MINISTRY OF EDUCATION AND SCIENCE OF UKRAINE
STATE INSTITUTION OF HIGHER EDUCATION
"UZHHOROD NATIONAL UNIVERSITY"
FACULTY OF DENTISTRY
DEPARTMENT OF THERAPEUTIC DENTISTRY

Horzov L.F., Danko E.M.

"THERAPEUTIC DENTISTRY"
Part 1

Educational and methodical textbook
for independent work of fourth-year students
of the dental faculty from therapeutic dentistry

Uzhhorod – 2021

UDC: 616.314.17-008+616.311.2-002(076)

Therapeutic dentistry. Part 1. Educational and methodical textbook for practical classes in therapeutic dentistry for fourth-year students of the dental faculty./ Horzov L.F., Danko E.M. – Uzhhorod; 2021 – 84 p.

Authors:

Horzov L.F. – Ph.D., Associate Professor, head of the Department of Therapeutic Dentistry.

Danko E.M. – Senior Lecturer of the Department of Therapeutic Dentistry.

The study guide is designed for studying the program in the discipline "Therapeutic Dentistry" by 4th year students of the dental faculty. This manual is designed to deepen students' knowledge of the classifications, etiology and pathogenesis of inflammatory and dystrophic periodontal diseases, features of clinical and additional methods of examination of periodontal patients; clinic, treatment and prevention of periodontal diseases.

Adopted by the Scientific Council of the Faculty of Dentistry of the state institution of higher education "UzhNU" Protocol N 6 of 17. 06.2021

Reviewer: MD, prof. Department of Therapeutic Dentistry Kuligina V.M

CONTENT

INTRODUCTION

"Periodontology" is the study of the tooth-supporting tissues, the ***"periodontium."***

The periodontium is made up of those tissues that surround each tooth and which anchor each tooth into the alveolar process (Greek: para = adjacent, odus = tooth). The following soft and hard tissues constitute the structure of the periodontium:

- *Gingiva*
- *Periodontal Ligament*
- *Root Cementum*
- *Alveolar Bone*

Periodontal diseases

There are numerous diseases that affect the periodontium. By far the most important of these are plaque-associated gingivitis (gingival inflammation without attachment loss) and periodontitis (inflammation-associated loss of periodontal supporting tissues).

- **Gingivitis** is limited to the marginal, supracrestal soft tissues. It is manifested clinically by bleeding upon probing of the gingival sulcus, and in more severe cases by erythema and swelling, especially of the interdental papillae (Fig. 3).
- **Periodontitis** can develop from a pre-existing gingivitis in patients with compromised immune status, the presence of risk factors and pro-inflammatory mediators, as well as the presence of a predominately periodontopathic microbial flora. The inflammation of the gingiva may then extend into the deeper structures of the tooth-supporting apparatus. The consequences include destruction of collagen and loss of alveolar bone (attachment loss). The junctional epithelium degenerates into a "pocket" epithelium, which proliferates apically and laterally. A true periodontal pocket forms. Such a pocket is a predilection site and a reservoir for opportunistic, pathogenic bacteria; these bacteria sustain periodontitis and enhance the progression of the disease processes (Fig. 4).

Gingival Recession

Gingival recession is not actually a "disease," but rather an anatomic alteration that is elicited by morphology, improper oral hygiene (aggressive scrubbing), and possibly functional overloading.

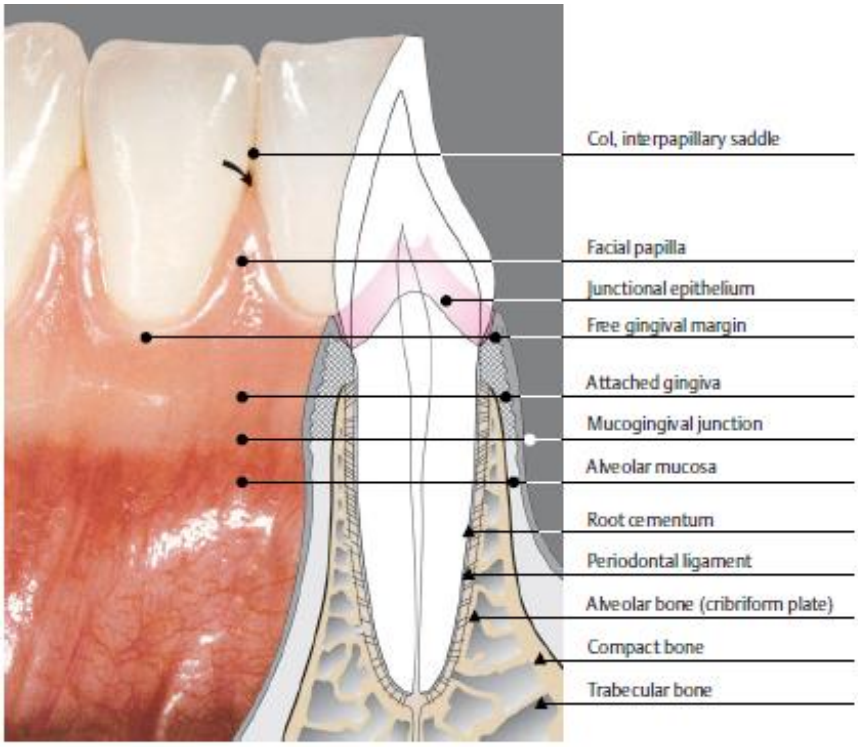
- Teeth are not lost due to classical gingival recession, but patients may experience cervical hypersensitivity and esthetic complications. If gingival recession extends to the mobile oral mucosa, adequate oral hygiene is often no longer possible. Secondary inflammation is the consequence. In addition to classical gingival recession, apical migration of the gingiva is often observed in patients with longstanding, untreated periodontitis, and it may be a consequence of periodontitis therapy in elderly patients.

These three periodontal disorders – gingivitis, periodontitis, gingival recession – are observed world-wide; they affect almost the entire population of the earth to greater or lesser degree. In addition to these common forms of oral pathology, there are many less frequently encountered diseases and defects of the periodontal tissues. All of these diseases were comprehensively classified at an international World Workshop in 1999.

Topic 1. Structure and functions of periodontium.

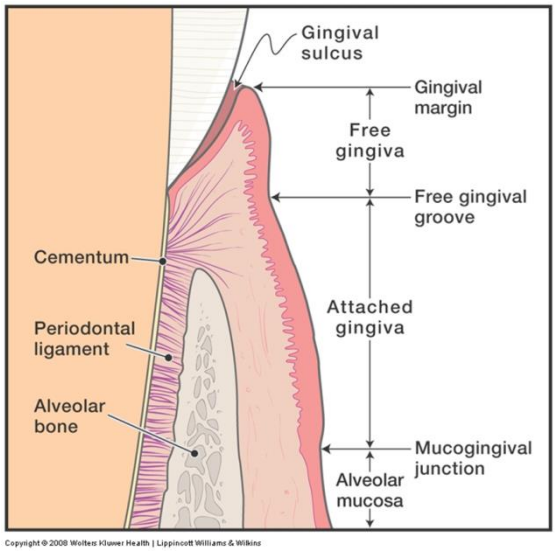
The following soft and hard tissues constitute the structure of the periodontium:

- *Gingiva*
- *Periodontal Ligament*
- *Root Cementum*
- *Alveolar Bone*



Gingiva - is the mucous membrane that covers the alveolar bone of both jaws and teeth in the neck area.

The gingiva is demarcated clinically into the *free marginal gingiva*, which includes *gingival sulcus* (space between the teeth neck and oral sulcular epithelium – Clinical depth: 0.5–3 mm) ca. 1.5 mm wide; *the attached gingiva*, which may be of varying width; *alveolar mucosa membrane* and *the interdental gingiva*. Healthy gingiva is described as "salmon" pink in color; in Blacks (seldom also in Caucasians) the gingiva may exhibit varying degrees of brownish pigmentation. Gingiva exhibits varying consistency and is not mobile upon the underlying bone. The gingival surface is



keratinized and may be firm, thick and deeply stippled ("thick phenotype"), or thin and scarcely stippled ("thin phenotype"; Müller & Eger 1996, Müller et al. 2000).

Junctional Epithelium—Epithelial Attachment— Gingival Sulcus

The marginal gingiva attaches to the tooth surface by means of the junctional epithelium, an attachment that is continuously being renewed throughout life (Schroeder 1992).

The gingiva consists of three tissues:

Junctional epithelium

Oral epithelium

Lamina propria (connective tissue)

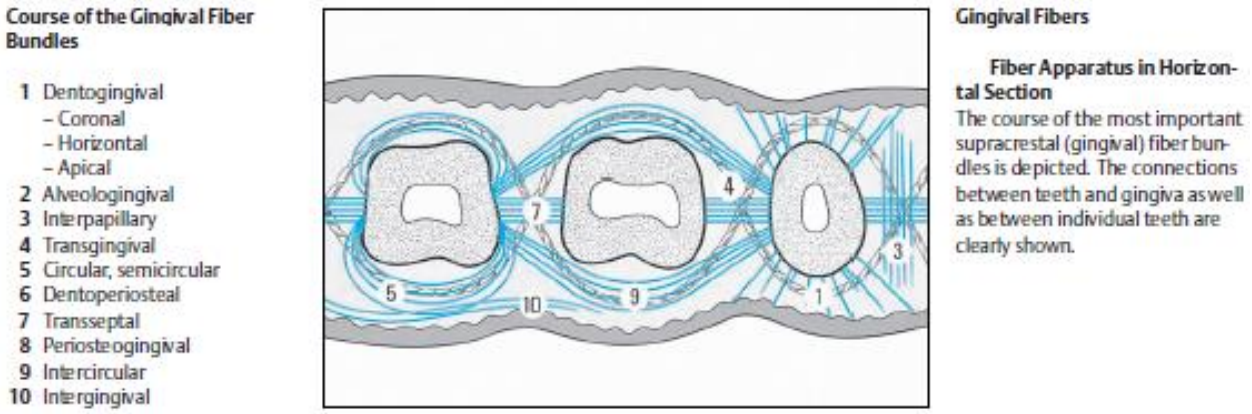
The *junctional epithelium (JE)* assumes a key role in maintenance of periodontal health: It produces the epithelial attachment and therefore creates the firm connection of soft tissue to the tooth surface. It is quite permeable, and thus serves as a pathway for diffusion of the metabolic products of plaque bacteria (toxins, chemotactic agents, antigens, etc.). There is also diffusion in the opposite direction, of host defense substances (serum exudates, antibodies, etc.). Even when the gingivae do not appear inflamed clinically, the JE is constantly transmigrated by polymorphoneuclear leukocytes (PMNs) moving towards the. The red arrows depict the migration of daughter cells from the basal layer toward the gingival sulcus.

Connective Tissue Attachment

The *fibrous connective tissue* structures provide the attachment between teeth (via cementum) and their osseous alveoli, between teeth and gingiva, as well as between each tooth and its neighbor. These structures include:

• Gingival fiber groups

• Periodontal fiber groups (periodontal ligament)



Gingival Fiber Groups

In the supra-alveolar area, collagen fiber bundles course in various directions. These fibers give the gingiva its resiliency and resistance, and attach it onto the tooth surface subjacent to the epithelial attachment. The fibers also provide

resistance to forces and stabilize the individual teeth into a closed segment. The periosteogingival fibers are also a component of the gingival fiber complex. These connect the attached gingiva to the alveolar process.

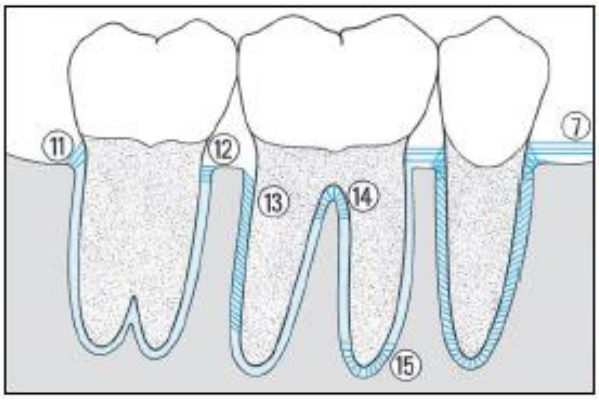
Periodontal Fiber Groups, Periodontal Ligament

The periodontal ligament (PDL) occupies the space between the root surface and the alveolar bone surface. The PDL consists of connective tissue fibers, cells, vasculature, nerves and ground substance. An average of 28,000 fiber bundles insert into each square millimeter of root cementum! The building block of a fiber bundle is the 40–70 nm thick collagen fibril. Many such fibrils in parallel arrangement make up a collagen fiber. Numerous fibers combine to form collagen fiber bundles. These collagen fiber bundles (Sharpey's fibers) insert into the alveolar bone on one end and into cementum at the other (Feneis 1952). The most ubiquitous cells are fibroblasts, which appear as spindleshaped cells with oval nuclei and numerous cytoplasmic processes of varying lengths. These cells are responsible for the synthesis and break-down of collagen ("turnover").

Cells responsible for the hard tissues are the cementoblasts and osteoblasts. Osteoclastic cells are only observed during phases of active bone resorption. Near the cementum layer, within the PDL space, one often observes string-like arrangements of epithelial rest cells of Malassez.

Course of the Periodontal Fiber Bundles

11 Crestal
12 Horizontal
13 Oblique
14 Interradicular
15 Apical



Periodontal Fibers

Fiber Apparatus in Mesiodistal Section
The anchoring of a tooth in the alveolar bone is accomplished via the dentoalveolar fibers of the periodontal ligament (PDL). Occlusal forces are absorbed primarily by the oblique fibers, which course from bone to cementum (**13**). The remaining fiber bundles (**11, 12, 14, 15**) counteract tipping and rotating forces.

Root Cementum

From a purely anatomic standpoint, root cementum is part of the tooth, but also part of the periodontium. Four types of cementum have been identified (Bosshardt & Schroeder 1991, 1992; Bosshardt & Selvig 1997):

Types of Cementum— Structure, Localization and Development

1. **Acellular, Afibrillar Cementum (AAC; red)** AAC is formed at the most cervical enamel border following completion of pre-eruptive enamel maturation, and sometimes also during tooth eruption. It is probably secreted by cementoblasts.

2. Acellular, Extrinsic-fiber Cementum (AEC; green) AEC forms both pre- and post-eruptively. It is secreted by fibroblasts. On the apical portions of the root, it comprises a portion of the mixed-fiber cementum.

3. Cellular, Intrinsic-fiber Cementum (CIC; blue) CIC is formed both pre- and posteruptively. It is synthesized by cementoblasts, but does not contain extrinsic Sharpey's fibers.

4. Cellular, Mixed-fiber Cementum (CMC; orange/green) CMC is formed by both cementoblasts and fibroblasts; it is a combination of cellular intrinsic- fiber cementum and acellular extrinsic-fiber cementum.

AEC and CMC are the most important types of cementum.

Acellular Extrinsic Cementum (AEC)

The AEC is primarily responsible for the anchorage of the tooth in the alveolus. It is found in the cervical third of all deciduous and permanent teeth. The AEC consists of tightly packed and splaying fiber bundles (Sharpey's fibers), which are embedded in the calcified cementum. The collagenous structures of cementum and dentin intertwine with each other during root formation and before calcification. This phenomenon explains the tight connection between these two hard tissues. AEC is the type of cementum that is desired following regenerative periodontal surgical procedures.

Cellular Mixed-fiber Cementum (CMC)

The CMC is also of importance for the anchorage of the tooth in its alveolus. But it is only the acellular extrinsic-fiber cementum portion (AEC) within the mixed cementum, into which the Sharpey's fibers secreted by fibroblasts insert and therefore affix the tooth. CMC is layered vertically but also horizontally to the root surface. The portions secreted by cementoblasts contains high numbers of cementocytes. The CMC is also tightly affixed to the dentin because of the intertwining of the collagen fiber bundles during tooth formation. The CMC "grows" faster than AEC.

Alveolar Process—Alveolar Bone

The *alveolar processes* of the maxilla and the mandible are tooth-dependent structures. They develop with the formation of and during the eruption of the teeth, and they atrophy for the most part after tooth loss. Three structures of the alveolar process may be discriminated:

- ***Alveolar bone proper***
- ***Trabecular bone***
- ***Compact bone***

Compact bone covers and contains the alveolar processes. At the entrance to the alveoli, the alveolar crest, it blends into the cribriform plate, *the alveolar bone proper*, which forms the alveolar wall and is approximately 0.1–0.4 mm thick. It

is perforated with numerous small canals (Volkmann canals) through which vessels as well as nerve fibers enter into and exit the periodontal ligament space. *The trabecular bone* occupies the space between compact bone and alveolar bone proper. The distance between the marginal gingiva and the alveolar crest is referred to as the "biologic width" of 2–3mm.

Blood Supply of the Periodontium

All periodontal tissues, but especially the periodontal ligament, have a copious blood supply even in the healthy state. This is due not only to the high metabolism of this cell- and fiber-rich tissue, but also to the peculiar mechanical/functional demands on the periodontium. Occlusal forces are resisted not only by the periodontal ligament and the alveolar process, but also by means of the tissue fluid and its transfer within the periodontal ligament space (hydraulic pressure distribution, dampening). The most important afferent vessels for the alveolar process and the periodontium are:

• In the maxilla, the anterior and posterior alveolar arteries, the infraorbital artery and the palatine artery

• In the mandible, the mandibular artery, the sublingual artery, the mental artery, and the buccal and facial arteries.

Lymph vessels follow for the most part the blood vascular tree.

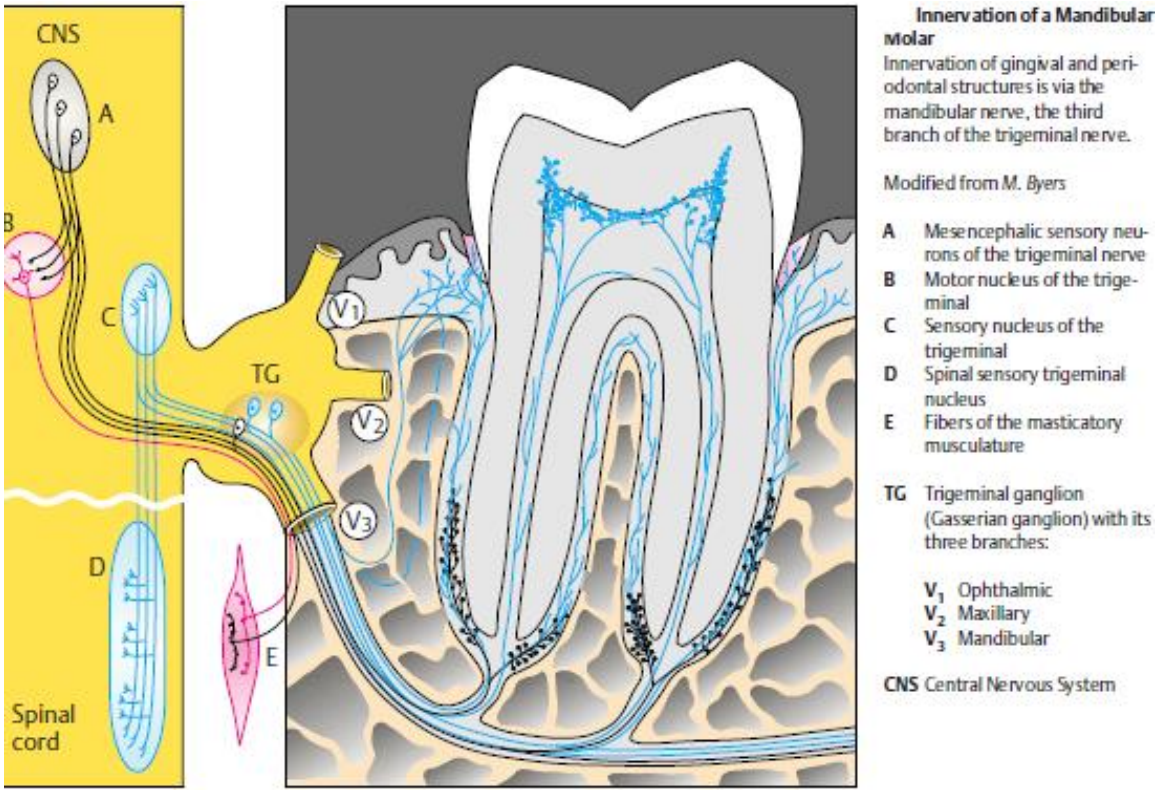
Innervation of the Periodontium

The sensory innervation of the maxilla occurs via the *second branch of the trigeminal nerve*, and that of the mandible via the third branch. The following description of the neural distribution within the periodontal structures is based upon investigations by Byers (1985), Linden et al. (1994) and Byers & Takeyasu (1997).

The periodontium, especially the gingiva and periodontal ligament, contains "Ruffini-like" mechanoreceptors and nociceptive nerve fibers, in addition to the ubiquitous branches of the sympathetic nervous system. The functions of these innervations are coordinated with those of the dental pulp and the dentin.

The stimulus threshold of the mechanoreceptors, which react to tactile (pressure) stimulus, as well as to the stretching of the periodontal ligament fibers, is very low. In contrast, the painsensing nociceptive nerve endings have a relatively high threshold. It is via these two separate afferent systems that "information" about jaw position, tooth movements, speech, tooth contact during swallowing and chewing, minor positional alterations (physiologic tooth mobility), pain during unphysiologic loading, as well as injuries are transmitted. In this way, various mechanoreceptors transmit "conscious reactions" via trigeminal ganglia to the sensory nucleus of the trigeminal in the central nervous system, while unconscious

reflexes transmit to mesencephalic sensory neurons. These various receptors are localized in varying regions of the periodontal structures: At the level of the middle



of the root, one finds more receptors for up-take of "conscious reactions," whereas in the apical region there are more receptors for the unconscious reflexes whose signals transmit to the mesencephalic sensory neurons.

The junctional epithelium as well as the epithelia of the free and attached gingiva, neither of which are vascularized, are served by a dense network of nociceptive and tactile nerve endings. The same is true for the subepithelial, supracrestal gingival connective tissue.

Somatosensory perception in certain gingival diseases (e. g., ulcerative gingivoperiodontitis), as well as pressure and pain sensation during probing of the healthy gingival sulcus or periodontal pockets are the clinical manifestations of the innervation of gingival tissues.

Periodontal functions

- support-holding (periodontal, gum, choroidal prosthesis)
- shock absorbing (fibers of periodontology, vessels - resistance to chewing pressure)
- barrier (epithelium gum - morphological integrity of periodontal tissues)
- trophic (innervation and microcirculation in periodontal tissues)
- reflex (nerve receptors)
- plastic (high regenerative capacity of periodontal tissues)

Topic 2. Classification of periodontal diseases

International classification of gingival and periodontal diseases (ICD – DA, 1994, WHO):

К 05.0 Acute gingivitis
К 05.1 Chronic gingivitis
К 05.10 Simple marginal
К 05.11 Hyperplastic
К 05.12 Ulcerous
К 05.13 Desquamative
К 05.18 Else diseases
К 05.2 Acute periodontitis
К 05.3 Chronic periodontitis
К 05.30 Simple
К 05.31 Complex
К 05.38 Else
К 05.4 Periodontosis
К 05.5 Else
К06.0 Gum recessio

CLASSIFICATION OF PERIODONTAL DISEASES (N.F.DANILEVSKY, 1994)

I. Inflammatory diseases

1. Papillitis, gingivitis

Forms: catarrhal, hypertrophy, necrotizing ulcerous, atrophic
Course: acute, chronic
Deep of injury: soft tissues, osteoporosis of interdental septa
Spreadness: localized, diffuse

2. Localized periodontitis

Forms: catarrhal, hypertrophy, ulcerous, atrophic
Course: acute, chronic
Deep of injury: soft tissues, osteoporosis of interdental septa
Stage of the diseases progression: initial, I, II,III stages
Spreadness: localized

II. Dystrophic-inflammatory diseases

1. Generalized periodontitis

Course: chronic, exacerbation, stabilization
Stage of the diseases progression : initial level, I level, II level, III level
Spreadness: diffuse

2. Periodontosis (Parodontosis)

Course: chronic

Stage of the diseases progression : initial level, I level, II level, III level

Spreadness: diffuse

III. Progressive idiopathic diseases

1. Diseases, induced by blood diseases (leucosis, agranulocytosis, etc.).
2. Histiocytosis X (Letterer-Zive disease, Hend-Shiller-Krischen disease, eosinophylic granuloma).
3. Diseases, induced by substance exchange disorders (PapiylloneLefevre syndrome, Niemman-Pick disease, Gaucher`s disease).
4. Induced by hereditary diseases (Down syndrome, akatalasia, desmodontosis).

IV. Productive processes Parodontomas - non-malignant, malignant.

Topic 3 . Clinical methods of examination of patients with periodontal diseases.

The correct diagnosis of the disease can only be based on thorough examination of the patient. Therefore, mastering the complex methods for evaluation of patients with diseases of oral cavity is the basis for establishing the correct diagnosis and further individual effective treatment. As in all fields of clinical medicine,examination of the patient in therapeutic dentistry is divided into two parts:

1) Taking a history (*subjective examination*), during which the patient provides doctor with all diagnostic information about itself;

2) *Objective examination* (visual examination, palpation, percussion, probing) using basic and extra (laboratory, instrumental) methods.

Subjective examination – included all personal information from patient (anamnesis of the disease/ life, present systemic/chronic diseases).

It includes:

- *QUESTIONS ABOUT COMPLAINS*: Do your have bleeding of the gum? If Yes, how often: sometimes, often, constant, during brushing the tooth, during biting, without any cause? Do your have puss from the gum? Do your have painless in the gum? Do your have mobility of the tooth? Difficult eating with this condition? Do your have predispose to production (formation) plaque and calculus? Change position of the tooth? Have your space between the tooth? "Stay food between the tooth after chewing?

- *QUESTIONS ABOUT HISTORY OF LIFE* q Style of the life q Borne diseases, trauma, surgery q General diseases q Allergic anamnesis q Bad habit`s q Which medicines you take in past and present

Objective examination :

1. ***Extra-oral****:*

- *Examination of the face* - Configuration o Symmetric o Height of down 1/3 part of the face ; Protrude of the chin ; Line connection of the lips ; Size of the mouth slit o Severity of wrinkles ; Condition angle of the mouth; The presence of physiological rest of the mandible Condition of the skin; Condition of the eyes.
- *Palpation Lymphatic nodes* - Size; Consistence ; Connection with skin; Solidary(united) between nodes; Painless Region.
- *TMJ clinical examination*

2. ***Intra-oral:***

- ***Examination of vestibule of oral cavity***. *Vestibule of oral cavity*—the region between the lips and cheeks and the teeth. The fold of tissue created by the vestibule between the lip and teeth is called the vestibular or mucolabial

(mucobuccal) fold. *Occlusion* is examined when dental arches is closed. There are *physiological occlusion* (such condition when person can normally bite, eat and articulate) and *pathological occlusion* that do harm in patients feeding and articulating. *The gingiva* are examined most easily with the mouth partially closed and the lips retracted with the fingers, a tongue blade, or plastic lip retractors. The free gingiva is soft, slight in pink color without any pathological changes. The *attached gingiva* is keratinized and appears paler than other oral mucosa. This tissue usually is firm, stippled, and firmly attached to the underlying bone. The *alveolar mucosa* extends from the attached gingiva to the vestibule. In contrast to the attached gingivae, alveolar mucosa is not keratinized and is darker in color. *The linea alba* is a horizontal ridge (often hyperkeratinized) that is located bilaterally on the buccal mucosa at the level of the interdigitation of the teeth (Fig. 39). The orifice of the Stensen duct is superior to the linea alba, adjacent to the maxillary 6-year molars. Gentle palpation of the parotid gland results in the expression of serous saliva from the duct. Saliva should be able to be expressed from the duct. *Form and attachment frenulum of the lips* - Normal - a thin triangular fold of mucous with a broad base on the lip, which ends in the midline to 5 mm from the edge of the gum - Short (strong) - mounted on top of the interdental papilla - Medium - fixed at a distance 1-5 mm from the top of the interdental papilla - Weak - fixed in transitional fold

- ***Examination of oral cavity itself.*** *Tongue examination. Floor of the oral cavity* should be examined thoroughly and palpated bimanually to check for any abnormalities. Lateral border of the tongue and floor of the oral cavity are the favorite areas for development of oral cancer. The *sublingual salivary glands* can be palpated laterally and frequently will be seen to bulge into the floor of mouth. The main sublingual duct joints the submandibular (Wharton) duct to empty into the oral cavity at the sublingual papilla near the base of the *lingual frenulum*. Direct visual inspection of the *hard palate* is accomplished most easily with the use of an intraoral mirror. The hard palate, similar to the attached gingiva, normally is less pink than other oral mucosal sites because of its increased keratinization. In contrast to the hard palate, the *soft palate* is nonkeratinized and salmon-pink in color. It is easily visible on direct examination by depressing the posterior tongue with a tongue blade and instructing the patient to say "Ahhh." *Examination of the teeth* should be the final part of the oral examination.

Investigation periodontal status

Colour, shape, size, consistency of the gum; Characteristic line of gingival margin; Condition dento-gingival junction; Character and contain of pocket; Present bleeding of the gum; Reaction gum on palpation; Index estimate condition of periodontal tissues

Structure changes in region of gingival sulcus(GS)

Gingival pocket (falce) – space between tooth and gingiva, which appear through edema or hypertrophy gingiva without loose of connection epithelium

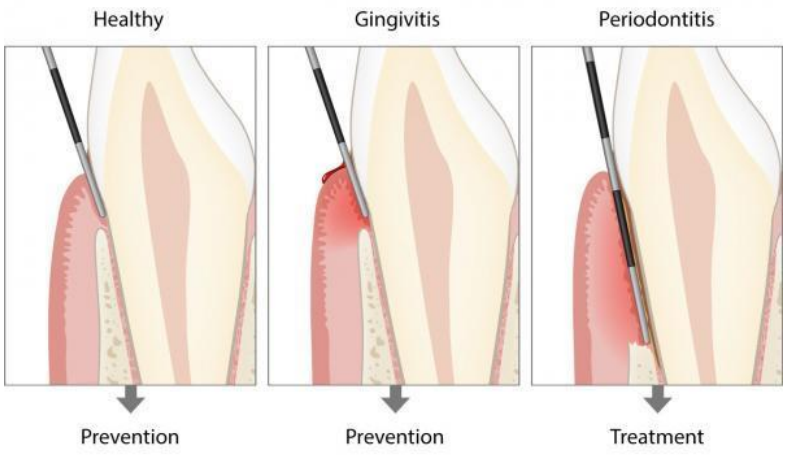
Periodontal pocket (true) – space between tooth and gingiva, which appear through destruction all periodontal tissue with loose of connection epithelium, apical migration CE and his transformation in epithelium of periodontal pocket SUPRABONE By horizontal destruction of the bone INTRABONE (BONE) By vertical, angular resorption of the bone. Checking of periodontal pocket is used by method – periodontal probing.

Diagnostic lesion of furcation

Furcation – divided region roots of multirooted tooth. Probe for diagnostic furcation, cavity, groove - Hu-Friedy; Furcation probe (Naberes, Hu-Friedy) with mark 3, 6, 9, 12 mm.

Depth of pocket (DP) – distance between gingival margin and point, where stop apex of probe (nonprecision measurement).

Clinical level of connection – distance between cemento-enamel border and point where stop apex of probe (precision measurement).



Gum recession – distance lower cementoenamel border and margin of the gum *Classification recession (Miller, 1985) I class* – narrow and widening localized, limitation on vestibular surface by

mucogingival line without violation interdental papilla; *II class* – narrow and widening localized, limitation on vestibular surface with lesion movement mucosa without violation interdental papilla; *III class* –widening with lesion movement mucosa with viollation interdentall papilla; *IV class* –loose hard and soft tissue by perimeter of the tooth.

Diagnostic mobility of the tooth *I DEGREE* – tooth movement in vestibulo-oral direct; *II DEGREE* - tooth movement in vestibulo-oral and mesio-distal direct; *III DEGREE* – tooth movement in vestibulo-oral, mesio Diagnostic mobility of the tooth with periotester.

Topic 4. Index assessment of the condition of periodontal tissues and oral hygiene.

- ***Diagnostic gum bleeding (index Muhllemann, Son, 1971 in modification Kouel, 1975)***

Probing gingiva periodontal probe in region 1.6 1.2 2.4 4.4 3.2 3.6 (tooth of Ramford`s) *0* – bleeding absent during probing; *1* –bleeding present after 30 seconds; *2* – bleeding present after probe or during 30 second; *3* – bleeding during action by air stream.

- ***Diagnostic papilla bleeding (Index PBI)***

Probing gingival sulcus all tooth with periodontal probe from base of papilla till apex. Estimation – 20-30 seconds after probing Index PBI = Number of bleeding= amount all degree /quantity papilla of examination.

Diagnostic papilla bleeding (Index PBI) 1 - One bleeding point; 2- Thin bleeding line or some bleeding points; 3 - Interdental triangle fill in blood; 4 - Strong bleeding after probing

Index estimation of periodontal tissue Hygienic index

- Index of Fedorov-Volodkin(1970)
- Index of Green-Vermillion (1964)
- Gingival index
- Test of Shiller-Pisarev (glicogenic test)
- Index of gingivatis of Silness-Loe(1964)
- PMA index in modification of Parma (1960)

Periodontal index

- Periodontal index on Russel (1956)
- CPITN (1982)
- Complex periodontal index on P.A. Leus (1987)

- ***Hygienic index (HI) by Fyodorov – Volodkina (1970)***.

As a hygienic cleaning test of teeth utilize coloring of vestibula surface of six lower frontal teeth by Shiller-Pisarev solution (1 g crystalline iodine, 2 g potassium iodide, 40 ml distilled water) or other iodinated solution. Than plaque is acquired deep-brown color. Criteria of estimation, marks (quantitative estimation): *1 mark* - absence of tooth crown colouring; *2 marks* - ¼ of tooth crown colouring; *3 marks* - ½ of tooth crown colouring; *4 marks* - ¾ of tooth crown colouring; *5 marks*- colouring of all tooth surface.

Calculate the formula:

$$HI = \frac{\text{sum of 6 scores;}}{6}$$

Interpretations of results :
1,1 – 1,5 mark - good HI;
1,6 – 2,0 marks - satisfactory HI;
2,1– 2,5 marks - unsatisfactory HI;
2,6 – 3,4 marks - bad;
3,5 - 5,0 marks - very bad

- ***OHI-S Green-Vermillion (1964)***

Consists of teeth plaque index (DI-S) and calculus and tartar index (CI-S) For determination of HI examinated vestibular surfaces 16, 11, 26, 31 and lingual surfaces 36,46 after painting them by Shiller-Pisarev solution.

Calculation formula:

$$\mathbf{HI} = \frac{\text{sum of dental debris scores}}{\text{Sum of surfaces}} + \frac{\text{sum of dental calculus scores}}{\text{sum of surfaces}}$$

Results` interpretation:

0 to 0,6 scores	low	good oral hygiene level
0,7 – 1,6	middle	satisfactory
1,7 – 2,5	high	unsatisfactory
2,6 and more	highest	bad

Evaluation scale:

Scores	Dental debris (Debris-index)	Calculus – index (CI)
0	Debris is absent.	Calculus is absent.
1	Dental debris covering 1/3 surface of tooth crown	Supragingival dental calculus covering 1/3 surface of tooth crown.
2	Dental debris covering 2/3 surface of tooth crown	Supragingival dental calculus covering 2/3 surface of tooth crown or subgingival dental calculus like separate conglomerates.
3	Dental debris covering more than 2/3 surface of tooth crown	Supragingival dental calculus covering more than 2/3 surface of tooth crown or subgingival dental calculus covering the cervical part of tooth crown.

Gingival indexes

- ***Papillary-marginal-alveolar index (PMA) by Parma modification (1960)*** Estimation of gum state near each toot.

Estimation criteria, marks : *1 point* - inflammation of gingival papilla (P); *2 points* - inflammation of marginal gum (M); *3 points* - inflammation of alveolar gum (A) Estimation formula:

$$\boldsymbol{PMA} = \frac{\text{Sum of marks of each tooth}}{3 \ * \ \text{number of checking teeth}} * 100$$

Criteria of index PMA estimation: *to 25%* - easy-level gingivitis; *25-50%* - middle-level gingivitis; more than *51%*- hard-level gingivitis

- ***Schiller-Pisarev test:***

Glycogen: accumulates in clear at inflammation
Solution: 1 g of iodine + 1 g of potassium iodide + 40 ml distilled water
Color of gums (color): - yellow
- light brown
- brown

Color	**Sample**	**Score**
absent	negative	1
yellow	weakly positive	2
brown	positive	3

- ***CPITN index (1982)***

For estimation index of need treatment the diseases of periodontium must examination tissue around the 10 tooth (17,16,11, 26, 27, 31, 36, 37, 46, 47). During examination periodontal tissues 17 and 16, 26 and 27, 36 and 37, 46 and 47 get the most hard situation.

Topic 5. Functional methods of examination of patients with periodontal diseases.

1. **Test of Kulazhenko** - is the definition the rate of hematoma formation on the mucous membrane at the action of negative pressure, which is determined by special device for vacuum treatment of periodontitis - ALP-02
2. **Capillaroscopy, biomicroscopy** - lifetime study of the capillary network of the periodontium and the mucous membrane of the oral cavity with capillaroscope, biomicroscope
3. **Rheoparodontography** – bloodless lifetime method research blood supply and blood circulation of vital tissues organism based on the registration of pulse oscillations of electrical resistance of tissues
4. **Photoplethysmography** - study of the state of blood circulation in periodontal tissues,
based on the registration of the optical density of its tissues reflective.
5. **Polarography** - determination of oxygen balance in periodontal tissues.
6. **Echoosteometry** - this method is based on bone conduction fabrics

Test for Kulazhenko: interpretation of results normally, in the frontal area of the gums hematoma occurs in 50-60 s, in the lateral areas - for 70-80 seconds. With periodontitis, the time of hematoma formation decreases3-5 times depending on the severity and course of the disease.

Application of polarography: Analysis of the polarogram allows to determine the rate of oxygen uptakeperiodontal tissues, the state of microcirculation and transcapillary metabolism. Thisthe method is used both for diagnosis and in the dynamics of treatmentperiodontitis

Topic 6. X-ray diagnosis of periodontal disease.

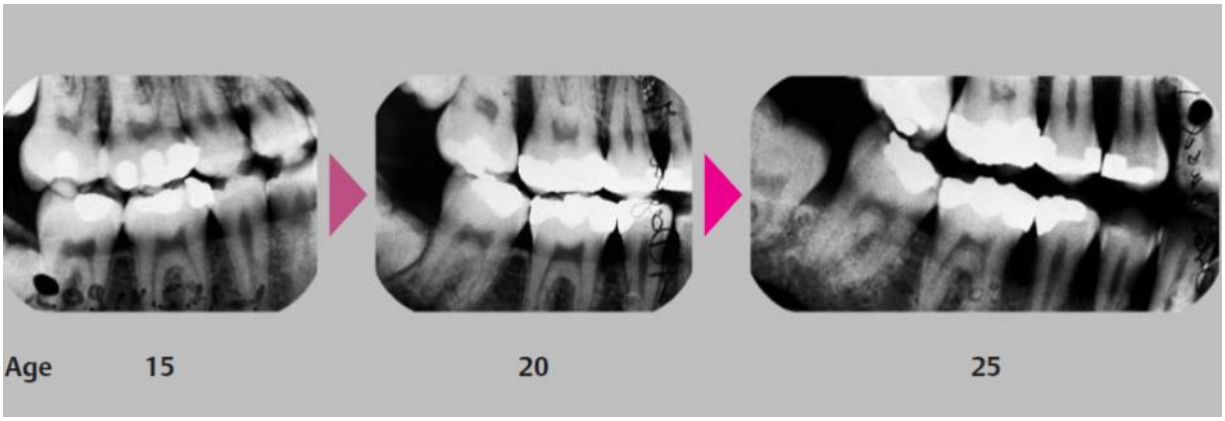
The clinical data described thus far must be supplemented by a radiographic examination. Comparative studies have shown that clinical measurement of probing depths and attachment loss does not always provide a complete and precise picture of the periodontal destruction. On the other hand, a diagnosis of periodontitis should never be made solely on the basis of radiographic findings. A radiograph depicts only a two-dimensional alteration of the interproximal bone. Additional changes in the hard tissues (caries, endodontic complications, reconstructive considerations) will be critical during treatment planning.

Intra- and Extraoral Radiographic Techniques

• **Screening**—**Panoramic radiography**: The slightly magnified film can provide a good overview of the dental structures, and often clarify incidental observations.

• **Single film survey**: The panoramic film cannot yet completely replace the classical radiographic survey, especially in complex cases. For the optimum depiction of periodontal structures, the parallel, right-angle technique, using the long cone is recommended.

Horizontal Bitewing Film

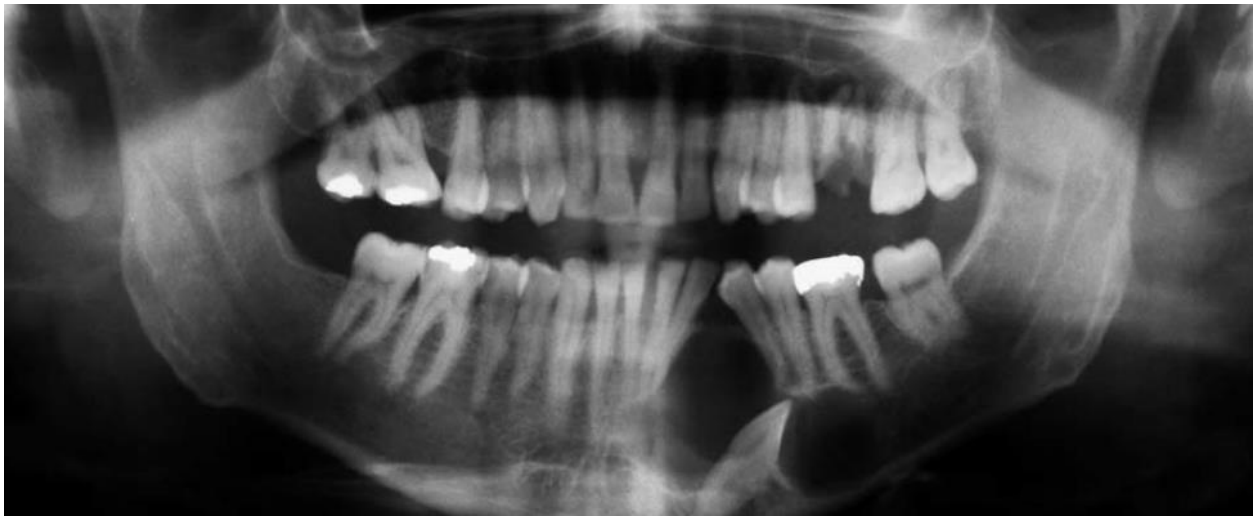


Dependent upon caries activity, bitewing radiographs are indicated in children and adolescents every 1–2 years. The case depicted here shows a 15-year-old patient with scarcely detectable juvenile periodontitis (LJP; new: type III A), but at 20 years of age revealed (local) “visible” periodontitis, and at age 25 a generalized periodontal problem. Regular use of a periodontal probe could have prevented this circumstance!

Vertical Bitewin Radiograph

In most periodontitis patients, four radiographs of this type can provide a satisfactory overview of the interdental bony defects (including caries and calculus accumulation) in the posterior segments. In addition, the above-described initial juvenile periodontitis, and above all the more severe and advanced types of bone loss can be depicted with vertical bitewing radiographs.

Panoramic Radiograph— Incidental Findings



Even though the diagnostic sharpness of individual periapical radiographs cannot be achieved, the panoramic radiograph is an excellent screening mechanism, especially in patients with a severe gag reflex. Frequently, unanticipated anatomic circumstances are detected.

Radiographic View of Pathologic Alterations and Their Causes

Distribution and localization of periodontal bone loss, the hard tissue defect:

- Changes in the entire dentition
- On individual teeth (septa) and root surfaces.

Type of bone loss:

- Demineralization (maintained matrix, reversible)
- Resorption at the alveolar margin
- Horizontal bone loss (with narrow septa)
- Vertical bone loss (with wide septa; Fig.195)
- Vertical cup-like bone loss.

Extent of destruction:

- Distance from bone to the CEJ
- Furcation involvement

- Remaining attachment in relation to root length.

Etiology of destruction:

- Supra- and subgingival calculus, iatrogenic irritants
- Tooth (root) form and position (niches).

Radiographic Survey for Periodontal Diagnosis

In a complete dentition, a minimum of 14 periapical radiographs is necessary to depict all interdental and interradicular septa. The 14-film series should be enhanced by two or four bitewing films in patients with multiple restorations or crowns in order to ascertain any iatrogenic problems.

Topic 7. Laboratory methods of examination of periodontal patients

Laboratory methods of examination of periodontal patients playimportant role in the stages of disease diagnosis and establishment of concomitantpathology.

Clinical blood tests are a mandatory method examination of patients with periodontal diseases. Using clinical blood test dentist may detect signs of blood diseases or other systemic somatic diseases with manifestations of periodontitis, which requires further, the pain is deeper and more thorough examination of the patient. Clinical blood tests may indicate an exacerbation chronic process in the periodontium (increase in the number of leukocytes, neutrophilic "shift to the left", acceleration of erythrocyte sedimentation rate and other); detect signs of blood diseases or other systemic somatic diseases with manifestations of periodontitis, which requires deeper and more thorough examination of the patient. Also, if surgery is planned on periodontal tissues (open curettage, gingivectomy, patch surgery and other), it is necessary to determine the time of bleeding and the ability to clot blood, thrombin time, platelet count and more.

Patients with signs of diabetes mellitus (dry mouth, progressive mobility of teeth, periodontal abscesses that) must be prescribed ***blood test for glucose***. If the indicator exceeds the norm (3.33-5.55mmol) the patient should consult an endocrinologist. In order to determine the cause of the disease and identify various common diseases and pathological conditions of the body, which are common accompanied by changes in blood and urine parameters of the dentist directs to biochemical research .To determine the available microflora of periodontal pockets, its type, pathogenic in all patients with periodontal pathology is necessary to carry out microbiological research, especially for the purpose of definition sensitivity to antibacterial drugs.

There are several ways to collect material for research:

1. **Smear-imprint**. Dehydrated with ethyl alcohol dry slideapplied to the surface of the SOPR, which is investigated and easypressed. Then the glass is air dried, fixed and painted,using the methods of Romanovsky-Gimza, Papanikolaou or others.

2. ***Smear-reprint*** is used when the elements of the lesion of mucosa are located in places inaccessible to obtain a direct fingerprint.In this case, a sterile student rubber band is first applied to the surface pathological area, and then by contact the imprint is transferred to degreased slide .

3. **Smear-scraping** . The contents of the surface of the affected element are taken trowel, spatula, curette or cotton swab and a thin uniform the layer is applied to a degreased dry glass slide.

4. **Rinse.** Among the cytological methods of research in generalized periodontal tissue diseases determine the migration of leukocytes into the mouth cavity and the number of epithelial cells. This test was in the clinic for the first time proposed by MA Yasinovsky in 1931 to determine the degree reactivity of the elements of the reticuloendothelial system. Migration of neutrophilic leukocytes through the gums papillae and oral mucosa indicate their participation in the process phagocytosis. Migrated leukocytes perform the functions of macrophages, taking part in non-specific protection of an organism. To determine Yasinovsky's test in Senatorova's modification rinse the mouth with 10 ml of saline (0.9%NaCl solution) 8 times.

5. **Histological examination** pathological focus of biopsy - lifetime tissue collection. The biopsy is performed in complex cases of differential diagnosis in the presence of hypertrophy periodontal tissue, etc. In this case, following the rules of asepsis and antiseptics, under local anesthesia, cut a piece with a scalpel tissues 3-5 mm in size with submucosal base and underlying tissues. It should be emphasized that biopsies must include clinically no altered periodontal tissue. Biopsy material is made into a test tube with fixing solution (10% solution of neutral formalin or ethylalcohol) and sent to the histopathological laboratory. In the direction be sure to indicate the date of the biopsy, passport and anamnesticpatient data, results of objective examination and preliminary diagnosis.

6. **The microbiological method** of research is carried out for definition the nature and type of available microflora from periodontal pockets, as well as for determination of its sensitivity to antibiotics and other drugs. Method of carrying out: immediately before taking the material the mouth should be rinse with warm water, clean the surface of the element of damage (erosion, ulcers and others) with a sterile gauze swab. Material for research take a fried and cooled microbiological loop or sterile dental spatula, then sow it on special nutrient media or sent to the laboratory. You need to know the general rules when picking up material for microbiological research:- material is taken on an empty stomach;- no drugs are used before taking the material;- before this manipulation the patient should not brush his teeth;- the material must be taken with sterile cotton wool or microbiological loop from the depth of the periodontal pocket;- the taken material should be sent immediately to the laboratory (sometimes sowing on special media is carried out directly in the dental office).

Topic 8. Errors and complications in the diagnosis of periodontal disease.

Diagnosis - a branch of clinical medicine that studies the content, methods and successive stages of the disease recognition process. In the narrow sense - it is a process of disease recognition and assessment of individual biological and social characteristics of the patient, including targeted medical survey, analysis of the results and their generalization in the form diagnosed. Diagnosis includes three interrelated sections: semiotics, methods of examination of the patient and methodological bases of diagnostics.

Semiotics - the study of the symptoms of the disease and their diagnostic value. A symptom is any sign of a disease that is recognizable. Every the disease is characterized by the presence of individual symptoms. Depending on method of detection of symptoms are divided into subjective and objective, from localizations - general and local, from the degree of manifestation - explicit and implicit. By diagnostic significance of symptoms is divided into nonspecific, specific and pathognomonic (characteristic of only one disease). For diagnoses are also used syndromes – pathogenetically due to a set of symptoms that reflect the overall picture disease.

Methods are used to determine the individual symptoms of the disease diagnostic examination. Applying the methodological basis of diagnosis, the doctor conducts analysis and synthesis of data obtained using basic (clinical) and additional (paraclinical) research methods, establishes the diagnosis disease in a particular patient and prescribes appropriate treatment.

A diagnostic error is considered to be the action in which the disease the patient was not recognized under the actual preconditions for it recognition. There are objective and subjective diagnostic reasons errors.

The objective causes of diagnostic errors include:

- constant changes, development of basic principles of theoretical and clinical medicine and periodontology as its section, namely the change of views on etiology, pathogenesis and treatment of diseases; imperfection nomenclature and classification of periodontal diseases;
- lack of the necessary technical diagnostic base, low quality of methods research, insufficient organization of medical and diagnostic process in a medical institution;

- the complexity of clinical manifestations, features of the disease, individual characteristics of the patient.

The subjective causes of diagnostic errors include:

- poor knowledge of the doctor of modern diagnostic methods, incorrect interpretation of research data, low level of medical thinking (doctor's qualification, ability to train a doctor, accumulation practical experience in diagnosis, improving its theoretical and practical information base; personal characteristics of the doctor - indecision or self-confidence);
- increase of means and methods of diagnostic research, their complication; reassessment of the value and informativeness of any diagnostic method, namely instrumental or hardware;
- narrow specialization of doctors.

Diagnostic errors are possible in each link of the diagnostic process:

- errors in collecting complaints and finding out the anamnesis of life and disease (possible influence of any factors on the nature and course of inflammation process in periodontal tissues, lack of generalization of anamnesis data);
- errors in drawing up a plan of clinical and laboratory examination, conducting it and interpreting the results:

a) latent symptoms of the disease, not fully detected in application of additional methods of inspection, atypical course, prolonged asymptomatic course;

b) when determining the depth of the gingival and periodontal pockets factors that affect the accuracy of the measurement are the gingival teeth deposits, overhanging edges of seals, the presence of a carious cavity in cervical region, uneven pocket depth, insufficient the inclination of the probe with a significant convexity of the crown of the tooth;

c) no panoramic radiography or orthopantomography has been performed, incorrect assessment of radiographic data, uncritical attitude to conclusion of radiologists;

d) no general clinical laboratory research methods are performed;

e) doctors of other profiles (therapist, endocrinologist, etc.), and is possible as an underestimation of conclusions consultant and revaluation.

Errors in diagnosis:

- non-compliance with the generally accepted classification of periodontal diseases.

Errors in the differential diagnosis:

- out-of-syndrome;
- intra-syndrome (degree of disease development): initial, I, II, III;

Errors in determining the final clinical diagnosis:

- underlying disease;
- concomitant disease.

The main result of the diagnostic process of diagnosis and prognosis of the disease. Periodontal diagnosis should be developed, because it serves as a basis for the doctor to develop strategies and tactics of action aimed at improving the patient's condition and treatment.

In periodontology, the doctor should use a synthetic diagnosis, which, in contrast to the diagnosis of the disease, includes information about a particular patient, and in contrast to the patient's diagnosis, has a clear scheme of construction.

Topic 9. Etiology and pathogenesis of periodontal diseases. Local disease-causing factors, mechanism of their influence.

The most common diseases of the tooth-supporting apparatus are plaque-induced, usually chronic, inflammatory alterations in the gingiva and the subjacent periodontal structures.

Gingivitis may persist for many years without progressing to periodontitis. With good oral hygiene and effective professional removal of plaque and calculus, gingivitis is completely reversible.

Periodontitis usually develops out of a more or less pronounced gingivitis. Periodontitis is only partially reversible (see periodontal healing, p. 205/regenerative therapies).

The reasons why gingivitis develops into periodontitis (or does not) are still incompletely understood. As with all infections, it appears that the proliferation of pathogenic microorganisms, their toxic potency, their capacities to invade tissues, and above all the individual host response to such infections are the determining factors.

Etiology of Periodontitis— Interaction between Dental Plaque and the Host

Bacteria

1 The primary etiologic factor for the existence of periodontitis is pathogenic microorganisms within the subgingival biofilm.

Host

2 The genetically determined non-specific and specific immune responses, as well as systemic syndromes and diseases influence the existence and the clinical course of periodontitis.

3 “Habits” and the patient’s own approach to general health will influence plaque formation and host immune response, both systemically and particularly with regard to oral health.

4 Social circumstances influence the systemic and psychic well being of the patient. Problems in the socioeconomic arena lead to negative stress.

5 Psychic burdens and stress influence the immune status.

Dental Plaque—Development

Within minutes after completely cleansing the tooth surface, a pellicle forms from proteins and glycoproteins in saliva.

A Association: Through purely physical forces, bacteria associate loosely with the pellicle.
B Adhesion: Because they possess special surface molecules (adhesins) that bind to pellicle receptors, some bacteria become the "primary colonizers," particularly streptococci and actinomyces. Subsequently, other microorganisms adhere to the primary colonizers.
C Bacterial proliferation ensues.
D Microcolonies are formed. Many streptococci secrete protective extracellular polysaccharides (e.g., dextrans, levans).
E Biofilm ("attached plaque"): Microcolonies form complex groups with metabolic advantages for the constituents.
F Plaque growth—maturation: The biofilm is characterized by a primitive "circulatory system." The plaque begins to "behave" as a complex organism! Anaerobic organisms increase. Metabolic products and evulsed cell wall constituents (e.g., lipopolysaccharides, vesicles) serve to activate the host immune response . Bacteria within the biofilm are protected from phagocytic cells (PMN) and against exogenous bacteriocidal agents.

- **Microorganisms and Periodontal Disease**

The oral bacterial microbiome includes over 700 different phylotypes, with approximately 400 species found in subgingival plaque. The subgingival microflora in periodontitis can harbor hundreds of bacterial species but only a small number has been associated with the progression of disease and considered etiologically important. Subgingival plaque from deepened periodontal pockets is dominated by gram-negative anaerobic rods and spirochetes. Strong evidence has implicated *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitan* to the pathogenesis of adult periodontitis. In addition, *Bacteroides forsythus*, *Prevotella intermedia*, *Peptostreptococcus micros*, and *Fusobacterium nucleatum* have been strongly linked with the progression of adult periodontitis.

- **Tobacco Smoking**

There is accumulating evidence for a higher level of periodontal disease among smokers. Tobacco smoking exerts a substantial destructive effect on the periodontal tissues and increases the rate of periodontal disease progression. Risk factors including tobacco smoking modify the host response to the challenge of bacteria in microbial dental plaque. Smokers with periodontal disease seem to show less signs of clinical inflammation and gingival bleeding compared to nonsmokers. That could be explained by the fact that nicotine exerts local vasoconstriction, reducing blood flow, edema, and clinical signs of inflammation [28]. Nicotine acetylcholine receptor has been found to play an important role in the development of nicotine related periodontitis.

- **Diabetes Mellitus**

One of the important oral signs of diabetes is gingivitis and periodontitis. Patients with undiagnosed or poorly controlled diabetes mellitus type 1 or type 2 are at higher risk for periodontal disease. There are many studies that demonstrate an association between diabetes and an increased susceptibility to oral infections including periodontal disease. Periodontitis also progresses more rapidly in poorly controlled diabetics, and early age of onset of the disease is seen as a risk factor for more severe diseases. Conversely, most well-controlled diabetic patients can maintain periodontal health and will respond favorably to periodontal therapy.

Despite discrepancy regarding this issue in the scientific literature, it seems that the effect of glycemic control is related to the mode of periodontal therapy. Many studies addressed the effect of periodontal treatment on glycemic control of diabetes patients.

- **Cardiovascular Disease**

The biological plausibility of the association between periodontal diseases and cardiovascular diseases is well studied and it includes some of the following possible mechanisms: high concentrations of cholesterol and the action of oral bacteria in the process of atherosclerosis or the participation of acute-phase proteins that may increase in chronic periodontitis. Several biological mechanisms have been proposed to explain the relationship between periodontal diseases and cardiovascular diseases. Therefore, periodontitis can probably elicit a systemic

- **Drug-Induced Disorders**

Some medications significantly decrease salivary flow. These include antihypertensives, narcotic analgesics, some tranquilizers and sedatives, antihistamines, and antimetabolites. Other drugs, particularly those in liquid or chewable form that contain added sugar, alter the pH and composition of plaque, making it more able to adhere to tooth surfaces.

Drugs can be a contributing factor in periodontal diseases. Drugs such as anticonvulsants, calcium channel blocking agents, and cyclosporine may induce gingival overgrowth.

- **Stress**

Patients with inadequate stress behavior strategies (defensive coping) are at greater risk for severe periodontal disease. Stress is associated with poor oral hygiene, increased glucocorticoid secretion that can depress immune function, increased insulin resistance, and potentially increased risk of periodontitis. Men who reported being angry on a daily basis had a 43% higher risk of developing periodontitis compared with men who reported being angry seldom. Studies have

found some periodontal disease indicators such as tooth loss and gingival bleeding to be associated with work stress and financial strains.

Topic. Papillitis.Classification Danilevsky clinic, diagnostics, treatment, prevention.

Papillitis is an inflammation, limited by the area of one or two gums papillae. Clinically-morphological features distinguish (MF Danilevsky, 1957) catarrhal (acute, chronic), ulcerative (acute, chronic), hypertrophic (chronic, acute) papillitis.

Catarrhal papillitis. Often arises as a result of acute injuries - prick, blow, slaughter, injury to the gums with fish bones, toothbrush nappies, tools for treating teeth, biting solid objects, etc. The most common reasons for locals are unsatisfactorily filled seals on the contact surfaces of the teeth. Especially clear papillitis is manifested in the absence or incorrect formation of the contact point, or when the filling material protrudes beyond the tooth border and partially fills the interdental gap, hanging over the gingival papilla. Catarrhal papillitis in case of contact with a contact point may be accompanied by pain.

Acute catarrhal papillitis. Patients complain of pain that occurs during eating, especially hard. Painful, persistent or lasting for a certain period of time after irritation.

Objectively detect hyperemia, edema of the gingival papilla, changes in its contours, relief, smoothing of the tops. Often, as a result of severe edema, there is a gingival pocket, the integrity of the tooth-epithelial attachment is not disturbed. In the case of papillitis of traumatic origin, traces of a previous injury (eg erosion, abrasions) are detected.

The papillitis of the chemical (medicinal) origin originally occurs as a catarrhal, but then can quickly grow into ulcerative. During the action of acids, the affected papilla decreases in volume as a result of dehydration, and becomes whitish in color. Later, as a result of hemorrhage, the color changes from bright red to brown, and sometimes to dark brown.

Chronic catarrhal papillitis. Objectively, during the examination of the contours of the gingival papilla, they are smoothed, his edema, pastoseness and cyanoticity are observed. During palpation, the gums papilla bleed, expressed a symptom of vasoparezu. The inflammatory process can be affected only by marginal gums or whole papillae as a whole.

The ulcerative papillitis is rarely seen in comparison with other forms. The most common cause of this form is the prolonged action of mechanical stimuli, among which the leading role belongs to the hung edges of seals. Less commonly, such

papillitis is a consequence of a chemical trauma. The ulcerative papillitis has a course on the type of alterative decubital process.

Patients complain of pain in the affected area of the papilla, the intensity of which depends on the nature of the course of the process - acute or chronic. There is an unpleasant smell from the mouth, with an acute course of the disease, a slight increase in regional lymph nodes may be observed. The general condition of the patients, as a rule, is not disturbed.

Gum papilla is hyperemic, swollen, at the apex there is a section of necrosis of grayish color . When cautiously probing this area, necrotic tissue is easily removed and exposed to the ulcer surface, which easily bleeds during mechanical stimulation. In the case of chronic papillitis, congestion is less pronounced, the tip of the cyanotic gum papilla is as if "cut" by the ulcer and the interdental gap is shining. The amount of necrotic tissue and the spread of dirty gray color is less than in acute ulcerative papillitis, since it is washed away by saliva during the course of the disease.

Hypertrophic papillitis has a chronic course, often aggravated. In children and adolescents, unlike adults, there is a relatively high rate of occurrence. The development of hypertrophic papillitis is associated with the presence of local stimuli (mainly gum trauma in the process of localization of carious cavity on contact surfaces, sealing defects). At an early age, hypertrophic gum growth is often observed in areas of anomalies of the position of individual teeth. In adolescents during puberty, the so-called "juvenile papillitis" may develop, the development of which may not be linked to local irritating factors.

Patients often do not complain about pain because it is weakly expressed and occurs only from mechanical stimuli. Gum papilla develops and fills the carious cavity on the contact surface of the tooth or is placed in the interdental gap. The gum tissue is relatively dense, slightly hyperemic, the edge of the toe has festonchist outlines. During mechanical irritation of the papilla (palpation, precautionary instrumentation) there is a slight bleeding. Hypertrophied papilla, placed in the carious cavity, repeats its outline, sometimes stands beyond its borders. In the process of localization of hypertrophic papillitis in the area of the roots of large angular teeth (temporary or permanent), the gingival papilla acquires a lobar structure, reminiscent of its appearance of raspberry, cauliflower.

Gum debridment is constantly damaged by hard food, antagonists, resulting in a chronic inflammatory process may become aggravated, and on the tip of the papilla ulcers are formed. This is manifested by more pronounced pain and increased

bleeding. Characteristic rapid progression of the pathological process, sometimes inflamed hypertrophied papillae can cover crowns of teeth to their height and length. This can lead to partial injury to the papilla. In the future, there may again be a loss of manifestations of inflammation, a transition to a chronic course and a relative stabilization of the pathological process.

Depending on the severity of the growth of the gingival papilla, I, II or III degree of hypertrophic papillitis is distinguished. In the first stage, the papilla is enlarged within 1/3 of the height of the crown of the tooth, at II - within 1/2 and at the third degree covers more than 2/3 of the height of the crown of the tooth.

Treatment of catarrhal gingivitis and catarrhal papillitis should be complex, including etiotropic, pathogenetic and symptomatic treatment.

Etiotropic treatment is to identify and eliminate general and local harmful factors. Particular attention is paid to eliminating local stimuli such as plaque, dental, inferior seals, dentures, carious cavities, root teeth, and the like. Carry out a thorough professional cleaning of teeth and oral hygiene. It is important to teach the patient the rules of rational hygiene of the oral cavity and to convince him of their use, because in the presence of gum disease patients often stop regular teeth cleaning. In the case of acute inflammation, it is advisable to use soft toothbrushes, the villi of which will not further irritate the gums. Assign mouth rinses to antiseptic, hygiene or hypertonic solutions, depending on the nature (acute or chronic) of the course of the disease.

Pathogenetic treatment consists of a medication effect on various pathophysiological links of the gum inflammation process. In acute inflammation, there is a need to suppress a rather severe pain in the gums. For this purpose, rinse with solutions of citral, propolis (for 20-50 drops of alcoholic solutions per 1 cup of rinse water). For applications on painful areas of gums use anesthetics (anestezin, lidocaine, trimecain, etc.), derivatives of anthranilic acid (mefenamino acid, mefenamine sodium salt). In the case of severe pain, analgesics are prescribed.

Anti-inflammatory therapy. When choosing medication should take into account the mechanism of development and the phase of inflammation. Most anti-inflammatory drugs affect not one but several pathogenetic components of inflammation, so they can be administered at different stages of inflammation.

Since the microflora plays an important role in inflammation of the gums, it is shown the use of antibacterial drugs in the form of rinses, appliqués on the affected

areas of the gum. For this purpose, antiseptics (ethcradine lactate, chlorhexidine bigliuconate, ethonium, myramistin, iodine preparations, etc.), nitrofuran derivatives (furatsillin, furagin, etc.), and, more rarely, antibiotics (tetracyclines, amoxicillin, macrolides, lincosamines (clindamycin), etc.) and sulfanilamide preparations . Also widely used antibacterial drugs for both local and general treatment in the case of generalized catarrhal gingivitis. Natural antimicrobial drugs are also used: novoiminin (0.1% alcoholic solution of St. John's wort), 0.25% chlorophyllipte solution (contains a mixture of chlorophyll from eucalyptus leaves), 0.2% sanguitarine, tincture of Japanese sophora, etc. They act predominantly on gram-positive microorganisms, including staphylococci resistant to penicillins. Today, the use of strong antibacterial agents (eg, triclosan) can lead to a significant violation of the saprophytic microflora of periodontal tissues. In the event of such a situation (it may be in weakened children) for correction of microflora it is expedient to use probiotics (lactobacilli, etc.).

In the early stages of inflammation, remedies that have the ability to stabilize the lysosomes and at the same time interfere with the formation of inflammatory mediators (mefenamine sodium salis, salicylates (choline salicylate, holisal)), proteolytic inhibitors (tracilol, contrikal) are shown; drugs that stimulate the formation of anti-inflammatory agents (salicylates, prodigiozan, calcium pantothenate, vitamins C, P, etc.). The use of anticoagulants (heparin, fibrinolysin), antiplatelet agents and non-steroidal anti-inflammatory drugs (sodium salicylate, mefenamine sodium, sodium diclofenac, mesulide, etc.) are indicated for the regulation of microhemocirculation disorders.

In the reparative phase of inflammation, in order to stimulate protective mechanisms and regenerative processes, prescribe drugs that enhance phagocytosis (lysozyme), preparations of pyrimidine bases (methyluracil, pentoxyl), vitamins (ascorbic acid, vitamin P, galasporbin, etc.), endogenous RNA and DNA (sodium nucleate), means of plant origin, etc. To enhance the action of medicinal drugs, their administration can be aerosol inhalations (especially in children with diffuse spread of inflammatory process in the gums), and with chronic inflammation - by electrophoresis, ultraphonophorezus. It is possible to independently use physical methods of treatment, for example, laser irradiation, UV irradiation, etc.

In the case of sensitization in the pathogenesis of acute inflammation (if necessary, especially during generalization of the process), hypoxibles (antihistamines, vitamin C, calcium preparations). They are used according to the generally accepted scheme, as the general treatment of the catarrhal inflammatory process in the gums. In children, the value of general treatment increases, and if necessary,

prescribe detoxification agents, vitamin therapy, and the like. Immunomodulatory (immunal, immunological, etc.) and general tonic drugs (drugs of eleutherococcus, magnolia, ginseng) are prescribed if necessary (and only after consultation with general specialists in the detection of immunodeficiency states).

In the treatment of chronic catarrhal gingivitis, the need for a broader use of analgesics disappears; therefore, mainly hypertensive 2% solutions of sodium chloride or sodium bicarbonate are used for rinsing. Apply astringents, tanners: infusions and decoctions of medicinal plants (St. John's wort, sage, chamomile, oak bark, etc.), solutions for the oral cavity Stomatophyte and Stomatophyte A; phytoncides (phytodontist, romazulan, yuglon, salvin, maraslavin, etc.). More often used physical methods of treatment - electrophoresis of various drugs, therapeutic irrigation, hydromassage, etc.

Topic 10. Clinic, diagnosis of catarrhal gingivitis and hypertrophic gingivitis.

Gingivitis is the inflammation of gums, conditioned by the unfavorable action of local and general factors, that flows without violation of integrity of tooth-gingival connection. Catarrhal gingivitis. Patients grumble about the unpleasant feeling in gums, feeling of itch. Odor nuisance from a mouth, by curvature of taste, sanguifluousness of gums during a meal or cleaning of teeth. The general of patients suffers small, however the periods of intensifying can be accompanied by indisposition, lowgrade temperature.

Acute catarrhal gingivitis is characteristic for the period of eruption and changes of teeth. It is observed at acute infectious and other inner diseases.

Chronic catarrhal gingivitis differs in the protracted languid motion; complaints are poorly expressed. At an objective inspection mark an edema, hyperemia, cyanosys clear, a mechanical irritation is accompanied by bleeding. On teeth is enhanceable maintenance of soft dental raid(patients avoid to clean teeth as a result of the pain feeling and sanguifluousness of gums). The amount of gingival liquid increases, the positive test of Schiller is determined also - Пісарева, increase emigration of leucocytes for Yasinivsky; mionectic firmness of capillaries is at a vacuum test for Kulagenko and other It follows remember, that catarrhal gingivitis can be the symptom of general disease, the early exposure of that has certain curatively, is a prophylactic value.

Treatment of catarrhal gingivitis includes: - removal of local irritating factors(moving away of dental sedimentations, stopping of carious cavities, first of all II, V of class for Blek, moving away of the unrationally made prosthetic appliances of т. д.); - local, and in case of necessity and general ethiotropic (antimicrobial) therapy; - local use *of antiinflammatory facilities*; - increase of local heterospecific resistance; - physical therapy methods. Preparations of choice for empiric etiotropic therapy are ethonij, chlorhexidine, «Paragel», «Metrogyl-Denta», Hinoxidin, Dioxidin as preparations of choice for empiric etiotropic therapy. For antiinflammatory therapy of generalized of catarrhal gingivitis recommend to use non steroid antiinflammatory facilities: to the mephenaminat natrium, nimesulid, movalis, indomethacin, Ibufrofenum, diclofenac and other. Them it follows to use in the holiatry of catarrhal gingivitis only in that case, when rational local interference, antibacterial and imunomodulatine therapy not able to stop an inflammatory process at gums. For general tonic treatment appoint vitamins And, C, E, ascorutin, polivitamins in combination with microelements – triovit, duovit, revit and other Physiotherapy. For fixing of results of medicamental therapy it is necessary to conduct the row of physical therapy events : auto-, hydro-, vibratory massage, hydropathy, electrophoresis, photoradiotherapy and other metods.

Hypertrophic gingivitis

This is a *chronic inflammatory process* in the gums, accompanied by proliferative phenomena with the growth of fibrous structures of the lamina propria of the gingival mucosa and the proliferation of the basal layer of the epithelium. It develops very slowly, exacerbations and remissions are possible.

Among the *etiological factors* are local irritants, as well as violations of some systems of the body, especially endocrine. Hypertrophic gingivitis often occurs during puberty and in women during pregnancy. It can occur with hypovitaminosis c, metabolic disorders, in particular carbohydrates. It is important to survive certain drugs (diphenine , diphenyl-hydantoin , which are against convulsive drugs).

There are *localized and generalized forms*. The localized form is caused by the action of local damaging factors, the generalized form is observed in concomitant diseases. According to the degree of severity, there are I, II, III degree depending on the degree of hypertrophy.

I degree – gingiva covers the crown of 1/3

II degree – gingiva covers the crown of 1/2

III degree – gingiva covers the crown of 2/3

Hypertrophic gingivitis is localized mainly on the vestibular surface of the frontal gums. Lesions of the oral surface most often occur in blood diseases (hyperplastic syndrome in leukemia). Particular attention should be paid to this, as it may be the only early clinical symptom of leukemia.

According to clinical and morphological features, there are 2 forms of the disease: ***edematous*** (inflammatory) and ***fibrous***.

Edematous (granulating) form

Complaints:

Growth of the gums, unusual appearance of the gums, bad breath, bleeding and pain in the gums, which is aggravated by eating and chewing. May exacerbate.

Objectively:

Changes in the relief of the gums due to hyperplasia of the gingival papillae and roller-like thickening of the marginal part of the gums. The shape of the gingival papillae is incorrect, the appearance of the gums in the advanced stages is distorted.

The gingival papillae and gingival margin are hyperemic with pronounced cyanosis, the alveolar part of the gums is in a state of chronic catarrhal inflammation. Pronounced swelling and loose gums, they lag behind the necks of the teeth. Pain on palpation of the gums, bleeding on examination of the gingival pocket is determined.

Due to severe edema and hyperplasia formation of different depths false gingival pocket filled with serous fluid, but dentogingival of ' unity is not broken.

This form of GG has the peculiarities of the course in adolescents during puberty: it is characterized by a tendency to frequent exacerbations, rapid

progression, excessive signs of inflammation and excessive hypertrophy of the gingival papillae.

Fibrous form

Complaints:

At insignificant hypertrophy of gums are absent, or on an unusual kind of gums. The disease develops slowly and imperceptibly for the patient, practically does not give exacerbations.

Objectively:

Changes in the relief of the gums due to hyperplasia of the gingival papillae and roller-like thickening of the gingival margin.

Gum papillae pale pink, close to the necks of the teeth. At inspection of a gingival furrow bleeding is not revealed. There is no exudate in the fake pockets. Hypertrophied areas of the gums on palpation are dense and painless. There may be hypertrophy of the gums not only from the vestibular but also from the oral surface of the teeth.

Data of additional methods of inspection at HG

- The Shillev-Pisarev test is sharply positive at the edematous form.
- Indicators of all hygienic indices indicate unsatisfactory hygienic condition of the oral cavity.
- Formalin test (Kotzchke 2) is negative (if there is no violation of the integrity of the gum epithelium due to injury).
- Decrease in stability of capillaries at vacuum test according to Kulazhenko (10-20 sec).
- There are no changes on the radiograph, with a long course of the disease, osteoporosis of the vertices of the interalveolar septa is possible.
- Mandatory ' if omitted is a blood test to exclude displays of hyperplastic syndrome in diseases of the blood.
- The amount of gingival fluid is sharply increased in the edematous form.

Pathomorphological changes in HG

In the ***granulating form*** - parakeratosis in the superficial cells of the epithelium, vacuolar dystrophy in the cells of the spiny layer. Proliferation of the basal layer of the epithelium. In its own plate - the growth of fibrous structures, round-cell leukocyte infiltrates. Granulation tissue is formed and grows. Capillary dilation, venous stasis.

In the fibrous form - a violation of the keratinization of the epithelium. In own plate of gums growth of dense collagen fibers. Lymphocytic infiltrates around blood vessels . The walls of blood vessels are thickened.

Differential diagnosis of HG

Intra - syndromic - between two forms, chronic and acute, severity.

- with symptomatic HG in periodontitis ;
- with symptomatic gingivitis on the background of somatic pathology (blood diseases, endocrine system, metabolic disorders);
- with symptomatic gingivitis with hypovitaminosis C.
- fibrous form - from fibromatosis of the gums, epulis and other tumor-like diseases.

	Catarrhal gingivitis	**Hypertrophic gingivitis**
Etiology	Plaque microflora, infectious diseases in the past medical history, impact of the stressors, vitamin and microelement deficiency, internal organ pathology, excessive alcohol consumption and smoking tobacco	Endocrine diseases, pregnancy, puberty, menopause, blood diseases (leukemia), the use of certain drugs (diphenylamine, cyclosporine).
Color of the gums	Congestive hyperemia in chronic course, bright hyperemia in acute course	Congestive hyperemia in granulating form, absence in fibrous form
Gingival bleeding	When brushing teeth and eating solid foods	When brushing teeth in granulating form, absence in fibrous form
Periodontal pocket	Edema-related periodontal pocket	Hypertrophy-related periodontal pocket
General condition	General weakness, fever in acute condition	Normal

Topic 11. Clinic, diagnosis of ulcerous-necrotic gingivitis.

Ulcerative (ulcerative-necrotic) gingivitis is an inflammatory process in the mucous membrane gum, which is characterized by the predominance of an altered component, a violation of integrity
and tissue necrosis. It is observed less often than other forms of gingivitis.

In etiology, the leading role is played by fuzzy-peptidic symbiosis: *fusobacterium* (gram-negative anaerobic sticks) and *spirochetes. Acute ulcerative-necrotic* gingivitis develops like the rule, against the background of reducing the immunological reactivity of the organism as a result of the suffered acute respiratory viral diseases, infectious diseases of bacterial etiology, with acute herpetic stomatitis, as well as deficiency of ascorbic acid.
Development the disease contributes to reducing the local immunity of the oral cavity, the presence of local traumatic factors, lack of hygienic care of the oral cavity.

Ulcerative gingivitis *usually has an acute course*. Depending on the prevalence process and severity of general clinical manifestations distinguish between light, medium and severe səˈvi(ə)r degree of disease.

In the prodromal period *(1-2 days),* the patient has an increase in temperature body to 37-38 C, weakness, headache, loss of appetite, sleep disturbance.

At first signs of acute catarrhal gingivitis are observed: swelling, hyperemia and bleeding of the gum mucosa. Subsequently, against the background of pronounced catarrhal changes occurs necrosis of the *gingival papilla* and marginal edema.

Patient complains of severe pain and bleeding of the gums, which increase when eating food, rotten smell from the mouth, elevated salivation.

The onset of the disease is manifested by the lesion of the gum mucosa in the area 1-3 teeth that corresponds to a slight degree of gravity. On the surface of the gum on the background of edema and pronounced hyperemia is a *dirty-gray necrotic plaque*. After it is removed it's exposed sharply painful bleeding ulcerous surface. Ejaculatory papillae lose conical shape,
compacted

There is a significant amount of soft plaque, very unpleasant smell from the mouth cavity, increased salivation, as well as an increase in regional lymph nodes.

The average degree of gravity is characterized by the diffuse spread of the process along marginal gums, higher body temperature (from 37.5 to 39 C) and an increase in signs of intoxication. A patient may be affected by sleep, nausea and diarrhea. When progressing
the process of interdental papillae can be completely necrotic

The severe course of acute ulcerative-necrotic gingivitis is observed, as a rule, at weakened patients. Necrosis can spread to the alveolar part of the gum and other parts.
Sometimes the disease begins with lesions of retro-molar areas and palatine tonsils (angina Vensana).

The general condition of these patients is significantly disturbed due to an increase in body temperature
(more than 390C) and severe intoxication.
Duration of the period of pronounced clinical manifestations in acute ulcerative gingivitis is, on average, 7 to 10 days, depending on the severity of the disease, timeliness and quality of assistance.

X-ray changes in the periodontal tissues during the acute course of ulcer-necrotic gingivitis is absent.

Differential diagnosis of acute ulcerative gingivitis is carried out with necrotic changes in gum mucus in idiopathic diseases with progressive lysis periodontal tissues (hereditary neutropenia, histiocytosis), as well as in diseases of the blood (leukemia).

Treatment depends on the characteristics of pathogenesis, severity, the nature of the course process and age of the patient.

Local treatment:
1. Anesthesia (10% lidocaine gel, Cimistad gel or other anesthetics in the form of applications).
2. Removal of necrotized tissues (application of proteolytic enzymes - trypsin, chymotrypsin, terylitin 1-2 times a day, ointment "lingezin", which, in addition to the enzyme, contains also antibiotic; antiseptics - chlorhexidine, hexytidine).
3. After the removal of necrotic tissue, antibacterial therapy is performed (metronidazole or complex drugs containing chlorhexidine and metronidazole *"Metrogil-denta";* at a severe course of the ulcerous process it is expedient use broad spectrum antibiotics - lincomycin, tetracycline, in the form of instillations or applications).
4. Anti-inflammatory therapy (NSAIDs - 0.1-0.2% aqueous solution of mefenamine sodium salt,
1% gel or jelly "Dickler", 1% emulgel "Voltaren", herbal means origin - flowers of chamomile, calendula, sage, nettle leaves, *artificial lysozyme "Lisobact*")
5. After cleaning the surface from necrotic and fibrinous plaque and after
The removal of acute inflammation requires the use of *keratoplasty (*oil solutions of *vitamin A and E, multivitamin complex "Aevit", rosehip oil, caratolin, sea*

buckthorn oil, as well as biogenic stimulants*: kalanchoe juice, jelly "Solkoseril",* they are used in the form of applications*).*
6. Training of methods of individual care of the oral cavity, hygienic control for their implementation and recommendations on the choice of hygiene products.
7. Elimination of local factors contributing to the development of ulcerative-necrotic
gingivitis (performed after the completion of tissue epithelization).

General treatment with a mild severity of acute ulcerative gingivitis is in removing the phenomena of intoxication at home (repeated drinking of vitamin and enveloping liquid - apple-carrot juice, berry or milk kissel, mozz). Food should be be energetically complete, soft consistency (buckwheat, oatmeal porridge), protein foods that are easy digested (fish, poultry meat), limited use of refined carbohydrates.
Also prescribed orally complex vitamin preparations that contain ascorbic acid, vitamins A, P, E and B (Decamivet, Macrovit, Vitrum).

Topic 12. Clinic, diagnosis of localized periodontitis.

Localized periodontitis is a disease that characterized by inflammation and progressive destruction of periodontal tissues (gum, periodontal disease, interalveolar septum, cement root) in the site one or several teeth, and may result in loss damaged teeth.

Clinical characteristic of localized Periodontitis is presented as acquired symptomatic complexes: symptomatic gingivitis, periodontal pockets, destruction of root tooth cement, osteopathy, breaking of

Classification (MF Danilevsky 1994):

Form: catarrhal, hypertrophic, ulcerative ,atrophic

The course: acute, chronic

Depth of defeat: soft tissues and alveolar bone

*Degree of developmen*t: initial, I degree, II degree, III degree.

Prevalence of the process: focal

Initial degree of localized periodontitis depends on the nature of the course of symptomatic gingivitis (acute or chronic). Clinically determined symptomatic light gingivitis (papillitis), dental deposits, gums pockets, traumatic occlusion.

X-ray defining a violation of a compact plate of vertebrae of alveolar partitions (destruction), insignificant resorption of vertices of partitions and expansion , periodontological cracks in the cranial region.

At I stage localized periodontitis is clinically is defined symptomatic gingivitis, periodontal pockets up to 4 mm deep with moderate serous-purulent content, abnormal tooth movement and degree, expressed traumatic occlusion. Radiologically determined resorption of interalveolar partitions to 1/3 length root, extension of the periodontance gap.

For II degree of localized periodontitis characteristic symptomatic gingivitis of the lung or moderate severity (marginal gingivitis), periodontal pockets up to 6 mm with serous-purulent contents, traumatic occlusion, movement of teeth I-II stages. Radiologically determined expansion periodontance gap, resorption of interalveolar partitions up to 1/2 of the length of the root. Often availability caries of cement root.

With localized periodontitis III degree the severity of clinical manifestations is reduced to severe symptomatic gingivitis, severe traumatic occlusion, depression of periodontal pockets up to 6-8 mm, tooth movement of II-III stages. On the X-ray - resorption of bone tissue is determined by 2/3 and more

length of the root, extension of the periodontance gap, decalcification of root tooth cement

Chronic localized periodontitis is characterized by chronic symptomatic gingivitis (catarrhal, hypertrophic) with moderate content serous or serous-purulent periodontal exudates pockets

- Surgical methods: curettage - at depth periodontal pockets 3-5 mm; gingivotomy - at depth of pockets more than 5 mm.(II – III degree)

	Localized periodontitis
Etiology	Mechanical trauma (inappropriate preparation and fixation of the fixed dentures), occlusal trauma (malocclusion, premature removal of molars), etc.
Color of the gums	Congestive hyperemia in chronic course, bright hyperemia in acute course
Gingival bleeding	Minor bleeding and tender palpable
Dental calculus deposits	Supra- and sub-gingival
Tooth roots baring	Baring according to the severity
Periodontal pocket	Periodontal pocket of various depth
Teeth mobility	Class I-III pathological mobility
The state of regional lymphatic nodes	Enlarged, tender palpable in the acute course
Findings of additional studies	Radiography shows destruction of the compact plate, resorption of the interalveolar septa on the bounded area of the jaw

Symptom complex in periodontitis :

- symptomatic gingivitis

- periodontal pocket
- under ' gingival tartar
- traumatic occlusion
- progressive resorption of alveolar bone.

Symptomatic gingivitis.

It most often manifests itself in the form of catarrhal gingivitis - in about 70% of patients. Symptomatic catarrhal gingivitis may be chronic or acute.

If the patient develops symptomatic hypertrophic gingivitis - there is a granulating (edematous) form. The course is often chronic, but possible exacerbation, in which there are more pronounced signs of gingivitis.

At development of symptomatic ulcerative gingivitis its chronic course is most often observed, thus insignificant necrosis of tops of gingival papillae or marginal edge of gums is noted. Necrotic surfaces are separated from the surrounding gingival mucosa by a demarcation line in the form of a hyperemic strip. Sometimes there is an acute course with more pronounced necrotic phenomena and the formation of deep periodontal pockets with serous-purulent or purulent exudate.

Periodontal pocket. Are required ' necessarily a sign of periodontitis .

In the initial stages of periodontitis allegedly preserved the integrity of periodontal of ' unity, however histopathological studies reveal a violation of his integrity. In the clinic, this can be confirmed by formalin testing Parma (Kotzchke 2) .

Further due to destruction of circular called ' bandages dental cement between the tooth and gums inner surface formed recesses of different depths - periodontal pocket. Their depth varies from 2 to 10 mm . As a result of the destruction of the ' unity of the epithelial attachment cuticle lost tooth mechanisms that restrain proliferation and maturation of immature epithelial attachment. As a result, the epithelium grows into the depth of the connective tissue of the gums and its differentiation into the multilayered squamous epithelium of the gums. At the bottom of the periodontal pocket proliferates granulation tissue. The periodontal pocket is filled with exudate - serous, serous-purulent or purulent.

Thus, the walls of the periodontal pocket: tooth cementum, ingrown multilayered gum epithelium, granulation tissue at the bottom.

Content periodontal pockets, fluid, under ' gingival tartar, dental plaque.

Traumatic occlusion.

The *primary traumatic occlusion* - develops against the background of an intact periodontal due to excessively second -largest or the usual trend for chewing pressure.

Secondary traumatic occlusion develops against the background of periodontal disease, when even the usual occlusal load exceeds the compensatory capacity of the periodontium, becoming a traumatic factor. Due to the mobility during the action of masticatory pressure, the teeth are deflected, as a result of which their physiological abrasion does not occur. Thus, teeth with intact periodontium are erased, while in areas with affected periodontium, the teeth retain

bumps on the masticatory surface and the cutting edge. Thus, these bumps when closing the dentition are the first to come into contact with the teeth-antagonists, which causes traumatic overload of such teeth and leads to the formation of so-called traumatic nodes.

The *mobility* of the teeth leads to the displacement of the teeth vestibularly or orally and their extension from the holes. The movements of the lower jaw change, because the patient reflexively protects the moving or displaced teeth from the action of masticatory pressure. This leads to disruption of chewing tone ' muscles and changes in the temporomandibular joint.

The movement of teeth leads to the loss of interdental contacts and violation of the integrity of the dentition. The dentition ceases to function as a single system.

The degree of mobility of the teeth does not always correspond to the severity of periodontitis . At a chronic current - degree of mobility, as a rule, coincides with degree of severity of a disease (I degree of mobility at I degree of severity, etc.). In the acute course of generalized periodontitis, the degree of mobility is always higher than the degree of severity (in the first degree of severity of periodontitis, mobility can reach II or even III degree).

Progressive resorption of alveolar bone.

The main radiological symptoms of periodontitis :

- destruction of the vertices of the interalveolar septa
- destruction of the cortical plate of the interalveolar septa
- decrease in the height of the interalveolar septa
- uneven resorption of interalveolar septa in different departments
- vertical, horizontal and lacunar resorption
- osteoporosis of the bone of the alveolar sprout
- large-loop pattern of bone tissue
- widening of the periodontal gap, especially in the marginal parts
- formation of bone pockets.

The severity of periodontitis is set solely on the radiograph:

- at the initial stage - destruction of the cortical plate of the interalveolar septa and a slight decrease in their height
- at the I degree - decrease in height of interalveolar septa to 1/3 of their height
- at the II degree - decrease in height of interalveolar septa within 1 / 3-2 / 3 of their height
- at the III degree - decrease in height of interalveolar septa by 2/3 of their height and more.

Topic 13. The mechanism of formation of gingival and periodontal pockets. Clinical manifestations of generalized periodontitis.

It is characterized by the development of chronic symptomatic gingivitis with scanty clinical symptoms. In periodontal pockets scanty serous or serous-purulent exudate. The mobility of the teeth usually corresponds to the degree of bone resorption. The general condition of the body is unchanged. Radiologically - resorption of alveolar bone is more uniform, without formation of deep bone pockets. The phenomena of osteoporosis are weakly expressed.

Exacerbation of periodontitis .

Occurs when the body's reactivity decreases (for example, against the background of comorbidities). Clinically there is an exacerbation of symptomatic gingivitis, accompanied by severe pain, edema, redness, bleeding. The number of secretions from the periodontal pocket increases , they are purulent in nature. Sometimes on the background of the formation of deep pockets in the bone ' which tissues are formed of single or multiple periodontal abscesses. With ' are symptoms of intoxication (fever, headache, malaise, etc.). Tooth mobility is higher than the severity of the disease. Radiologically : uneven progressive resorption of alveolar bone, formation of bone pockets, severe osteoporosis of the bone. The periodontal fissure is expanded for a considerable extent.

Laboratory indicators.

Capillaroscopy and biomicroscopy . At a chronic course there is a desolation of capillaries, turbidity and cyanoticity of a capillary background, increase in number of functioning capillaries, venous stagnation, expansion of capillary loops, slowing down of a blood-groove. In the course of exacerbations than specified, there is excessive povnokriv ' I capillaries.

Vacuum test for Kulazhenko . Normally, a hematoma is formed in about 30-50 seconds. With generalized periodontitis, this time is reduced to 7-10 seconds in chronic and up to 3-5 seconds - in acute.

Migration of leukocytes into the oral cavity by the method of Yasinovsky . Norm - leukocytes up to 150 in 1 ml of flushing fluid, at least 80% of them are alive, the number of squamous epithelium does not exceed 50. In chronic periodontitis, the number of leukocytes doubles, the percentage of living neutrophils is below normal. The number of desquamated epithelial cells increases to 100. With exacerbation, the number of leukocytes increases 4 times, the percentage of living neutrophils is below normal. The number of squamous epithelial cells reaches 150.

Cytological examination. In chronic periodontitis, the content of active phagocytes is sharply reduced. With an acute course, the percentage of active neutrophilic granulocytes increases, which can be explained by a certain increase in the protective properties of periodontal tissues.

Microbiological research. In the case of chronic periodontitis , cocci, spindle-shaped rods, spirochetes, yeast-like fungi, and protozoa are found in periodontal pockets . In the acute course, the number of microorganisms

detected by microscopic or bacteriological studies increases by 5-10 times, dominated by cocci or rod-shaped microorganisms.

Protein fractions of blood. In patients with chronic and acute GP there is a decrease in albumin, an increase in globulins, especially γ -globulins.

Phagocytic activity of leukocytes. Normally, the percentage of phagocytic neutrophils is 83-86%, and the phagocytic number is 5-6. These indicators in the chronic course of GP are at the lower limit of normal, in the acute course - at the upper limit.

Clinical course

Initial degree.

Complaints of bleeding gums when chewing, brushing teeth; discomfort in the gums, itching and paresthesia of the gums. Sometimes there are no complaints, the pathological process without subjects ' of objective signs.

Anamnesis. It is quite difficult to find out when the disease started. As a rule, pain and bleeding bother the patient for several years, periodically increase and decrease.

Objectively . Local traumatic factors. Symptomatic catarrhal or hypertrophic gingivitis. Primary traumatic occlusion in places of low-quality prostheses, fillings, etc. Periodontal pocket 1-2 mm deep . Massive deposits of ' gingival tartar (especially on the lingual surfaces of the lower front teeth and upper molars vestibular surface - that is, in places of ductless glands). The deposits at ' gingival plaque in the interdental spaces and periodontal pockets. The necks of the teeth are not exposed. The mobility of the teeth is physiological. Positive formalin test.

Radiologically . Osteoporosis of the upper and middle third of the interalveolar septa, destruction and violation of the integrity of the compact plate, a slight expansion of the periodontal gap near the tops of the interalveolar septa.

I degree.

Complaints differ in chronic and acute course: in chronic - bleeding gums when brushing teeth, bad breath; with acute - severe pain, significant bleeding gums, hyperesthesia of the hard tissues of the teeth in the neck.

Clinical signs. Symptomatic gingivitis of diffuse nature. Hyperemia of the gums with a cyanotic tinge in the chronic course, bright redness - with acute. Decrease in height of tops of gingival papillae owing to what interdental spaces "gape". Exposure of the necks of the teeth, resulting in the development of hyperesthesia of the hard tissues of the teeth. Periodontal pockets 2-3 mm deep , the content - serous-purulent, its number depends on the course. Massive deposits of ' gingival tartar, to a lesser extent - under ' gum . Expressed mobility of the lower front teeth (I degree - in chronic, II-III - in acute). Displacement of individual teeth, mainly in the frontal part of the mandible, secondary traumatic occlusion.

Radiologically . Complete destruction of the cortical layer at the apex and partially in the lateral areas of the interalveolar septa. Osteoporosis of the spongy substance of the interalveolar membrane within the middle third. Resorption of interalveolar septa up to 1/3 of their height (root length). Expansion of the periodontal gap within the upper third of the interalveolar septa.

II degree.

Complaints. In the chronic course of the process - scanty symptoms - bad breath, slight bleeding and sore gums, displacement and mobility of the teeth, the appearance of gaps between the teeth. At an exacerbation pain and bleeding are considerably expressed, spontaneous bleedings are possible.

Clinical signs. Symptomatic diffuse gingivitis with a pronounced decrease in the height of the gingival papillae and the "gaping" of the interdental spaces. Periodontal pockets 3-5 mm , filled with serous-purulent exudate, granulation tissue grows at the bottom. Massive postponement of over- and under ' gingival tartar. The necks and roots of the teeth are exposed by 2-3 mm , elongation of the clinical crown of the tooth. Hard tissue hyperesthesia . The mobility of the teeth (I-II degree in chronic course, II-III - in acute), is most pronounced in the area of the front teeth. Fan-shaped difference of frontal teeth.

With an acute course and the formation of a bone pocket, the formation of a *periodontal abscess is possible* . Its development is accompanied by almost constant pain in the affected teeth, sharp pain when biting the tooth, redness and swelling of the gums in this area. The general condition may be disturbed, body temperature rises.

Radiologically . Resorption of interalveolar septa within 1/3 to 2/3 of the tooth root length. Destruction of the cortical plate. Significant expansion of the periodontal gap in the middle third of the interalveolar septa. Diffuse osteoporosis of the interalveolar septa. Bone pockets may form near teeth with overhanging fillings.

III degree.

Complaints. In the chronic course, they are reduced to bad breath and significant mobility of the teeth, which causes an aesthetic defect and makes it difficult to eat. With exacerbation - severe pain and bleeding gums, possible deterioration of the general condition (due to intoxication of the body due to significant purulent discharge from periodontal pockets), possible enlargement of lymph nodes.

Anamnesis. Loss or removal of teeth due to their pathological mobility.

Clinical signs. Diffuse symptomatic gingivitis. The gums are congestively hyperemic , bleeding when touched. Periodontal pockets 8 mm or more deep , sometimes reaching the apex of the root. Their content is serous-purulent or purulent, there is a massive growth of granulation tissue in them. Massive postponement of over- and under ' gingival tartar. The necks of the teeth are exposed by 5-8 mm , the elongation of the clinical crown of the

teeth. Hard tissue hyperesthesia . Mobility of teeth of the II-III degree. Displacement of teeth in different directions. Defects of dentitions due to removal of movable teeth. The development of single or multiple periodontal abscesses is possible.

Radiologically . Absorption of alveolar bone reaches 2/3 of the length of the root and more. The cortical plate is destroyed almost throughout. The periodontal fissure is dilated throughout. Osteoporosis of the bone of the interalveolar membrane, which is still preserved. Bone pockets reaching the apex of the root are often identified.

Stabilization of the process appears after a comprehensive treatment. The patient has no complaints. Clear pale pink, tightly covering the roots of the teeth. The roots are exposed depending on the degree of periodontitis . There is no discharge from periodontal pockets and bleeding gums. No dental plaque. Teeth are stable or splinted with permanent splinting structures. These are manifestations of clinical stabilization (remission). Hygienic indicators within the norm. Pisarev-Schiller test is negative. RI is approaching 0. Kulazhenko's test within 50-60 seconds. Clinical and radiological stabilization in addition to clinical well-being characterized by the absence of foci of osteoporosis in , sealing spongy substance (osteosclerosis) of alveolar bone .

Topic 14. Clinic, diagnosis of distrophic periodontal disease. – parodontosis

Is a dystrophic periodontal disease, is quite rare (5-8% of dystrophic-inflammatory periodontal diseases). It begins imperceptibly, develops and progresses slowly, lasts for years.

Characteristic features:

- symptomatic atrophic gingivitis
- exposure of necks and roots of teeth
- formation of wedge-shaped defects
- moderate traumatic occlusion
- lack of periodontal pockets
- pathological mobility does not develop for a long time, it is manifested only in the late (II-III) stages of the disease.

The main radiological symptoms of parodontitis :

- osteosclerosis of the bone of the alveolar sprout
- fine-loop drawing of bone tissue
- uniform decrease in the height of the interalveolar septa (horizontal resorption)
- sclerosis of the tooth cavity, formation of denticles , petrifications
- hypercementosis near the tops of the roots
- cortical plate is stored for a long time

Initial degree.

Complaints. Has scanty clinical symptoms: some patients complain of itching, numbness in the gums, hyperesthesia of the hard tissues of the teeth.

Objectively . The mucous membrane of the gums is pale, there is no normal luster. Blunting of the tops of individual gingival papillae, most often in the area of the front teeth. The gums are dense, painless, do not bleed on examination. A slight (up to 1 mm) exposure of the necks of the teeth, but the violation of the integrity of periodontal of ' unity is not observed. Hypersensitivity of hard tissues to all types of stimuli (chemical, temperature, mechanical). Minor traumatic occlusion. Tooth mobility is not observed . Pisarev-Schiller test is negative.

Radiologically . Areas of osteoporosis are preceded by areas of osteosclerosis. Uniform decrease in the height of the interalveolar septa to ¼ their height. Periodontal fissure without changes. The cortical plate is preserved throughout.

I degree.

Complaints. More pronounced signs of itching, a feeling of aches in the gums, hyperesthesia of the hard tissues of the teeth.

Objectively. Clear pale, dull, dense and painless. Exposure of necks on 1 - 1,5 mm . Gaps in the interdental spaces. Formation of shallow wedge-shaped defects on the front teeth. Traumatic occlusion, overload of individual teeth. Tooth mobility is not observed. Pisarev-Schiller test is negative.

Radiologically . Osteosclerosis of the alveolar bone. Uniform decrease in the height of the interalveolar septa to 1/3 of their height. Periodontal fissure without changes. The cortical plate is preserved throughout.

II degree.

Complaints are similar.

Objectively. Clear pale, dull, dense and painless. Exposure of tooth roots up to 5-6 mm . The interdental spaces gape. Development of wedge-shaped defects on the vestibular surface of the teeth. Hyperesthesia of the hard tissues of the tooth. Slight delay on ' gingival tartar and plaque pigmented. Traumatic occlusion, overload of individual teeth. Tooth mobility can be manifested in the first degree. Pisarev-Schiller test is negative.

Radiologically . Osteosclerosis of the alveolar bone. Uniform decrease in the height of the interalveolar septa to 2/3 of their height. Periodontal fissure without changes. The cortical plate is preserved throughout.

III degree.

Complaints of hypersensitivity of the exposed necks of the teeth. Displacement and divergence of teeth.

Objectively. The gums are dense, anemic, do not bleed - atrophic gingivitis is most pronounced. Slight delay on ' gingival plaque and pigmented coating. The necks of the teeth can be exposed by 5-8 mm . Pathological mobility is either absent at all, or there is mobility of I-II degree. Fan-shaped divergence of front teeth is possible. Wedge-shaped defects develop on the vestibular surfaces of the teeth. Pisarev-Schiller test is negative.

Radiologically . Osteosclerosis of the alveolar bone. Uniform decrease in the height of the interalveolar septa by 2/3 of their height and more. Periodontal fissure without changes. The cortical plate is preserved throughout.

Differential diagnosis is performed with atrophic gingivitis, premature and senile periodontal atrophy.

Topic 15. Clinic, diagnosis of progressive idiopathic periodontal diseases

The development and course of disease does not meet the basic understanding of the clinical course of different pathogenesis of periodontal diseases. Prediction of these diseases as different and quite complex.

Common to idiopathic disease is localization process. Above mentioned disease entities in the clinic are rare, such as 0,5-0,7% in the general structure of the pathology of periodontal tissues, as studied not enough detail.

In the group of idiopathic diseases include symptoms and syndromes of various common diseases, mostly children and adolescents: blood disease, diabetes, eosinophilic granuloma, syndromes Gende-Schiller-Krischena, Papiyona-Lefevre, Osler, disease Letterera-Sive, Ytsenko-Cushing, histiocytosis X.

Classification (Danilevsky MF, 1998):

1. Diseases that accompany the disease blood: leukemia, agranulocytosis, cyclic neutropenia.
2. Histiocytosis X: disease-Letterera Zive, disease Gende-SchillerKrischena, eosinophilic granuloma (Taratynova disease).
3. Diseases of the accompanying metabolic: Niemann-Pick disease, Gaucher disease, a syndrome Papiyona-Lefevre.
4. Periodontal lesions in the case of immune deficiency states are: AIDS, aggressive periodontitis:
- Localized;
- Generalized.
5. Periodontal lesion that is diagnosed with congenital diseases: Down Syndrome, akatalaziya.

General regularities of changes in the body with progressive idiopathic diseases:

1. The development of the disease at an early age, soon after the eruption of primary teeth;
2. Progressive course that leads to loss of milk and permanent teeth;
3. Pathological process is localized in alveolar appendix and other parts of the skeleton and in the organs involved in hematopoiesis (spleen, lymph nodes, etc.).

1.Periodontal syndromes that accompany blood disorder:

1.1. *Leukemia* - a systemic blood disorder that is characterized by progressive hyperplasia and dysplasia of leukocytes in the organs of blood, the appearance of foci ekstramedulary hematopoiesis and maturation process violation.

Over the course:
- Acute;
- Chronic.

Changes in the periodontal tissues and mucous membrane:

- Myeloid form: hemorrhagic diathesis in the form of bleeding in the gums and mucous membranes (petechiae, ecchymosis, hematoma, hemorrhagic blisters), color change interdental papillae and gum edge from bright red to dark, ulcerousnecrotic lesion of the gums and oral mucosa.
- Lymphoid form: hyperplastic process by leykemoid gum tissue infiltration diffuse nature. Gingival hyperplasia more pronounced on the oral surface, and areas that are subject to local traumatic factors. Gingival hyperplasia is usually combined with hypertrophy of the lymphoid system: tonsils, lymphoid structures posterior pharyngeal wall.

1.2. *Neutropenia* - a group of diseases, the main feature of which is permanent or periodic reduction of neutrophils in the blood and bone marrow.

Etiology: genetic vulnerability factors differentiating young forms of neutrophils.

- Hereditary neutropenia;
- Cyclical neutropenia.

Changes in the periodontal tissues and mucous membrane: dystrophininflammatory changes rapidly-progressing nature which occur in milk and permanent occlusion.

1.3. *Agranulocytosis* - hematological syndrome characterized by a sharp decrease or absence of neutrophils in the peripheral blood.

For pathogenesis :

- Myelotoxic;
- Immune.

Changes in the periodontal tissues and mucous membrane: necrotic changes of the primary lesion of the gums with the formation of films or dirty whitish-gray character. The lesion occurs on the background areaktive pale mucous membrane around it is no visible demarcation inflammatory reaction. The main differences of periodontal lesions in case of agranulocytosis is without pus , even in the presence of deep periodontal pockets and bone, the advantage of necrotic phenomena without severe inflammatory reaction.

1.4. *Periodontal syndrome in diabetes –millitus* - it`s complex of symptoms inflammatory-dystrophic changes in periodontal arising on a background of diabetes.

Characterized by:

- Rapid progression;
- Active productive process in the form of granulation;
- Lots of dental layers;
- Funnel-and crateriform type of bone destruction alveolar bone, which does not cover the body jaw.

1.5. *Periodontal syndrome in disease of Izenko-Cushing* (characterized by multiple lesions of endocrine glands with primary pituitary lesion involving staff in the process of adrenal, gonads) – complex symptoms inflammatory-dystrophic changes in periodontal arising against the disease.

Characterized by:

- Rapid progression;
- Bleeding and gum hyperplasia interdental papillae;
- Tendency to abscess formation.

2. ***Histiocytosis*** - a group of diseases of unknown etiology, accompanied by impaired endogenous metabolism and accumulation of histiocytes in metabolic products.

2.1. *Disease-Letterera Zive* - acute progressive systemic histiocytosis, which is typical for the formation of foci of atypical histiocytes proliferation in the skin, bones and internal organs. These diseases development in 2-3 years old. Changes in the periodontal tissues and mucous membrane: changes in periodontal tissues with rapidly progressive dystrophic-inflammatory in nature, characterized by the development of acute catarrhal gingivitis with marked edema

and hyperemia. Rapid accession ulcerous-necrotic process, there are periodontal pockets with abundant granulations. There alveolar bone resorption process, pathological mobility of teeth, leading to tooth loss.

2.2. *Disease Gende-Schiller-Krischena* - chronic progressive systemic histiocytosis, characterized by infiltration of various organs and tissues, histiotsytar atypical elements, characterized by a triad of symptoms:

- Multiple defects in flat bones;
- Exophthalmos;
- Diabetes insipidus.

It occurs mainly in children 4-5 years in adults in very rare cases.

Changes in the periodontal tissues and mucous membrane: changes in periodontal tissues with rapidly progressive dystrophic-inflammatory in nature, characterized by the development of acute catarrhal gingivitis with marked edema and hyperemia, gum edge covered stratification characteristic orange-yellow. Rapid accession ulcerous-necrotic process, there are periodontal pocket.

2.3. *Disease of Taratynov* - a disease characterized by the presence of bone infiltrates consisting of histiotsytarnyh elements, eosinophils, leukocytes and neutrophils and plasma.

Diagnosed in children aged 1 to 15 years. Changes in the periodontal tissues and mucous membrane: changes in periodontal tissues with rapidly progressive dystrophic-inflammatory in nature. Clear become cyanotic, swollen, formed periodontium socio pocket without purulent discharge. As a result of destruction of

alveolar bone formed bone pockets, teeth are moving. After removal of teeth with holes proliferating granulation, bone formation occurs. On X-ray find isolated pockets of destruction of alveolar bone of the jaw bone and body with indistinct contours with no signs of sclerosis. Unlike generalized, localized bone defect in the body of the jaw.

3. Periodontal syndromes that accompany metabolic:

3.1. *Niemann-Pick disease* - a disease caused by hereditary metabolic lipid (sfinholipid), in which the accumulation sfinhomiyelin in the liver, brain, spleen, adrenal glands, kidneys, lymph nodes, skin, blood mononuclear cells. Hereditary disease with autosomal recessive inheritance is rare, mostly in young children. Development of disease associated with deficiency in tissues of a specific enzyme - sfinhomiyelinaza. Clinical manifestations occur in infants less than in older children. The leading symptoms are hepato-splenomegaly, failure of the child of food, intermittent vomiting, sudden weight loss. There are a violation of pyramidal system due to CNS lesions. Changes in the periodontal tissues and mucous membrane: changes in periodontal tissues with rapidly progressive dystrophic-inflammatory in nature. There proliferative processes in the gums, destroyed communications apparatus of milk teeth, appears at ix premature abnormal mobility, leading to premature loss of teeth.

3.2. *Disease of Gaushe* - an inherited disease characterized by accumulation of cells in hlikotserebrozyd of phagocytic mononuclear cells. The disease is inherited by autosomal recessive. Development caused by an inherited deficiency of hydrolytic enzyme hlikotserebrozydazy-β-hlyukozydazy.

The course:

- Acute;
- Chronic.

Changes in the periodontal tissues and mucous membrane: changes gums are rapidly progressive proliferative nature, accompanied by the formation ofperiodontal pockets, resulting in mobility and tooth loss. Radiological findings show focal or diffuse areas of osteoporosis with thinning compact plate.

3.3. *Syndrome Papiyona-Lefevre* - syndrome characterized by progressive alveolar bone lysis (lose) and pronounced cornification palms and soles. The disease is congenital and inherited by autosomal recessive. It is a complex symptomokopleks neuroendocrine and metabolic disorders. The disease develops in early childhood and is characterized keratodermiya palms and soles.

Changes in the periodontal tissues and mucous membrane is characterized by lesions only in alveolar bone process. Changes in the periodontal

tissues with rapidly progressive dystrophic-inflammatory in nature, initially in the form of gingivitis, there is a quick pathological mobility of teeth, the formation of periodontal pockets of exudate and the tendency to abscess formation.

4. Periodontal lesions in immunodeficiency states.

4.1. *AIDS* - a disease agent which is the human immunodeficiency virus that infects T-lymphocytes-helpers (CD4), which leads to immunodeficiency. As a result of developing opportunistic infections caused by saprophytic microflor. Periodontal lesions in AIDS are divided into two groups:

- HIV - gingivitis (catarrhal, ulcerative); catarrhal - characterized by lots of gum lesion almost complete absence of these local stimuli periodontal tissues, the inflammatory process has the form of narrow strips on the marginal part of the ash
- "linear erythema gum." ulcerative - gingivitis occurs at low reactivity, and is a sign of progression of severity of the immune system. Characteristic is the almost complete absence of reactive inflammation of tissues around the necrotic gums. Gingivitis is flabby clinical course and no positive trend in the application of conventional treatment regimens.
- HIV - periodontitis - gets no rapid progressive dystrophic-inflammatory process. Characteristic is a small number of local stimuli in the affected areas. Periods of exacerbation characterized by deterioration of the patient with AIDS, with the acquisition of symptomatic ulcerative gingivitis.

4.2. *Aggressive periodontitis* - the presence of periodontal lesions, characterized by a noticeable rate of progression of the disease process. The criterion for aggressive periodontitis is (R. Page, H. Schroeder, 1982):

- A young age from puberty to 35 years;
- The rapid destruction of tools epithelial attachment to the further development of destructive changes in periodontal;
- The number of dental plaque does not meet severity of illness;
- Destruction of periodontal tissues is generalized in nature;
- Aggressive periodontal not a continuation of the generalized form of juvenile periodontitis, and there is actually in period;
- Loss of alveolar bone process is very fast;
- During the active phase of the observed manifestations of acute inflammatory process in remission symptoms of inflammation almost never occur;
- Species composition of microflora of periodontal pockets is variable and does not meet the severity of the disease;

5. Periodontal lesion that is diagnosed with congenital diseases:

5.1. *Down's syndrome* - chromosomal disease, which is based on abnormal karyotype as trisomy of chromosome 21 pair. The clinical picture of this syndrome

represented a combination of mental retardation and a set of neurological, cardiac and changes in the musculoskeletal system.

Changes in the periodontal tissues: changes in periodontal tissues diagnosed in 90% of patients presented inflammatory and inflammatorydegenerative processes

5.2. *Akatalaziya* - hereditary abnormality of exchange, which is characterized by the absence of catalase enzyme in the blood and tissues. The disease is inherited by autosomal recessive. Changes in the periodontal tissues: It`s occur an early age and progresses rapidly. The clinical picture presented catarrhal gingivitis gum with his transition into ulcerous-necrotic or gangrenous process. Because bone lesions have deep pockets, bone, resulting in rapidly progressive loss of teeth.

Topic 16. General principles of treatment of patients with periodontal diseases. Preparing a treatment plan.

Treatment Strategies

In order to determine the appropriate treatment or combination of treatments that best fit their unique situation, patients must work closely with their dental professionals.

Treatments for periodontal disease may include:

- Professional cleaning to remove tartar and plaque buildup
- Scaling and root planing to the depth of the pockets, sometimes with local anesthesia
- Use of a locally or systemically applied antibiotic drug to reduce the bacterial load in the pockets and help lead to reduction of the inflammation
- A daily oral hygiene treatment plan for between professional visits
- Surgical repair of the diseased tissues
- Removal of the damaged tooth

Local antimicrobial delivery into periodontal pockets may be further classified as providing either nonsustained or sustained subgingival drug delivery. Nonsustained subgingival drug delivery provides high pocket concentrations of the antimicrobial agent for only short time periods. Subgingival irrigation with antiseptic agents lacking substantivity for oral tissues (povidone-iodine) is examples of nonsustained subgingival drug delivery. Sustained subgingival drug delivery provides retention of the within periodontal pockets. Controlled drug release can be provided with subgingival irrigation of agents intrinsically substantive for tooth root surfaces (aqueous tetracycline) or pocket placement of commercial antimicrobial fibers, gel or films.

Locally applied antimicrobial agents should be safe, stable, substantive, efficaceous, cost effective, patient compliant, achieve effective concentrations. Factors affecting the bio-availability of an antimicrobial agent are solubility, pH and ion-binding capacity, delivery vehicle-drug interaction and metabolism.

Supragingival irrigation

Home irrigation devices allow the patient to deliver medicaments into the periodontal pocket at home on more frequent basis than is practical with professional gingival irrigation,,the ability of the device to gain a access to the depth of periodontal pocket and the manual dexterity of the patients are the limiting factors . The mechanism of action of irrigation occurs through the direct application of a pulsed or steady stream of water or other solution. Studies have found pulsation and pressure to be critical components of an irrigation device. The pulsation creates two zones of hydrokinetic activity. The impact zone is where solution initially contacts the surface and flushing zone is where solution reaches into the subgingival sulcus. The outcome of hydrokinetic activity is subgingival

penetration . Home irrigation devicees include supragingival and subgingival devics. Irrigation with a standard jet tip is generally called supragingival irrigation. Tip is placed coronal to gingival margin. Oral irrigation devices with traditional jet tip results in greater access of medicament to periodontal pocket when compared with rinsing alone. A 90 degree angle of application to the tooth surface provides 71% penetration in shallow pockets. These devices may be useful in delivering of medicaments in cases of gingivitis with shallow pocket depths, they are less useful in delivering medicaments in periodontitis patients with deeper pockets. They are mainly used for full mouth irrigation.

Subgingival irrigation

Irrigation with the soft, site specific tip is often called subgingival irrigation. This also refers to placement of tip, which is placed slightly below the gingival margin. These devices generally include blunt end metal cannula that the patient inserts into the periodontal pocket, this increases the depth of penetration of fluid but has the potential for injury owing to the metal tip. The subgingival tip is generally used for the localized irrigation of specific site, such as a deep pocket, furcation, implant, or crown and bridge. Studies have shown that it can deliver solution into a pocket of 6mm or less up to 90%of its depth. In pockets greater than 6mm, the depth of penetration has been shown to 64%. Professional subgingival irrigation device include a wide range of powered and manually operated irrigations. Irrigation using a syringe with blunt end cannula attached to an oral irrigator can penetrate to 71.5% of the pocket depth in pocket 3.5to 6 mm deep. Vehicles tested for sustained periodontal pocket delivery of antimicrobial agents include solution pastes, hollow fibre, acrylic strips, monolithic fibres, resorbable cellulose, collagen and biodegradable gel

Local antimicrobial agents

A local route of drug delivery can attain 100-fold higher concentrations of an antimicrobial agent in subgingival sites compared with a systemic drug regimen. For example, local placement of a tetracycline- releasing ethylene vinyl acetate monolithic fiber can yield tetracycline concentrations in excess of 1300 Fg/ml in gingival crevicular fluid over 10 days. In comparison, repeated systemic doses of tetracycline- HCl can only provide tetracycline levels of 4-8 pg/ml in gingival crevicular fluid. Disadvantages of local antimicrobial treatment of periodontitis include difficulty in placing therapeutic concentrations of the antimicrobial agent into deeper parts of periodontal pockets and furcation lesions. Personal application of antimicrobial agents by patients as a part of their home self-care procedures is frequently compromised by the patient’s lack of adequate manual dexterity, limited understanding of periodontal anatomy, and poor compliance and performance with recommended procedures.

The task of professionally applying local antimicrobial agents in periodontitis patients with numerous advanced lesions distributed throughout their

mouth is time-consuming and labor-intensive. Nonsustained subgingival drug delivery is limited by a only brief exposure of the target microorganisms to the applied antimicrobial agent. Antimicrobial agents locally applied into periodontal pockets do not markedly affect periodontal pathogens residing within adjacent gingival connective tissues and on extra-pocket oral surfaces (tongue, tonsils and buccal mucosa), which increases the risk of later reinfection and disease recurrence in treated areas. Local agents used for irrigation includes chlorhexidine, povidine iodine, stannous fluoride ,hydrogen peroxide.

Antibiotics

The physical removal of biofilm has proven to be the most effective method for treating periodontal disease. The use of adjunctive antibiotic therapy, either systemic or topical, is controversial. Some studies show superior results with antibiotic use while others show no clinical difference. There is a general consensus that antibiotics should not be used as a monotherapy in the treatment of periodontal disease. Antibiotics as a stand-alone treatment are ineffective at diminishing intact subgingival biofilms.

Antibiotic therapy is generally used as a follow up treatment after conventional mechanical therapy. Aggressive periodontitis may use systemic antibiotics as an adjunctive therapy.

There are many systemic antibiotics on the market. The most commonly used include tetracycline, ciprofloxacin, metronidazole and the penicillins, including amoxicillin and amoxicillin/clavulanate acid (Augmentin®). Tetracycline is bacteriostatic, targets both gram positive and gram negative organisms, and has become bacterial resistant. Ciprofloxacin is bactericidal, targets gramnegative rods, and may cause gastrointestinal discomfort. Amoxicillin and Augmentin are both bactericidal, with Augmentin targeting a more narrow spectrum than amoxicillin. Augmentin was developed due to amoxicillin's bacterial resistance from penicillinase enzyme sensitivity.

Of the many systemic antibiotics available, there is no consensus as to an ideal dose and duration. The choice of antibiotic should be made on an individual basis. In addition to serious adverse effects, like anaphylactic shock, microbial resistance is a growing concern. Other issues with oral antibiotic administration are patient adherence and adequate absorption from the gastrointestinal tract.

Understanding that the periodontal disease process may be initiated by bacteria but the individual's host response was critical to the progression of this disease led to the FDA approval of doxycycline at a sub-antimicrobial dose (20mg twice daily). When administered at this low dose, doxycycline does not cause the long term side effects seen with other systemic antibiotics. Randomized double blind placebo controlled trials demonstrated reduction in probing depths, improvement in clinical attachment levels and decreased bleeding on probing when used as an adjunct with scaling and root planing.

A recent review evaluating non-surgical chemotherapeutic strategies for the management of periodontal disease determined that "systemic antibiotics reach the

periodontal tissues by transuduction across serum, then cross the crevicular and junctional epithelia to enter the gingival sulcus." By the time the systemic antibiotic reaches the gingival sulcus it no longer has an adequate concentration to achieve the desired antimicrobial effect. This supports the fact that the mechanical disruption of biofilm must be included in the treatment of periodontal disease.

Atridox® is a 10% doxycyline hyclate gel and is prepared by mixing powder and liquid from two syringes. The antibiotic is administered into the gingival sulcus through a cannula. Absorption lasts up to 21 days, while therapeutic drug levels in the gingival crevicular fluid start to decline at 7 days. The most notable drawback is the high level of clinician skill needed to deliver this therapy as the material tends to come out of the pocket as the syringe is being pulled out of the sulcus. The majority of the time, more than one site can be treated depending on the depth and size of the pockets.

Arestin® is comprised of spheres embedded with 2% minocycline HCl that is slowly released and holds the therapeutic dose in the gingival crivicular fluid for 14-21 days. The most notable drawback for Arestin is the delivery dose. The syringe holds pre-set doses that may not be sufficient for every site. This results in the need to reapply in the same pocket.

Currently, resorbable antibiotics such as Atridox® and Arestin® are the topical antibiotics of choice.The American Academy of Periodontology (AAP) supports that local adjuncts, when compared with scaling and root planning alone, provide limited improvement. Locally administered antibiotics still require a strict health history review to verify there are no known allergies. Even though these medications are applied topically, as opposed to oral administration, the same precautions apply.

Antiseptics

Unlike topical controlled-released antibiotics, oral rinses do not penetrate deep into the gingival sulcus. Despite this limitation they do show benefit when used adjunctively for gingival inflammation. Oral rinses are also of great value in post surgical healing. Substantivity is a crucial component when considering the effectiveness of a mouthrinse. This term refers to the adherent qualities of a mouthwash and its ability to be retained. Saliva has a natural flushing property making it difficult to maintain an antimicrobial effect. Research shows a significant antibacterial effect up to 7 hours after mouthrinses with high a substantivity property.

First generation antimicrobials include phenolic, sanguinarine, qua- ternary compounds. Listerine® and its generics are phenolics, which possess the only ADA Seal of Acceptance among the first generation antimicrobials. Listerine contains 26.9% alcohol, alters the bacterial cell wall, and has 36% gingivitis reduction. Cepacol® and Scope®, quaternary ammonium compounds, contain 14% and 18.9% alcohol respectively, increase bacterial cell wall permeability causing cell lysis, and reduces gingivitis approximately 15%.1

Second generation antimicrobials include cetylpyridinium chloride (CPC) and chlorhexidine (CHX). A commercial name for CPC is Crest® Pro-Health®, which contains 0.07% CPC. Bacteria cells are killed by cellular pressure, resulting in a similar efficacy as Listerine. Chlorhexi- dine has many commercial products including the availability of a nonalcoholic version by Sunstar Americas, Inc. Peridex® by 3M Espe and Periogard® by Colgate® Professional are two examples of popular chlorhexidine-based products. Their active ingredient is 0.12% chlorhexidine. Cell death results from altered osmotic equilibrium. CHX efficacy in the reduction of certain aerobic and anaerobic bacteria has been shown to be as high as 97% after 6 months of use. CHX has 29% gingivitis reduction. The gingivitis reduction percents listed above for both first and second generation antimicrobials are based on efficacy data published by manufacturers.

Other antimicrobials include oxygenating, chlorine dioxide, and zinc chloride agents. Peroxyl® is an oxygenating agent with the active ingredient of hydrogen peroxide. It has anti-inflammatory properties as well as a bubbling action to clean and alleviate discomfort. Short term studies have produced controversial findings. Oxyfresh®, a 1% chlorine dioxide agent, has minimal plaque reduction. It is a stable, free radical and an oxidant with algicidal, bactericidal, cysticidal, fungicidal, sporicidal, and viricidal properties. Oxyfresh is primarily used for the treatment of halitosis. Breath Rx® is a zinc chloride agent designed to odorize sulfhydryl groups with zinc ions. It claims to be a scientific bad breath treatment specifically designed to help treat the causes of bad breath and the symptoms.

Antimicrobial mouth rinses have been linked to several side effects; some more serious than others. First generation compounds like Listerine can cause a burning sensation and bitter taste. Chlorhexidine can cause supragingival calculus build-up and staining. Research has demonstrated permanent damage to enamel through erosive pH levels and abrasive antimicrobial toothpastes.1 Carcinogenic changes have been linked to the use of oxygenating agents and mouth rinses containing alcohol.

Nutraceuticals

As antibiotic resistance becomes more of a concern, health care pro- viders looking for alternate adjunctive periodontal therapies for their patients. Some examples of nutraceuticals include herbal and nutri- tional supplements and the future of this type of therapy is promising. There are approximately 500,000 plant species, with only 1% having been photochemically investigated. Herbal plant extracts have been shown to reduce the level of biofilms influencing the level of bacterial adhesion. This has shown results with the reduction of periodontal disease. Some herbs such as Coptidis rhizome extract and Hamamelis virginiana, are used as bactericidal agents against oral bacteria while others such as cranberry, Polygonum cuspidatum and Mikania are used to inhibit adhesion.

The use of probiotics in the control of periodontal pathogens is emerging. Probiotics are “live microorganisms, which when administered in adequate amounts confer a health benefit on the host.” Simply put, they are healthy bacteria

that displace unhealthy or pathogenic bacteria. A reduction in gingivitis and dental plaque has been shown with the administration of L. reuteri Prodentis® gum chewed twice daily in patients with moderate to severe gingivitis.14 GUM® Perio Balance®, marketed by Sunstar Americas, is a once daily lozenge with L. reuteri Prodentis® that claims a reduction in moderate to severe plaque and bad breath. EvoraPlusTM from Oragenics, Inc. is another new probiotic for oral health and is used once daily. This supplement contains a combination of three bacterial strains Streptococcus uberis KJ2, S. oralis KJ3, and S. rattus JH145, and claims a reduction in periopathogens within the periodontal pocket.

Topic 17. Professional hygiene of the oral cavity, its components, professional oral hygiene products, and methods for removing dental deposits.

Almost all middle-aged people suffer from inflammation of the gums. Currently, the number of dentists, who consider professional hygiene as a powerful stage in the fight against modern dental diseases is significantly increasing. In the corresponding literature there is no single terminology that objectively characterizes the dental deposits. Under the same name, often mean different structural formations.

All dental deposits can be grouped as follows:

Non-mineralized (soft) dental deposits.

- Cuticle;
- Pelicula;
- Dense dental deposits (dental plaque);
- Soft dental deposits.

Mineralized dental deposits.

- Supragingival calculus;
- Subgingival calculus.

CUTICLE or reduced epithelium of the enamel organ, shortly after the tooth eruption is lost, therefore, does not play a significant role in the physiology of the tooth.

PELICULA (acquired cuticle) is formed on the surface of the tooth shortly after its eruption, is a derivative of glycoproteins of the oral cavity. When removing the pelicula by abrasive method it is quickly restored if the tooth is in contact with saliva (20-30 minutes). The pelicula is an unstructured formation, which is tightly fixed to the surface of the tooth. Bacteria in the pelicle are not found. The diffusion and permeability of the surface layer of the enamel depends on the state of the pelicule, the change in the composition and properties of the pelicule may contribute to the development of caries.

DENTAL PLAQUE is located above the tooth pelicula, it is colorless, therefore, coloring solutions are used for its detection. The dental plaque is formed by adsorption of microorganisms on the surface of the enamel, tightly attached to it and grows due to the permanent layering of new bacteria. In addition, it contains epithelial cells, leukocytes and macrophages. In dental plaque formation, carbohydrates play a major role, which contribute to the adhesion of the plaque to the tooth surface.

SOFT DENTAL PLAQUE clearly visible without coloring with special solutions. It accumulates at night, during the rest of the speech and chewing

apparatus, in persons who do not take regular care of the oral cavity. Soft plaque, unlike dense (mineralized), does not have a permanent structure. It consists of organic and inorganic substances that settled on the surface of the enamel as a result of the destruction of the cell congregation of envelope epithelium, which are rejected, the oral mucous membrane, leukocytes, microorganisms, residues of food, dust. Soft plaque is the cause of the smell of the mouth, changes in taste, as well as the main center of mineralization and the formation of solid dental deposits.

MINERALIZED DENTAL DEPOSITS (dental calculus) is a hardened mass that forms on the surface of natural and artificial teeth, as well as dentures. Depending on the ratio with the gums edge, supragingival and subgingival dental calculus are marked out. Supragingival dental calculus is located above the crest of the gingival margin, it is easy to detect on the teeth surface. It is usually white or whitishyellow, solid or clay-like consistency, easily detached from the tooth surface by scratching. Its color often depends on the action of tobacco or food pigments. Most
commonly, supragingival dental calculus is localized on the cheek surfaces of the upper molars, on the tongue surfaces of the anterior teeth of the mandible. Supragingival dental calculus refers to the salivary type (formed from salivary mineral substances).

Subgingival dental calculus is located under marginal gums and usually in periodontal pockets. Subgingival calculus is not visible at the visual inspection of the oral cavity. Careful periodontal probe is required to determine the location and length of subgingival calculus. It is usually dense and solid, dark brown or greenish-black color and tightly attached to the tooth surface. Frequently, supraand subgingival calculus are found in patients. Subgingival calculus refers to a serum type (since it has been proven that the source of minerals for it is gums liquid that resembles serum).

Professional oral hygiene - a system of therapeutic and prophylactic measures performed in a dental clinic, aimed at prevention of the onset and progression of oral cavity diseases.

There are four stages of professional hygiene of the oral cavity:

- Controlled tooth brushing;
- Removing toothpatches;
- Grinding and polishing;
- Fluorination.
- Controlled cleaning

Thoroughly examination of the patient with compulsory calculation of caries intensity indices, hygienic index (IG), assessment of the condition of the oral

mucous membrane of the cavity and periodontal tissues. It is necessary to determine the type of bite teeth, the presence of active risk factors for dental diseases.

Next, it is necessary to form a positive motivation in the patient to follow the rules of oral hygiene. It is important to inform the patient that the success of treatment of periodontal disease, preservation of healthy teeth, fillings and restorations depends on the quality of oral care.Then it is necessary to form a positive motivation for the patient to comply with the rules of hygiene of the oral cavity. It is important to inform the patient that the success of the treatment of periodontal diseases, the maintenance of healthy teeth, fillinfs and restorations depends on the quality of oral health care. It is advisable to conduct a controlled toothbrush cleaning. The patient brushes the teeth with an individual toothbrush, and then is carried out staining of the remaining plaque. (Plaviso (Voco), a tablet «Dent» (Japan), «Dinal» (Russia), etc. are used as a liquid plaque indicator.). Simultaneously with controlled toothbrush cleaning procedure, the sequence of movements of the toothbrush, gum massage are memorized. The selection of personal hygiene products is carried out, recommendations for selecting a toothbrush, toothpaste, tools for cleaning interdental gaps and rinses are given. In addition, recommendations for proper nutrition and possible use of chewing gum are given.

Removal of tooth patches. First of all, it is mandtory to carry out irrigation of the oral cavity with solutions of antiseptics (chlorhexidine, furacilin, metragyl, propolis, etc.) or infusions of medicinal herbs (St. John's wort, chamomile, sage, calendula, eucalyptus).

Methods of removing dental deposits:

- mechanical (instrumental)
- chemical
- hardware
- combined.

Tooth patch must be removed from all dental surfaces until they become smooth. After removal - polish the hard tissues of tooth. Polished surface of the crown, the cervix, the root of the tooth must be covered with fluoride-containing varnish or applications of remineralizing solutions. Depending on the condition of the parodont, a treatment band (self-hardening, not hardening, on a glutinous basis) to the area of the gum are applied. When removing tooth patches, safety precautions should be followed (safety glasses, gauze mask, rubber gloves).

Topic 18. Manual and hardware methods for removing dental deposits. Rules of the apparatus, contraindications, advantages, disadvantages of the hardware method.

The removal of all stains, deposits and concrements comprises the first phase initial therapy. It is also an important preventive measure in the healthy periodontium, and the
most significant post-operative measure following completion of periodontitis therapy. Thorough tooth cleaning is performed during each recall appointment (maintenance
phase).

The prevention/treatment/maintenance therapy trio "without end" is the sole responsibility of the dental hygienist. It also demands rationalization, standardization and work simplification, as well innovation in the development of new instruments (ultrasonic devices, Air-Scaler etc.).

Difficult-to-remove stains resulting from medicaments (e. g., chlorhexidine), tobacco, beverages (tea, wine) and foodstuffs as well as dental plaque can be removed using instruments
that provide a water-powder spray (e. g., Cavitron- Jet). The powder that is used in the water spray must be minimally abrasive for dentin and restorative materials (Iselin et al. 1989). Furthermore, the spray should never be directed perpendicular to the tooth surfaces, and should usually be used only on enamel, with constant movement of the tip. Such devices do not guarantee perfect cleaning in interdental spaces or niches. The spray with normal abrasive
powder should not be directed into pockets. With the new, "mild," minimally abrasive agents and fine tips, effective cleansing can be achieved, in certain circumstances, even subgingivally (e. g., glycine powder from Espe, with the EMS Airflow Handy 2; p. 282; Petersilka et al. 2002).

After the removal of soft deposits, calculus becomes visible. Calculus is an excellent substrate for plaque accumulation and must be completely removed. Numerous power-driven instruments are available: Ultrasonic apparatus (e. g., Cavitron) as well as Air-Scaler that can be attached to the air-water supply of the dental unit (e. g., Titan-S, Satelec; Sonicflex KaVo etc.; Hermann et al. 1995). However, the most important and most precise means for removal of concrements remains: hand instruments.

Powder-Water Spray Device (Cavitron-Jet)

The powdered abrasive consists primarily of sodium bicarbonate (NaHCO3) which can remove tough deposits and stains when
used with a water spray. The water-powder spray requires use of a high-speed evacuator.

Right: “Jet Shield” This “mini-evacuator” is affixed directly to the working end of the Cavitron-Jet.

Stabilized Power System (SPS) with Ultrasonic Scaler Tips (Cavitron Thru Flow Inserts— TFI)

In modern instruments, the water coolant is directed through the instrument tip in a groove on the instrument head. Ultrasonic scalers work at between 25,000–50,000 cycles per second, with very small amplitudes.

Right: Various ultrasonic scaler tips; from left to right: TFI-1000, TFI-9, TFI-1, TFI-7.

Air-Scaler (Titan-S, “Sonic Scaler”)

The air-scaler has a regulable frequency of maximum 6,000 Hz and thus is considerably slower than an ultrasonic instrument. The motion of the tip of the instrument

is between 0.08–0.20mm; relatively slow.

Right: Three tips for the Titan-S device.

Additional manufacturers:

• KaVo • Satelec, and Others

Supragingival Tooth Cleaning—Hand Instruments, Prophy Pastes

In addition to ultrasonic devices, hand scalers and curettes remain the most important instruments for periodontal therapy and prophylaxis. For the removal of soft deposits

and stains, hand instruments are enhanced by the use of brushes, rubber cups and polishing strips along with cleaning and polishing pastes.

It is not the manufacturer that is critical for successful treatment, rather the shape of the instrument, especially its degree of sharpness, and above all the manual dexterity of the dental hygienist (scaling technique)! For the removal of supragingival deposits, chisels, straight and angled scalers and also lingual scalers are effective. In premolar and molar segments, also on difficult-to-reach areas, grooves and depressions on the crown, as well as exposed root surfaces, the removal of supragingival concrements may require curettes in addition to scalers, usually without anesthesia.

Scalers

For supragingival calculus removal and for concrements that are located only a few millimeters below the gingival margin, sharpedged, pointed scalers in various shapes are indicated:

- Zerfing Chisel ZI 10 (white)
- Zbinden Scaler ZI 11, 11 R+L (blue), straight and paired
- Lingual Scaler ZI 12 (black)

Right: Working end of the Zerfing chisel (45° sharpening angle!) and the lingual scaler.

Curettes

For difficult-to-reach areas and for subgingival accretions, the scaler armamentarium must be enhanced by curettes:

- Universal Curettes ZI 15 (yellow) 1.2mm wide
- Anterior Curettes GX 4 (orange), Deppeler
- Posterior Curettes M 23 A (red); both are ca. 0.95mm wide; Deppeler

Right: Working ends of a pair of universal curettes.

Standardized Prophy Pastes—RDA

Prophy pastes are available according to abrasiveness. The standardization is achieved on the basis of dentin abrasion, measured by radioactivity.

All are fluoridecontaining: RDA Value Abrasiveness Color

- 40 mild yellow
- 120 normal red
- 170 moderate green
- 250 heavy blue

Right: Finger cups with color-coded prophy pastes.

Topic 19. Local medicamentous treatment of periodontal diseases, group of drugs, methods of application, indications.

Drug therapy plays an important role in the local treatment of periodontitis. Rational and timely use of it makes it possible to reduce or eliminate inflammatory processes in periodontal tissues, to influence the microflora of periodontal pockets, to improve trophic and metabolic processes, to prevent the progression of the pathological process in the periodontium. The choice of drugs depends on the form of symptomatic gingivitis, the presence of microflora in periodontal pockets. It is also necessary to take into account the patient's possible intolerance to pharmacological agents and comorbidity.

Requirements for dosage forms of drugs

a. Not to irritate the periodontium and the mucous membrane of the oral cavity;
b. To weaken the infection of periodontal pockets;
c. To stimulate the regeneration and healing of periodontal pockets;
d. To strengthen the resilience of the periodontium;
e. To eliminate hypoxia.

The basic principle of drugs effect

a. Effect on microflora;
b. Reduced vascular permeability;
c. Sclerosing of the tissue;
d. Dilution of non-viable tissues, purulent exudate;
e. Stimulation of regenerative and metabolic processes.

Groups of drugs used for local treatment of periodontitis:

• Antiseptics;
• Antibiotics;
• Anti-inflammatory drugs;
• Vitamins;
• Enzymes;
• Biogenic stimulants;
• Drugs containing calcium, phosphorus, fluorine.
• Antihistamines and antiallergic drugs.

Antiseptic drugs are widely used in the local treatment of periodontitis as disinfectants, anti-inflammatory, deodorizing agents in the form of applications, instillations, irrigation, oral baths. Atomic oxygen in antiseptics enhances the bactericidal effect. Free chlorine is actively working with the microorganisms. Gram-positive and gram-negative bacteria, viruses, amoebas, fungi are sensitive to chlorinated drugs.

Antimicrobial drugs have broad spectrum of action, affecting strains of microbes resistant to antibiotics, have bacteriostatic and bacteriocidal effect on gram-positive and gram-negative bacteria.

In the presence of fungal microflora in periodontal pockets, antifungal drugs are used. In the presence of purulent exudate in periodontal pockets, broadspectrum antibiotics are used, in the form of solutions, suspensions, water-oil emulsions, pastes.

To enhance and prolong the action of antibiotics, they are used in combination with sulfanilamides.

Anti-inflammatory drugs are widely used in periodontal practice. There have been developed drugs consisting of equal parts of antibiotics, anti-inflammatory drugs and vitamins. Steroids are used less frequently, according to indications (only in exacerbation phase, since they suppress local immunity..

Phytogenic drugs are characterized by anti-inflammatory, deodorizing, analgesic effect. They are used in the form of solutions, decoctions, suspensions for irrigation applications and instillations in periodontal pockets. Some of them also have a sclerosing effect for the treatment of concomitant hypertrophic gingivitis.

In order to eliminate metabolic and microcirculation disorders in periodontal tissues, *vitamin* preparations are used in the local treatment of periodontitis. The reasonable use of antioxidants in the complex treatment of periodontitis includes the use of vitamin A and vitamin E in a mixture of vegetable oils.

To regenerate bone tissue, appropriate growth stimulants are used, which are either introduced into the periodontal pockets or used for application in the form of pastes. In order to stimulate reparative processes in the periodontium, derivatives of pyrimidine bases, which are characterized by anabolic and anti-catabolic activity, are used. These drugs have no toxic effect. They are used in combination with antibiotics, vitamins.

In the process of local treatment of periodontitis, the drugs introduced into the periodontal pockets are quickly washed out of them, which reduces the therapeutic effect of the drugs. After surgical interventions, such as curettage of periodontal pockets, the wound surface is practically not protected from injuries when eating and from infection with the contents of the oral cavity. These disadvantages can be avoided by using periodontal dressings.

Therapeutic periodontal dressings are used for the deposition of drugs in the gums, periodontal pockets, interdental spaces in medical treatment and after surgical treatment of periodontal diseases. They usually contain a variety of drugs,

which expands the possibilities of drug treatment. The basis of most medical dressings is zinc oxide, dentin, white clay.

CONCLUSIONS

By complementing the study at the Faculty of Dentistry with the analysis and study of the information array of this study guide, each student will be able to achieve the goal of studying the discipline of "Therapeutic Dentistry", namely to be ready to work in a dental clinic, to develop the ability to diagnose and treat periodontal diseases.

CONTROL TASKS AND QUESTIONS

1. The purpose and objectives of local drug therapy in the treatment of generalized periodontitis.
2. Preparations of plant origin. Mechanism of action, indications, representatives, method of application.
3. Antiseptics. Mechanism of action, indications, representatives, method of application.
4. Nonsteroidal anti-inflammatory drugs for topical use in the treatment of periodontal tissue diseases. Mechanism of action, indications, representatives, method of administration.
5. Antienzyme drugs (proteolysis inhibitors, anticoagulants) for topical use in the treatment of periodontal tissue diseases. Mechanism of action, indications, representatives, method of administration.
6. Keratolytics, cytostatics for topical use in the treatment of periodontal tissue diseases. Mechanism of action, indications, representatives, method of administration.
7. Enzyme preparations for topical use in the treatment of periodontal tissue diseases. Mechanism of action, indications, representatives, method of administration.
8. Antiprotozoal and antifungal agents for topical use in the treatment of periodontal tissue diseases. Mechanism of action, indications, representatives, method of administration.
9. Keratoplastics for local use in the treatment of periodontal tissue diseases. Mechanism of action, indications, representatives, method of application.
10. Antibiotics for topical use in the treatment of periodontal tissue diseases. Mechanism of action, indications, representatives, method of administration.
11. Specify the types of mucogingival interventions on periodontal tissues and indications for their implementation.
12. State the indications for gingivotomy, gingivectomy.
13. Name the principles of preparation of patients before surgical interventions on periodontal tissues.
14. Name the stages of surgical methods of treatment of periodontal diseases.
15. Determine the tactics of managing a periodontal patient in the postoperative period.
16. Make a choice of surgical interventions for periodontal tissue diseases.
17. Identify indications and contraindications for surgical treatment of periodontal diseases.
18. To make a plan of preoperative preparation.
19. Provide adequate anesthesia.

20. To know the technique of frenulotomy, plastic surgery of the short frenulum of the tongue and lips, vestibuloplasty, gingivoplasty, curettage, gingivotomy, gingivectomy, flap surgery, directed tissue regeneration.
21. Provide recommendations to periodontal patients in the postoperative period.
22. To determine the indications and contraindications for orthopedic and orthodontic interventions in periodontal diseases.
23. Identify traumatic occlusion in the oral cavity of a patient with periodontitis
24. To know the technique of selective grinding, splinting of mobile teeth
25. Provide recommendations for oral hygiene after orthopedic and orthodontic interventions for periodontal disease.
26. Basic principles of treatment of patients with generalized periodontitis.
27. The concept of complex treatment of patients with generalized periodontitis.
28. Types of treatment of patients with generalized periodontitis.
29. The main stages (Phases) of complex treatment of a patient with generalized periodontitis.
30. The main points of the plan of complex treatment of a patient with generalized periodontitis
31. Indications for surgical, orthopedic and physiotherapeutic treatment of a patient with periodontal tissue pathology.
32. Local drug therapy as an element of complex treatment of a patient with generalized periodontitis of chronic course. The main objective.
33. General pharmacotherapy in the complex treatment of patients with generalized periodontitis. The main objective.
34. Surgical methods of treatment in the complex therapy of a patient with generalized periodontitis. The main objective.
35. Orthopedic methods of treatment in the complex therapy of a patient with generalized periodontitis. The main objective.
36. Physiotherapeutic methods of treatment in the complex therapy of a patient with generalized periodontitis. The main objective.
37. Define the concept of diet and diet therapy.
38. Indicate the role of food in the formation of a full load on the dentition.
39. Name the rules of healthy eating.
40. Determine the role of a balanced diet in the diet therapy of patients with periodontal disease.
41. Name radioprotective foods in the diet of patients with periodontal disease.
42. Define the concept of "Physiotherapy of periodontal patients".
43. List the benefits of physiotherapy treatment
44. List the types of therapeutic physical factors

45. What determines the reaction of the body to the action of the physical factor?
46. What are the stages of this reaction?
47. List the mechanisms of formation of the body's response to the action of physical factors?
48. What are the general contraindications to physiotherapy?
49. Name the general principles of treatment of periodontal patients.
50. Specify the stages of treatment of patients with inflammatory, inflammatory-dystrophic and dystrophic lesions of periodontal tissues.
51. Name the features of elimination of local traumatic factors in patients with gingivitis, generalized periodontitis and periodontal disease.
52. Indicate the features of professional oral hygiene in patients with gingivitis, generalized periodontitis and periodontal disease depending on the clinical situation.
53. Name the medicines for local treatment of patients with gingivitis, generalized periodontitis and periodontal disease, taking into account the principles of treatment of periodontal patients.
54. Specify the features of surgical interventions on periodontal tissues in patients with gingivitis, generalized periodontitis and periodontal disease.
55. Specify the features of eliminating traumatic occlusion in patients with gingivitis, generalized periodontitis and periodontal disease.
56. Name the features of the use of physiotherapeutic methods of treatment in patients with gingivitis, generalized periodontitis and periodontal disease.
57. Specify the features of the general treatment of patients with various forms of gingivitis, generalized periodontitis and periodontal disease, taking into account the principles of treatment of periodontal patients.
58. Name the features of diet therapy in patients with dystrophic-inflammatory and dystrophic changes in periodontal tissues.
59. What are the general principles of treatment of patients with periodontal tissue diseases?
60. Name the criteria for successful treatment of periodontal diseases.
61. Give the mistakes that are possible in the preparation of a treatment plan for a periodontal patient and its realization.
62. ization of it.
63. Give errors and complications in the appointment of pharmacotherapy.
64. What complications are possible with the use of systemic drug therapy?
65. What are the features of clinical manifestations of periodontal disease in patients with endocrine system pathology?

66. Specify the features of clinical manifestations of periodontal disease in patients with cardiovascular disease.
67. Name the features of clinical manifestations of periodontal disease in patients with pathology of the digestive system.
68. Specify the features of clinical manifestations of periodontal diseases in patients with pathology of the hematopoietic system.
69. Name the features of clinical manifestations of periodontal diseases in patients with immunodeficiency states.
70. List the public preventive measures.
71. Highlight the purpose of sanitary and educational work.
72. List the forms of sanitary and educational work
73. List the indices that should be determined during the examination of periodontal patients.
74. Explain the concept of individual oral hygiene.
75. Explain the concept of professional oral hygiene.
76. Describe the concept of prevention of periodontal disease.
77. What is the etiological approach to the prevention of periodontal disease?
78. How is realized pathogenetic prevention of periodontal disease?
79. Specify the levels of prevention of periodontal tissue diseases.
80. What are the types of prevention of periodontal disease?
81. What are the stages of the algorithm of oral hygiene in periodontal patients?
82. What are the features of the algorithm of individual oral hygiene in gingivitis (catarrhal, hypertrophic, ulcerative-necrotic)?
83. What are the features of the algorithm of individual oral hygiene used in periodontitis and periodontal disease?
84. What are the features of individual oral hygiene in various diseases of periodontal tissues burdened by somatic pathology?
85. What is the role and importance of individual oral hygiene in the prevention of gum disease?
86. What is the role of professional oral hygiene in the development of periodontal diseases?
87. What personal oral hygiene products are used for periodontal tissue diseases (gingivitis, periodontitis, periodontal disease)?
88. What are the mechanisms of action of preventive oral hygiene?
89. What methods are used for individual oral hygiene?
90. Organization of work of periodontal office (department)
91. How is the periodontal department equipped?
92. What are the sanitary and hygienic standards required for the arrangement of periodontal office couples.

93. What medical staff is provided for work in the periodontal department.
94. What medical procedures are performed by a dental hygienist.
95. What set of instruments and devices is provided in the periodontal office.
96. What is the medical support of the periodontal office 8. What, in addition to medicines, should be a set of tools for emergency care....
97. What are the main tasks of medical examination you know
98. The main organizational form of medical examination is
99. What groups of population are subject to dispensary registration (and the selection of patients).
100. What medical measures are used during dispensary registration?
101. What groups of dispensary patients are defined according to the existing legislation - What are the stages of dispensary registration
102. Indicate what documentation should be filled out when registering a patient with periodontal tissue disease.
103. What is the procedure for removing patients from dispensary registration in periodontal disease?
104. What is dispensary registration?
105. Name the stages of medical examination.
106. Who and where carries out the selection of patients for dispensary observation?
107. Name the groups of persons subject to medical examination.
108. What documentation is drawn up for each person subject to medical examination?
109. What is the frequency of medical and health measures for various periodontal pathologies?
110. What are the criteria for evaluating the medical examination at different stages of its implementation?

LIST OF REFERENCES

Main literature

1. Stomatology: textbook: in 2 books. Book I / M.M. Rozhko, Z.B. Popovych, V.D. Kuroiedova et al.: edited by M.M. Rozhko. – Kyiv: AUS Medicine Publishing, 2020. – 792 p.: color edition.
2. Periodontal and Oral Mucosa Diseases: textbook. Vol. 2 / A.V. Borsenko, L.V. Lynovytska, O.F. Nesyn et al.; edited by A.V. Borysenko. - Kyiv: AUS Medicine Publishing, 2018. – 624 p.; color
edition.
3. Stomatology: textbook: in 2 books. Book 2 / M.M. Rozhko, 1.I. Kyrylenko, O. H. Denysenko et al.; edited by M. M. Rozhko. – Kyiv: AUS Medicine Publishing, 2018. – 960 p.; color edition.

Additional literature

1. Esthetic Dentistry: A Clinical Approach to Techniques and Materials 3rd Edition by Kenneth W. Aschheim. Publisher: Mosby; 3rd edition, 2014. – 600 p.
2. Esthetic and Restorative Dentistry: Material Selection and Technique 3rd Edition by Douglas A Terry, Willi Geller. Publisher: Quintessence Publishing Co Inc.; 3rd edition, 2017. – 776 p.
3. Dental Composite Materials for Direct Restorations Softcover reprint of the original 1st ed. by Vesna Miletic. Publisher: Springer; 1st edition, 2018. – 327 p.
4. Posterior Direct Restorations 1st Edition by Salvatore Scolavino, Gaetano Paolone. Publisher: Quintessence Pub Co; 1st edition, 2021. – 264 p.
5. Smile Design Integrating Esthetics and Function Essentials of Esthetic Dentistry Volume Two by Jonathan B. Levine. Elsevier Health Sciences, 2015. – 240 p.
6. Cohen's Pathways of the Pulp 12th Edition by Louis H. Berman, Kenneth M. Hargreaves. Publisher: Elsevier; 12th edition, 2020. –992 p.
7. Newman and Carranza's Essentials of Clinical Periodontology, 1st Edition. Michael G. Newman & Irina F Dragan & Satheesh Elangovan & Archana K. Karan. 2022. – 352 p.
8. Newman and Carranza's Clinical Periodontology, 13th Edition. Michael G. Newman & Henry Takei & Perry R. Klokkevold & Fermin A. Carranza. 2019. – 944 p.