphyromonas gingivalis, Treponema denticola.

Arylsulfatase Campylobacter rectus.

Neuraminidase Porphyromonas gingivalis, Bacteriodes forsythus, Prevotella melaninogenica.

Fibronectin-degrading enzymes Porphyromonas gingivalis, Prevotella intermedia.

Phospholipase A Prevotella intermedia, Prevotella melaninogenica.

The following microorganisms are significant in causing fast-acting (aggressive) periodontitis (up to 35) in adults

Actinobacillus actinomycetemcomitans Bakt. intermedius, Fuzobacterium nucleatum, peptostreptococcus micros, Triponema denticola Selenomonas species intermedius. Porphyromonas-phospholipase stores, which allows it to injure the cell membrane; it also produces indole, scatol isovalerian acid, producing an unpleasant odor in the mouth (galitosis). Bacteroid-resistant to Antimicrobtherapy, resistant to antimicrobial treatment, which begins after 4 days, changes its structure and reduces the effect of treatment.

Inflammation and degradation of the periodontal tissue is mainly caused by bacterial products i.e. chemotoxins antigens and mutagens. As a result of their influence, leukocyte migration to the milk ego increases. At the expense of edema, epithelial attachment with the tooth breaks down, paving the way for the absorption of Gram-positive bacteria and

their products in the area of the tooth-gum joint in the apical direction in the area of the bottom.

Micro-organism life products i.e. a large number of hydrolytic enzymes collagenase, elastases lead to gum breakage and Nishi, resulting in the formation of a periodontal pocket.

In the periodontium, progressive inflammatory changes are accompanied or surpassed by dystrophic changes. Depending on this, microorganisms in one or another HIL multiply in the tissues. The periodontal pockets have facultative anaerobic or aerobic microflora in the initial development of the process, which then releases obligate anaerobes.

The microflora in the periodontal pocket and gum ego when viewed through a microscope is as follows:

In moderation: the predominance of cocci, small motionless rods gingivitis increase in the number of moving and motionless rods, a decrease in the number of cocci

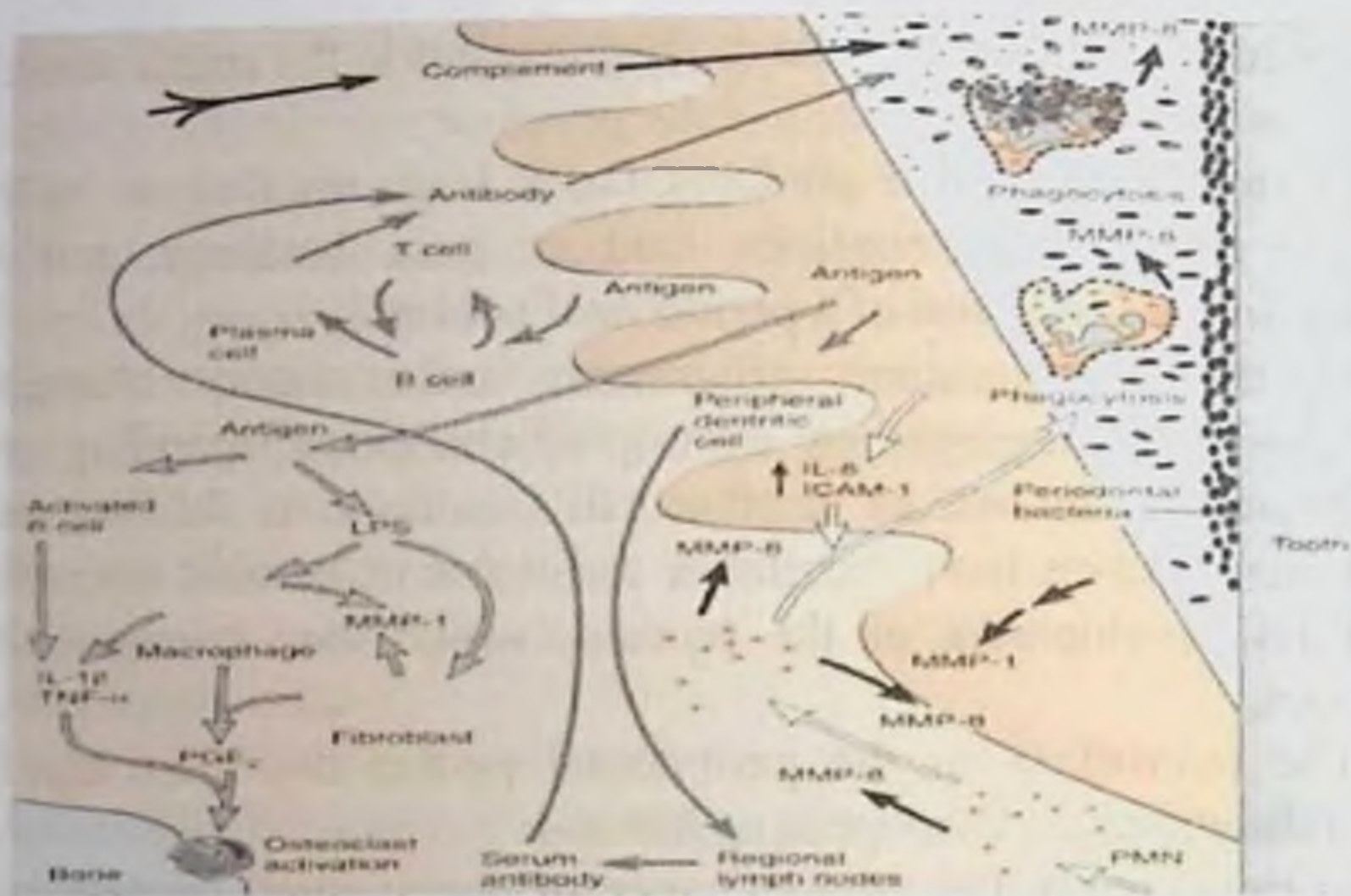
Periodontitis: the predominance of specific forms of rod-shaped and bacterial in motion

In the origin of the local adolescent periodontium it is considered that some microorganisms have a lot of influence in particular *Actinobacillus*, *actinomycetem comitans*.

In the periodontal pockets of fast-acting and treatment-resistant periodontitis, in addition to the aforementioned triggers, there will also be microorganisms that have a different homogeneous aggressive character.

In the etiology of periodontium diseases, the composition and properties of saliva, dental its chemical composition and calcification are of great importance. Mixed saliva has been found to contain more than 60 enzymes. In particular, there has also been found to be a periodontium pathology resulting from a lack of catalase in tissue and saliva.

There is speculation that polymorphic nuclear leukocyte tissue, in addition to producing an enzyme, also recruits immunocomponent cells to the Sox using a chemotoxic reactant. The effect is called by bacterial, like the antigen-antitelo reaction mechanism and the complementary system. This reaction is influenced by cellular and humoral immune mechanisms.



The results of the study of the role of pathological staphylococci in inflammatory reactions of periodontium diseases, that is, by finding formed in relation to pathological staphylococci and other microorganisms, make it possible to determine whether purulent-inflammatory reactions are associated with the action of pathological staphylococci.

Pathogenic microflora with antigen substations in the Periodontal pockets and dental has a sensitizing effect on periodontium tissue. This leads to an increase in the intensity of tissue alteration and the formation of tissue auto antigenlame, as well as a microorganism and tissue complex. The immune system responds to these antigens. In the first cases, a protective immune response occurs without homeostasis disruption, which is maintained until the functional state of T - and V - lymphocytes is impaired. In other cases, T-lymphocyte suppressor function attenuation resulting from chronic effects determines the clinical symptoms of the disease as well as the nature of "spontaneous" withdrawal. Tooth decay and stones cannot always be considered a local factor, since their formation and activity will depend on the state of reactivity of the body (mineral and protein content of saliva, fluid, its enzymatic activity, immunoglobulins and clinical system. The development of a pathological process in a periodontium can be visualized as follows. Infectious agents are osteoclastlamy activating bacterial toxins (lipopolysaccharides, lipotenic acid dipeptide.) are produced. Polymorphic core leukocytes, platelets, monocytes attracted

to the foci of inflammation produce prostoglandin-producing macrophages. Prostoglandins are able to directly activate osteoclastlam, as well as acting on macrophages to force the production of the osteoclastlamy activator - FAO factor. Local and systemic factors together lead to the development of periodontitis with pronounced inflammatory - destructive changes. Hence, the activity of inflammatory processes in the periodontium is characterized by increased growth of the microorganism in the periodontal pockets, changes in composition and increased aggressive properties. The process of bone resorption forms the basis of the mechanisms of the development of periodontium, while a large role in tissue degradation is played by complement, since it is activated using an antigen - antitelo complex, which not only enhances the synthesis of E, but also leads to resorption of bone tissue.

Hyperfunctions in the periodontium appear as a complication of periodontitis in cases where partial tooth loss causes to crumple, and the ratio of Root to crown portion length does not overlap. Overloading of the backbone tissue is observed with impaired microcirculation in the periodontium tissue.

Tooth decay and Stone cannot be considered the only local factor affecting the tissues of periodontium since the formation and activity of depends on the state of reactivity of the body (changes in mineral and protein metabolism of saliva, milk fluid, enzymatic activity, the presence of immunoglobulation and the quinine system).

Below are the etiological factors that influence each other of tooth decay and macroorganism.

Bacteria:

1. The main cause of periodontitis is pathogenic microorganisms of dental.

Macro organism.

2. The origin and course of periodontitis can be influenced by genotypic immune reactions (specific)systemic diseases (diabetes mellitus) and syndromes.

3. Harmful habits (smoking, alcohol, drugs, nutrition) and the attitude of patients towards their own health affect the accumulation of tooth decay and immune reactions.

4. The physiological and psychological state of the patient depends on the social conditions. For example, social and economic problems lead the body to a state of stress.

5. The immune state is negatively affected by emotional tension and stress.

As a result, the balance between the damaging factors (bacteria) and the body's protective forces is disturbed. If the bacterial flora is highly virulent, and the body's reaction is sluggish (immunodeficiency condition), the periodontitis develops rapidly and proceeds aggressively.

Microbiology. Today, more than 530 species of bacteria have been identified in the oral cavity, the bulk of which are in ecological balance and do not cause disease. Opportunistic or facultative pathogenic microorganisms are sometimes observed in periodontitis or fungal infections. It is not yet clear whether these bacteria are the cause of the disease or whether the pathological process is favorable conditions for their reproduction. Nospecific dental (mixed flora) calls experimental gingivitis in 7 days. Gingivitis quickly recovers when tooth decay is removed. Various types of periodontitis, especially aggressive type, manifested by rapid inflammation, are associated with a specific appearance.

The mouth-larynx is an open ecosystem with permanent bacteria. They try to occupy all surfaces. On smooth, unprocessed surfaces (hard tissue of the tooth, restorative materials, implants, prostheses), bacteria can live only in the form of a. In the norm, there is a balance between the formation and decay of biofilm. The decay of the biofilm is caused by the mechanism of self-cleaning of the lunge and tongue, proper nutrition and hygiene rules.

Tooth decay can take from several hours to several days to appear and is divided into the following phases:

A few minutes after the tooth surface is cleaned, a pellicle of glycoproteins and protein is formed.

A. Approach. Under the influence of physical forces, bacteria sink to the surface of the pellicle.

B. Adgesia. Primary colonization of some bacteria occurs, notably streptococcus and actinomycetes. To do this, on their surface there are special bacteria (adhesins) that bind to pellicular receptors. Microorganisms are then attached to the "primary columns".

C. Bacterial growth begins.

D. Microcolonies are formed. Most streptococci produce extracellular polysaccharides (dextrans, Levans) that are protective.

E. Bioplyonka ("compound cabbage"). Microcolonies form groups conducive to bacterial metabolism.

F. Tooth growth and maturation Bioplyonka is characterized by a primitive "rotation".

Tooth decay begins to behave like a whole organism. The number of anaerobic microorganisms increases. Cell wall components and metabolic products (lipopolysaccharides, vesicles) enhance the body's immune response. Inside the the bacteria will be protected from phagocytic cells and exogenous bactericidal preparations.

Gum surface tooth decay and its spread under the gum. The first bacteria that accumulate on the surface of the tooth (on the gums) are mainly (*Streptococcus* sp., *Actinomyces*). In the following days, the colonization of Gram-negative cocci, as well as rods and filamentous bacteria, begins. The products of the metabolism of bacteria affect the tissues, exudation and migration of polymorphic-nuclear leukocytes (PMYaL) begins in the gum pit. This causes the connective epithelium to soften. The distance between the tooth and epithelium is opened so that bacteria can enter under the gums. Gingivitis develops, a gum pocket occurs. When hygiene measures are not applied in time, dental accumulates and the primary protective reaction of macroorganism begins. In normal oral hygiene, when the interdental spaces are cleaned, the dressing is interrupted and the gums remain healthy.

Natural factors that lead to retention of tooth decay. Natural factors of retention not only cause dental to accumulate, but also make it difficult to implement hygienic measures. Includes retention factors:

- cement-enamel compound, enamel drops;
- irregularities and recesses in the field of furcation;
- fissures and cracks;
- neck caries root caries;
- tightness of teeth.

Tartar is not pathogenic. But its uneven surface is favorable conditions for the retention of pathogenic bacteria. The cement-enamel compound is also uneven, rough-here it can be seen under a microscope. Enamel drops and tumors damage the normal attachment of the epithelium. Cracks, fissures and recesses in the furcation area are characterized by a retention zone. Carious Sox accumulate large amounts of bacteria. Dental congestion makes oral hygiene and self-cleaning system difficult.

Yatrogenic factors of dental Carache retention. Restorative practices can be more harmful to the oral cavity than benefit when improperly implemented, from simple fillings to prosthetics. A properly performed restoration, on the other hand, prevents periodontium diseases.

These include prosthetic defects (dentures dipped into the gum, a complete removable prosthetic base with the gum suckers pressed), dental treatment defects (an excess of amalgam or composite material), paste drop into the Intertribal interval, prosthetic Monomer effect, orthodontic apparatus misinterpretation.

Another of the causes that generate dental are the anatomical features of the teeth, the location of the in the tooth arch, and the carious injury. These causes can cause papillitis, gingivitis, less often periodontitis.

Irregularities are always found when viewed microscopically in fillers and coronks, which look without flaws from macroscopic humor. If such irregularities are under the gums, the periodontium tissue can scratch. The hanging edges of the filler and the Corolla create additional conditions for the accumulation of dental Carache, and gingivitis develops. The composition of Karash changes, the amount of Gram-negative anaerobes increases, which leads to the development of periodontitis.

The gum surface tooth decay or bioplyonka slowly begins to spread under the gum, into the gum pit and into the pocket. As a result of an increase in the depth of the pocket along with Gram-negative bacteria, the number of anaerobic gram-negative bacteria increases. Bioplyonka can be limed, and as a result, a dark, hard, hard removable ("whey") tartar is formed. In addition, an agglomerate of soft, independent, motile bacteria can also be seen in the pocket. During the lambing period, the number of periodontopathogenic bacteria increases dramatically. They include *Actinobacillusactinomycetem comitans*, *P. gingivalis*, *T. forsythia*, *spiroxeta* et al. Despite such changes in the composition of, even in the acute period of periodontitis cannot be called a "high-specific" infection, since different bacteria can be encountered in different areas of the oral cavity in different patients and even in the same patient himself.

Entry of bacteria into tissues. When the organism's immune response is slow, when the periodontitis is rapid and aggressive,

pathogenic bacteria can penetrate the epithelial pocket and the connective tissue under it and maintain life activity for a long time. Most often this occurs in deep parts of the pocket, where inflammatory infiltrate (protective mechanism), which develops near the edge of the gum, does not harm to bacteria. Periodontogenic bacteria produce virulence factors that block phagocyte chemotactic control. Over time, bacteria are found and destroyed by the active immune system. If bacteria enter in small quantities, a small foci of necrosis appear in the tissue, if in large quantities, an acute purulent abscess develops.

The origin of periodontium diseases is also influenced by general factors that reduce the reactivity of the body. Examples of these are the lack of vitamins C, B, A, E. Lack of ascorbic acid in the body in the first place affects the connective tissue elements of periodontium especially collagen fibers, the permeability of capillaries and intercellular structures increases, the formation of bone tissue decreases, stagnation of periodontium tissues in relation to infection decreases. When Vitamin A is not enough, the barrier function of milk weakens, which leads to inflammation. As a result of Vitamin E deficiency, changes are observed in the blood vessels of the periodontium, nerve fibers. As a result of the deficiency of B and C vitamins, damage to periodontium tissue (gingivitis, periodontitis) and stomatitis in the mucous membrane of the oral cavity have been observed in experimental animals.

Inflammation of the gums begins in the area of the gum pit, in which the composition of the milk fluid changes. In Milk fluid, the number of PMYAL increases, in which, under the influence of endotoxins, degranulation of cells occurs, and the lysosome exits. Lysosome enzymes (protease, hydrolase, lysozyme) interact with surrounding structures to promote alteration.

As a result of degranulation of fat cells (macrophages), the cells and plasma mediators of inflammation come out. Bradykinin (plasma mediator) causes histamine release, which increases vascular permeability, while the kallikrein-kinin system of the plasma mediator disrupts blood clotting by activating the chemotaxis of PMYAL and factor XII (Hageman factor). Cell mediators include histamine, serotonin, prostaglandin, lymphokinin, which is produced by PMYAL, mast cells and basophils. Biologically active substances dramatically increase vascular permeability, microcirculation is disrupted, blood flow is impaired, thrombus formation is accelerated, vasculitis occurs, hypo

coagulation and hyper fibrosis, secondary hypoxia occur. These changes lead to the depolymerization of intercellular substances in the epithelium of the gum pit, vacuoles, cracks appear, favorable conditions for further penetration of bacteria and their toxins appear.

Violation of microcirculation increases tissue permeability to ham, resulting in a bulge of vascular walls and perivascular tissue with protein components (albumin, fibrin-fibrinogen, immunoglobulin G, A, M). Solid inflammatory infiltrates are formed, consisting mainly of lymphocytes and plasmatic cells, and the structure of the gums changes. Depolymerization of the main substance of connective tissue occurs. The bacteria produce the enzyme hyaluronidase, which disrupts the periodontium tissue. The enzymes collagenase, aminopeptidase, elastase, phosphatase, phospholipase are involved in collagen degradation of tissues and bacteria. The weakening of fibroblast function disrupts the process of collagen production. Violation of the protective mechanism leads to a weakening of the regeneration process, the appearance of pathological granulatory tissue and the deepening of inflammation to the alveolar bone. Microbial sensitization triggers autoimmune processes. The effect of microorganisms on the periodontium and the response reaction of the organism determine the degree and character of damage to the periodontium. It causes destruction of the alveolar bone and various pathogenetic immune processes: anaphylactic, cytotoxic, immune complex reactions, etc.

In anaphylactic reaction, IgE produces plasmocytes that are fixated or sensitized with tissue basophils. The continuation of the antigenic effect leads to the fact that on the surface of the cell the antigen-antitana complex is characteristic, and tissue basophils are degranulated, from which histamine and other inflammatory mediators such as this are released. Leukocyte infiltration is associated with these reactions, in which enzymes that lead to the breakdown of periodontium tissue are released.

Vitamin deficiency. Tissue disorder, vitamin C, B, A, E deficiency also have an effect on the pathogenesis of periodontium diseases. Hypo- and avitaminosis C is accompanied by inflammatory, destructive changes in the periodontium tissue. Vitamin C deficiency in the body is observed in a violation of the process of formation and formation of collagen fibers, the tissues relax, the permeability of the intercellular substance and capillaries increases. Mature collagen fibers undergo

destructia. In addition, at a lack of vitamin C in the body, the formation of bone tissue slows down, the resistance of periodontal tissue to infection decreases.

Changes in periodontium tissue caused by Vitamin A and B deficiency in the body also cause periodontium diseases. In the processes of inflammation of the gums, serum vitamin A decreased by 75% in patients, the stronger the inflammation, the less vitamin A. However Edelberg (1991.) also studied the effects of vitamin A in the clinic and concluded that it does not affect any gingivitis index. In E-avitaminosis, however, the oil free radical oxidation increases to form periodontal pockets, bone atrophy, and the periodontium vascular system changes.

Atherosclerosis of the vessels. A.I.Evdakimov considers parodontosis to be a dystrophic process, which is the result of atherosclerotic changes to the periodontium vessels resulting from both general and local atherosclerosis. The pathomorphological research done by the same author shows changes in the walls of arterial vessels in the periodontium (perivascular sclerosis, thickening of the vessel walls, especially the middle floor, hyalinosis, narrowing of the fissure).

In addition to atherosclerotic changes in the periodontium vessels, the nature of cholesterol in atherosclerosis itself is also important.

Nervous-dystrophic nature of parodontosis

It is clear that atrophic - degenerative changes in tissues are the result of a lack of blood supply. However, nutritional disorders are also observed as a result of the inability of nutrients flowing into the tissue itself to assimilate if blood supply is good. In doing so, nutritional disorders are seen as a neuromuscular factor.

The effects of the nervous system have been studied not only in controlling the metabolism of substances in tissues, but also in maintaining the structure of tissues, accelerating the process of regeneration, and it has been proven that the nervous system depends on these processes. Therefore, a violation of the trophic process caused by reflectorally acting on nerve derivatives can be caused by a pathological process, i.e. dystrophy.

Decrease in the reactivity of the body

From a decrease in the reactivity of the body, as well as a lack of strength of protective and compensatory mechanisms, it is possible to observe the development of a dystrophic process in the periodontium.

This is seen in physicochemical changes, first of all in extracellular structures (GAG, collagen fibers), which in turn leads to changes in vascular permeability disorders, circulatory disorders, tissue hypoxia, metabolism.

Hypoxia

Many literature records that hypoxia in periodontium tissue occurs in the form of dystrophic - inflammation of parodontosis. One of the indicators that characterize the degree and specificity of oxidation - reduction processes in tissues is the oxidation - reduction potential (OQP). It shows that the ratio of oxidized and reversible forms indicates the absorption of oxygen in tissues during the metabolism process. E.V. Krechina (1988.) and N.K. Loginova (1994.) Studied OQP and showed it to decline in periodontium diseases, indicating that unoxidized productling remains in tissues. With the development of the Dystrophic process, OCP also begins to decrease. As a result, the barrier function of the periodontium tissue decreases, leading to pathological manifestations (bone resorption, gingivitis, periodontal pocket formation) characteristic of periodontium disease in combination with general and local factors.

Endocrine disorders

Many scientists have studied the relationship between periodontium diseases and endocrine disorders. In the hypo - and hyperfunctions of the thyroid gland, The Shield gland, the gonads, it was found that periodontium diseases occur.

Especially in diabetes periodontium changes have been studied. With periodontium angiopathy, net and kidney angiopathy is among the first and most common surgeon - 90-93% of diabetics are diagnosed with periodontium changes. In the pathogenesis of parodontal syndrome in diabetes mellitus, periodontium anigiopathy is central. It is of great importance in the violation of small vessels, first of all, dysproteins and an increase in the amount of GAG in the blood. Pathomorphological changes of vessels in diabetes D.A. Zerbino (1977 y) has a specific character according to his research: the vessel crack does not disappear, but the walls are injured. On the basis of diabetic microangiopathy, plasmorrhagia processes lie. It causes primary plasmatic injury of the basal membrane of the stream, and later wall sclerosis and hyalinosis. These changes are inflamed, which has nothing to do with it. Hence, microcirculatory disorders are of a primary nature. Against the background of impaired transcapillary exchange and increased

permeability of periodontium - connective tissue structures, the microflora in the gum fissure induces inflammatory-destructive changes, the load generated in periodontium tissues further grouping them.

Diseases of the digestive system

Gastric and duodenal ulcer disease is considered to be one of the causative agents of periodontium disease. In almost all types of OIC (gastrointestinal diseases), one form or another of periodontium diseases occurs. Due to the change in the functions of the thyroid and the thyroid gland, calcium metabolism, hormonal control is disturbed. Clinical-laboratory research shows that pathology of the digestive system (in 85-91% of cases) is observed accompanied by inflammatory forms of periodontium diseases, and in 9-14% - by parodontosis.

Diseases of the blood and hematopoietic system

In anemias, dystrophic changes in periodontium tissue are observed, and in acute leukemia of children and adults, milk sucker hyperplasia is observed. In safe neutropenia (acyclic and persistent), there are no changes to itself - resorption and osteoporosis of bone tissue. In periodontitis, the rate of formation of prothrombin and thrombin decreases.

Periodontium, in relation to healthy people, occurs more often in people with diseases of the digestive system, vascular system, endocrine system, liver, kidney, nervous system diseases, rheumatic diseases (collagenoses), hypertension and allergic diseases.

The WHO committee of experts on parodontology has proposed a scheme of the etiology of gingivitis and parodontitis, relating the concept of "time factor" to the time, consistency, microbial composition, and marginal periodontium variation of tooth Carache formation.

Psychosomatic factors

In the development of periodontium diseases, xerotomies, taking drug drugs (sedatives, tranquilizers, neuroleptics) are also the causes.

Systemic factors include genetic predisposition (adolescent periodontium diseases), sexual maturation disorders, deviations in polymorphic - nuclear leukocyte structural organization, the following from adverse drug substances: corticosteroids, immunodepressants, hydantoin, heavy metal salts, peroral pregnancy prevention pills, ciclosporin. In all forms of periodontium surgeon the functional state of the connective tissue is damaged.

Immunological disorders

Many scientists have found that in periodontium diseases, specific and non-specific oral defense factors are damaged, indicating immune disorders of the T and V lymphocyte systems. Even if parodontitis and parodontosis are not a typical autoimmune disease, immune system pathology plays a big role in the inflammatory - destructural processes of periodontium. However, in the case of inflammation, the autoimmune component in the mechanisms of degradation of the periodontium tissue is also relevant as a zveno. A Normal organism has a very sensitive immunological apparatus that can recognize foreign antigen. This feature is genetically given through generations, so the predisposition to certain diseases is manifested in one generation by passing the disease in the dominant type.

Experienced and comorbid diseases, hormonal disorders

These conditions lead to a decrease in the local barrier resistance of the periodontium and the development of autosensibilization and immunopathological processes accompanied by resobcia of the alveolar barrier-bone tissue of the jaws

Diagnosis of periodontium diseases.

Traditional methods of oral examination are used when examining patients with periodontic disorders. The purpose of examination in diseases of the pardontic tissue is to assess the general condition of the body, determine the maxillary and general etiological and pathogenetic factors, determine the type, severity and nature of the course of the disease. A comprehensive examination allows you to make the correct diagnosis and carry out the necessary treatment measures and carry out Disease Prevention.

Verification starts with a questionnaire. The doctor asks for the patient's complaints, the cause of the appeal, social conditions, occupational risk factors, chronic diseases, allergic status, diet and characteristics. When collecting Anamnesis, it is necessary to pay attention to bruxism (nocturnal tooth wheezing), whether there is pain in the chakka-lower jaw joint, bleeding gums, bad breath when eating and cleaning teeth (the reason for this can be not only diseases of the oral cavity, but also diseases of the gastrointestinal system, ENT-organs, respiratory organs). The reason for the arrival of an unpleasant odor from the oral cavity may be the arrival of purulent detachment from the periodontal pocket, ulcerative-necrotic gingivitis, improper oral hygiene,

carious teeth, prostheses, leaks in chronic odontogenic inflammatory processes.

Visual examination is of great importance during the examination process, when the oral cavity is examined and the condition of the periodontium tissue is determined. In a visual examination, attention should be paid to the color of the skin covers, asymmetry in the face at the expense of scars, edema, whether there is a facial nerve paresis, the condition of the lymph nodes under the lower jaw and chin, facial proportions, the appearance of the lip-nose and chin folds. Facial proportions are disturbed in prikus anomalies, tooth friction, and the absence of teeth.

The examination of the oral cavity begins with immunity, in which attention is paid to the color of the mucous membranes, the tightness of the grooves, the depth of the suture, the condition of the prikus and teeth.

Methods and diagnosis

The purpose of clinical examination methods is to accurately determine the patient's diagnosis of the disease. This in turn helps to properly treat or prevent the disease. In periodontology, oral examination is the primary branch of examination of a patient. Verification methods are in turn divided into basic and additional verification methods. Basic verification methods are conducted in a way that relies on subjective and objective information. Subjective examination methods begin with anamnesialization from the patient.

Anamnesis collection

Examination of the patient begins with the collection of (anamnesis vitae) and disease Anamnesis (Anamnesis Morbi). Awal is determined by the patient's complaint, the reason for going to the doctor, the presence of professional and chronic diseases, diet, harmful habits, etc.

During the collection of Anamnesis in periodontium diseases, it should be noted that there is mainly bruxism. During the collection of Anamnesis, the patient is asked referral questions in many cases, and the information necessary for the diagnosis is collected.

Review. When studying dental status, it is necessary to focus on the position of the entrance to the oral cavity, especially on the lip grooves, the depth and shape of the oral entrance. Taking into account the influence of local and general factors in the development of inflammation in the marginal tissues of the periodontium, dental

placement (presence of location anomalies, congenital partial adentia, diastema, and tremas) should be considered. Early tooth decay, especially molars, often leads to traumatic bites (pricuses). The absorption of adjacent teeth, the growth of the anthonistlami also leads to the loading of the periodontium. Great importance should also be attached to the act of swallowing. When swallowed in Normal condition, the tongue should not be worn on the front teeth. Violation of the act of swallowing can be the only reason why the anterior bite, especially the lower teeth, protrudes forward, which in turn leads to a violation of articulation and, as a result, to the loading of the periodontium tissue and the development of the pathological process.

Among the pathologies of the smooth pricus, the deep pricus is of fundamental importance, since in this case pathological changes develop in the periodontium of the anterior teeth of the upper and lower jaw. Such changes are also found in the open prikus and other prikus anomalies, but relatively less so.

Inflammatory processes in the gums are accompanied by redness, swelling, bleeding, deskvamacia and scarring. In addition, atrophic and hyperplastic processes, gallstones and gallstones, abrasions are also detected. To identify tooth decay and stones, they are painted using a Schiller - Pisarev solution or 1-2% methylene breast. for this purpose, erythrosine tablets are also used, which dissolve in the saliva and stain tartar.

Ponoram enlarged radiography and orthopantomagraphy. Rather than intra-oral X-ray, orthopantomagraphy helps to objectively assess the state of bone tissue of alveolar tumors in periodontium diseases. The X-ray shows inflammatory changes of bone tissue (resorbcia) in periodontium disease. They are accompanied by the destruction of the porous bone and the formation of bone pockets, starting with the injury of the cortical plates.

Periodontosis is characterized by sclerotic changes in the bone tissue of the jaws on an X-ray. The height of the inter-alveolar barrier can decrease evenly. To determine the Radiological appearance in the absence of an orthopantomographer, we recommend x-ray on the 654456 tooth socket. This allows, on the one hand, to determine the changes in bone tissue in the oxae of all tooth groups, and on the other hand, to determine the symmetry of the formation.

Measure pocket depth. It is carried out using a special graduated probe and a antift Guttaperchali antifreeze is the safest. The is inserted into the pocket until there is resistance in the direction of the tooth axis, the place where the scarf is worn is assessed as the bottom of the pocket. When there are bone pockets, the shaft is inserted to the bottom of the pocket.

Now, computer tomography is used to determine the character of a periodontal pocket, especially in the study of pocket topography.



The pocket depth dental formula defines Arabic numerals opposite each tooth, while the degree of crumpling is Roman numerals. Hence, the degree of parodontid surgeon is determined on the basis of radiographic examinations.

Morphological examination of biopsy material. This method is important when making an accurate diagnosis. In some cases, however, (eosinofd granulema, traumatic disorders, collagenoses, idiopathic forms of periodontium injury) is one of the main indications in the diagnosis.

Bacteriological method. With this method, it is possible to determine the microflora of 67ilk pockets, as well as leukocyte migration according to the Yasinovsky method in the process of cytological changes and treatment. Neutrophil leukocyte migration through the oral mucosa indicates involvement in phagocytosis.

In the cytological examination, the material for the print-grease is obtained using rubber strips. For general vision, it is painted according to the Romanovsky - Gimza method, and for nucleic acid, glycogen

Paradontology

detection, it is painted using special methods, for example in Brashe and Mac-Manus. Preparations are seen under an immersion systemic microscope. For cytomorphological and citochemical BA trapping, the study of the number and qualitative composition of cell elements (neutrophil leukocytes, epithelial cells), the distribution of glycogen and nucleic acid in cells is used.



IV. CLASSIFICATION, CLINICAL PICTURE, DIFFERENTIAL DIAGNOSIS AND TREATMENT OF PERIODONT DISEASES.

Modern Periodontology has gone a long way of development. For many years, there was no single systematized classification of periodontium diseases, so that dozens of classifications appeared.

At the XVI plenary meeting of the Scientific Association of dentists in 1983, the classification proposed by the WHO was approved.

I. Gingivitis is an inflammation of the gums and is the result of an unfavorable effect of both the maxillary and general factors, and this tooth-gum combination proceeds intact.

Type: catarrhal, hypertrophic, ulcerative-necrotic.

Duration: acute, chronic, lameness, period of remission.

Weight class: light, medium heavy, heavy.

Distribution: local, generalised.

II. Parodontitis is an inflammation of the periodontium tissue characterized by destruction of the periodontium and bone.

Course: acute, chronic, laminated (including abscessed), remission.

Weight class: light, medium heavy, heavy.

Distribution: local, generalised.

III. Parodontosis is dystrophic damage to the periodontium.

Course: chronic, remission.

Weight class: light, medium heavy, heavy.

Distribution: generalized.

IV. Idiopathic diseases of periodontium are manifested by lysis of periodontium tissue (Papiona-Lefevre syndrome, histiocytosis-X, uncompensated diabetes mellitus, neutropenia, etc).

V. Periodontomas are tumor derivatives of periodontium (gum fibromatosis, etc.).

In recent years, a fast-paced type of periodontium has been isolated. Four types are distinguished, depending on age and nature of discernment:

- prepubertat (under 11,)

- local adolescence,

- generalised adolescence (ages 12-21,)

- adult fast-moving periodontitis (21-35 years old).

In 1993, the European Federation of periodontologists proposed a new classification, which was further modified in collaboration with the

American Academy of periodontologists at the time of the International Symposium in 1999-2000.

Classification of periodontium diseases (APA and YePF International Symposium, 1999/2000).

I-Type. Milk diseases

A. Gum diseases associated with tooth decay

V. Gum diseases that are not associated with tooth decay

Type II. Chronic periodontitis

V. Generalized

Type III. Aggressive periodontitis

V. Generalized

Type IV. Periodontitis in the outcome of systemic diseases

A. Related to blood diseases

V. Related to genetic diseases

S. Related to other common diseases

V-Type. Necrotic diseases of the periodontium

A. Ulcerative-necrotic gingivitis

V. Scar-necrotic periodontitis

Gingivitis is characterized by the following diagnostic manifestations, regardless of various clinical - morphological forms.

1. Soft tooth decay and the presence of tartar on the gums.

2. Iodine positive reaction.

3. Lack of tooth - gum pocket due to intact tooth - gum ligament.

4. Milk bleeding.

5. Sickle clinical - morphological changes in milk. Catarrhal gingivitis -

edema and change are disorders of the mucosal process of the epithelium, lymphocyte - leukocyte - plasmacytic infiltration, vascular reactivity. In hypertrophic gingivitis-swelling of the fibrous elements connecting the gums lymphoplasmic infiltration, dilation of the vessels; and in the form of fibrosis-the predominance of fibroblast proliferation,

6. The X-ray shows that the interdental barriers are not affected by destructions.

7. The general condition of patients has not changed, except for acute and chronic forms of catarrhal and ulcerative gingivitis.

IV.1. CATARRHAL GINGIVITIS. CLINIC, COMPARATIVE DIAGNOSIS, TREATMENT, PREVENTION.

Gingivitis is an inflammatory process of the gums, which occurs as a result of the negative influence of general and maxillary factors, without a violation of the integrity of the tooth-gum joint. The following types of gingivitis are distinguished: catarrhal, hypertrophic, ulcerative, atrophic and desquamative. The first three species are widely distributed.

Catarrhal gingivitis is more common in children, adolescents, and people under 30 years of age. The main causes of origin are changes in the reactivity of the body and general somatic diseases (cardiovascular, gastrointestinal, endocrine, infectious, blood diseases and radiative lesions) a decrease in the resistance of periodontium tissue under the influence of harmonic changes in the ham. Maxillary factors include unsatisfactory oral hygiene, as a result of which the appearance of microbial prosthetic and filler defects, dental-jaw anomalies, soft tissue anomalies.

The severity of gingivitis is determined by the general changes in the body and the spread of the inflammatory process. For mild Grade gingivitis, damage to the gums is characteristic, for the middle grade – the damage to the free (marginal) milk, for a heavy grade – the damage to the entire milk, the attached (alveolar) milk. In gingivitis, the integrity of the gum-tooth joint is not disturbed, but at the expense of the Intertribal tumor, the gum arch appears to deepen. In local gingivitis, inflammation of the gums is detected in 1-2 tooth sacs. The cause of such a limited inflammation is the maxillary factors. In generalized gingivitis, the inflammatory process spreads to the socket of all teeth of the upper and lower jaw. In the upper jaw Sox and the Sox of the ham front teeth of both jaws, especially in young people, a generalized symmetrical inflammatory process can occur against the background of General diseases.

Local (poor oral hygiene, mild dental caries and an excess of tartar, dental - jaw abnormalities and defects, fillings and prosthetics defects, occupational diseases and) and develops under the influence of general factors (diseases in different systems of the body).

Catarrhal gingivitis can be acute and chronic. Acute catarrhal gingivitis is observed in children with acute infection and other somatic diseases, during the period of tooth rupture and replacement. Timely

treatment measures allow you to recover without complications. Catarrhal gingivitis is similar to the chronic inflammatory process in adults. At the onset of gingivitis, pain is not observed, so patients do not see a doctor, and due to dental bleeding, they do not clean the teeth, begin to take soft eavesdroppers. This worsens the changes in the milk: the bleeding of the milk increases, the appearance of the milk and the color changes Ham. When examining a patient with acute catarrhal gingivitis, bright hyperemia of the mucous membrane of the gums is detected. The surface of the Milk is smooth, shiny, swollen and bleeds when probed.

Chronic catarrhal gingivitis is differentiated by its long duration. Patients sometimes see a doctor. Most often, chronic catarrhal gingivitis is detected during examinations or at the time when a dentist is consulted for the purpose of treating or shaving a tooth. The main complaints are bleeding gums during meals or when cleaning teeth, unpleasant taste in the mouth, smell, itching of gums. If treatment measures are not taken, the bleeding of the gums becomes permanent, in the morning it begins to be observed on its own. During lambs, there is pain in the gums, increased bleeding, and a taste of blood in the mouth. Subfebrile kharorat and holiness can be observed. In chronic catarrhal gingivitis, the gums are lighter in color, swollen, thickened Bolish, easily bleed when probed.

In acute gingivitis or chronic gingivitis lambs when the oral cavity is examined, there is light-colored hyperemia of the gums, smoothness of the surface of the gums, glare, swelling and bleeding in the palpation. In chronic catarrhal gingivitis, milk bleeding is observed when the mucous membrane is swollen, hyperemized, pronounced cyanosis, thickened, probed. The gum tumor looks like a periodontal pocket, but it is not, the integrity of the tooth-gum compound is preserved. On the teeth – a large amount of soft tooth decay, tartar is detected as a result of unsatisfactory hygiene of the oral cavity. In chronic gingivitis, patients do not have a change in their general acuity, no changes in peripheral blood flow are observed. There is no change in bone tissue on the X-ray, only when it is long there is an expansion of osteoporosis and periodontal fissure in the alveolar barrier. Inflammation can be local (around 1 or 2 tooth gums) or diffuse. The degree of severity of gingivitis depends on the general changes in the body and the spread of the pathological process to the gums. Mild gingivitis is characterized by

inflammation of the gum suckers, while moderate ones - the gum suckers and marginal milk, severe gingivitis - the alveolar milk, including alveolar milk, are also damaged.

In catarrhal gingivitis, the accumulation of glycogen in epithelial cells allows.

Schiller-Pisarev test positive. In mild gingivitis, the RMA index is 30%, in the middle level of gingivitis - 30 - 60%, in the severe level of gingivitis - more than 60%.

The reduction in the time of hematoma formation indicates a decrease in the stagnation of capillaries.



Differential diagnosis. Chronic catarrhal gingivitis should be distinguished from mild-Grade periodontitis. In contrast to periodontitis, catarrhal gingivitis retained the tooth-gum compound, and no change in bone tissue is observed on the X-ray.

Treatment. In acute catarrhal gingivitis, the use of hot antiseptics consists in the removal of dental under appliqué anesthetic, constant rational hygiene. Acute catarrhal gingivitis begins as an additive in acute respiratory diseases, gastrointestinal diseases, and quickly goes away with the treatment of the underlying disease.

Treatment of chronic catarrhal gingivitis includes:

- loss of etiological factor;
- restoration of the barrier function of the epithelium;
- exposure to pathological peoples of the inflammatory chain;
- normalization of the exchange process and microcirculation in milk.

To do this, it is necessary to pay attention to:

1. Loss of maxillary provoking factors: removal of bacterial and hard above-tooth residues, filling of carious pores, etc.
2. Train your teeth to clean properly and control them with staining solutions and pills.
3. Ingredients for the selection of dental hygiene products, the use of an electronic toothpick.
4. Professional oral hygiene.
5. During the period of study of oral hygiene, 0.06% chlorhexidine solution is used for oral baths in the morning and in the evening for 3 minutes after tooth cleaning in order to eliminate bacteria from dental.

Removing bacterial training the patient to the proper hygiene of the oral cavity leads to the loss of inflammation: the milk enters the pale pink color, hardens, does not bleed. However, despite the satisfactory hygiene of the oral cavity, sometimes the swelling, hyperemia, bleeding of the interdental gum tumors are preserved. In such cases, anti-inflammatory drugs should be placed in milk in the form of an applique or in the form of a healing Bond for 2-3 hours: 5% butadione 3% acetylsalicylic acid mazi, indomethacin maz, containing metronidazole and chlorhexidine glucanate preservative, Metrogil Denta in the form of gel. The appliques are placed after drying the milk with cotton swabs and separating it from the saliva using cotton rollers, the duration is 10-15 minutes. Depending on the degree of severity of the inflammatory process, the applique can be put for 7-20 days from 1-2 max per day.

For the treatment of chronic catarrhal gingivitis, decoctions from natural antiseptics such as chamomile flowers, calendula, schalfey leaves, zveroboy grass can be used. To reduce gum bleeding, appliques with intoxicating logaxilus leaves and flowers, nettle leaves, millipede grass, decoctions from the medicinal krovoxlebka root can be applied. The gums can be appliqued with a solution of high-bleeding vicasol, 5% aminacaprionic acid. Once the inflammatory process has subsided, restorative treatment treatments can be carried out.

In catarrhal gingivitis, physiotherapy (ultrasound light, aeronotherapy, calcium electrophoresis to the collar socket) aims to increase the overall reactivity of the body. It is advisable to carry out massage, HydroMassage, darsonval, medicinal electrophoresis (vitamin C, papaverine, 5% solution of aminocaprionic acid, 5% solution of calcium chloride) in order to increase the maxillary resistance of tissues,

normalize microcirculation and eliminate dimming in tissues. The course of treatment consists of 10-15 treatments, the duration of the treatment is 20 minutes. These drugs improve the metabolism, reduce the fibrinolytic activity of the saliva, promote the regeneration of epithelial cells by having a papaverine deagrigrative effect.

IV.2. ULCERATIVE-NECROTIC GINGIVITIS. CLINIC, COMPARATIVE DIAGNOSIS, TREATMENT, PREVENTION.

Ulcerative-necrotic gingivitis is caused by a decrease in the resistance of the gum tissue to autoinfection of the oral cavity as a result of changes in the overall reactivity of the organism. It develops in more young people in general diseases (influenza, angina), disorders of the balance of proteins and vitamins in food, after psychoemotional stress and when it is difficult for the wisdom tooth to crack. In the last case, not only in the gums, but also in the mucous membrane of the lunge, a ham wound-necrotic process is observed. Factors that weaken the overall resistance of the body include smoking, unhealthy habits such as alcohol products, Ham. Under these conditions, microorganisms multiply easily and increase in aggressiveness. Mainly dominated by anaerobic bacteria – spirochetes and fuzobacteria. The presence of Fusobacteria in addition to streptococci and staphylococci indicates a violation of the resistance of the tissue. Unsatisfactory hygiene of the oral cavity, broken and carious teeth, the presence of residues on the surface of the teeth leads to damage to the gums. Most often, ulcerative-necrotic gingivitis is generalized. According to the clinical course, acute and chronic types are distinguished. The degree of severity of gingivitis is determined depending on the spread of the inflammatory process and the latency of the body's intoxication.

The main complaints in acute ulcerative-necrotic gingivitis are: pain in the gums, especially during feeding, the arrival of the smell of humus from the mouth, excessive salivation, bleeding gums; sometimes ham pain in swallowing. When it is severe-headaches, an increase in body temperature, weakness, profuse sweating, a decrease in work activity can be observed.

When examined objectively, it is possible to see foci of necrosis and decay at the apex of intertribal gum surges against the background of hyperemia of the edges of the gums, and these tumors are covered with a gray fibrinous film. It is seen to be covered with large amounts of tartar and soft carash. Light, medium and heavy types differ depending on the degree of weight.

At the mild level, the general condition of the organism may not change. In the oral cavity, necrosis of the gum tumors and the gum Coast is detected. A clear border is visible between the damaged and healthy

gums. At the mid-severe level of ulcerative-necrotic gingivitis, partial alveolar gum is damaged along with gum tumors and marginal gum. At the same time, the general condition of the body also worsens, the body temperature rises to 37.5-38 C.

At severe levels, gum tumors marginal and alveolar gums are damaged. In this case, ulcerative-necrotic gingivitis is accompanied by intoxication, an increase in body temperature by 38-39 C degrees, an increase in the size of the maxillary lymph nodes, and sometimes painful. The patient is pale, the skin covers are earth-colored, headaches, loss of appetite, pain in the joints and muscles are observed. The mucous membrane of the gums is swollen, reddened, covered with gray fibrinous plaque, while bleeding wounds can be seen when raised. Many gum tumors have undergone necrosis, and this is more commonly seen in the molar, premolar, and retromolar tooth socket, and in low cases in the anterior tooth area. Soft and hard tooth sediments are often detected on the surface of the frontal teeth of the lower jaw facing the tongue. In the early stages of the disease, pain and bleeding in the gums are observed, and it is for this reason that patients do not clean their teeth with sometimes doctors recommend not to clean the incisors with and instead rinse the mouth more often with decoctions of gyro, hydrogen peroxide. But this is a wrong recommendation, because in this case, the hygiene of the oral cavity is even worse, under dental caries, favorable conditions for the growth of anaerobic microbes and the further spread of ulcerative-necrotic jarane appear. Decoctions from decoctions-slow is an antiseptic, does not have pronounced antimicrobial properties, while frequent rinsing of the mouth with them leads to a violation of the natural protective mechanism of the oral cavity and saliva.

Diagnosis of ulcerative-necrotic gingivitis is not a challenge. From laboratory examination methods, a bacterioscopic examination of the blood planter and the material obtained is carried out. In most patients, no change in blood flow is detected, sometimes there is an acceleration of EChT (up to 20 mm/s), an increase in the amount of leukocytes (up to the upper limit of the norm – 8-9ming), a shift of the leukocyte formula to the left. Cocci are visible in smear microscopy, but fuzobacteria and Spirochetes are present in large amounts of ham. Cytology, on the other hand, is characteristic of the nospecific inflammatory process: the drug contains a large amount of decomposed leukocytes, phagocytic neutrafiles and lymphocytes are practically absent. The X-ray shows an

expansion of the extremities of the periodontal fissure and a focal osteoporosis of the cortical plates. After 2-3 weeks after the onset of ulcerative gingivitis, changes in the bone begin. \

The necrotic process often begins in the retromolar soxa, less frequently-in the palate murmurs (Venson's angina).



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When examining the oral cavity, a large number of dental caries (hard and soft) usually attract attention. The skin of the face is more R colorless in color, the patient is injured with scattered Lange scales, the ends of the suckers seem to be cut off. The affected gums will not have an epithelial floor and will be covered with yellow fibrinous plaque.

When you want to take Karash, there is severe pain, blood flow increases. A demarcation line separates the milk necrotic sacs from the healthy milk.

Regions lymph nodes enlarged, painful. there is also headache, insomnia, loss of appetite. In the blood, blood-shaped elements change, ECHT increases, leukocytosis, leukocytosis there is a shift of the formula to the left.



Ulcerative gingivitis is subject to a comparative diagnosis of necrotic changes in blood diseases (cyclic neutropenia, agranulocytosis, panmyelopathy, antitelo deficiency syndrome), damage with heavy metal salts, vascular diseases, herpetic infection, VICH - infection.

Acute ulcerative-necrotic gingivitis can go into its chronic type if not treated in time. In this, patients do not have pronounced complaints, their general morality does not change. In objective examination, post-necrosis defects, edema, hyperemia, and decay are prominent in the interdental gum tumors. Necrotic defects in gums are not reversible.

When diagnosing ulcerative-necrotic gingivitis, it is necessary to be able to distinguish it from changes in the gums found in blood diseases (leukosis, agranulocytosis). There are similarities in the clinical course of these diseases: it usually occurs in more young people, changes in the gums develop after infectious diseases (influenza, angina), weakness, rapid fatigue, increased body temperature and necrosis of the gums are observed. The difference symptoms are blood clots in the mucous membrane of the oral cavity in blood diseases, specific infiltrates in the gums, spontaneous bleeding of the gums, immature elements of the blood in the blood taxil, leukocytosis, anemia. With timely treatment in ulcerative-necrotic gingivitis, recovery is observed after 5-14 days, but acute leukosis is difficult to heal.

In agranulocytosis, similar changes in Ham are observed, in which granulocytes are lost in the blood. Properly assembled for differential diagnosis, Anamnesis has great achamicity, the development of agranulocytosis is associated with the intake of drugs or with radiation. In agranulocytosis, in addition to gums, the soft palate Ham is damaged, necrosis foci appear on the unchanged surface. There is a very decrease in granulocytes in the blood, sometimes complete loss, anemia, thrombocytopenia.

It is necessary to distinguish between bismuth-containing and lead-containing gingivitis with Maxillary and general changes similar to ulcerative-necrotic gingivitis. In lead gingivitis, a gray line appears on the edge of the gums, aseptic necrosis develops in the gums as a result of lead sinking into the vascular wall, impaired blood circulation. Such patients are found to work with lead in their Anamnesis. Common signs may include paraliches, leaded sledges. In the milk of patients receiving bismuth drugs, a dark-gray line appears as a result of the deposition of

bismuth sulfide. In lead and bismuth intoxication, dark spots appear on various Sox of the mucous membrane of the oral cavity.

Ulcerative-necrotic gingivitis can develop Ham in AIDS, in which lymphadenopathy is characteristic of ham. Before the ulcerative-necrotic process, damage to the lymph nodes begins. Necrotic changes in aids are long-maintained, difficult to treat, accompanied by candidiasis and leukoplakia.

Wound damage to gums is considered one of the signs of radiation sickness. In case of damage with ionized radiation, hemorrhagic syndrome is observed in the oral cavity: bleeding of the gums, petechiae, foci of blood clots under the mucous membrane. Wounds and large foci of necrosis appear in the blood transfused Sox.

In patients with diseases of the cardiovascular system, ulcerative-necrotic changes occur in the gums and mucous membranes of the oral cavity as a result of a lack of blood circulation. Against the background of trophic disorders, wounds with Necrotic Carache appear, the surrounding mucous membrane – flowing, which smells of humus if applied from the mouth. In cardiovascular failure, ulcerative damage is slow, there is no inflammatory reaction, the mucous membrane of the palate, the edges of the gums, the lips and skin coverings are streaked and bruised.

Treatment. Complex, individual, etiopathogenetic, sequential, based, should be symptomatic. In the treatment of ulcerative-necrotic gingivitis, it is necessary to eliminate the foci of inflammation, limit the spread of the pathological process from the gums to other Sox of the mucous membrane of the oral cavity. To increase the overall reactivity of the body, to eliminate the symptoms of intoxication, muxim is prescribed.

Treatment plan:

- Bonomi selected personal hygiene items and trained in oral hygiene (toothbrush, toothpaste, flosses, etc.).
- Cleaning the surface of the gums from tartar, removing necrossed tissue.
- Washing the infected area with the help of local proteolytic enzymes, antiseptics, antimicrobial drugs and applying medicines.
- Stimulation of reparative processes in milk (use of keratoplastics).

- General treatment procedures-the recommendation of antimicrobial drugs, in severe cases, antibiotics, antihistamine drugs, drugs that restore immunity, vitamins.

- Prescribing physiotherapeutic procedures (laser therapy. UFN, UYUCH et al.

- Follow the diet and quality.

- When the inflammatory process decreases, it is recommended to sanitize the oral cavity (treat caries, relieve complications), orthopedic treatment if necessary.

Treatment of ulcerative necrotic gingivitis should be aimed at relieving local foci of inflammation, reducing pain, preventing the pathological condition from passing from the gums to other mucous membranes of the oral cavity, removing necrotic tissues, reducing intoxication and prescribing antimicrobial therapy, strengthening substance exchange and regenerative processes in the gums, preventing the recurrence of the disease, increasing local and general immunity.

Patients who have a severe common ulcerative-necrotic process will need an examination of a hematologist, therapist, immunologist, endocrinologist, dermatovenerologist. It is necessary to carry out clinical-laboratory tests to diagnose blood diseases, distinguish between cardiovascular and venereal diseases.

Treatment of ulcerative-necrotic gingivitis should be complex and should be carried out actively from the first day of application of the patient. Upon diagnosis after examinations, the patient receives oral irrigation from the first day of arrival and is treated with a soft cotton swab and turundas on the surface of the teeth, with a hot antiseptic solution to the interdental intervals (1% hydrogen peroxide solution, 0.02% furacilin solution, 0.02% ethylridine lactate, 0.06% chlorhexidine solution). Hot-state solutions accelerate the process of phagocytosis and increase leukocyte emigration. Creating unfavorable conditions for anaerobic infections in the treatment of ulcerative necrotic gingivitis is highly recommended. To do this, tooth sediments, necrotic tissues in the gums are removed under anesthetic. In a limited non-widespread inflammatory process, a type of applicative anesthesia (2% novocaine solution, novocaine with urotropine, 2% gel and solution of lidocaine, 10% spray of lidocaine, 2% solution of trimecaine, 5% mixture of anesthetic in glycerin) can be used.

In common ulcerative-necrotic gingivitis, it is necessary to use a type of injection anesthetic: infiltration or conductive anesthesia with a solution of lidocaine, trimecaine, novocaine. After anesthetic, soft caraches and tartar are removed on the floor, shaking the treated surfaces with a hot solution of antiseptics. Dental sediments should be carefully removed in a hole that prevents the infection from spreading to the deep tissues of the periodontium. The gum tissue that has undergone necrosis with an acute excavator is then removed. Proteolytic enzyme appliques are used to completely remove necrosis-affected Sox, which accelerate the release of necrotic masses by dissolving has an anti-inflammatory and stimulating effect.

Applications are used with the enzyme in the form of hot anesthetics: trypsin, chymotrypsin, lysozyme, deoxyribonuclease, ribonuclease, hyaluronidase – in the ratio of 10 ml anesthetics and 10 mg enzymes (one maxal per day for 10-15 minutes). For an antibacterial effect, especially anaerobic flora, various drugs are used in the form of solutions and ointments when applying to milk. For example, chlorhexidine solution together with metronidazole. The metronidazole tablet is dissolved in chlorhexidine and the resulting mixture is placed in milk for 15 minutes.

At home, the patient is advised to thoroughly process the oral cavity, use a soft-fiber dental floss and toothpaste with the drug which contains enzymes and freezing properties. Dental mechanical to remove in a way, teeth should be cleaned 2-3 times a day. After cleaning the tooth, it is recommended to carry out an oral bath with a 0.06% chlorhexidine solution for 2-3 minutes.

On the second day, when the patient arrives, the oral cavity is again treated with the help of a hot antiseptic solution, when necrosis foci appear again in the milk, proteolytic enzymes are removed from the application Hall under anesthetic. Then appliques made from drugs with antimicrobial effects are placed on milk.

If the treatment work is carried out correctly for 2-3 days, the pain in the gums decreases, body damage falls, general morbidity improves. Keratopastic preparations are used, which enhance reparative processes after the gums are completely cleared of necrosis: an oil solution of vitamin A and e, carotolin, chamomile oil, vinylin; aerosols ("Panthenol", "Ginozol", "Olazol"), greases (in the form of solcoseryl gel, 10% methylurotsil 5% actovegin). Oil solutions, flavors can be

applied in the form of a medicinal bond. Complete epithelization occurs by 4-7 days, and this will depend on the course and spread of gingivitis.

General types of treatment along with maxillary treatment are carried out in ham. The yield of treatment measures depends on the scale and severity of the inflammatory process. If the body temperature rises from 38 C degrees, it is necessary to use sostins of the anti-inflammatory drug nosteroid. This group of drugs has a strong slimming effect on the biosynthesis of prostaglandin, a mediator of inflammation, pain and fever. In severe cases, glucocorticoids are recommended.

Antihistamine drugs are recommended for desensitization purposes. They weaken vascular permeability, edema and hyperemia. Vitamins C (Ascorbic acid) and R are recommended for all patients. They strengthen the vascular wall, weaken exudation and promote tissue regeneration.

In cases of severe ulcerative-necrotic gingivitis, antibiotic therapy against pathogenic microflora is recommended. When choosing an antibiotic, it is worth paying attention to the fact that it is quickly absorbed and enters the milk intake, forming a high concentration in the milk fluid. Such a feature is possessed by the tetracycline group, especially monocycline hydrochloride. It is effective against Gram-positive and Gram-negative strains, spirchetes, rickets, Mycoplasma, neutralizing the action of microbial strains and collagenase, which becomes characteristic in the inflammatory process. It turns out that the concentration of this drug in milk fluid is 5-7 times higher than in serum. Monocycline Hydrochloride is cabulated from 50 mg for 2 maxal, 7 days a day. Of the antibiotics in the tetracycline series, rondomycin Ham is effective and does not have the same opposite effect as photosensitization, it is recommended to take from 0.15-0.3 g 2-3 maxal 7 days a day.

Antibiotics belonging to the macrolide group have a high antibacterial activity against anaerobic microorganisms. The antibiotics used at all times have an antibacterial effect on the Gram-negative and Gram-negative microflora, disrupting protein synthesis in the shell of the microbial cell: ampicillin, ampiox, Oxacillin sodium, lincomycin hydrochloride, it is recommended to take for 4-6 days.

Among antibacterial drugs, metronidazole has its own acamity. It has a high activity against anaerobic bacteria and Spirochetes and, when

ingested, is proportional to all antibiotics, Ham is released into the milk fluid, 0.25 g 3 maxal per day is taken for 4-7 days.

In order to increase the maxillary immunity of the oral cavity, an immunomodulatory drug imudon is recommended. It increases the phagocytic activity of macrophages, increases the amount of lysozyme, secretor a immunoglobulin in the saliva, thus having a healing and prophylactic effect. It is kept in the mouth until it is completely dissolved, it is possible to take 6-8 tablets a day for 10 days. It can be taken with antibiotics.

In ulcerative-necrotic gingivitis, laser therapy, UFN, aeration from physiotherapeutic procedures are recommended.

Parchez should be rich in versatile protein and vitamins, not affecting the mucous membrane of the mouth. It is recommended to drink more liquid. Hot dishes, cashews, alcoholic beverages and smoking are prohibited.

Acute inflammatory process caries and its complications are treated, proper oral hygiene is taught. When choosing fillers, especially in milk – touching Soha, Composite agents – microphilic and hybrid-are recommended, compared to Amalgam and sement, agents with low microbial adgesia and column dressing properties.

IV.3. HYPERTROPHIC GINGIVITIS. CLINIC, COMPARATIVE DIAGNOSIS, TREATMENT, PREVENTION.

Chronic inflammation in the gums is a process that manifests itself with more proliferation. Hypertrophic gingivitis is rare in adults (3-5%), more common in children of pubertal age (12-15 years) and adolescents (18-20 years).

Hormonal disruptions in the development of hypertrophic gingivitis have an acute aetiology, due to which the types of gingivitis are distinguished in the alloxide – "adolescent gingivitis" and hypertrophic gingivitis in the fetus. The development of gingivitis in adolescence and in the fetus due to maxillary factors can lead to endocrine disorders. To the maxillary factors: unsatisfactory hygiene of the oral cavity, pathology of the prikus, improperly made orthopedic and orthodontic structures, dental TSH, hanging of the edge of the fillings.

Complaint. To the aesthetic defect of milk, swelling in milk, bleeding, pain in taking food

Anamnezi. Young people in the prepubertate and pubertrate period, when taking pregnant, citostatic, contraceptive, anti-seizure drugs, in deep bite and dense arrangement of teeth

Course of the disease. Hypertrophy of the gums occurs against the background of chronic catarrhal gingivitis.

Appearance. The appearance of the face is unchanged, the lymph nodes are not enlarged, the mouth opening is free

The appearance of the oral cavity. In the swollen form of hypertrophic gingivitis, the gums are reddened, swollen, false gum pockets, bleeding are detected. In the form of fibrosis, the gums are light pink, the gums are solid, the tooth surface can cover from $\frac{1}{3}$ to $\frac{3}{4}$ qism.

X-ray. Alveolar tumor resorption of bone tissue and interdental suchc tissue is not observed.

Hypertrophic gingivitis usually develops in the socket of the frontal teeth of the upper and lower jaw. It occurs in more pregnant people (46-49%) due to increased estrogen levels in 4-5 months. This type of gingivitis is manifested by an increase in the size of the gum suckers in a barrel shape, bleeding easily. The reason for this is the unsatisfactory hygiene of the oral cavity and the presence of inflammation until pregnancy.



Hypertrophic gingivitis often occurs under the influence of local factors. These include improper prosthetics, filling defects, prikus pathology, uncannulated oral cavity, etc. Of the common factors, hormonal changes are of great importance (juvenile gingivitis, fetal gingivitis, climacteric gingivitis), as well as prolonged use of medicamentous drugs, vitamin deficiency (especially vitamin C), blood diseases, diseases of other organs and systems are distinguished from etiological factors. Most authors classify hypertrophic gingivitis into 2 forms:

- šišli
- * Fibrosis.

Contact hyperplasia of the gums (due to prosthetics) is common and is mainly diagnosed in women. Causing such changes, hormonal changes that occur during menopause are indicated. The clinical picture is manifested by edema and hyperemia of the gum. Hypertrophic gingivitis also develops in fibroblasts when the drug diphenin is taken, which enhances collagen synthesis. Hyperplasia does not disappear until the reception of the drug is completely stopped, manifested by the growth of the gums from the vestibular and oral side in the socket of the frontal teeth. Inflammatory symptoms are more pronounced when the enlarged gums are held hard, swelling and hyperemia are sluggish, and the hygienic condition is unsatisfactory.

Hypertrophic gingivitis is typical for myelomonoblast, monoblast types of acute myeloid leukosis. Initially manifested by a painful swelling of the gums. Gum suckers grow and quickly clog the dental Corona. Hemorrhagic syndrome Ham develops along with hyperplasia

of the gums. Blood clots are prominent in langes, tonsils, mucous membranes of the palate and gums.

Two types of hypertrophic gingivitis are distinguished: vitreous and fibrosis. The vitreous type is manifested by the enlargement of the gum suckers, a bluish tinge, a glossy surface, bleeding when touched, and the formation of false tooth-gum pockets. Epithelial attachment is intact. The honeycomb remains when the suckers are pressed using a tool. Patients are disturbed by pain during feeding, teeth cleaning, bleeding gums. In the fibrosis type of hypertrophic gingivitis, the gums are overgrown, hard in palpation, the color of the mucous membranes has not changed, the surface is uneven. False gums-there will be Dental pockets, milkosti tartar, bleeding gums are not observed. In the fibrosis type, patients are disturbed only by the unusual appearance of gums.

Three levels of hypertrophic gingivitis differ depending on how advanced milk hypertrophy is:

Hypertrophy of Light-Gum suckers closes $1/3$ of the height of the dental Corona;

Middle level- $1/2$ of the height of the tooth Corolla;

Heavy grade-closes more than $1/2$ of the height of the dental Corona.

It is found that hypertrophic gingivitis develops after catarrhal gingivitis. Under the influence of maxillary and general factors, the process in the gums becomes chronic, at the morphological pace of inflammation, the process of proliferation begins to prevail.

Fetal gingivitis-the tumor form develops from catarrhal gingivitis. During pregnancy, the amount of hormones in milk fluid - estrogen and progesterone-increases. Under the influence of these, the gum tissue becomes more sensitive to the microorganism.

Estrogen and progesterone increase vascular permeability, resulting in edema, which stimulates the synthesis of prostaglandin and other inflammatory mediators.

Adolescent gingivitis is caused by the action of harmonic changes in The Shape of the tumor. Unsatisfactory oral hygiene also occurs when breathing through the mouth.

Clemacteric gingivitis-depends on the increase or decrease in the amount of estrogen in the body. This leads to a decrease in the degeneration of cells and the loss of protective function of the mucous membrane of the gums.

Hypertrophic gingivitis can also develop in special diseases (leukemic reticulosis, myeloleicosis).



Thus, hypertrophic gingivitis is manifested by various degrees of lameness of the chronic inflammatory process, vascular reaction, swelling of the stroma in the subepithelial sacs of the gums and mucoid edema in the deep layers.

If hypertrophic gingivitis persists for a long time, the tooth-gum joint begins to disintegrate, the gum pocket is formed, the destruction of bone tissue of the alveolar tumor begins. Gingivitis passes into parodontitis, but hypertrophy of the gums remains. Significant fragmentation of the gums can lead to changes in the position of the teeth.

Differential diagnosis.

Hypertrophic gingivitis should be distinguished from the following diseases:

- milk fibromatosis,
- milk hyperplasia in leukosis,
- milk hypertrophy in periodontitis,
- epulide.

Gum fibromatosis is a tumor-like derivative, manifested by diffuse growth of the gum and gum suckers. Rarely observed, it occurs mainly in young people and middle-aged people. The gums from the clinical picture are manifested by the fact that they increase in size and partially or completely close the dental Corona. Mainly the vestibular part of the gums is damaged, but changes can be observed in the oral part. The surface of the tumor is often smooth, the color of the mucous membrane is light pink. In cases where fibromatosis is prolonged on an X-ray,

changes are observed in the interdental barriers and the edge of the alveolar tumor. From a microscopic Gill, gum fibromatosis is a tumor consisting of connective tissue with low blood vessels, solid fibers.

In leukosis, milk hyperplasia occurs in 5% of cases, in addition to the enlargement of the tonsils, infiltrates appear. The hypertrophic process occurs suddenly and is accompanied by a rapid increase in the volume of the gums. Symptoms of the main disease, accompanied by an increase in the size of the gums, Ham is observed: increased body temperature, weakness, enlarged lymph nodes. A blood count confirms the diagnosis.

In periodontitis, gum hypertrophy is often circumscribed and is the effect of a traumatic factor (tooth or coronal edge, absence of a touching surface). In periodontitis, there is a periodontal pocket, there is a twitching of the teeth, on an X – ray-destructive changes in the bone tissue of the interdental barriers are observed.

When acute gingivitis develops, a differential diagnosis with epulide is carried out. Epulide is also a tumor derivative and is mainly observed in women during pregnancy, as well as in adolescents. More alveolar settles in the front of the tumor. The derivative is bluish-reddish with a broad base. In the regions where the epulide is located, osteoporosis is observed in the bone tissue of the alveolar tumor.

Treatment. General treatment measures are carried out with an endocrinologist, hematologist, psychiatrist and other specialists, depending on the underlying disease, and mainly vitamins are recommended: ascorutin, vitamin E, as well as sedative preparations, depending on the indication.

Maxillary treatment measures:

1. Removal of dental sediments by cleaning (abrasions, under-gum and over-gum tartar), loss of maxillary influencing factors (non-rationally prepared fillings, non-removable dentures, orthodontic devices, etc.).

2. To teach oral hygiene and to control the way it is done.

3. The use of anti-inflammatory, anti-edema and sclerosing drugs.

When choosing drugs for maxillary treatment, the type of hypertrophic gingivitis should be taken into account. For mouthwash and oral baths, a 0.06% solution of chlorhexidine, a 0.02% solution of furacilin, a 1% solution of dioxidine, etc. are used in the turban type. To normalize anti-inflammatory and vascular-tissue permeability, 5%

butadione mazi, 3% acetylsalicylic Acid, 10% indomethacin mazi, 1% heparin mazi are used in the application form. Glucocorticoid applique is used when the tumor is enlarged.

It is also advisable to rinse the mouth after 2 maxal teeth are cleaned per day with tincture of zupturnum herb, kalankhoye juice, oak bark decoctions, applique from oil-alcohol decoction of vagotil, galaskorbin, propolis and chistotel tincture on milk for 10-15 minutes.

Sclerosing therapy is aimed at reducing or losing milk hypertrophy. It is used in the fibrosis type of hypertrophic gingivitis and also in the tumor type after taking anti-inflammatory treatment measures. For this purpose, a 40-60% solution of glucose is injected into the hypertrophied gum sucker using a thin injection needle from the top of the sucker towards the base. After 0.1-0.3 ml of the solution is injected, ischemia is observed in the gum sucker and persists even after the needle is removed. The course of treatment consists of an injection that is done 6-8 times per suction, the interval between injections is 1-3 days.

As a sclerosing agent, a 0.25% solution of calcium chloride, a 10% solution of calcium gluconate, 700 ethyl alcohol (3-4 times), lidase solution (64 yed.) along with trimecaine (4-5 injections), a 25% solution of magnesium sulfate is applied. All drug vests are administered with anesthetics in a 1:1 ratio. 6-7 teeth are injected into the socket at a time. As a keratolytic agent, 25% dimexide solution, 25% chlorine zinc, vagotil are used, which are placed in the Sox of hypertrophied suckers in turundas separated from the milk by a cotton swab.

Physiotherapeutic methods are widely used in the treatment of hypertrophic gingivitis: in combination with an applique in the bleeding and swelling of the gums, electrophoresis is carried out with a 10% solution of calcium chloride, a 5% solution of potassium iodide, lidase, heparin. The duration of the treatment is 15-20 minutes, every day or day ora, the course of treatment is 12-15 treatments.

In order to reduce the inflammatory process in the gums, diadinamotherapy is recommended in the swollen type of hypertrophic gingivitis. As a result of the physicochemical changes that occur under the influence of diadinamic currents, the permeability of the cell membrane changes, blood and lymph circulation, tissue trophic improvement, inflammation and edema decrease.

In the fibrosis type of hypertrophic gingivitis, diathermocoagulation of hypertrophied gum suckers is recommended.

The treatment is carried out under appliqué or injectable anesthesia. As an electrode, a sterilized root needle is used, which is inserted into the gum sucker to a depth of 3-5 mm. Coagulation is carried out with a voltage of 6-7 W for 2-3 seconds. Up to 3-4 dots are coagulated on each suction cup. In one session, 4-5 milk sucker coagulations are performed.



Gum hyperplasia (fibrosis thickening of diffuse or focussed gum tissue) is manifested in the appearance that the gum suckers have grown. The color of the gums will be normal or bruising. Milk suckers are dense, shiny, sometimes rough - this is the surface, does not bleed when probed. At the beginning of the procedure, there will be no clinical manifestations of inflammation in the gum tissue. However, it can occur later with the formation of false pockets.



Severe levels of chronic generalised periodontitis include pain in the gums, difficulty chewing, bleeding of the gums, flutter of the frontal teeth of the upper and lower jaw, and Veersymon shift. Patients refuse

tooth cleaning as a result of pain and bleeding gums, which further worsens oral hygiene and leads to increased inflammation.

At the examination-signs of inflammation in the parodont tissue: the mucous membrane of the gums is hyperemized, swollen, tooth necks, even the root is bare, individual teeth are found to have a degree II-III tremor. The depth of the periodontal pockets reaches 5-8 mm. When the Milk is pressed, the pus is released. On the teeth – many tooth sediments, under the gums and above the gums, tartar is visible. On X – ray-Level III of bone tissue destruction, resorption occupies 2/3 of the length of the tooth root, sometimes the bone tissue of the alveoli is completely resorbed.

In the process of intermediate to severe grade periodontitis, abscesses may form. The lameness of chronic periodontitis will be associated with a decrease in the resistance of the body and functional tension of the teeth in the aloxia after the transferred diseases (against the background of URVI, pneumonia, cardiovascular failure). Chronic periodontitis lambs develop severe pulsating pain, exacerbated during chewing. Body temperature rises to 37.5-38.0 C, weakness, headaches are observed. In the objective examination: the mucous membrane of the gums is dark red, bleeding, pus is released from the edge of the gums in the palpation. In the socket of one or more teeth, an abscess is detected, in the blood – leukocytosis, EChT acceleration, lymph nodes in the same socket are enlarged and painful.

The reason for the appearance of abscess is preserved round plaque, deeply located tooth sediments and Road Y'qliq for pus to flow. The abscess can be located in the root tip socket, in the middle, or in the longitudinal Aldi socket. Sometimes the abscess spontaneously ruptures to form a leaky path near the edge of the gum. In the soxa, where the abscess is located, inflammation in the gums is more pronounced, deep periodontal pockets, pus leakage and tooth decay are detected. On an X-ray, bone resorption is visible, chronic periodontitis manifests itself 15-20 days after lamination. Resorption is manifested in the alveolar tumor bone in the form of uneven destruction of the hemispherical edges. The serous-purulent exudate-filled periodontal pockets are a foci of highly pathogenic infection and are considered the body's foci of staphylococcal and streptococcal sensitization. Proof of this is a positive test for anitistreptolysin-0 high titer and streptococcal allergen. During the period of remission, there are no complaints, as a result of surgical

procedure, the tooth neck is opened and hyperesthesia of the dental tissue occurs. The mucous membrane of the gums is light pink, tightly adhered to the surface of the tooth, there are no periodont pockets. Oral hygiene is satisfactory in axvol, the RMA index=0 is equal, the periodontal index Ham decreases due to inflammation and the absence of periodontal pockets.

Differential diagnosis. Acute local periodontitis should be distinguished from the period of lameness of acute peak periodontitis and chronic peak periodontitis. Acute or chronic peak periodontitis is characterized by exacerbating, persistent simulating, or pulsating pain especially when the teeth are in contact with each other during lamination. The feeling of "lengthening" of the tooth is clearly manifested. The permeable twisted mucous membrane in the sore tooth socket is swollen, reddened, painful in palpation. A carious hole is detected in the tooth, tooth decay, pain is observed in percussion. On X-ray, it can be seen that in acute peak periodontitis – the tissues next to the root tip are unchanged, in the lamination period of chronic peak periodontitis-bone tissue in the root tip socket is eroded.

In acute periodontitis, the pain gradually increases, increasing when chewing. Signs of inflammation, such as swelling and redness, settle on the banks of the gums. The probe detects a gum pocket. The fact that the periodont tissue is constantly traumatized by an artificial corona, a suspended filler, a prosthetic clammer with a partially solvable plate serves for the origin of chronic local inflammation. The started inflammatory process will go deeper and lead to resorption of the tooth lung. Chronic local periodontitis is characterized by the penetration of food debris between the teeth, a feeling of friction, bleeding gums when the teeth are cleaned and during feeding. After the remnants of food are removed between the teeth, the feeling of friction disappears. At the examination, milk edema and redness are detected, at the palpation, milk bleeding, gum pockets, slight tremors of one or two teeth, at the teeth sediments. The X-ray shows inter-dental barrier resorption, destruction of bone tissue, bone pockets as the process of destruction intensifies. Complete or asymmetric reorption of the tooth lung is sometimes observed.

Chronic local periodontitis should be distinguished from chronic papillitis and chronic fibrosis pulpitis. Chronic papillitis has symptoms similar to local periodontitis: ingestion of food debris between the teeth.

a feeling of friction, pain when eating. Local periodontitis is characterized by the twitching of one or two teeth as well as the occasional lambing of the Jaran. In papillitis, moderate swelling, redness or bruising (cyanosis) of the interdental gum sucker is observed. The gum sucker is infiltrated, the epithelial lining is thinned, erosion and erosion sometimes occur. The tooth does not move, an unpleasant sensation appears on percussion, pain appears from cold influences. In chronic papillitis, there are no signs of osteoporosis, resorption of bone tissue on an X – ray-at the tip of the inter-dental barriers. In local periodontitis, the gums are reddened, swollen in one or two tooth sockets, these changes can spread to the alveolar tumor mucosa. The probe detects gum and bone pockets, gum UST Iva tooth sediments. In horizontal percussion, pain is observed, the movement of one or two teeth that appear from time to time, sometimes their displacement. X – ray-an alveolar tumor in one or two tooth sacs shows a spreading destruction along the root.

For chronic fibrosis pulpitis, it is characteristic to have pain when eating and when the temperature changes. Spontaneous pain characteristic of acute pulpitis is observed from Anamnesis. The mucous membrane of the gums in the affected tooth socket is unchanged, a carious hole is detected in the tooth, the cavity of which is filled with a large amount of softened dentin, the root of the tooth may be opened, the visible pulp is red. In X-ray chronic fibrosis pulpitis, no change in bone tissue is observed, Sometimes thinning may appear around the root tip.



Mild chronic periodontitis should be distinguished from chronic catarrhal gingivitis. Unlike gingivitis, periodontitis has a broken tooth-

gum Joint, there is a gum pocket, changes in bone tissue are observed on X-ray.

The active course of chronic periodontitis should be distinguished from periodontosis. Chronic periodontitis is characterized by inflammation of the gums, periodontal pockets, inflammation of the interdental barrier, as well as by the destruction of the gums.

During remission, many signs characteristic of periodontitis can be observed: no inflammation, no periodontal pockets, tight adhesion of the gums to the tooth, opening of the tooth root, and hyperesthesia. In such cases, differential diagnosis is made depending on the Anamnesis and the changes that develop in the bone tissue on the X-ray. In parodontosis, the gums are whitish, strongly adhered to the tooth surface, do not bleed, there are no periodontal pockets, the gum recession manifests differently along the tooth row. On an X – ray-a decrease in the length of the Intertribal obstruction without foci of osteoporosis is observed in the Horizontal Type.

In periodontitis during remission (as a result of X-ray imaging before treatment and 1-2 years after treatment), there is no resorption of osteoparose foci and Inter-alveolar obstruction, there is a hardening of bone tissue.

Chronic periodontitis in lameness should be distinguished from periostitis in the case of abscess, in which a connection with the eroded tooth is determined.

Treatment the treatment of periodontitis should be complex, individual, etiopathogenetic, sequential, based and symptomatic.

Treatment of acute local periodontitis should be aimed at eliminating the factors caused, inflammation and gum pockets. The factors that caused acute local inflammation of the periodontium should be carried out under the applique anesthetic, removing hanging fillings, restoring the touch point, removing traumatized artificial Coronas, uncomfortable orthopedic devices, cleaning dental chimneys (abrasions and tartar). To relieve inflammation when the etiological factor is eliminated, antiseptic and antibacterial drugs can be used in the form of mouth rinses, mouth baths, appliques. From anti-inflammatory drugs, nosteroid anti-inflammatory drugs can be used in appliques or healing ligaments. In chronic local periodontitis (when there is a bone pocket), surgical treatment is recommended.

The origin of the inflammatory process in the periodontium is caused by pathogenic microorganisms in the gums, which manifest themselves as the reactivity of the body changes. Therefore, in the treatment of chronic periodontitis, it is necessary to choose treatments that affect the microflora in dental and the general body.

Etiotropic (antibacterial) therapy, focused on the loss of causative factors;

Pathogenetic therapy, along with symptomatic agents affecting various pathogenetic stages of the inflammatory process in the periodontium as well as reducing tooth decay, depth of the periodontal pockets, slowing bone tissue resorption;

Means of strengthening the protective system of the patient's body;
Rehabilitation treatment.

It is necessary to use complex treatment methods with the attachment of general and maxillary methods of treatment. To increase the effectiveness of treatment, individual planning of complex treatment should be carried out, taking into account the type, severity and general condition of the disease. Also, the correct sequence of treatments.

Etiotropic treatments for periodontium diseases include antibacterial drugs. The indication for their use is: pus leakage from the periodontal pockets, abscesses, leaks, intoxication.

Conservative and surgeon treatments differ. Conservative treatments include professional oral hygiene, Maxillary and general antibacterial therapy, anti-inflammatory treatments.

It is necessary to create favorable conditions for the chewing function of the affected periodontium for which it is necessary to perform oral counting: treatment of caries and its complications, replacement of poor-quality fillings, restoration of the touching point. When treating the carious hole of teeth touching the gum, it is advisable to use microphilic and hybrid fillers, the property of which is poor in the adhesion of microbes than fillings made of cement. Especially in the middle and heavy stages of periodontitis has a priority. At the mild level of periodontitis and gingivitis, the use of various fillers is better than resin, but more composite ones.

Professional oral hygiene is the main stage of treatment and is performed before surgical, orthodontic and orthopedic treatments.

This includes the removal of hard tooth sediments, tooth decay, cleaning and smoothing of the tooth root, antibacterial processing of

pockets. To remove sediments, special tools or an ultrasound machine are used, the special pump of which allows you to align the socket of furcation defects and align the root of the tooth.

Before and during the extraction of sediments on the tooth surface, the mucous membrane of the oral cavity is treated with a warm solution of antiseptics. Appliqué or instillation anesthetic methods can be used to painlessly pick up sediments on the tooth surface.

In the treatment of periodontitis, the stage is the training of oral hygiene and the control of its quality. Further stages of treatment can only be passed if the patient fully adheres to oral hygiene.

Upon removal of caroches on the tooth surface, a biopolymer film "Diplen Denta" should be applied to the gum edge, of which there are different types, containing "X" - chlorhexidine, "M" - metronidazole, "L" - lincomycin, "LX" - chlorhexidine and lidocaine, and a solcoseryl dental adhesive paste, the "Gingitek" gum bandage can be applied. At this stage, it is necessary to determine the supracontact sites using black paper and, if there is an indication, scrape them with diamond bores and treat the teeth with fluoride or remineralizing solution.

The next stage of treatment is aimed at reducing the activity of microorganisms in dental caraches, which are mainly anaerobic and are detected when the periodontal pocket is 4mm and deeper (moderate to severe levels of chronic periodontitis). At mild levels of chronic periodontitis, periodontal pocket handling can be considered a method of treatment, while at medium and severe levels it is a preparatory stage for surgical removal of the pocket.

It is now better to use antiseptics for the treatment of medicamentosis in the periodontal pocket, since in comparison with them, tolerance in bacteria develops more slowly than in antibiotics, and allergic effects are also observed less often.

One of the methods of treatment of periodontium medicamentosis is the maximum use of antibacterial drugs, which has several advantages:

- formation of the maximum concentration of the drug in the foci of inflammation;
- reducing the effect of the oral cavity on the microflora;
- attenuation of the effect of the drug on the body.

The main requirements for drugs used for maxillary treatment:

1. The periodontal pocket is maintained for a long time.

2. Anti-fungal antibacterial effect on pathogenic infections in the pocket.

3. Good permeability of the drug

Dimexide (dimethylsulfoxide) is an organic substance with antiseptic and nonspecific anti-inflammatory properties. It is lightly passed, reduces inflammatory edema, has an anesthetic effect, is easily absorbed into tissues and easily passes through the cell membrane. Dimexide is used for mouthwash (0.25% solution), periodontal pocket washing (1% solution), pocket injection with TURUNDA (2% solution), and drug dissolution.

In the treatment of periodontium diseases, chlorhexidine Ham, which is produced in the form of a solution for rinsing the mouth and gel, is effective. Another antiseptic, miramistin, is a cation detergent, which increases the permeability of the bacteria cell wall and leads to cytolysis. Gram-positive and Gram-negative, aerobic and anaerobic, spore-forming and has a strong effect against candidiasis, viruses, spirochetes, reduces the resistance of bacteria to antibiotics, increases the functional activity of immune cells, enhances the maxillary immune response and does not call an allergic reaction.

Metronidazole muxim is important in the antibacterial treatment of periodontitis. It has effects against many anaerobic bacteria, spirochetes, and can be used with antibiotics. The drug is peroscable, but its concentration in milk fluid will be 2 times less than in blood serum. When injected into deep periodontal pockets (7 mm), its impact strength is greater.

It is not enough to apply only hygienic measures (removal of milkusti and milkosti tartar) due to the variety of pathogenic microorganisms in the gum tissue, since some microorganisms have the property of invasion into the parodont tissue, and since others penetrate into the cell, it will not be possible to eliminate them mechanically.

Systemic-acting antibiotics are also recommended along with maxillary antiseptics to eliminate pathogenic microorganisms. There are strict indications for their use: pus leakage from the periodontal pockets, abscess, leaks, destruction of alveolar tumor bone tissue, intoxication, pre-and post-treatment of surgeon.

Antibiotics of the tetracycline group have a high effect and good results have been obtained, from which monocycline hydrochloride, tetracycline, rondomycin are widely used.

One antibiotic used to treat the complicated types of periodontitis is lincomycin, which has the property of acting on microorganisms that are stagnant in Gram-positive microorganisms, some anaerobes and other antibiotics. Its mechanism of action is due to the suppression of protein synthesis in the bacterial cell. Lincomycin increases the body's nonspecific reactivity and has the property of folding in bone tissue. Clindamycin, which is similar in mechanism of action, chemical structure and antimicrobial action to lincomycin, also has an effective therapeutic effect.

Macrolides are now also used effectively in periodontium diseases. They are considered a specific infection of the periodontium which also affects strains of bacteria. Drugs such as erythromycin, azithromycin, roxithromycin, belonging to the macrolide group, have been widely used.

After antibacterial therapy, Maxillary and general anti-inflammatory treatment is carried out on an instructional basis. Anti-inflammatory agents act on various stages of inflammation, eliminating symptomatic gingivitis, periodontal pockets.

Pathogenetic treatment.

Affects the following:

- ingestion of lysosomal enzymes (hydrolase, protease);
- lowering the activity of inflammatory mediators – histamine, quinine, prostaglandin;
- reduce exudation, edema, normalize vascular tone, reduce the permeability of the vascular wall;
- microcirculatory effect on flow, improving the metabolism in periodontium.
- increase Maxal immunity.

In pathogenetic treatment, medicines, orthopedic remedies, surgeon and physiotherapeutic methods are used.

Enzyme preparations have a good effect on treatment: trypsin, chymotrypsin, lysozyme, lysoamidase, etc. They dissolve tissues that have undergone necrosis without damaging healthy tissues, accelerate the regeneration process, have an anti-inflammatory effect. An indication for use is the presence of a large amount of purulent exudate in the periodontal pocket at severe levels of periodontitis.

Heparin also has a good effect on the exudation stage of inflammation. It normalizes the exchange of substance and gas in

tissues, reduces vascular permeability, reduces the activity of hyaluronidase, slightly dilates blood vessels, reduces blood viscosity, adgesia and aggregation, accelerates blood flow, normalizes microcirculation, has an effect on allergies and promotes tissue regeneration. Because of these properties, it has anti-inflammatory and anti-edema effects. In the form of the periodontal pocket is placed under the link, while insertion using electrophoresis gives an even better effect. Cases of impaired blood clotting are an indication against the use of heparin.

In the treatment of the inflammatory process, steroid drugs – glucocorticoids-are widely used. They have an effect on all phases of inflammation: alteration, exudation, proliferation, desensitizing, anti-allergy. Glucocorticoids have a direct effect on the foci of inflammation and are completely stored in the same place in the maxillary application. They are not just signs of an early stage of inflammation (enlargement of the capillaries, edema, fibrin deposition, etc.), slowing down the regeneration process and osteogenesis in the foci of inflammation - relatively late signs, so there are restrictions on application. It is advisable to use glucocorticoids in the acute stage of inflammation, it is not recommended to use when the process is slow, pus is flowing, abscesses are formed.

When using glucocorticoids, their rapid absorption from the mucous membranes can affect the metabolism and cause harmful effects: impaired renal gland function, steroid diabetes, edema, increased arterial blood pressure, osteoporosis. For this reason, other anti-inflammatory agents have been found for use in periodontology.

Nonsteroidal anti-inflammatory agents that have been isolated from a group of non-narcotic analgesics have analgesic, desiccant, and anti-inflammatory effects. These drugs are widely used for their low harmful effects, even if they do not have an active action, such as glucocorticoids, affecting two phases of inflammation: exudation and proliferation. By slowing down the synthesis of prostaglandins and inflammatory mediators (histamine, serotonin, quinine, norepinephrine), it affects the exudation phase of inflammation, in which redness, swelling and pain are reduced. Nosteroid anti-inflammatory agents reduce hyaluronidase activity, normalize microcirculation, prevent the formation of microthrombs. Fibroblasts affect the proliferation phase by decreasing activity and collagen synthesis.

Nosteroid anti-inflammatory drugs are used for Maxillary and general treatment procedures in the form of gel (3% acetylsalicylic 5% butadione emulsions), tablets and solutions. It is not recommended to take nosteroid anti-inflammatory drugs enteral at a mild level of parodontitis, at an intermediate level it is possible to take it during the preparation period for surgery, and at a severe level it is advisable to take enteral. In cases of pus, abscess, it should be used together with antibacterial drugs.

Vitamin drugs are used in different ways. They strengthen the vascular wall, reduce exudation, enhance the regeneration process. The most commonly used vitamins are vitamins A, C, P, E and B rice.

Drugs used for maxillary treatment, which are sent to the periodontal pocket, are quickly washed out and the healing effect is reduced. When the periodontal pocket is cureted its surface can scratch when food is ingested and the risk of infection is high. To prevent such situations from happening, a periodontal ligament is used, these ligaments can be curative and insulating.

Healing ligaments are used in the milk, in the periodontal pocket, in the conservative and surgical treatment of periodontitis for the accumulation of drugs in the interdental intervals.

Drugs with different mechanisms of action are introduced into the healing bonds: vitamin A, C, P, E and B group, enzymes, corticosteroids, sulfonamides, antibiotics, etc., due to which they have an antibacterial, anti-inflammatory, desensitizing, effect and stimulate the regeneration process in tissues. The main function of healing ligaments is to prolong the effect of drugs, the ligaments are placed for 2-3 hours.

Insulating periodontal pockets protect the periodontium tissue from various influences: oral fluid, trauma in the process of taking food. Insulating periodontal ligaments should be hardening, but elastic, keep in the oral cavity for a long time (more than a day) and not press on the tissue.

General treatment in periodontitis should be aimed at normalizing the reactivity of the body, eliminating inflammation, metabolic and microcirculatory changes, and increasing the maxillary resistance of periodontic tissue.

To normalize the reactivity of the body, it is necessary to treat general somatic diseases, correct the state of the immune system, observe a balanced, a mode of Labor and rest. Immunomodulators are

used to correct the immune system, especially at intermediate and severe levels of generalised periodontitis.

Periodontium diseases can often develop against an allergic background or against the background of changes in the resistance of the body. For this reason, nonspecific or specific desensitizing drugs are recommended. In the form of a tablet of calcium gluconate to nonspecific desensitizing drugs, 10% calcium chloride solution, 10% sodium thiosulfate solution. In addition to the desensitizing effect, calcium preparations reduce the permeability of the vascular wall and are involved in the formation of bone tissue.

Antihistamine drugs: suprastin, pipolfen, tavegil, diazolin, fencarol, among others, reduce tissue edema, eliminate allergic reactions, have a sedative and anti-inflammatory effect.

One pathogenetic treatment for generalised periodontitis is hyperbaric oxygenation (GBO). Oxygen saturation of Periodontium tissues and reduction of hypoxia, respectively, the metabolism and energy balance in cells are normalized, reparative processes, including osteogenesis, anaerobic microorganisms die, microcirculation in parodont tissue is improved. GBO is recommended before surgery (3-4 treatments) and after (6-7 treatments). Contraindications to gbo: poor-quality tumors, heart failure, respiratory and lumbar organ diseases, any hernia, thrombophlebitis, epilepsy, hypersensitivity to oxygen.

To improve the oxygen saturation of Periodontium tissue, it is advisable to use Gbo together with cytochrome-Sbilan, a respiratory enzyme. This drug is well absorbed into cells, is well absorbed when introduced in any way, accelerates the oxidation-recovery process and oxygen utilization.

To normalize the perexis oxidation process of fats and activate antioxidant protection, ozono therapy is recommended, for which water or oil saturated with ozone is used for appliqué or rinsing.

Physiotherapeutic methods are widely used in the treatment of periodontium diseases. In complex treatment, in the Prevention of the disease, in rehabilitation, it is used to influence the pathogenetic stages of the inflammatory process. Valuable features of physiotherapy are an increase in the protective forces of the body and the nospecific reactivity of tissues. Usually, physiotherapy procedures are performed when tooth decay is eliminated and the acute inflammatory process is stopped.

Physical methods of treating periodontitis are used before and after surgical procedure, as well as as holding therapy with the aim of prolonging the period of remission.

Ultraviolet radiation-has an anti-inflammatory, desensitizing, enhancing the processes of metabolism and regeneration. It is used in pronounced inflammation of the Periodontium tissue.

Laser therapy-as a rule, has a multidisciplinary healing effect: anti-inflammatory, due to the normalization of vascular wall permeability and microcirculation; analgesic; thrombolytic; enhances the processes of metabolism and oxidation in tissues; a helium-neon radiation laser is used, which stimulates the regeneration process and the general and local immune defense of the body.

Magnetic therapy-a permanent magnetic field is used. It has the properties of strengthening anti-inflammatory, spasmolytic, analgesic, reparative processes.

Hydrotherapy is the treatment of rinsing the oral cavity using special tool equipment under pressure 1.5-2.0 atm of saturated liquids or aqueous solutions. Decoctions and decoctions made from medicinal herbs, sea water, mineral waters, oxygen, water saturated with dioxide have a more healing property.

Orthopedic treatment is one of the main stages in the complex treatment of periodontium diseases. To do this, a patient with periodontium disease:

- must distribute chewing pressure evenly throughout the preserved teeth;
- eliminate the pressure that occurs when the teeth are taken or the position changes;
- by combining individual zvenos of the chewing system, it is necessary to create a functional unit that is lost for the entire tooth row.

For the complex treatment and rehabilitation of periodontium diseases, special methods of orthopedic treatment are used, which are divided into the following groups:

- selected grinding;
- normalization of the occlusion relationship of teeth by treating anomalies and defects with orthodontic methods;
- temporary prosthetics (immediate-prosthesis) when frontal teeth are taken, as well as when teeth holding the height of the PRIKUS are taken;

temporary shinning, duration from 1 week to 2-3 months (indication – tooth decay);

orthopedic treatment by permanent shine.

Permanent tire structures can be decelerating and Unbreakable, when choosing them, attention should be paid to the condition of the tissues of the coiling teeth and their antogonists periodontium the degree of twitching, the magnitude of the defect of the tooth row. Shinning structures or prostheses should ensure immobilization of the teeth that have been moved, do not pose a challenge to carry out treatment and practices in the periodantium tissue, be able to meet aesthetic requirements.

Permanent shinning can be included in the ranks of rehabilitative procedures, and it can be carried out 3-5 months after surgical treatment of periodontitis.

VI. ATYPICAL CONDUCTIVE TYPES OF PERIODONTITIS

The local recession of milk is allocated to aloxida gurukh within the periodontic diseases. This is manifested by atrophy of the periodontal tissue and a decrease in the height of the gums on the vestibular surface and the opening of the tooth root. The procedure is most often observed in the socket of frontal teeth and pile teeth, but there is no inflammation of the gums and pockets.

Local recession occurs most often in children and adults and, as a rule, gives birth to a knock-off from aesthetic humor, sometimes a hyperesthesia of the hard tissues of the tooth is observed.

The main reason that leads to the local recession of milk is the trauma of the coastal periodontium. In the case of maxillary factors (grinding or not at all present of the dental equator, dental dystopia, pricus pathology), it is possible to distinguish between occlusion trauma with a bite of food and trauma from the result of nonrational hygiene of the oral cavity (the use of a hard tooth tube, vertical and especially horizontal movement of the tube).

Several manifestations of local recession differ:

- partial recession of the vestibular or oral surface with preserved dental gums;
- recession of the vestibular or oral surface on the shaft, which is a partial retraction of the interdental milk;
- a gum recession in two tooth sockets located side by side on the shaft, which is a complete retraction of the interdental milk.

Four types of recession are distinguished:

- 1) wide and high;
- 2) wide and short;
- 3) narrow and high;
- 4) narrow and short.

Treatment of local recession consists of two stages.

The factors caused in the first stage are eliminated: oral hygiene training, dental equator restoration, orthodontic treatment, oral counting. The second stage is the surgeon method, which consists of reconstructing the height of the periodontal compound and forming a tooth-gum compound. Various ussus of Milk plastic are used: sliding the locknut to the lateral side, transplanting the locknut, coronal shifting of the locknut. Currently, the method of using various drugs that enhance

the regeneration of bone tissue with coronal displacement is used in the treatment of local recession of the gums by the method. After the practice of attention should be paid to:

1) rinse the oral cavity with a 2% solution of chlorhexidine bigluconate for 2 minutes a day. Chlorhexidine bigluconate has a healing effect for 12 hours, binding to the mucous membrane of the oral cavity and the surface of the tooth. Due to the presence of a wide range of action, 75% of the oral cavity loses bacteria and resists the formation of tooth decay for 12 hours;

2) brush teeth with extirpation using soft fiber dental floss. In the period after the practice of surgery, the use of dental floss and dental cleaners is prohibited;

3) control over the hygienic condition of the oral cavity for 6 months. Re-examination 3, 6, 12 months after surgical treatment.

Fast-moving periodontites

Inflammatory diseases of periodontitis include an increasing number of atypical suppressor species, one of which is fast-suppressing periodontitis. It is characterized by a severe course of the disease, the development of a pathological process, and in 2-5 years, and sometimes a few months, the alveolar tumor ends with the lysis of bone tissue and the loss of a tooth. Four types are distinguished depending on the course of the fast-moving periodontitis and the age at which it occurs:

- prepubertat (under 11);
- local adolescence;
- diffuse adolescence (12-21 years old);
- adult fast-acting periodontitis (21-35).

On the basis of the etiology and pathogenesis of this disease lies a special microflora, mainly gram-negative anaerobes, which are resistant to antibiotics, have high activity and have the property of entering the parodont tissue. The activity of these microorganisms depends on the toxic substances they produce (endotoxins, enzymes, chemotactic factors, etc.) related. On the basis of the etiology and pathogenesis of all fast-moving periodontitis, two main factors lie: proteolytic activity of microorganisms and functional deficiency of polymorphic-nuclear leukocytes, which leads to impaired chemotaxis and attenuation of phagocytosis.

Primary impairment of functional activity of polymorphic-nuclear leukocytes is observed in 60% of patients. This is caused by a congenital

defect of neutrophils, which leads to a decrease in the amount of receptors sensitive to toxic substances in the periodontal pocket. Secondary disruption of polynuclear function occurs as a result of the aggressive action of microorganisms located in the periodontal pocket. Fast-acting periodontitis is characterized by a change in the immunological status of the patient. Violation of cellular immunity in the humoral Ham leads to a decrease in the adgesia of immunoglobulins and the dressing of immune complexes. The inflammatory process is slow. The degree of humoral immunity depends on pedigree, age, gender, and nationality. Sometimes the genetic congenital deficit of immunoglobulins occurs among Caucasians, Arabs and eureans. Changes in cell immunity are accompanied by a decrease in the ratio of T-lymphocytes and T-helper Ham to T-suppressors, activation of V-lymphocytes.

In fast-moving periodontitis, there is swelling in the connective tissue of the gums, the breakdown of fibrous structures, a violation of microcirculation, followed by microthrombin dressing. The state of tissue basophils changes, degranulating types appear, which increase collagen lysis and resorption of bone tissue. Tissue basophils influence cell immunity by establishing control over T lymphocytes. For this reason, these cells play an important role in the emergence of autoimmune diseases. Fast-acting periodontitis is accompanied by The Desolation of metabolic processes in the periodontium tissue. Each type of fast-moving periodontitis has its own characteristics.

In severe cases of prepubertate early onset is accompanied by a violation of all its stages in the process of phagocytosis. In this case, genetic disorders of the function of polymorphic-nuclear leukocytes have, while there is a slow response to the invasion of microorganisms in dental a slowdown in chemotaxis, insufficient production of a bactericidal factor, a decrease in adgesia and digestive properties. The clinical signs of prepubertal period rapid periodontitis will be similar to those of desmodontosis. Against the background of cases when arrhythmias, hormonal status are established and the formation of periodontium has not reached its end, the onset of the disease in early childhood is the cause of its severe course. The process develops rapidly and leads to tooth loss.

Treatment-complex, Maxillary and general. Oral hygiene, maxillary antibacterial and anti-inflammatory treatment, fluorine,

calcium preparations, complex multivitamin preparations are recommended. It is necessary to come to the dentist's appointment three times a year so that the disease is not re-provoked.

Yuvenil periodontitis is included in idiopathic diseases and is also described under the name "desmodontosis-periodontosis". Yuvenil parodontosis can be local (LYuP) and diffuse (TYuP). According to the literature, Lup is more common among Indian, Armenian, Afghan, yevrey and tatar nationalities. The course of the disease is characterized by rapid destruction of bone tissue in the socket of the central cranium and the first molar teeth. Lupus is three times more common in women than in men. This is due to the fact that hormonal changes are more pronounced in girls during the pubertal period. The disease is acute, can begin with an increase in body temperature. The maxillary signs are manifested by redness, swelling and pain. This process quickly spreads to the tissue of the parodontis, causing the formation of periodontal pockets, bone resorption, tooth decay, and leads to a fall. The root of the resulting teeth will be thinned out. In some cases, the disease can go away without any signs and accidentally manifest itself on an X-ray, while a deep invasion of pathogenic microflora is visible on microscopic examination. Polymorphic-nuclear leukocyte dysfunction in yuvenyl periodontitis is more due to the influence of external factors, in particular with aggressive microflora.

(LYuP) *Bacteroidesgingivalis*, *Capnocytophagaspp.* when detected and in response to their invasion, the immunological status changes, the amount of antibodies in the saliva and gum fluid decreases and does not remain completely after, which leads to the rapid development of the process and the destruction of the parodont tissue. Scattered periodontitis is characterized by an ASL predisposition. The disease most often occurs in Caucasians, eureans, Arabs, Tatars.

The difference between TYuP and ordinary periodontitis is the rapid development of the disease and the presence of signs of resorption of the alveolar tumor bone on an X-ray. The initial period of the disease passes without awareness due to the fact that it is without signs, signs of inflammation and general changes in the body are not observed. Sometimes the disease develops after local yuvenil periodontitis and often begins in the first molar tooth socket, sometimes in the anterior tooth socket. As the process in the periodontium progresses, symptoms characteristic of this disease appear: tooth decay and abscesses. The

examination reveals a violation of the relief of the gum crust as a result of filtration and swelling of the mucous membrane of the gums. The color of the exposed tooth roots does not change. In deep gum pockets, purulent exudate is collected. From time to time, the process is laminated in the form of abstinence, and sometimes the gums are necrosis. As a result of this, the tooth will soon fall out and a defect will occur in the tooth row. X-ray shows bone resorption in the first molar and anterior tooth socket. The resorption is vertical, causing the alveoli to erode completely. On X-ray examination, carious changes are not detected in juvenile parodontitis, but tartar is visible in most cases.

Adults may develop rapidly developing periodontitis (KTRP) in people who have undergone local or diffuse juvenile periodontitis. Adult fast-progressing periodontitis occurs between the ages of 30-62, making up 10-17% of the total patients with periodontitis characterized by the appearance of new, strongly pathogenic and more aggressive microorganisms (*Porphyromonas gingivalis* from the group of bacteroids). They affect polymorphic-nuclear leukocytes, altering their morphology, losing their ability to move towards the foci of inflammation, especially deep periodontal pockets. The amount of lysosomal enzymes entering into interaction with surrounding tissues increases, the permeability of tissue and vascular bars is impaired, as a result of which the signs of alteration increase. Frequent relapses (2-4 times a year), short periods of remission are characteristic of the disease. Sometimes during the relapses of the disease, signs of increased body temperature, loss of body weight, depression are observed. KTRP develops against the background of stress in many cases, taking pregnancy, breastfeeding, climax and is more severe in climax periods. The development and severity of adult periodontitis is influenced by smoking and endocrine disorders (diabetes mellitus).

The process of CTRP relapse is accompanied by the formation of gum tumors and hyperemia, bleeding, tooth decay, periodontal pockets with a depth of 8-10, sometimes 14 mm. There is a rapid and uneven loss of alveolar bone tissue and the formation of bone pockets. X-ray examination reveals uneven destruction of bone up to 2/3 of the tooth root, in severe cases, bone destruction can reach the root tip, signs of osteoporosis appear.

Fast-developing periodontitis develops for 2-5 years, characterized by the spread of the process and the state of depression. There are types

of ham that go symptom-free, in which tooth flutter, gum recession and X-ray are only detected when changes appear. Morphological examination reveals a violation of the epithelial lining structure in the form of cracks, erosions. Basal floor cells lose their characteristic properties to the violation of the basal membrane. On the mesh floor of connective tissue, interstitial edema, neutrophil-leukocyte infiltrate, blood vessel rva lymph capillaries expand, their cavity is observed. The connective tissue matrix of milk is abundant in the immature type of neutrophils, which is caused by their defect I, and the autoimmune response process of organism.

The treatment process of fast-developing periodontitis is continuous, difficult and requires an integrated approach to the choice of treatment measures. Comprehensive and general treatment measures should be aimed at strengthening the protective system of the periodontium tissue and the whole organism.

Premature tooth loss causes disruption of occlusion Relativity, which leads to an exacerbation of the pathological process in the periodontium tissue. Therefore, the effectiveness of CTRP treatment depends on the joint actions of the parodontologist and orthodontist.

General treatment measures are monitored by a therapist, hematologist, endocrinologist, gynecologist and other professionals, depending on the pathological processes identified.

Complex multivitamin drugs, an imudon tablet to enhance the protective property of the mucous membrane of the oral cavity, nosteroid anti-inflammatory drugs and antibiotics are recommended in the course of intense inflammation, when abscesses appear.

Maxillary treatment measures include professional oral hygiene, the use of antibacterial and anti-inflammatory drugs in the form of appliques and ointments. It is advisable to use ozone, which activates the oxidation process of lipids and the antioxidant property of the body, and has an anti-inflammatory effect.

Nonspecific smear syndromes in periodontium tissue.

Desmodontosis (periodontosis) is a chronic diffuse lesion of the Dystrophic nature of the periodontium, which proceeds without inflammation. In 1949, idiopathic disease of the parodont periodontium was announced by the American Academy of parodontology, which is accompanied by non - inflammatory, degenerative destruction of the parodont periodontium tissue, with the formation of tooth decay, tooth-

gum pocket. Prior to them, Wotlieb named it periodontosis in 1920 as "diffuse atrophy of the alveolar bone". Francia's researchers, on the other hand, proposed the name desmodontosis, because, the purpose of this is that the tooth turnover in the disease (Lig. circulari dentis) and the periodontium was an emphasis on injury, damage to the fibers.

Etiology is uncertain. It is included in hereditary diseases and enzymopathies.

Characteristic features: the periodontium is symmetrically damaged (incision and the first molars), the absence of gum - top and gum-bottom stones, the presence of deep pathological pockets, from which the serous-purulent exudate is distinguished is characteristic. Twitching of the teeth, a traumatic occlusion that causes secondary bleeding. Hypoplasia is observed in the teeth. Teeth are less sick with caries. Once the tooth is sucked out, it is normal for the cell to finish.

In histological examination, structural changes in the epithelium are not visible. Hypervascularization of the mucous membrane, thickening of the walls of the capillaries, lymphoplasmocytic infiltration, thickening of collagen fibers, hyaline sclerosis. In the periodontium, edema, inorganic, hyalinosis of collagen fibers, cement are resorbed. The osteoclastic reactant is negative. Compensatory reactivity of the periodontium tissue is not observed.

In X-ray examinations, bone resorption in the disease does not correspond to the clinical picture of the disease. Diffuse resorption of bone tissue is observed.

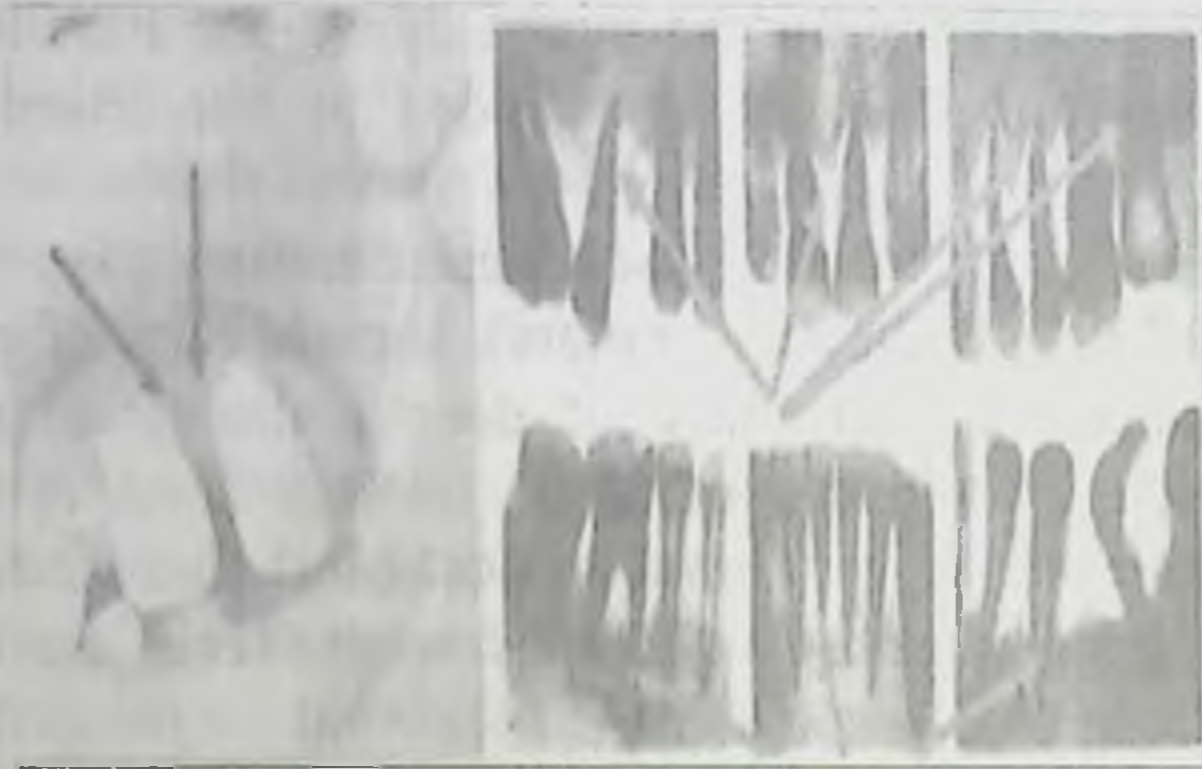
Comparative diagnosis is carried out with jarocative traumatic periodontitis, with post - jarochate osteolysis of bone tissue, with damage to periodontium in Down disease, with ciccyclic neutropenia, hypophosphatasia, histiocytosis, Papion-Lefevra syndrome.

Treatment of the patient mainly consists in treating the symptoms of the disease (symptomatic).

Tooth-gum pocket cleaning-curettage, depulcation of the bite followed by gingivotomy surgery, formalinized transplants such as ingestion, coagulation, etc. Tyrocalcionine is useful because it stops bone absorption. In deepening processes, teeth are obtained.

Eosinophilic granuloma-occurs in diffuse and furnace forms. In the oral cavity, gingivitis, periodontal and bone pockets, tooth decay are detected. Oral 661 in the initial stages of the disease

manifestations in the cavity are considered the first and only symptoms of the disease. Radiological bone tissue osteoporosis and absorption are detected. In its furnace form, a pronounced compensatory reactionary process occurs in a demarcated character pattern.



In pathomorphology, reticular cells, eosinophil Assembly, lymphomacrophagal infiltration can be seen.

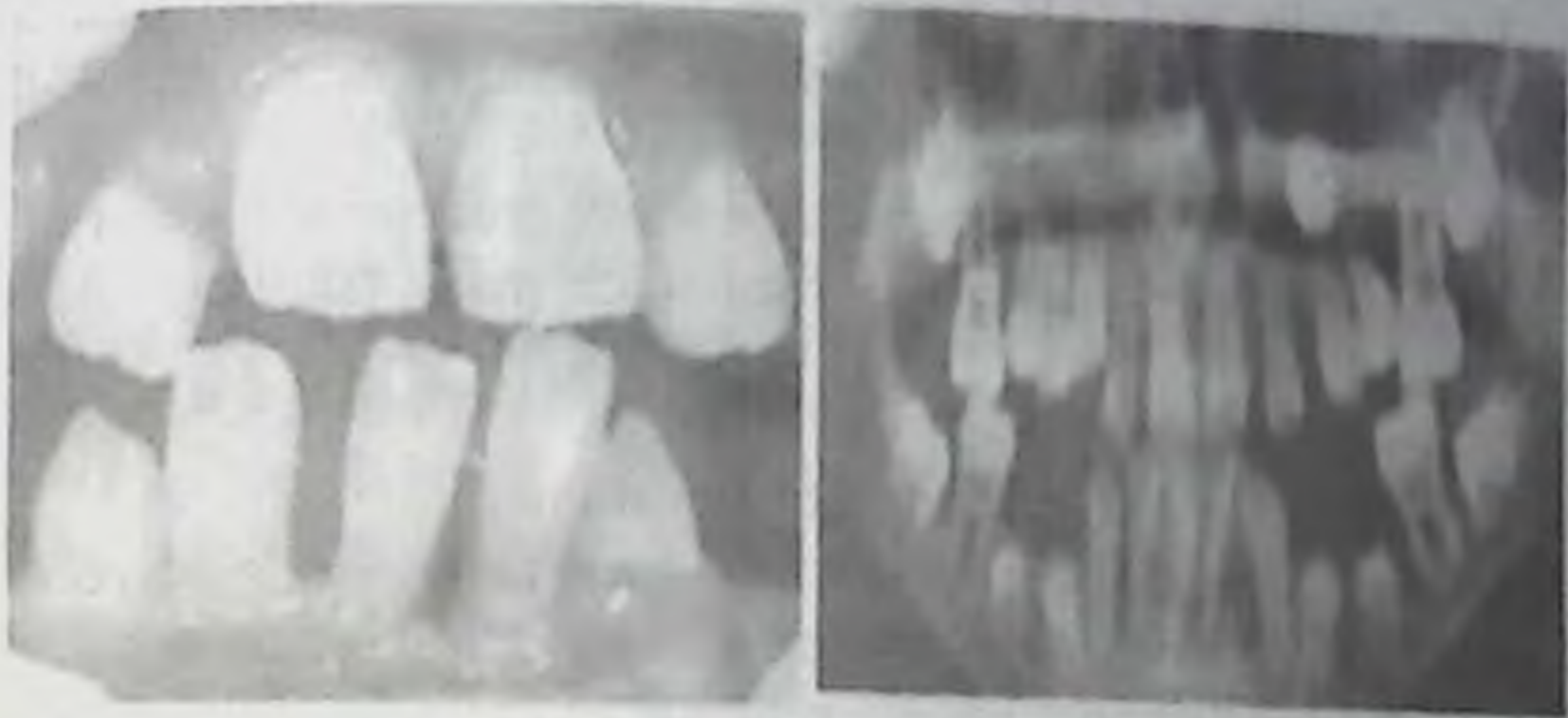
Severe clinical course of Letterer-Sive may include et urination, elevated temperature, leaking skin, inactivity, loss of weight and appetite, and hepato and splenomegaly. A generalised periodontitis in the oral cavity is noted, the culmination of bone absorption. On an X - ray examination-osteoporosis, the absorption of alveolar obstructions, even the absorption of the jaw bone in the migraine, where the disease is located, is observed.

Henda-Schüller-Krischen. It belongs to reticulogistiocytoses. It is accompanied by diabetes mellitus, exophthalmia, non-tumor derivatives in the bone, slowing growth and development. Obvious changes in the oral cavity are observed in the form of a diffuse periodontitis clinic. On an X-ray, a bone tissue destructor is detected in the jaw alveolar tumor.

The advanced stage of Letterer-Sive disease is characterized by fever, discoloration of the skin covers, adinamia, weight loss, loss of appetite, Hepato - and splenomegaly. In the oral cavity, scattered periodontitis manifestations are observed with increasing osteolysis of bone tissue. On an X-ray, the displacement of diffuse and focussed alveolar barriers of the jaw body, osteoporosis is detected.

Papion-Lefevra syndrome is characterized by the fact that periodontitis is accompanied by severe forms, damage in the form of

hyperkeratosis of the skin of the hands and feet, which is transmitted through autosome - recessive Xol. Rare



Due to the destruction of the bone tissue of the alveolar tumor, milk and permanent teeth fall out before the time.

Rathbun syndrome (hypophosphatasia) - autosome - recessive xolda is inherited. Due to the lack of alkaline phosphatase, it leads to severe mineral disturbances in the body skeleton and premature loss of the bite.

Periodontal syndrome in hypophosphatemia (phosphate diabetes, vitamin-D resistant rickets). It is diagnosed against the background of rickets-based disease, which occurs against the background of crooked legs and stagnant gingivitis. Gingivitis passes into periodontitis at a rate of, with the recently ruptured lower and upper cranial teeth falling out at a rate of. The child is low in height, crooked arm legs, early fall of milk teeth. On X-ray, sparse bone tissue becomes a coarse fibrous structure. The amount of calcium in the blood is in moderation, the amount of alkaline phosphatase has increased. Ossification disorders in the field of bone metaphyses. In the jaw bones there is alveolar bone dystrophy in the area of the milk pile and premolars, and in the area of the permanent dental follicle, the bone shell is broken.

For the treatment of histiocytosis, the dentist must necessarily act with oncologists and hematologists, since they (oncologist, hematologist) take general treatment measures for hormonal drugs, citostatics protein anabolizers, antibiotics, desensitizers fluorine and other organism. The dentist is engaged in the use of local treatment measures - the use of orthopedic treatments to obtain tartar (after aplikacion is anesthetic), curettage of dental gum pockets, sometimes

removal of the bite. Treatment is carried out (symptomatic), depending on the symptoms.

T.N. Modin and G.B. Ospanova (2001) produced an individual algorithm of measures for the complex treatment of Ktrp, which is complicated by prikus pathology.

1. Elimination of the acute inflammatory process: antibacterial and anti-creeping therapy, removal of dental caraches (skilling and root grinding).

2. Teaching oral hygiene.

3. Treatment of caries and its complications.

4. Surgical procedure: closed and open curettage, gingivectomy, loskut operations using osteoplastic materials to close the bone defect, as well as on an instructional basis – removal of epithelial lines 6 months after frenuloplasty, vestibuloplasty, locknut operation.

5. Orthodontic treatment is by means of non-solvable orthodontic equipment.

6. Temporarily tight tire after the end of active orthodontic treatment with the help of insoluble orthodontic equipment.

7. Surgical practice with the aim of correcting and re-modeling the Marginal milk and closing the recession.

8. Solution of insoluble orthodontic equipment, preparation of equipment with a retentive hood.

9. Selected grinding.

10. Professional Hygiene.

11. Permanent glazing on the floor using non-removable metalloceramic structures, shinning bügel prostheses, fiber-compatible composites.

12. Aesthetic restoration of dental coronks.

13. Re dispensary examination every 3 months.

CTRP timely diagnosis, individual complex treatment makes it possible to stop the acute conductive process in time, reduce the destruction of the parodont tissue and achieve stagnant remission. In order to prevent relapses, it is necessary to be on dental observation (no less than 3 times a year), carry out holding therapy and constantly stimulate patients to oral hygiene.

VII. LOCAL TREATMENTS FOR PERIODONT DISEASES. PHYSIOTHERAPEUTIC TREATMENTS Prevention RECITURA

In the local treatment of periodontitis, regardless of the severity of the type of disease, it is imperative that anti-licking treatments are carried out first. This should be started after tooth decay, complete removal of the stones. The treatment complex consists of therapeutic, physiotherapeutic, surgical and orthopedic methods. As a result of treatment, it is completed by bringing periodontitis from its active stage to a state of remission, that is, stabilization, but even during remission, it will be necessary to support the healing agents that harmonize the processes of nutrition, substance exchange of periodontitis. Before treatment, a treatment plan is drawn up for a specific patient, in which appropriate, appropriate and specific treatment procedures are introduced, which are applied alternately according to the plan. The most basic and important work is the cleaning of tartar (above the gums and, in particular, under the gums), since the good benefit of subsequent treatment treatments ensures BSR, the usefulness of both surgical or orthopedic treatments, to what extent exactly this work is complete. Of course, teeth to be treated, teeth worthy of removal, roots to be taken, improperly placed fillings, prostheses, devices, clammers and cockroaches to be replaced - the oral cavity must be sanitized. If there are granulemes at the root ends of the tooth, and the same patient has a general autoimmune system of the body, diseases associated with disorders (kidney diseases, rheumatism, rheumatoid conditions, anemia, etc.), then these teeth should be treated or removed again — stomatogenic foci should be lost (of course, there are tooth-milk periodontal, bone pockets). Even then, stomatogenic foci remain in the pathological tooth-gum pockets, and they are G. 3. Based on the microbiological examinations of balyanskaya, it is determined that at least 15 cm of gum (chronic generalized periodontitis), licking in bone tissue, and this can negatively function as a chronic stomatogenic infectious furnace. 8 purpose of dental-gum pathological pocket treatment in the local treatment of periodontitis: 1. licking in the tissues of the parodont is the loss or reduction of disorders of the microcirculation process that are causing destructive changes: 2. loss of edema; 3. reducing microflora in negatively affecting tooth-gum pockets; 4. normalization of tissue substance exchange, oxidation -

recovery processes, the regenerative capacity of periodontium tissue. The first task is to identify the tooth surfaces that cause occlusion surgeon (trauma) and sharpen them to lose and, if necessary, to carry out appropriate orthopedic treatment and perform surgical procedures such as curettage, gingivotomy, sometimes some performed with depulcation of teeth. To accelerate the end of the pocket (regeneration), resorcin, zinc chloride, chlorhexidine, bactericide are used in hypertrophic gingivitis, with abundant blood flow from the gums. They have a bactericidal privaag, which prevents the growth of granulatory tissue and epithelium, helping to finish the pocket. A mixture of resorcin (resorcin 0.1 g and glycerin 10 ml) with glycerin is used to massage (massage) gums. 20% Li resorcin, on the other hand, has a keratolytic effect: its crystals burn. They are inserted into the pathological gum pocket in the turun using a bent probe or root needle. Solutions of zinc chloride (25-50% solution, vagotil (1-2% ionovocaine in 1:2 amounts), 3% chlorhexidine may also be used. They are inserted into the pockets once every 1-3 days. Glucocorticoids, heparin, butadione, salvin, maraslavin, indometacin (indocide), 25% dimexide solutions are used as dehydrating (curable) topical remedies to relieve swelling in the gums. Glucocorticosteroids have exudation, proliferation, anti-allergy, desensitizing and immunodepressive effects. Glucocorticosteroids are used with antibiotics. In the treatment of periodontitis, they are used in the form of Paste, bone or healing ointment, prednisolone ointment (dermosolone), creams ("Lidercort", "Fluorocort", "Kenalog"), ointments "Sinax", "Flutsinar", "Lorinden" and others. For example, when there are milk abscesses, opening them, they give a good result if hydrocartisone – biomyacin (bomicin 50000 ulchov units – YeD, 1 g of anesthetics, 0.125 g of hydrocartisone or 0.125 g of prednisolone, white clay – belaya glina – 6 g, peach oil – paste formation) is sent inside. But their use in the long term can reduce the reparative process, burn 9 without benefit. They are mainly intended for use in proliferative cases, in the short term. Heparin stops blood burning. Microcirculatory processes reduce disruption, thus acting against licking and swelling. Marslavin fights against licking (due to its antimicrobial, vascular shortening, anti-edema effect) in a keratoplastic, anti-proliferation manner. The course of treatment consists of 10-12 sessions, each pathological pocket is inserted, inserted 8-10 times using turun Dental gum pockets are used with antibiotics, antiseptics, sulfanilamides,

nitrofurantoin (such as Furacilin, furazolidone, furagin), decoctions made from medicinal plants, extracts, enzymes, protein anabolizers and other anti-licking drugs. Thus, in mild periodontitis, pocket sludge is used to eliminate licking in the 3.5 mm gum by tooth decay, when the stones are taken, the oral cavity is sanitized and anti-licking in the gum, drugs that correspond to each patient. Full compliance with the rules of oral cleanliness is ensured. In middle parodontitis (parodontal pocket sludge 5 mm), it is necessary to perform curettage or perform measures using ultrasound (ultrasound, using ultrasound dastak – scalers on some modern dental equipment), dental operations on the gums and, in particular, under the gums, after the stones are completely removed (painlessly removed), to finish the pathological pocket, to meiorize the exchange of substances. In severe periodontitis (the thickness of the periodontal pockets is more than 5 mm), a locknut operation is sometimes performed. Orthopedic treatments include pathogenetic treatments with the cessation of tooth decay, loss of traumatic articulation, and ensuring the fullness of dental cathores. Well-prepared dental prostheses are directly related to the good result of treatments in which tires enter another treatment complex.

The method of treatment and medication are selected at the expense of taking into account the severity of the condition of chronic periodontitis and the clinical course of the disease. Complex treatment treatments (therapeutic and orthopedic treatments) are aimed at eliminating inflammation of the periodontium tissue, with methods of loss of the periodontal pockets, stimulation of reparative osteogenesis. Taking into account conservative and surgical methods of treatment, an individual treatment method is developed for each patient.

Conservative treatment.

1. Local treatment.

- Bemomi training in oral hygiene with personal hygiene items (toothbrush, paste, flosses).

- Dental fillings with caries, replacement of hanging filler banks and rational prosthetics, forming full-fledged contact points, filling teeth, replacing poor-quality dental coverings.

- Treatment of periodontal pockets and the pathological process on the mucous membranes by cleaning the surface of the gums and dental Carache with the help of antiseptic drugs.

- Loss of traumatic occlusion by sharpening, correction of bite pathology by the method of orthopedic and orthodontic treatment.

- To carry out a type of local anti-inflammatory treatment, depending on the type of inflammation (the course of the disease, referring to the general condition of the body).

For these purposes, the following medicines are used: antiseptics, antibiotics, non-steroidal anti-inflammatories, sclerosing and improving tissue hemodynamics herbal medicines, enzymes, hormones, Vitamin applicability, Drug Administration, oral baths, healing ligaments.

- Physiotherapy (electrophoresis, darsonval, phonophoresis, milk HydroMassage

- Non-traditional types of treatment - Phytotherapy, apitherapy, cryotherapy, etc.

Glycocorticoids, heparin, butadione, salvin, maraslavin, indometacin (indocide), 25% dimexide solutions are used as dehydration (curable) topical remedies to relieve edema in the gums. Glucocorticosteroids have exudation, proliferation, anti-allergy, desensitizing, and immunodepressive effects. Glucocorticosteroids are used with antibiotics. In the treatment of periodontitis, they are used in the form of a liquid or healing ointment: prednisolone ointment (dermosolone), creams ("Lidercort", "Fluorocort", "Kenalog"), "Sines", "Flucinar", "Lorinden", etc. For example, when there are milk abscesses, opening them, they give a good result if hydrocortisone - biomicin (bomicin 50000 ulchov unit - ED, 1 g of anesthetic, 0.125 g of hydrocortisone or 0.125 g of prednisolone, White Clay - 6 g, peach oil - up to the formation of paste) is sent inside. But long-term use of them can reduce the reparative process, burn without benefit. They are mainly intended for use in proliferative cases, in the short term.

Heparin relieves swelling, bruising in the gums. Microcirculatory processes reduce disruption, thus acting against inflammation and edema.

Maraslavin fights in a keratoplastic, anti-proliferation manner, as it has anti-inflammatory (antimicrobial, vascular shortening, anti-edema effect). The course of treatment consists of 10-12 sessions, each pathological pocket is inserted 8-10 times using turun.

Dental gum pockets are used with antibiotics, antiseptics, sulfanilamides, nitrofurans in-line drugs (such as furacilin, furazolidone,

furagin), decoctions from medicinal plants, extracts, enzymes, protein anabolizers and other anti-inflammatory drugs.

2. General treatment treatments should include:

- fight against infection-reduce intoxication-increase immunity
- to stimulate the normal ingestion and regenerative processes of the metabolism in the parodont.

For this, anti-bacterial infusion disintoxication, desensitization therapies are used, and hormone therapy and vitaminotherapy are also prescribed. For immunity tikiash, an imudon-polyvalent complex is prescribed. It increases salivary phagocytic activity. Depending on the severity of the disease, comprehensive antibiotics are prescribed, which increase the effectiveness of local treatment, reduce the degree of development of complications arising in surgical and conservative treatment. Knowing the magnitude of the role of an anaerobic microorganism in the development of periodontitis, it is advisable to use the drug 5 - nitroimidazole. Nosteroid anti-inflammatory drugs are used, they stop the activity of prostoglandins, reduce the resorption of bone tissue, hormonal drug The schemes of treatment complexes are as follows:

1. Scheme for the treatment of mild chronic and recurrent periodontitis 1. Healthy oral cavity (tooth decay, taking stones, removing teeth that need to be taken). 2. When the Milk is hidden, when the powder becomes darsonvalization or the use of the milk electrophoresis method after vacuum treatment with the fluctuorization or Kolajenko method. 3. When blood flows from gums – to conduct milk electrophoresis with ascorbic acid. 4. Dessensibilizing treatment-to recommend taking one of the pipolfen, diprozines, with chloride calcium. 5. Hygienic recommendations. 6. The finger performed by the patient uzi is sieving, stroking (massage, performed at home). 7. Orthopedic, surgical procedures (correction of occlusion tooth surfaces, preparation of prostheses) when necessary. 8. General treatment: a) good and complete ovulation; B) drinking multivitamins; C) active movement and relaxation, body conditioning.

Indications for the treatment of periodont diseases by physiotherapy methods (V.S. Ivanov, 1989). Diagnostic treatment method gingivitis: catarrhal, chronic hydrotherapy, electrophoresis, darsonvalization, diadinamotherapy, COUF (day), automassage, vacuum treatment, laser. Acute Medicamentous processing. CUF

hydroaeroinotherapy, laser. Hypertrophic, swollen gingivitis electrophoresis, darsonvalization, diadinamotherapy, hydrotherapy, vacuum therapy, all types of massage, CUF. Fibrosis form treatment of gum suckers with dotted diathermocaogulation, iskra darsonvalization, hydrotherapy, paraffin. Wound gingivitis CUF, rinsing with drugs, laser. Periodontitis: rinsing with acute and recurrent drugs, mold, UVCh-treatment, microwaves, fluctuorization, laser. Chronic hydrotherapy, darsonvalization, automassage, hydroeroionodavolation, electrophoresis. Periodontoz Automassage, vibromassage, HydroMassage, darsonvalization, electrophoresis. Idiopathic periodont diseases physiotherapy is not used. Periodontomas physiotherapy is not used. Thus, it is known to you that the treatment of periodont diseases is a complex, multifaceted, work that requires patient and doctor to be patient.

VIII. GENERAL TREATMENT OF PERIODONT DISEASES. Recipe.

It is known from historical manbas that clinical manifestations of parodont diseases were first covered by the French physician Foshar (1746). But until then, the famous Abul Kasim from our eastern doctors made and applied special tools for the removal of tartar, considering that it was important to remove tartar in the treatment of periodont diseases as early as the 10th century.

Abu Ali Ibn Sina (980-1037), in addition to writing full parodont diseases in his famous work, the laws of Tib, proposed to improve the general condition of the body to treat them, improve oral hygiene and put a yolk oil mixture with honey on the gums, and perform a treatment such as chewing wax.

For the general treatment of periodontitis, it is necessary to check with laboratory methods of the patient for common diseases that play a large role in its origin - diseases of the transcaucasus, cardiovascular, kidney and urinary tract, diabetes mellitus, rheumatism, liver and other diseases of the internal organs and systems, determine whether they exist or not, and treat the disease found. Most researchers cite factors that confirm that common chronic diseases that occur in the body are of great importance in the origin of periodont diseases. Therefore, in addition to the general diseases mentioned above, which can occur in a patient with periodontitis, the vegetative nervous system, all diseases associated with metabolism, hypo and avitaminoses, blood diseases and other similar diseases must be treated together with periodontitis.

A patient with Parodont disease will be given comprehensive and complete clinical and laboratory tests by the dentist, and, according to the data obtained, will perform appropriate treatment work together, consulting doctors (therapist, endocrinologist, hematologist, pediatrician, neuropathologist, psychoneurologist, etc.) who work in the necessary specialists.

General treatment of periodont diseases should be started with psychotherapy (exposure to patient zinc). It is necessary that the doctor is able to convince bemomi that the periodont disease in the patient can be cured or stop its development, give much more relief.

Aphorism in medicine, which says "it is necessary to treat the patient, and not the disease in the patient," is very appropriate to treat parodont diseases.

Whether the treatment gives a good result, whether the patient has a general morbidity, living conditions, working conditions, eating patterns and what the doctor prescribes or recommends for the treatment of the disease, is timely and complete, in other words, an attachment to the patient's literacy, intellect, World Vision, culture.

General remedies should be aimed at increasing the total capacity (reactivity) of the patient's body and revitalizing the processes of substance exchange in his body. Complex treatment of periodontitis includes the use of vitamins, antibiotics, stimulant (nonspecific, non-specific) treatments, autogemotherapy, treatments aimed at tissue regeneration-reparative regeneration, desensitizing, hyperbarooxygenotherapy, immunostimulation or immunodepressive therapies.

Antibiotics are used only according to certain guidelines. It is necessary to know in a microbiological way whether the microblading in the pathological pocket has more or less sensitivity to which antibiotics before application.

Stimulant (nonspecific, non-specific) liver-invigorating treatments are widely used in parodont diseases. V.P. Filatov's (1933) proposed method of blunt-force therapy is used. For example, 1 ml of a 10% solution of uterine placenta tissue extract (ampoule) under the skin in total, 10-15 maria injections will give a good result. Vitreous flesh, plasmol, splenins made from animals are also sometimes used.

Chonsuride (chondroitinsemaya acid) is useful for the formation, regeneration and epithelization of connective tissue along with hyaluronic acid. One vial of it is dissolved in 1 ml of 0.5% novocaine and injected into pathological pockets using turundas. Treatment lasts 10-15 days. Chonsuride does not benefit in the forms of hyperplastic-hypertrophic gingivitis, periodontitis and periodontosis.

Drugs that benefit more in parodontosis are vitreous body, hyaluronic acid, lidase.

Biosed mouthwashes made from medicinal herbs are also used - from 1-2 ml daily or daily, and 30 Ni'ekcias in total, such as insadol and piaskledin.

Stimulating treatment consists in increasing the body's immunobiological activity, eliminating vascular - nerve damage, in order to increase the reactivity of the body.

The following remedies are used in stimulating treatment::

a) Aloe extract - activates the metabolism.

B) activates Fibs - regenerative protection.

V) Peloidodestillyat-funkcin. Contraindication-new derivatives, the second half of pregnancy.

r) Plasmol (drug from human blood, desensitizing property). The course of treatment is 10 injections.

d) Follyutin (a protein - free drug, obtained from the placenta) - activates the regeneration process.

e) Splenin (protein - free drug, taken from the mole spleen) - m/o is taken from 2 ml per day. The course of treatment is 10 injections.

J) Regenerator (protein-free drug, stimulant) t/o is introduced on 1 ml of Har. The course of treatment is 10 injections.

Z) Prodigizon is a high molecular polysaccharide-nature agent. Provides stimulant, vascular permeability and tone. m / o is sent 0.5-0.6 ml (25-30 gamm). The course is sent with a break of 2-3 days from 6-8 injections.

Antireticular cytotoxic serum-A.A. Bogomols-activate the function of connective tissue, increase the resistance force of the body. Serum is diluted 10 times in isotonic sodium chloride solution. T / o is introduced from 0.1, 0.3, 0.5 ml 1 time in 2-3 days. The course of treatment consists of 3 injections. K) Autogematherapy-7-10 injections. According to the scheme.

L) Pentoxyl - leukopoiesis stimulator. It is prescribed in capsules for drinking from 0.2 to 2 mAh per day after meals. The course of treatment is 12 days

M) Metacil-pentaxil analogue, in the form of powder, is prescribed from 0.5 g to 2 mAh per day. The course of treatment is 15 days

H) Anabolytic steroids: retabolil, nerobol, thyrocalcitonin

o) Filatov whey, autoanticoagulants, Bogomolc buyicha ACS.

For the general treatment of periodontitis, vitaminotherapy is prescribed. Vitamins C, V, A, E, etc. are reduced in the body of patients with parodont diseases, necessitating their treatment. In addition to

them, vitamins VG, VB, B12, KZ, P are also used in their place. Vitamins C and P (rutin) are widely used in young people in particular when blood flows from milk. These vitamins reduce the permeability of the walls of the capillaries, preventing blood flow. Vitamin C acts on the formation of collagen, compacting the walls of blood vessels, increasing the activity of osteoblasts. Vitamin C is used with vitamin P. Vitamin P helps to recover from a violation of the permeability of capillaries, oxygenation - recovery processes occur in moderation, allowing the accumulation of vitamin C in the tissue. In much more micellar, vitamin C enters the body with food. Ascorutin (vit. P) 0.1 g, 2-3 times, and vitamin C in the composition of multivitamins from 2-3 pieces is given overnight. Vitamin B1 normalizes the nutrition of parodont tissue and the metabolism of carbon dioxide, proteins. In the form of a 5% - thiamine solution, 1-2% - novocaine is added and injectable, or milk electrophoresis (administered by Method to parodont. Vitamin K (nicotinic acid) is more commonly used in mucosal diseases, sometimes also in periodont's disease. In particular, in diseases of the gastrointestinal tract, liver, diseases of vascular spasms, when there are periodont changes in atherosclerosis, 0.005 grom 2 injections of nicotinic acid every day (after meals). In order for vitamin B6 (pyridoxine) to participate in protein metabolism, periodont diseases are used when accompanied by diseases of the stomach and 12-finger intestinal ulcer, hypochromic anemia, chronic hepatitis, cardiovascular atherosclerosis, diseases of the central nervous system. Pyridoxine (Be) is taken in 0.01 g of a tablet, 2-3 times a day, the way to drink biiian. Vitamin B12 (ciancobalamin, coamide) is used when periodont diseases occur in conjunction with liver (hepatitis, botkin's disease, cirrhosis), atherosclerosis, anemia, including anemia in pregnant women. It is injected subcutaneously or into a muscle from 30-100 MCG 2-3 times a week. In case of violation of local and general metabolism, V12 is sent along with vitamin C, V and ATF. Vitamin E reduces edema in the tissue (parodont) and also reduces the absorption of the alveolar bone tissue of the hyaluronidase. It is administered from 50 mg 3 times a day (oily solution) for 3-5 weeks. Also, oblepixa is introduced into pathological tooth-gum pockets in turundas due to the fat content of 1650 MCG/1 vitamin E.

A mixture of vitamins E and a (aevit) (fatty solution) is sent to pockets or muscle in turundas daily for 1 ml to 20 days to strengthen, revitalize the regeneration of Parodont tissues.

Vitamin A improves the function of epithelial tissue, is taken from 10 drops during meals or 2-5 drops for 3 weeks after meals. Vitaminotherapy gives a good result in the initial periods of periodont disease (gingivitis, mild periodontitis). Their use can mean pathogenetic treatment. However, a variety of vitamins B₁, VB, V₁₂ can give complications by causing an allergic condition. It is also good to use vitaminlam with antibiotics together.

If periodont diseases are complicated by allergic reactions, then antihistamine drugs (sodium thiosulfate, dimedrol, pipolfen, suprastin, tavegil) are prescribed. Antibiotics are prescribed mainly in the acute period of the disease. In this case, antibiotics with a wide spectrum of action are recommended from 1.0 or 1.5 GR per day. In periodont diseases, there is a weakening of the general protective forces of the body, and therefore the recommendation of immunostimulants gives good results.

Pyrogenal - microbial polysaccharides are used to animate the body's protective - adaptive function. In this, the body temperature rises, which is considered useful. Its administration to the muscle is gradually increased from 5 mcg to 20 mcg 3 times a week, all with bulib doing 20 injections. Patients may have complications - headaches, nausea, vomiting, joints, back pain, etc. These stop when treatment is complete. It cannot be used in pregnancy, diabetes, hypertension. Treatment is carried out in the hospital (stationary). Also, the likes of prodigizon, pentoxyl can be used when certain indications are available.

T.V. Nikitina (1975) performed well by sending retabolil, a member of the anabolic steroid group, to the muscle from 1 ml (5% fat solution) once a week (3-5 injections in total).

Hormones are also recommended in patients with periodont diseases, in which the functions of the endocrine glands are impaired. But their application is carried out with caution.

In Parodont diseases - when the parts of the tooth neck open and pain or unpleasant sensations arise from various external influences in the lands, fluoride preparations - 1-2% - solution of fluoride sodium are

applied in the method of application (20 minutes), milk electrophoresis.

Desensitization treatments in Parodont diseases are widely used to increase reactivity in parodont diseases of the body. For this purpose, calcium preparations - 10% chloride or gluconate calcium, glycerophosphate-calcium are used. Chloride calcium (10% solution) is drunk or injected 3 times a day (before meals) in 1 large (soup) spoon. Thiosulfate sodium also has a desensitizing, antitoxic and anti-inflammatory effect. Its 30% - solution is sent to Vienna (from 10 ml), at a rate of 6-10 injections. Decensibilizacia and anti-inflammatory glucocorticoids, as well as a 5-10% formalin solution (applicacia for 7 days), can also be used.

Also antihistamine drugs (thiosulfate, dimedrol, pinolfen, suprastin, elenium, tavegil, etc.

It is important to note that when periodont diseases are accompanied by General diseases of the body, all-time treatment is two - to-three-sided, for example, for the treatment of periodontitis in diabetes, with an endocrinologist, with periodont diseases in pregnant patients-with gynecologists, with psychoneurologists (and hokoza).

Research suggests that there may be different changes in the immune system in parodont diseases and that they cause parodont disease to be called, leading to different complications.

Shuna, immunotherapy began to be used. It was also anicized that immunotherapy should be carried out under the supervision of phakat patient hospitalization, laboratory tests and the same observations. For these purposes, levomizol (Z. Iwani, T. Lehner, 1977), diucifon (T.X. Safarov, 1986), immunol, immunomodulin etc.

General treatment of periodont diseases also requires the treatment of primary or comorbid diseases of the body. To do this, identify diseases and make them in their specialists (therapist, endocrinologist, allergologist, hematologist, etc.) complex treatment is recommended.

Basic treatment principles

1. Treatment will be focused on stabilizing the disease, based on loss of the inflammatory-destructive process in the parodont tissue and Prevention of disease complications. These treatments should aim to not only eliminate the primary symptoms of the disease and bring the

parodont back to normal, but also to prevent negative conditions that affect the patient's body in general and locally.

2. It should contain an etiotropic, pathogenetic and symptomatic treatment.

3. The treatment plan must be selected individually for each patient, as the parodont disease clinic varies and determines each organism based on its individual condition.

Whatever type of periodont, local treatment begins all the time with tooth decay, removal of stones. To do this, work with the help of the "Ultrastom" apparatus and the ultrasound tools based on it (for example, there is a special scalper - nakonechnik on dental equipment of the "Ergostar" brand) will work well.

In addition to it, work should be carried out such as the loss of fillers, orthopedic and orthodontic prostheses, defects of devices (parts of the filling that have entered or pressed the gums, artificial coatings that have penetrated deep into the gums and similar negative effects), the treatment of prikus pathologies.

Of course, following the rules of oral hygiene (cleanliness), rinsing the oral cavity with a solution of 0.06% chlorhexidine against germs (bathhouse), which reduces the flaking in the parodont, interrupts the Assembly. Again, rinsing the mouth with various antiseptics, decoctions of various medicinal plants also provides good benefits.

In the case of periodont diseases, it is necessary that a doctor helps with the choice of dental elixirs, pastes, give instructions - recommendations. In the case of periodont inflammation with tooth powder, tooth cleaning is not advisable. The teeth to be treated must be treated, the ones to be removed must be removed.

Once local adverse effects are transmitted, pathogenetic complex treatments are used that affect the various pathological changes that occur in the tissues of the parodont.

These methods should be aimed at improving the exchange process of a broken, derailed substance in the parodont, which affects the gums, bone tissue, microcirculation — blood vessels, nerve fibers, and thus.

Complex treatments that affect the pathogenetic joints include medications (mainly anti—inflammatory), orthopedic, jarrox, and

physiotherapeutic treatment factors. Of the drugs that affect inflammation, 3% acetylsalicylic ointment (maz) is used as an applicator or treatment option, 3%-salicylate sodium ointment, such as butadione ointment, decoctions of medicinal plants -1:10 Hairy xandelia, 1:20 coarse field tea, etc.

To normalize vascular-tissue permeability, heparin ointment (appliqué), mouth rinsing with 5% aminacaprionic acid or injection in the milk electrophoresis method will give a good result, electrophoresis, antibiotics, sulfonamide drugs, enzymes, corticosteroids (only carbons to the immunological state), immunomodulators can be used.

Orthopedic treatment

For the full-fledged conservative and surgical treatment of parodont, temporary and permanent glazing is widely used. Tires, plastic or composite ligatures made of ligature wires are used to tire moving teeth. For permanent glazing, removable and non-removable orthopedic structures are used. In a diffuse parodontitis, all teeth are introduced into the shin area, and in a local one, some tooth groups.

In the treatment of periodontitis, the periodont pocket is lost using jarroxic methods. By stopping the alveolar destruction of the bone, stabilization of the periodont state is achieved.

In clinical practice, V.S.It is convenient to apply the Ivanova classification:

1. Methods of jarring aimed at the loss of the periodontal pocket:
 - Kyuretaj
 - Gingivotomy
 - Gingivectomy
 - physiochurgical (surgical) methods (electrocoalulation, vacuum surgery, cryosurgery, laser surgery)
2. Parodontoplasty jarroxia:
 - Laxtak operacia-Gingivoplasty
 - Lahtak operacia for tikiash of reparative processes of parodont
3. Oral cavity correccia (frenulotomy, frenulectomy, vestibuloplasty).

The main function of surgical intervention depends on the depth of the parodontal pocket and the resorption of bone tissue:

Table 4.

Pocket depth treatment

3.5-4mm curettage

Open curettage up to 5 mm

Operation Cup laxtak from 5mm

Instruction to remove the tooth:

* bone tissue resorption makes up % of the root.

• The tooth has a whipping rate of 3.

Contraindications to the conduct of surgery:

- active phase of systemic osteoporosis

- blood disease (hemophilia)

- active form of tuberculosis

- oncological diseases

- fetal

- acute infectious diseases

- low hemoglobin content in the blood

Local contraindications:

- insufficient oral hygiene

- bite irreparable defect

- traumatic occlusion

- fibrosis altered gums

Preparatory events held before surgery:

- normalization of oral hygiene

- oral sanitation

- cleaning tartar and Carache

- alignment of occlusion surfaces of the tooth

- tire moving teeth

- conducting local and general antimicrobial and anti-inflammatory treatment procedures.

In addition to the conservative method of treatment, surgical methods of treatment are also widely used in the complex treatment of periodontitis. These techniques contribute to the loss of chronic foci of inflammation and the periodontal pocket, and the cessation of alveolar tumor bone tissue degradation and resorption.

According to the degree of incidence, the following surgical treatment methods are used:

- Curettage in mild Grade periodontitis.

- Open curettage in middle-level parodontitis.

- Lactic operacia in heavy-grade parodontitis.

The purpose of surgical treatment is to remove tartar from under the gums, granular tissue, remnants of the epithelium, fill bone tissue defects with osteoplastic materials and promote the restoration of bone tissue. In the treatment of chronic periodontitis, upper and lower jugular plastic, tongue plastic, oral cavity entrance plastic are important, since all this is considered to be the cause of the origin of periodontitis.

Surgical treatments have an important place in the complex treatment of periodont diseases. The purpose of this is to eliminate the periodontal pockets, to eliminate the foci of infection, to stop the destruction of the alveolar tumor bone tissue. Surgical treatments for periodont diseases can be combined as follows:

I. Methods of surgical treatment of periodontal pockets:

- curettage (indoor, outdoor);
- cryo - and electrojarrochlik;
- gingivotomy;
- gingivectomy.

II. Loskut operations.

III. Creating a mouthpiece immunity and moving the eaves.

Before surgical treatment, preoperative preparatory work is carried out, in which treatment procedures should be carried out in full, and oral hygiene should also be taught. During the preparatory period, the patient must be examined by a therapist, mandatory examination methods are blood sampling, determination of blood sugar levels, VICH infection testing, urine sampling, blood clotting time, platelet count. Milkusti tooth sediments are obtained by extentionality, oral cavity dating (filling of carious pores, removing hanging fillings, obtaining broken teeth), general and maxillary anti-inflammatory treatment procedures are carried out. Supracontacts are identified and removed if necessary. In the case of teeth in a state of loose and functional tension, a temporary tire is carried out.

Closed curettage is the elimination of the periodontal pocket. Mild level of periodontitis and pocket depth up to 3.5-4 mm, more often performed in cases where the vestibular or oral side retains a gum-tooth joint and no bone pockets. To eliminate the periodontal pocket, mycotic tartar, microbial caroches should be removed. It is

recommended to carry out closed curettage up to 2 times a year, more often it leads to thinning of the gums and the opening of the tooth roots in the interdental intervals. At the same time, 3-4 teeth are processed into periodontal pockets in the Sox, the interval between curettage is 2-3 days.

Method of conducting closed curettage:

1) treatment of the oral cavity with a warm antiseptic solution (1% hydrogen peroxide solution, 0.02% fibracillin solution);

2) anesthetic (infiltration or conductive (conductive) anesthesia);

3) removal of tartar and altered cement. First, tooth sediments are obtained with an excavator, a loop, a, sequentially treated on all surfaces of the tooth, and then the softened cement is removed to the point where the hardness is felt. The root of the tooth is smoothed with the help of an ultrasound scaler, finer and polar, shaking with a warm antiseptic;

4) periodontal pocket bottom processing. Granulations with a sharp curette or excavator are scraped off;

5) Pocket deoptimization. With the big finger, the gums are pressed to the surface of the tooth, the acute curette is inserted to the bottom of the pocket, and with the finger in control, the instrument is moved towards the dental corona, and the granulation and epithelial floor are cut, which impair the attachment of the nail to the tooth;

6) the operating area is washed with a warm antiseptic, the gum is pressed to the surface of the tooth and a protective bandage is placed.

So that the process of formation of connective tissue is not disturbed, it is not recommended to introduce drugs into the bladder, a blood clot is a good biological link. The regeneration and epithelization of the crack lasts 7-10 days, it takes 21 days for collagen fibers to form.

As a link, various healing sections are used, which have an anti-inflammatory, epithelial-enhancing property. A day after the surgical procedure, a control examination is carried out with the replacement of the ligament (every day for 5-7 days). It is impossible to probe the periodontal pocket for 3 weeks.

After surgery, the patient should clean his teeth with healing pastes every time after eating, rinsing with antiseptic solutions.

Complications that can be observed after closed curettage:

1. During chewing, pain in the teeth may appear in percussion, in severe cases lymphadenitis or retrograde pulpitis may occur. Such complications are a rough increase in curettage to practice and the body's response to operational trauma. In pulpitis, the tooth is depilated, in lymphadenitis, general anti-inflammatory treatment is carried out.

2. Bleeding during and after curettage-as a result of an inflammatory process around the wall of blood vessels or their injury during a surgical procedure. A 5% aminocaproic acid solution, 3% hydrogen peroxide, and various other hemostatic bonds are used when multiple bleeding is observed.

3. When the surface layer of dentin and the affected part of the cement are removed, temperature changes and hypersensitivity to tactile effects occur. Fluorine preparations and others are recommended in this.

Contraindications to closed curettage:

- pus leakage from the periodontal pocket.
- Periodontal pockets deeper than 4 mm.
- acute inflammatory diseases of the mucous membrane of the oral cavity.

The disadvantage of this method is the absence of visual control, as a result of which milkosti tartar and granulations may not be completely removed. Properly performed curettage makes it possible to eliminate the periodontal pocket as a result of the growth of connective tissue at the expense of scarring adhesions and blood clots.

Outdoor curettage. A modification of locknut operations has been proposed at the mid-severe level of periodontitis, and this method is known as open curettage.

Instructions for conducting open curettage:

- pocket depth 4-5 mm;
- granulation growth;
- milk does not stick to the tooth.

Open curettage is also performed in several stages, such as closed curettage:

1. Treatment of the oral cavity with an antiseptic.
2. Anesthetic (infiltration or conductive anesthesia).

3. With the help of a scalpel, an incision is made from the tip of the interdental gum suckers in the Sox of 5-6 teeth.

4. In an impenetrable way, the mucous-Bony curtain locknut (lip-lunge and tongue suckers of the interdental gum) is separated towards the depth of the pocket.

5. With the help of excavators, loops, tooth sediments and granulations are removed.

6. With scissors, granulations are trimmed, and the di epithelization of the locknut is carried out the changed part of the 1-1.5 mm milk is removed.

7. The wound is washed with an antiseptic solution, the locknut is placed in the previous place and an anti-inflammatory grease-based bandage is placed for 2-3 hours.

Curettage in the area of the other group of teeth is carried out after 7-10 days.

There are several advantages of open curettage over closed curettage: the periodontal pocket is eliminated under visual control (removal of tartar, granulations, di epithelization of the locknut). In the postoperative period, milk is treated with antiseptics.

Contraindications to open curettage: pus, the presence of an acute inflammatory process in the mucous membrane of the oral cavity.

To enhance osteogenesis after open curettage, orthotropic drugs are recommended. They are inserted into the bone pockets and into the operating wound under the mucous membrane locknut and fixed with a protective bond.

Gingivectomy as an emergency surgical procedure, it is used in periodontal abscesses, when there are deep and thin periodontal pockets.

Gingivectomy technique: under conductive or infiltrative anesthesia, the gum wall of the periodontal pocket is cut along its entire depth to the bottom of the pocket. With visual control, curettage is carried out, the surgeon is treated with antiseptics, sutures and a gum connection are placed on the frontal tooth socket. In the Frontal Sox, taking into account the appearance, The Vertical cross-section is performed from the pocket projection on the lateral, healthy tissue Sox, a triangular locust is formed, opening the entrance to the bone

pocket. Curettage is carried out, the locknut is put back in place, sutures and a gum tie are placed on The Vertical cross-section. The surgeon recovers at the base of the bone and no defect is formed at the edge of the gum.

In periodontal abscesses, the direction of the cross-section may be horizontal, 4-5 mm below the edge of the gums, treated with an antiseptic solution to the cavity of the abscess and applied sorbent appliqué.

The disadvantages of gingivectomy are the narrowness of the range of indications for its transfer, gum retraction, and insufficient visibility of operational injury.

Gingivectomy. There are 2 types of gum cutting along the depth of the periodontal pocket: simple and radical. Indication for normal gingivectomy: micellar pockets up to 5 mm deep, fibrosis changes of the gums, absence of bone pockets, horizontal resorption of bone tissue, hypertrophic gingivitis. An indication against gingivectomy is deep bone pockets, a narrow part of the attached gum.

The purpose of surgeon practice: to remove gum pockets to their entire depth.

Gingivectomy execution technique: treated with warm antiseptics to the oral cavity. After anesthesia, the depth of the pockets is measured and marked with dots on the alveolar tumor. The horizontal cross-section is made from the vestibular and oral side 1 mm above the points. The gum edge is removed and an open curettage is performed with visual control. The gums are removed at an angle relative to the surface of the alveolar tumor so that the bone tissue does not open surgeon is carried in an open manner in an iodoform turun or under a gum link.

Radical gingivectomy combines in itself the removal of the periodontal pocket and partial suturing of the alveolar tumor. Indication-vertical uneven resorption of the alveolar tumor in the pocket up to 5 mm deep. In radical gingivectomy, the gums and intra-bone pockets are processed. The operation technique is similar to a simple gingivectomy, except that the horizontal cross-section pockets are made 2 mm lower than the specified point. Since the depth of the pockets is different, the cross-section is not only horizontal, but can be arched. The edge of the gums is cut off, the bone and partial bone

pockets open. The changed bone tissue is removed with a milling cutter, boron, curettage spoon, the alveolar part is flattened and the remains of tartar, granulations are removed. Surgeon is closed with an iodoform buffer or healing protective bond that can be fixed to the teeth.

Disadvantages of gingivectomy: dissatisfaction with aesthetics humor, uneven elimination of bone pockets, since there is insufficient visual control during this practice, dental hyperesthesia. For this reason, gingivectomy is not widely used, it is recommended to be performed only on the socket of the lateral teeth. The postoperative period is carried out like an open curettage.

Locknut operations. Applied to eliminate periodontal pockets, correct gum edge defects, regenerate damaged bone tissue. Whole and divided locknut are forked. The entire locknut is a divided locknut, consisting of the epithelium, connective tissue and the osseous curtain composed of epithelium and connective tissue. In some operations, the isolated locknut is re-inserted into place (a simple locknut), while in others it is transplanted into another new Sox at the end of the surgeon practice (positional locknut). On the basis of simple locknut operations lies a radical operation on Vidmar-Neumann-Dzershinsk. Indications are diffuse periodontitis with moderate to severe grade, periodontal pockets 4-5 mm deep, bone pockets, bone tissue resorption vertically oriented, teeth flutter level I-II, thinned or fibrosis altered gums.

Technique of surgical practice: after anesthetic, two vertical cuts are made along the border of the operating field from the left and right side to the conductive fold from the edge of the gums, but not through the gum suckers. Then a horizontal cross section is made along the base of the interdental suckers from the vestibular and oral sides, using scissors, cutting the changed mucous membrane at the edge of the gums to a width of 2 mm. The mucous-Bony curtain locknut is isolated, and under visual control, the gums are removed with dental sediments, damaged cement, granulations extirpated, the tooth root is smoothed out. Slowly, the altered part of the alveolar tumor is treated, with Di epithelization of the inner surface of the locknut. The surface of the surgeon is washed with warm antiseptics, the locknut is put

back in place, sutures are placed in a vertical section and in a deaf tooth interval.

The practice of jarring in the Sox of up to 6 teeth can be performed at a time. Surgeon is recovered by the primary transaction, the seams are taken on 6-7 days. The disadvantages of the operation are the nudity of the tooth neck, hyperesthesia, a decrease in alveolar costal height. Therefore, the practice of Surgeon in such a style is carried out in very few cases. More often, various modifications of the gum edge are used, aimed at restoring the anatomical structure.

Currently, reparative and reconstructive methods are used in the treatment of periodontitis by jarring. In the reparative method, the infective furnace in the periodontium is eliminated, tissue destruction is stopped, in part by the principle of reparative regeneration, the tooth-gum is regenerated. For this purpose, means for accelerating regeneration processes are used in periodontium weaving.

Etiotropic treatment includes to expand the oral cavity and carry out the displacement of the larynx by surgeon. The narrowness of the lip, the short cleft of the tongue, the oral cavity increases the characatenes of the suckers and, when speaking, eating, causes the gum crust to tighten when the tooth is cleaned. This leads to trauma, inflammation and the formation of periodontal pockets of periodontitis. Narrow inclusions and short are diagnosed by pulling the lip in the apical direction. If the width of the attached gum is insufficient, the tooth neck will open. In order to expand the attached gum, the oral cavity is deepened, and in the case of shortened grooves – the plastic of the lab vat il grooves is performed.

All surgeon methods are characterized by the necessary stage of complex treatment of periodontium diseases. And the quality of treatment depends on the correct diagnosis, timely and correct implementation of adequate treatment measures.

IX. PREVENTION OF PERIODONT DISEASES. DISPENSARIZATION.

Prevention is a system of political, social, medical and hygienic measures aimed at keeping the oral cavity and the whole organism healthy and preventing oral diseases. The main goal of prophylaxis is – the causes that lead to the origin and development of the disease, which is to increase the body's resistance to the action of non-volatile factors of the environment and environment.

Limiting the intake of carbohydrates refined by pregnant women has a positive effect on the development of the fetus, hard tissues of the teeth and, in particular, periodontium. S.V. According to Tarmayeva, the growth of caries in pregnant women in KPU indksi was 25.4% in 7-8 months and 56.4% in 4-5 months after childbirth. Therefore, it is necessary to carry out the prophylaxis of dental caries and periodontium diseases from the antenatal period in pregnant women and children, since improper nutrition of pregnant people can lead to defects in fetal development and affect a woman's health during pregnancy and breastfeeding.

Natural feeding of a child, that is, breastfeeding, has a great acuity for him to grow up to mature in all respects. Together with breast milk, the child receives not only fast digestible food products, but also enzymes, hormones, as well as substances that contain protective factors (immunoglobulins, lysozyme, bifidoomil, etc.) receive Ham, which serve for the proper development of the child's dental-jaw system. As the child grows and develops, the diet changes depending on the body's demand. The main components of the diet are fruits and vegetables and Greens. In this, nutrition is beneficial, the chewing reflex is activated and positively affects the musculoskeletal apparatus of the tooth-jaw system.

Solid pieces of food are an exercise and cleansing factor that not only saturates the body, but also relieves the sloth to chew for the organs of the oral cavity. Teeth cleaning foods include: apples, radishes, turnips, carrots, cucumbers. For the tooth-jaw system, the consumption of hard and dry nutrients is a good exercise, which during long, intensive chewing secretes a lot of saliva and ensures that all the surface of the teeth is cleaned. Hard bread, pieces of meat, dried fish and sausage

products are cleaned all the surface of the teeth as a result of long chewing and abundant salivation.

The formation of soft tooth decay is hindered by hydrophobic oil solutions. When rinsing the mouth with vegetable oils, they are kept in the mouth until the next meal is taken and prevent the formation of soft tooth decay.

The effect of fluorine, on the other hand, is due to the fact that this microelement increases the maxillary immunity, reduces the risk of caries formation and improves the hygienic condition of the oral cavity.

Health as a way of life of children is closely related to the way of life of parents. Raising a healthy lifestyle in children and teaching the rules of personal hygiene are directly related to the parent's desire to raise a healthy child.

The effectiveness of dental preventive measures is determined by the level of dental wellness, while increasing this level is the main goal of prevention.

In recent years, the method of therapy has become widely used in dentistry. Ozonized solutions are used in the treatment of periodontium disorders, in the optimization of oral hygiene. Today, ozone is used in all areas of Medicine.

The effect of ozone is due to the action of free radicals of oxygen, which are formed when ozone is dissolved in water. It is because of its highly reactive hydroxyl radicals that ozone has a destructive effect on various micro organisms. Ozone also has the property of destroying aerobes, anaerobes, fungi, viruses, it distorts the integrity of the shell of micro organisms, reacts with its DNA molecule and stops the development of the microorganism. Low ozone concentrations affect lymphocytes and monocytes, increasing cytokine activity can have an immune correctional effect.

From the information mentioned above, it can be understood that periodontium diseases are an important medico-social problem, the solution of which depends on modern dental prevention. The first and main task for this is to bring preventive measures to every city, district and village.

All doctor-dentists are mainly engaged in restorative work remains, while dental caries and the unsatisfactory condition of oral hygiene are not addressed by either the doctor or the patient himself. It is necessary to revise the Polyclinic system due to the inability of doctors

to provide time for employment, preventive and dental bleaching work. It is necessary to organize treatment and prophylactic departments in every city Polyclinic and schools specialists can be involved in restoration work and other specialists for treatment and preventive work.

In the implementation of primary preventive measures, it is important to educate on hygiene, carry out dental bleaching and teach the rules of oral hygiene.

In increasing the level of dental health of the population, the cost – effective method of discharge is mass prophylaxis, using all methods and means of dental bleaching.

Three different manifestations of prophylaxis are distinguished: primary, secondary, tertiary.

The purpose of primary prevention is to maintain the wellness of the periodontium tissue, preventing the effects of environmental and social factors that can cause pathological changes in the periodontium

Primary prevention takes the following measures into its own drink:

1. Breastfeeding and rational feeding.
2. Exercise the chewing apparatus for the normal development of the periodontium
3. On the basis of indications – early orthodontic and orthopedic treatment.
4. To carry out stimulation work on cleaning the oral cavity.
5. Identification and elimination of risk factors associated with anomalies of the attachment of lip, tongue and small intact grooves.
6. Selective grinding of teeth to eliminate supracontacts.
7. Identification and treatment of bruxism.

Secondary prevention is aimed at early detection of periodontium diseases, preventing the development of the disease and the occurrence of complications.

Measures implemented for secondary prevention:

1. Elimination of traumatic factors (tartar, hanging fillings, poor-quality orthopedic and orthodontic devices anomalies, carious cavities).
2. Professional Hygiene, controlled teeth cleaning.
3. Treatment of gingivitis.

Tertiary prophylaxis is aimed at transferring periodontium diseases to a severe type, preventing recurrence of the disease, re-biting the function of the chewing apparatus. Tertiary prophylaxis embodies

therapeutic, Surgical, Orthopedic and physiotherapeutic treatments within itself.

Prevention of periodontium diseases requires the implementation of measures such as dental bleaching measures, mastering hygienic incontinence in rational nutrition.

Dental bleaching measures aimed at stimulating oral cleansing include various measures depending on the population player, based on WHO recommendations:

1. Pregnant women:

a) to inform fetal women about the possible origin of periodontium diseases;

b) encourage women to increase their dental wellness level;

C) to make recommendations for proper, rational nutrition.

2. Children under 5 years old:

a) to attract parents to interest their child in cleaning the oral cavity (to set an example themselves and control the cleaning of children's teeth);

b) limit the consumption of refined carbohydrates to children.

3. Children from 6 to 10 years old:

a) to provide information about oral hygiene and the autonomy and necessity of the diet of proper nutrition;

b) control the need to carefully clean the teeth and follow oral hygiene;

C) recommend effective methods of compliance with oral hygiene;

g) to empower parents in the presence of hidden "sugar" in the composition of some processed foods and sweets, and to give children advice on safe foods and sweets in the cause of dental and parodont periodontium diseases;

d) to provide information about the first signs of periodontium diseases, in particular, the appearance of bleeding gums during dental cleaning.

4. Children aged 11 to 16 years and older:

a) to explain the autonomy of the health of the teeth from the social nature;

b) promote the concepts of the linkage of the state of Health and oral cavity, the first signs of dental and periodontium diseases (bad breath coming, impaired sense of taste);

C) to explain that it is necessary to refrain from smoking, alcohol, drugs, which are considered dangerous to health, harmful habits.

5. Adults with periodontium disease:

a) to give an understanding of the diagnosis, the purpose of the treatment and the fact that it is necessary to come to the repeated reception of the dentist;

b) to provide information on the possibility of recurrence of the disease in a case of non-compliance with oral hygiene after treatment;

C) the knowledge of the proper cleaning of teeth of adults, the autonomy of rational nutrition, understanding that it is necessary to explain and deliver to children;

g) to change the misconceptions about oral hygiene, treatment of teeth and periodontium diseases.

In the prophylaxis of periodontium diseases, it is important to eliminate professional risk factors, normalize the ecology of the environment, promote general wellness measures aimed at the Prevention of infections and other diseases, increasing the overall fighting capacity of the body.

The following issues of prevention should be considered at the state level:

1. Of the oral cavity in all young gurus among

2. Organization of rational nutrition in workshops.

3. Organization of treatment rooms for hygiene and periodontium diseases.

4. Establishing the production of oral cleaning agents.

Individual prophylaxis (oral hygiene, rational nutrition, healthy lifestyle) should be carried out by the patient himself, and professional prophylaxis (oral counting, professional hygiene, guided orthodontic and orthopedic treatment, treatment of diagnosed periodontium disease, teaching oral hygiene, compliance and monitoring its effectiveness, conducting dental whitening among population) should be carried out by the doctor.

Measures for the Prevention of periodontium diseases are divided into public (1) and individual (2).

Public events in turn:

1.1. By Public Administration (Ministry of Health, Social Security, etc.) public events aimed at improving the health of the population.

1.2. Activities of a public nature aimed at raising the social situation by local governing bodies (city, village).

Individual events in turn:

2.1. Events that each individual holds individually.

2.2. There are events with an Individual appearance, which are held by the doctor-dentist in a dental Polyclinic, department, center or cabinet.

From the age of 18-20, compliance with hygienic rules in the care of the oral cavity in combination with mass events; in order to improve blood circulation in the, periodontium various exercises should be carried out, such as Hydro Massage, exposure to a sharply differentiated temperature.

At the age of 21-40 years, signs of periodontium begin and develop. At this time, the measures to prevent periodontium are considered:

1. Full compliance with oral hygiene, with the use of curative toothpastes.

2. Timely and complete suturing of defects of the tooth and tooth rows.

3. Loss or neutralization of Occupational malignancy.

4. Auto massage, vacuum therapy, Hydro Massage, clectrophoresis and b. receiving treatment.

5. Prevention of osteoporotic progressions in the alveolar tumor (Sa-Mg agents, Calcinol, calcium-D3-Nicomed and b.).

6. Electro, hypnosis and cases of hypertensive disease and irritability. prevention with.

And in patients over 40 years of age, it is advisable to carry out the following activities:

1. Oral hygiene.

2. Rationalization of work, rest, nutrition plan.

3. Balanced nutrition with antisclerotic orientation.

4. Oral cavity in the regular ravine.

5. Rational orthopedic treatment measures.

6. Eliminate hidden vitamin deficiency.

7. Elimination of periodontium tissue and hypoxia in the body.

8. Constant examination in a dentist-parodontologist.

One of the most necessary health care work is the Prevention of diseases - the implementation of prevention.

The easiest, executable task for the Prevention of dental, including periodontium diseases, maintaining the health of the human body is to fulfill the rules of oral cleanliness, to prevent dental and periodontium diseases.

The first to attach great importance to oral hygiene are Abul Kasim (916-1013), Abu Ali Ibn Sino (980-1037), the German I Aizenberg (1661-1729) from Europeans, and others.

YU.A. Fedorov, V.M. Koren (1973) have proven that people who follow oral hygiene rules have slightly fewer cases of periodontium (30.4%), and more than 48.5% in non-compliance. Oral harmony is the salivation in it, the health of the mucous membrane, the flatness of the tooth rows, completeness, the moderation of the oral corridor, the normality of the the specificity of the diet and the connection with the character of meals.

Improperly placed fillings, sharp edges of toothpicks, improperly prepared artificial dental prostheses, orthodontic apparatus and devices cause food clogging and rotting, mucosal injury and the connection with it to the origin of various irregularities.

In later years, periodontium in addition to decreasing organism reactivity in the origin of slime mold diseases, places more importance on local adverse effects (tooth decay, tartar, microbes and metabolic products, factors that injure various epithelium).

Goal from hygienic procedures in periodontium disease:

1. Prevention and treatment of gingivitis.
2. To create conditions for regenerative processes occurring in the periodontium tissue.
3. Reducing microbial exposure to periodontium tissue in orthopedic, orthodontic treatment.
4. Correction of disorders-aesthetic treatment.

Taking into account this, taking into account the specific and appropriate preventive measures for each patient, conducting, making preventive plans taking into account the general diseases of the patient's body, the doctor is one of the main tasks of the dentist.

Prevention of inflammatory diseases of the periodontium is dominated by oral hygiene, in particular the elimination of dental caraches. Various medikamentosis agents are used to weaken the speed and aggressive effects of tooth decay.

Dispensarization for the treatment of long-lasting periodontium diseases with chronic relapse - taking into account, from time to time it is necessary to carry out measures for the treatment of diseases of the

oral cavity organs, Kerak, internal organs, while conducting various clinical and laboratory tests for these patients.

So, dentists-therapists, orthopedists, children's dentists, as well as doctors of other specialties participate in the provision of comprehensive periodontological assistance. Polyclinics should have periodontological rooms and work in them dentists - periodontologists with certain special knowledge. Their main duties were to provide E.V. Borovsky et al. (1998) defines as follows:

1. Periodontological assistance should be provided at a high level.
2. Choosing the necessary medication for treatment in consultation with Doctor-therapists.
3. Organization of methodological work.
4. Putting the dispensary account on the road.
5. To incorporate science and practice innovations into the treatment of diseases.
6. New work procedures and improvement of treatment preventive work.

In the periodontological room, the dentist must have equipment, tartar, an ultrasound machine for removing caraches, a vacuum-apparatus (Kulajenko's apparatus), instruments designed to take tartar, kayakers for performing surgical procedures, scalpels, curettes, excavators, various buoys for detecting dental caraches (indexacia) (fuchsin bone, Lugol solution for Schiller-Pisarev fracture), containers for cytological, bacteriological examinations, devices, etc.

In polyclinics, there will be hygienic rooms, and there the patient who comes to seek help will be familiarized with the rules of dental cleaning, and in the hydrotherapy room, using a special device, the teeth, oral cavity will be washed with water supplied at a pressure of 1 - 2 atmospheres (various khushbuy - aromatic liquids can be mixed into it). It also acts as a hydromassage at the same time.

By dispensary, we must understand that certain diseases - long-lasting, diseases that cause various complications during their course (flowering-caries disease, a huge number of types of diseases of the mucous membranes of the mouth), including a patient with periodontium diseases, open special account books, put special marks "R" in the history of the disease, invite them from time to time,

X. LEVEL OF ORAL HYGIENE.

Methods for examining patients with Periodontal disease

Clinical pocket (pathological dentoalveolar pocket) - a condition in which the dimensions of the dentoalveolar pocket are slightly deepened (more than 2 mm) than usual, but the integrity of the tissues that make it up is not disturbed.

The Periodontal pocket is formed by a partial violation of the integrity of the tissues that make up the periodontal. The depth of such a pocket is much deeper than the norm. If the destruction of bone tissue is recorded in the alveoli, it is also known as "bone tin".

Special examination methods are used to determine the Schiller-Pisarev fracture, radiological, capillary-microscopic, functional examination methods, special indices.

The Schiller-Pisarev method shows the separation of glycogen in living tissues (based on the iodine-starch reaction). It is known that during the inflammatory process, a large amount of glycogen accumulates in the cells of the mucous membrane. with this method, we determine the presence of an inflammatory process on the mucous membrane of the gums, if any, its level. Schiller-Pisarev solution consists of 1 g of iodine crystals, 2 g of potassium iodide salt and 40 ml of distilled water. The specified is wrapped in cotton swabs and poured into the mucous membrane of the gums. If the resulting mucous membrane is eaten dark brown, this indicates the presence of an inflammatory process in the tissue. If this method is used after treatment and a positive result is obtained, the course of treatment is continued.

Happy to move. E. E. According to Platonov (1951), the injury of the periodontium is determined. Level 1 cramping - when cramping in the lunj-tongue (palate) or lab-tongue (palate) direction with respect to the adjacent tooth is less than 1mm, Level 2 will result in more than 1mm in the above directions, as well as cramping in the palate-distal direction.

At Level 3, the tooth moves in all directions, the tooth can also bend in the direction of the missing tooth.

Determination of the permeability of Milk capillaries (according to Kulajenko). Based on determining the timing of hematoma formation in milk. Vacuum diameter and pressure are constant. 720-740 mm wire. 20-40 mm on the column. when there is a residual pressure equal to the

wire column, the sterile is placed in the gum and the in the rubber hose is removed, as a result of which the milk is absorbed into the The timing of hematoma formation indicates the degree of stagnation of milk capillaries. In the norm, the frontal part of the jaw is formed at a duration of 50-60 sec, while in other parts it is formed for a relatively longer period.

Vacuum-carrying hematomas show a therapeutic effect by stimulating the enzymatic activity and regeneration of tissues in the field of pathological foci.

The exacerbation of the inflammatory process leads to the formation of several symptoms. Deepening of the periodontal pockets, increased dental flutter, pus discharge, etc. X-ray diagnosis helps in this that is, the depth of the pocket is determined by the presence of a bite and pus.

Indications for dental examination.

Today there are a number of indicators that characterize the condition of the teeth and mucous membranes of the oral cavity. These indicators, collected during mass research by regions, cities, regions and countries, give the data not only a basis for statistics, but also the prevalence, intensity of dental diseases, they do not give an idea of the inconvenience that can "create" for the population. On the basis of this data, preventive measures and treatment measures are being developed. Dentists in this area and their proper distribution by Gcal are in demand. In addition, on the basis of the statistics presented in the testimony, priority areas of dental care are determined.

Periodontal index (PI). In the study of the prevalence and activity of periodontium tissue, R. The periodontium index (PI) produced by Russel (1956) is widely used. The periodontium state in each tooth is determined on a scale of 0 to 8, with a focus on the degree of inflammation of the gums, tooth flutter, and the depth of the periodontal pocket. All points are divided by the number of teeth that have a sum (table). The resulting PI is characterized as follows: 0,1-1-the first stage of the disease; 1,5-4,0-the second stage, characterized by destructive changes; 4,0 - 8,0-the third stage of the disease The papillary-marginal alveolar indicator (PMA) is widely used in dental practice to assess the degree of inflammation of the mucous membrane of the gums and to observe and compare the dynamic changes in this inflammation. Despite the fact that there are ways to identify this indicator in various

modifications, the simple, easily possible method recommended by Ragta (1960) is as follows. With this method, to determine the degree of inflammation of the gums, the mucous membrane of the gums is stained with a protective solution of iodine and potassium iodide proposed by Schiller-Pisarev. This method is based on the reaction of glycogen (starch), a complex substance in the cells of the mucous membrane of the gums, with an iodine substance. In the course of inflammation, the amount of glycogen in the gum mucosa increases.

As a result of the iodine-glycogen reaction, the inflamed area of the mucous membrane becomes brown-brown.

PMA indicator assessment is assessed according to the following code criteria (in points):

0-no inflammation

1-inflammation only in the area of the gums (R)

2-milk has marginal inflammation (M)

The alveolar softening (a) RMA indicator of 3-boron gums is calculated by the formula: $RMA = \frac{\text{sum of points}}{3 \times \text{number of teeth}} \times 100\%$ where the number of teeth is relative to age: 6-11 years-24 teeth 12-14 years-28 teeth 15 and load; ori - 30 teeth

If there are no known teeth in the tooth row, then the sum of the points is divided by the number of teeth in the mouth.

Usually in healthy people. indicator -0. The indicator obtained as a result of the examination the more teeth, the stronger the inflammation of the gums.

PMA indicator evaluation criterion:

The PMA indicator is usually " what percentage of the mucosa of the gums in the oral cavity represents inflammation?"

30% or less-milking mild rate

31 -60% - moderate violence

61% and above-solid gingivitis

Punishment of oral hygiene. Today, the level of cleanliness of the oral cavity is the only indicator of the health of the teeth and periodontal system does not eat a secret. Therefore, from the point of view of preventing oral diseases, it is important to study oral hygiene and improve the level of cleanliness. There are a number of recommendations for assessing oral hygiene. Of these, we will look at some of the simple, convenient and more informative ways of sleeve, sleeve:

Determination of the hygienic index by the Fedorov-Volodkin method (1971).

This indicator is recommended to determine the hygienic condition of the oral cavity in children aged 5-6 years. Determination of the hygienic index by this method 43, 42, 41, 31, 32, 33 the vestibular surface of the teeth is examined. The corridor surface of the teeth is assessed using a special (Schiller-Pisarev, futsin, erythrosin) and the following code and scores of thoughts painted with the existing tooth surface.

Calculated by the following formula:

Blindnin this case, the Blind hygienic index; K_n is the sum of the index of all teeth; n is the number of teeth (6). In the norm, the hygienic index should not exceed 1.

At the same time, a 5-point system is used:

1 point-no dental care-the tooth is unpainted. 2 points-1/4 of the bite is painted.

3 points-1/2 tooth row is painted.

4 points-3/4 of the bite is painted.

5 points-the tooth surface is completely painted or covered with plaque.

The amount of points obtained to determine the hygiene indicator is divided by the number of teeth painted:

Evaluation criterion: indicator level: purity level;

1. 1.1-1.5-good

2. 1.6-2.0-satisfactory

3. 2.1-2.5-unsatisfactory

4. 2.6-3.4-bad

5. 3.5-5.0-judayomon

Assessment of the intensity of tooth decay is determined by a 3-point system: multi-staining of the tooth surface -3, less often -2, non-staining -

1. Calculated by the following formula: S_n/S_{om} in this case, is the hygienic index indicating the abundance of dental caries; S_n is the sum of the index of all teeth; n is the number of teeth (6).

G.N Pakhomov (1974) Fedorov-Volodkina index modifier, increasing the number of teeth to 12:6 1|1 6 6 321|123 6

Measure pocket depth. It is carried out using a special graduated probe and a antifreeze is the safest. The is inserted into the pocket until

there is resistance in the direction of the tooth axis, the place where the scarf is worn is assessed as the bottom of the pocket. When there are bone pockets, the shaft is inserted to the bottom of the pocket.



Now, computer tomography is used to determine the character of a periodontal pocket, especially in the study of pocket topography.

The pocket depth dental formula defines Arabic numerals opposite each tooth, while the degree of crumpling is Roman numerals. Hence, the degree of parodont is determined on the basis of radiographic examinations.

The reduced hygiene index OHI-s (Greene, Vermilion, 1969) studies the Carache of teeth in the socket of 6 adjacent teeth or two of the teeth in the smooth group (incision), (oral surfaces of the upper and lower teeth). If carash covers the tooth crown $1/3$ and less-I; $' / 2-2$; % - 3. -0 points if there is no carash on the surface of the teeth. If the carash is uneven on the surface of the tooth, then the most Place is assessed. In the norm, the hygiene index should not exceed 1, if there are many oral hygiene conditions are assessed as poor.

A Kotzschke fracture is used to examine fluid flowing from the periodontal pocket. The tooth-milk pocket is injected into a solution of the following composition in a cotton turun: 0.5 gr. benzidine, 10 gr. Polyethylene glycol, 15gr. Cefiral (1: 1000) and 3% n2o2 are mixed with each other. According to the amount of pus in the liquid in the pocket, cotton turun is colored green, bluish-green and bluish.

Currently, for epidemiological investigations, the WHO has proposed the index of need for treatment (CPITN - Community

Periodontal Index of treatment Needs). This index helps to accurately select treatment tactics by examining groups that are not numerically large. This takes into account the bleeding of the gums, the presence of tartar, the periodontal pocket and its depth. In periodontology, functional methods of investigations are widely used. Reoparodontography is performed to determine the functional and structural changes in the parodont blood supply system. Regional circulation in Parodont tissues can also be studied using the photoplethysmography method. Parodont microcirculatory flow functionally States it can be detected using biomicroscopy.

Determination of the level of oxygen capacity in tissues is carried out using polarography.

To determine the average level of oral intake for a particular group of people, it is necessary to divide the sum of hygienic indicators of all objects by the number under examination. Then the average level of oral cleanliness is determined for this group. The quality and effectiveness of hygiene products in conducting mass epidemiological examinations of the oral cavity hygienic toilet. In assessing and determining the place of oral hygiene in the occurrence of dental and periodontal diseases, objective indicators that provide information about the mixture and quality of dental appearance are very necessary. Recently, it has been proposed to determine the structure and surface of the nail, through the mathematical calculation of thinking and stones on the teeth in many ways, recognizing the important role of thinking involving the surface of the tooth in the occurrence of dental diseases and periodontal disease.

Of the indicators obtained using the recommended methods, the following are worthy of mention in the comprehensive assessment of the soft and hard layers formed on the tooth: a) to provide information about the surface layers of teeth; b) to provide information about the thickness of the surface layers of teeth; c) to provide information about the chemical, physical and microbiological properties of the surface. The data obtained on these indicators are mainly aimed at studying the structure and properties of plaque. Plaque, as we know, is a colorless look painted with special paints. The board line will be the same as the painted line. Brown Bismarck, fuchsin-based yeritma, red yeritrosin tablet (for chewing), Lugol yeritma are widely used for dyeing dental gums and gum stones. Schiller-Pisarev peanuts are commonly used extensively.

Green Vermilon is an indicator.

This method consists in presenting the hygienic index of the oral cavity in numbers by calculating the number of tooth rows. Green-Vermilon indicator (1964) gently look at the teeth and eat the stones to an individual assessment. To determine the green-Vermilion indicator, 6 tooth surfaces are painted from a number of teeth:

Dental pavement surfaces 16, 11, 26, 31

36, 46 criteria and codes for assessing the tooth surface of teeth. Assessment is carried out by System: 1.- A soft gaze extends 1/3 of the tooth surface to 2.- soft gaze 3, covering less than 1/3 of the tooth surface.- soft-looking toothbrush contains more than 2/3 tooth surface or entire tooth surface.

Tartar assessment criteria and codes: 0-tartar has not been identified. 1-Milkstone spreads (covers) up to 1/3 of the tooth surface. 2-gum stone covered more than 1/3 tooth surface, but less than 2/3. 3-gum Stones cover more than 2/3 of the tooth surface, or completely the entire tooth surface. At the same time, many deposits formed under the gums on the surface of the tooth neck. To calculate the indicator, find the sum of points from all painted surfaces and divide it by the number of painted teeth. The following formula is found by: the sum of the points
Ogievaluation criterion:

a) lung value: oral hygiene level: 0.0-1.2-good 1.3-3.0-satisfactory 3.1-6.0-bad

b) soft appearance and stones indicator 0,0-0,6-good 0,7-1,8-satisfactory 1,9-3,0-a poor indicator of the level of cleanliness of the oral cavity not only provides information about how clean the oral cavity is, but is also used by us to compare the effectiveness of one group of hygiene products with the second group and develop a When performing the above work, an indicator of the effectiveness of oral hygiene helps very well.

Tests:

1. The loss of a periodontal compound is called a condition that is accompanied by the action of the tooth root:

- a) chronic periodontitis;
- b) hypertrophic gingivitis;
- c) marginal descent of the parodontal;
- d) vertical defect of the alveolar bone

2. Is the medical bandage applied to numa?

- a) parodontal pocket separation;
- b) prolong the effect of the drug;
- c) protection of the wound surface from the effects of oral factors;
- d) isolation of pocket microorganisms to prevent infection.

3. Sanation of tooth rows is carried out:

- a) in the main treatment stage;
- b) during surgical treatment;
- c) before the periodontologist begins treatment;
- d) upon completion of surgical treatment

4. Loskut surgical treatment differs from open curettage:

- a) when removing granulation tissue;
- b) apply horizontal cutting, when removing the tape
- c) hypertrophic gum removal;
- d) in the formation of the mucous-Bony loscut.

5. Drugs used for the local treatment of periodontitis, which includes metronidazole:

- a) elizol;
- b) levomekol;
- c) corsodile;
- d) metrogil-Denta.

6. Mark the sequence of medical activities that take place in the periodontitis:

- a) removal of tartar;
- b) purpose of physiotherapy;
- c) preparation of oral hygiene;
- d) curettage of the periodontal pockets.

7. Ultrasound is used to remove dental deposits:

- a) low, not high frequency;
- b) high, not low frequency;
- c) both low and high frequency;
- d) neither high nor low frequency.

8. Using the curettage of the periodontal pocket:

- a) cuts do not apply;
- b) cleaning the gum cover;
- c) the cutting process is carried out on the edge of the gum;
- d) periodontal pocket transmission.

9. The main method of prevention of periodontal inflammatory diseases:

- a) tire teeth with a degree of cramping bo;
- b) curettage of periodontal pockets;
- c) proessional oral hygiene;
- d) local anti-inflammatory therapy.

10. Symptoms of hyperemia in periodontosis:

- a) violation of regional hemodynamics;
- b) structural changes in the vessel wall;
- c) fci of osteosclerosis and osteoporosis in the jaw bones;
- d) errors in conducting a differential diagnosis of the disease.

11. It is recommended when restoring the chewing surface of teeth in patients with periodontological pathology:

- a) removing a tooth from an occlusion to reduce the chewing load;
- b) creating a surface with multiple and uniform occlusive contact;
- c) to create an occlusive surface with multiple contacts to adapt the teeth to the chewing load;
- d) revert the formation of an occlusive surface that restores the anatomy of the tooth to prevent chewing load.

12. Intact parodont contains the gum borozdai

- (a) microbial Association
- b) exudate
- c) gingival fluid
- d) granulatory tissue

13. Gingivitis is a disease

- a) inflammation
- b) inflammatory and dystrophic
- c) dystrophic
- d) swelling

14. In clinical course, gingivitis differ

- a) Acute
- B) chronic
- c) in the acute stage
- d) general

15. Erythrosine solution is used

- a) in determining the hygiene index
- b) to determine the PMA index

- c) control teeth cleaning
- d) to determine the CPITN index

16. Lugol solution is used

- a) determination of the hygiene index
- b) to determine the PMA index
- c) control teeth cleaning
- d) to determine the CPITN index

17. Mild levels of catarrhal gingivitis include inflammation of the gums

- (a) papiller
- b) papillary and marginal
- c) marginal and alveolar
- d) alveolar, marginal and papillary

18. The quantitative strain of catarrhal gingivitis can be determined using an index

- a) PMA
- b) Green Vermilion
- c) parodontal
- d) CPITN

19. Catarrhal gingivitis differentiates

- a) with periodontitis
- b) with fibromatosis
- c) with parodontosis
- d) with herpetic gingivostomatitis

20. Hypertrophic gingivitis tumor form differentiates

- a) with periodontitis
- b) with fibromatosis
- c) with periodontal disease
- d) with herpetic gingivostomatitis

21. The fibrosis form of hypertrophic gingivitis is differentiated

- a) with periodontitis
- b) with fibromatosis
- c) with parodontosis
- d) with herpetic gingivostomatitis

22. The following treatment is carried out in the form of edema of hypertrophic gingivitis

- (a) gingivotomy
- (B) gingivectomy

- c) anti-edema
- d) treatment of loskutli jarrahlik

23. Surgical treatment of hypertrophic gingivitis in the form of fibrosis is carried out

- (a) gingivectomy
- (B) gingivotomy
- c) curettage
- d) treatment of loskutli jarrahlik

24. Clinical manifestations of ulcerative-necrotic gingivitis

- a) a large amount of soft plaque
- b) necrosis between suckers and in the gums
- c) milk hypertrophy
- d) cyanosis of the gum edges

25. Blood analysis tests should be performed in ulcerative necrotic gingivitis

- a) general clinical
- B) biochemistry
- c) for HIV infection
- d) for diabetes

26. Proteolytic enzymes are used

- a) bleeding in the gums
- b) in suppuration
- c) milk necrosis
- d) in milk retraction

27 Method of removing tartar

- a) removal of tartar from the oral surface of the tooth
- b) tooth surface polishing
- c) removal of tartar from the vestibular surface of the tooth
- d) removal of tartar from the spaces between the teeth

28. The multilayered flat epithelium of the Gingival crust consists of

- a) basal layer
- b) hairy layer
- C) granular layer
- d) keratinizing layer

29. Middle-level parodontitis differentiates

- a) with catarrhal gingivitis
- b) with hypertrophic gingivitis

c) with severe degree of parodontitis

d) with parodontosis

30. Alveolar milk is the name of:

a) mucous membrane of the transition layer

b) mucous membrane of the alveolar process

c) mucous membrane of the palate

d) all right

31. The barrier function of the parodont provides:

a) the stagnation of milk

b) large amounts of collagen fibers

c) minimum amount of collagen fibers

d) keratinized epithelium

32. The biological width of the Milk is equal to:

a) 1.71-2.42 mm

b) 2-3 mm

c) 1-1.5 mm

d) 0.3-0.5 mm

33. Indicate the width of the gum connective tissue joint

a) 1-1.07 mm

b) 0.5-0.7 mm

c) 2-3 mm

d) 1.5-2 mm

34. Show the flow of periodontosis diseases.

a) Acute

b) localized

c) general

d) chronic

35. Determination of the depth of the periodontal pocket is carried out:

a) clinical method

b) radiological method

c) histologically

d) all right

36. Parodontalchonts are detected in middle-level parodontosis

a) more than 3mm

b) more than 5mm

c) mre than 6mm

d) no periodontal pockets

37. Examination of a patient with periodontal disease includes:

- a) determination of the depth of the periodontal pockets
- b) determination of tooth mobility
- c) detection of gum recessions
- d) all right

38. Usually, gingival is present on the coat:

- a) gingival fluid
- b) granulated tissue
- c) tartar
- d) food residues

39. Periodontosis:

- a) inflammatory disease
- b) dystrophic disease
- c) tumor
- d) inflammatory and dystrophic

40. Blood analysis tests should be performed in ulcerative necrotic gingivitis

- a) general clinical
- B) biochemistry
- c) for HIV infection
- d) for diabetes

USEFUL LITERATURE

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