**МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ
РЕСПУБЛИКИ БЕЛАРУСЬ**

**УО «ВИТЕБСКИЙ ГОСУДАРСТВЕННЫЙ ОРДЕНА
ДРУЖБЫ НАРОДОВ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ»**

**КАФЕДРА ТЕРАПЕВТИЧЕСКОЙ СТОМАТОЛОГИИ
С КУРСОМ ФПК И ПК**

Терапевтическая стоматология для студентов 4 курса

Therapeutic Dentistry for the 4th year students

Рекомендовано Учебно-методическим объединением по высшему медицинскому, фармацевтическому образованию
в качестве учебно-методического пособия для студентов учреждений высшего образования, обучающихся
по специальности 1-79 01 07 «Стоматология»

ВИТЕБСК, 2020

УДК 616.31-084=111(072)
ББК 56.61я73
Т 35

Рекомендовано к изданию Научно-методическим
советом ВГМУ в качестве учебно-методического пособия
(«26» февраля, протокол № 2).

Рецензенты: 2-ая кафедра терапевтической стоматологии Белорусского государственного медицинского университета (заведующий кафедрой - д.м.н., профессор Т.Н. Манак);
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Т 35 Терапевтическая стоматология для студентов 4 курса = Therapeutic Dentistry for the 4th year students: educational and methodical edition / Н.Э. Колчанова, А.А. Пожарицкая, Ю.П. Чернявский, Н.А. Байтус. – Витебск: ВГМУ, 2020. – 262 с.

ISBN

Учебно-методическое пособие «Therapeutic Dentistry for the 4th year students» рассматривает один из важнейших разделов терапевтической стоматологии - периодонтологию. Изложены основные аспекты этиологии и патогенеза заболеваний периодонта, их клиническая картина. Представлена классификация заболеваний периодонта, основные и дополнительные методы диагностики, профилактики и лечения заболеваний периодонта. Пособие соответствует образовательному стандарту, типовой и учебной программам по дисциплине «Терапевтическая стоматология». Предназначено для студентов 4 курса стоматологического факультета, обучающихся на английском языке.

УДК 616.31-084=111(072)
ББК 56.61я73

ISBN

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7 SEMESTER

LESSON 1. TOOTH ROOT CARIES IN PATIENTS WITH GINGIVAL RECESSION: A CLASSIFICATION, CHARACTERISTIC, MECHANISM OF THE DEVELOPMENT, CLINICAL FEATURES, DIAGNOSIS, TREATMENT.

The questions to be studied for the learning of the topic:

1. Definition, epidemiology, predisposing factors of root caries in patients with gingival recession.
2. Etiology and pathogenesis of root caries.
3. Classification of root caries.
4. Features of clinical manifestations.
5. Planning of preventive measures.
6. The choice of tactics of treatment, methods of local treatment of caries of the tooth root.
7. Features of preparation cavities at tooth root caries, the choice of filling materials.

Question 1. Definition, epidemiology, predisposing factors of root caries in patients with gingival recession.

Definition. By definition EV Borovsky, tooth decay is a pathological process that take place after the eruption of the tooth at which the demineralization and softening of the dental hard tissues and the formation of cavities.

S. Hasen gives the following definition of caries cement (root caries). It's progressive lesion in any part of the tooth root surface, washed by the oral fluid, a result of disturbance the gingival attachment.

Epidemiological indicators of root caries in the world varies according to different authors: prevalence - from 4.7% to 100%; intensity - from 0.07 to 1.63; the average number of teeth with root caries - from 1.9 to 4.3; RCI - from 2% to 38%.

The prevalence of root caries can be calculated using the RDF index (W. Geurtsen, D. Heidemann, 1972-1993). The index of the prevalence of root caries is determined as the percent ratio the number of teeth with caries and (or) the seals at the root to total number of teeth in a given individual. Interpretation prevalence carried out as follows: 1-5% - very low; 6-15% - low; 16-30% - medium; 31-50% - high, 51% or more - very high.

Predisposing factors to development root caries.

1. Affect the root environment:

- ✓ unsatisfactory oral hygiene
- ✓ periodontal disease

- ✓ elderly age
- ✓ anatomical features of dental system

reduction of periodontal attachment.

2. Affect the crown environment:

- ✓ microorganisms plaque
- ✓ cariogenic foods
- ✓ insufficient intake of fluorine
- ✓ changes in oral fluid properties

the development of caries

The risk factors of caries root-surface in elderly people

1. Age.
2. General disease.
3. Change the quality of dental plaque.
4. Peculiarities diet (prevalence of soft carbohydrate food).
5. The neglect oral hygiene (or an inability).
5. Acceptance of drugs.
6. Inadequate salivation

Indicators of risk are gum recession, a thick layer of microbial plaque and reduce the amount of saliva.

Question 2. Etiology and pathogenesis of root caries.

Cement tooth consists of collagen fibers and amorphous minerals. They are arranged in horizontal layers. Cement seems soft when probed with a sharp tip, the color of its light yellow, the thickness ranges from 20 μm in the cervical area of up to 200 μm in the apical part of the root.

Desalination of cement is only possible if the exposed surface of the tooth root, i.e., with gingival recession (K06.0). In the cement is formed rectangular focus demineralization on the entire thickness of the cement. After unsheathing the tooth root cement can be lost due to mechanical destruction. The dentine reaction is observed in the form of sclerosis.

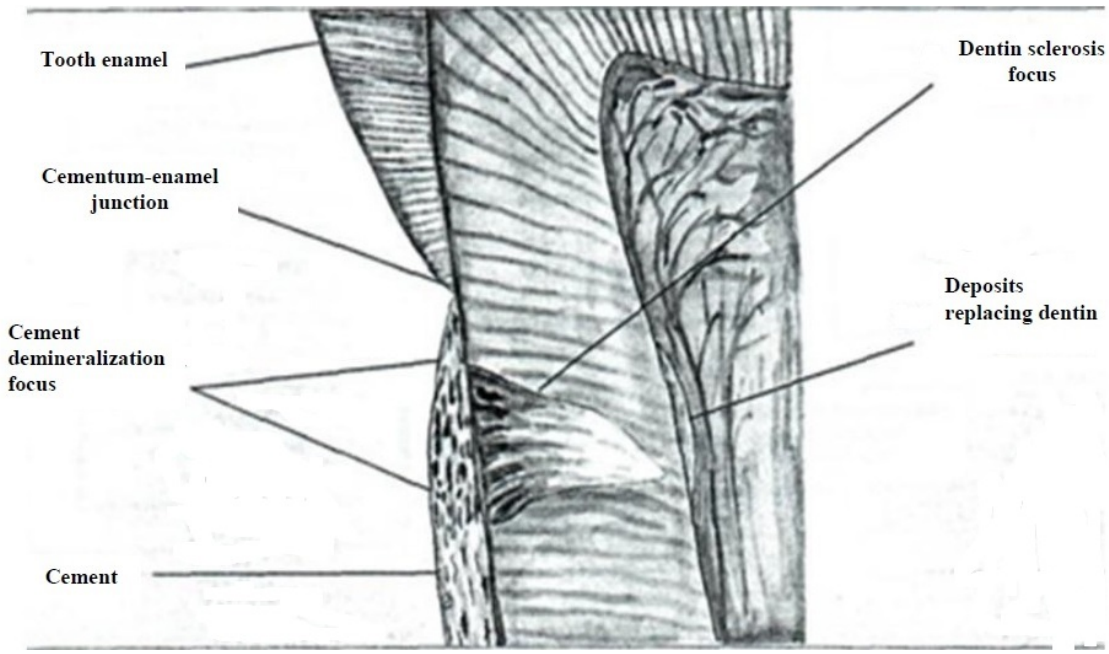


Figure 1. Microscopic changes in tooth tissues during caries of cementum (K02.2) at the spot stage

The main stages development of caries root-surface

1. Gum recession (an important sign for the possible occurrence of caries of cementum)
2. Microbial plaque
   - Actinomycetes
   - Str. Mutans
   - Lactobacillus
3. Demineralization
4. The horizontal direction of propagation of destruction (circular caries)
5. Slow cavitation (formation of carious cavity)
6. Inactive caries (subject to the removal of plaque)
7. Painless over (until the destruction of the tooth pulp)

Question 3. Classification of root caries.

1) Fejerskov and co-workers introduced a classification for diagnosing root-surface lesions which integrates activity assessment as well as assessment of surface integrity. The criteria were developed on the basis of empirical observations of experimental non-operative treatments of root-surface caries (Nyvad & Fejerskov, 1986).

Active lesions were described as soft or leathery and were usually found at plaque-retention sites next to the gingival margin or along the cement-enamel junction. Inactive lesions were typically located at some distance from the gingival margin, felt hard on gentle probing and often presented with a shiny. The color of the lesion was not helpful in

distinguishing between active and inactive stages. The following diagnostic categories were identified:

- inactive lesion without surface destruction
- inactive lesion with cavity formation
- active lesion without definitive surface destruction
- active lesion with surface destruction (cavitation), but cavity is estimated not to exceed 1 mm in depth (visually)
- active lesion with a cavity depth exceeding 1 mm, but not involving the pulp
- lesion expected to penetrate into the pulp
- filling confined to the root surface or extending from a coronal surface onto the root surface
- filling with an active (secondary) lesion along the margin
- filling with an inactive lesion (secondary) confined to the margin

2) Classification of root caries (L.N. Dedova, O.V. Kandrukevich, 2008)

Table 1. Classification of root caries (L.N. Dedova, O.V. Kandrukevich, 2008)

1. Dental caries	
1.1. Flow	1.1.1. Rapidly progressive 1.1.2. Progressing 1.1.3. Remission 1.1.4. Relapse.
1.2. Depth	1.2.1. Without defect hard tissue stain 1.2.2. Defect hard tissue cement
1.3. Tooth tissue	1.3.1. Cement 1.3.2. Dentin.
1.4. Topographic surface of the root	1.4.1. Vestibular 1.4.2. Oral 1.4.3. Approximal 1.4.4. Circular
1.5. Localization	1.5.1. Supragingival 1.5.2. Subgingival
1.6. Prevalence	1.6.1. Very low 1.6.2. Low 1.6.3. Medium 1.6.4. High 1.6.5. Very high

3) The classification of the clinical forms of the root surface caries (P.A. Leus, L.G. Borisenko, 2005).

A. Active carious lesions

A. 1. Without formation cavity
A. 2. With the formation of cavity
B. Suspend caries
B. 1. Without cavity formation
B. 2. With the formation of the cavity of any size
C. The secondary caries
C. 1. Active lesions along the edge of the seal
C. 2. Inactive lesion adjacent to the edge
D. Not refined root surface caries
D. 1. Without cavity formation
D. 2. With the formation of cavities

Secondary caries is formed along the edge of the seal.

Not refined tooth root surface caries may be temporary diagnosed when a precise definition of the degree of disease activity during the initial examination difficult.

4) The International Classification of Dental Diseases (ICD-DA, WHO 1995) root caries is considered in section K02 «Dental caries». Root caries in this classification termed "K02.2 Caries of cementum." Caries extending into dentine (K02.1) and Arrested caries (K02.3) also may be localized in the area of the roots of teeth with gingival recession. In practice, there are cases where the initial evaluation is not possible to determine the stage of development of caries. Such lesions are classified as "Dental caries, unspecified" K02.9 and are subject to monitoring to the final diagnosis.

Question 4. Clinical appearance of root caries lesions

Patients with dental root caries complain:

- On the lack of aesthetic (the localization of the cavity in the vestibular surface of the anterior teeth roots);
- Discomfort when eating, brushing teeth;
- Thermal pain, mechanical and chemical stimuli disappear immediately after the removal of the stimulus;
- Complaints relating to the presence of periodontal disease in the patient, resulting in the loss of periodontal attachment,
- Perhaps the absence of complaints (often painful sensations occur only in the development of inflammation of the dental pulp).

Recession of the gingival margin is an inevitable result of poor oral hygiene and loss of periodontal attachment with age (Baelum et al., 1991; Baelum, 1998). Even in populations with regular oral hygiene some recession occurs, and its pattern of distribution within elderly populations is very characteristic (Fejerskov et al., 1993). In today’s populations it is frequent that even adolescents experience some exposure of the cervical root surfaces in several teeth owing to inappropriate plaque control procedures.

As the gingival margin recedes the enamel–cementum junction becomes exposed. This region of the tooth is highly irregular and represents a particular bacterial retention site. Therefore, a majority of root caries lesions develop at this site.

It is occasionally claimed that root-surface caries may occur within a deep periodontal pocket. From a biological point of view this is not very likely, as the pH of the gingival exudate flushing the pocket is above 7. It seems more likely that in such cases the carious process has originated along the gingival margin. Gingival inflammation and swelling of gingiva may subsequently lead to the impression that the lesion is 'hidden in the pocket'

Root-surface caries comprises a continuum of clinical manifestations ranging from small, slightly softened and discolored areas to extensive, yellow–brown soft or hard areas, which may eventually encircle the entire root surface. The lesions may or may not be cavitated. However, even in the case of rather extensive lesions, cavitation does not necessarily involve the pulp.

As for enamel lesions, root-surface caries lesions may be classified as active or arrested (inactive) according to the following diagnostic criteria:

An active root-surface lesion is a well-defined, softened area on the root surface that shows a yellowish or light-brown discoloration. The lesion is likely to be covered by visible plaque. Some slowly progressing lesions may be brownish or black and reveal a leathery consistency on probing with moderate pressure.

An arrested (inactive) root-surface lesion appears shiny and is relatively smooth and hard on probing with moderate pressure. The color may vary from yellowish to brownish or black. In both active and inactive lesions, cavity formation may be observed, but in the latter case the margins appear smooth. No visible microbial deposits are seen to cover such lesions.

Although characteristic in their classical manifestations, there will be a range of transitory stages between active and arrested lesions. Thus, it is important to appreciate that when using the diagnosis arrested (or inactive). This is a reflection of a clinical judgment that no further progression of that lesion is expected to take place. This does not imply that there may not be minute niches within certain areas of the lesion that, if examined for example in a microscope, will show bacteria and very localized demineralization. However, if at the time of examination a lesion is judged to be arrested, the lesion is considered to remain clinically unchanged unless the patient's oral hygiene deteriorates at that particular site.

If there is doubt over whether to assign a lesion into the active or the inactive category, the surface texture of the lesion (soft/leathery or hard) is a more valid criterion than is the mere color of the lesion.

It is clinically important to distinguish between active and inactive lesions because root surfaces also respond to the dynamic metabolic

processes in the plaque. Thus, if these processes are interfered with, for example by regular plaque removal, active lesions may become arrested, with associated changes in surface texture and color of the lesions.

From a differential diagnostic point of view a root surface caries lesion is easy to distinguish from other root surface discolorations because the latter usually are widespread and ill-defined.

The differential diagnosis is carried with non-carious lesions: K03.1 Abrasion of teeth and K03.2 Erosion of teeth.

Question 5. Planning of preventive actions for patients with gingival recession and tooth root caries

Methods of individual prevention

Recommended activities in the first visit:

1. Definition of dental status.
2. Identification of the active and suspended caries of the root surfaces.
3. Detection of cariogenic factors (xerostomia, common diseases, unhealthy diet, reception of medicines).
4. Determination of plaque index (qualitative and quantitative).
5. Dental education and training of effective methods of cleaning teeth

At subsequent visits (at least 1-2 times per year) held:

1. Control of oral hygiene.
2. Professional hygiene.
3. Conservative treatment of root caries.
4. Processing with fluoride preparations of healthy root surfaces.
5. Assign courses mouth rinses with chlorhexidine or Listerine with poor oral hygiene.
6. Tips on healthy eating.
7. Selection of effective hygiene (F toothpaste, brushes, brushes for teeth, floss, toothpicks).

Recommendations for hygienic oral care implement:

1. The standard brushing method is modified by Stillman,
2. Additional hygiene products for cleaning concave surfaces of the tooth root - interdental brushes and toothbrushes onebeam.
3. Toothpastes, gels, and mouthwashes containing fluorine compounds, including in combination with antiseptics.
4. Toothpastes (or gels) with low abrasiveness.
5. In the case of dentine sensitivity in the exposed roots of the teeth - paste comprising means for reducing the sensitivity of dentin (potassium salt, hydroxyapatite, tricalcium phosphate, etc.).
6. Patients with decreased salivation - pastes, gels, mouthwashes containing substances that exist in the natural saliva (lysozyme, lactoferrin, saliva proteins).

7. Activities of professional oral hygiene with regular use of fluoride preparations and treatment of periodontal disease.

Question 6. The choice of tactics of treatment, methods for local treatment of root-surface caries.

At the dentist has a choice of conservative or surgical treatment of primary root-surface caries.

Methods topical application medicines:

1. Use of fluorine agents (lacquers, gels, solutions, etc.).

Method rinsing the mouth 0.05% sodium fluoride. The procedure is repeated daily 2 times a day after meals for one month.

Method of coating a fluorine-containing varnish. The procedure is repeated after a month and 12 months.

2. The use of fluorinated drugs in combination with antiseptics.

- ✓ 0.05-2% sodium fluoride;
- ✓ 4% titanium fluoride in combination with antiseptics (chlorhexidine 1.5%, 1% thymol, triclosan);
- ✓ Aminoftorid and 0.4% stannous fluoride.

3. Deep fluoridation. Application of the dentin-sealing liquid containing fluoride crystals high dispersion and copper ions:

- ✓ Crystals of high dispersion penetrate into dentinal tubules;
- ✓ Fluoride crystals of high dispersion on the tooth surfaces create high local concentration of fluoride ions;
- ✓ There is an increase rate and duration remineralization (months 6-12);
- ✓ Copper ions inhibit the proteolytic activity of microorganisms.

4. A combined method of remineralization therapy and fluorination. Using a 10% solution of calcium gluconate and 0.5-1% sodium fluoride solution. Applications therapeutic solutions spend every other day, three times.

5. Physical factors and fluorine preparations

Principles of therapeutic and prophylactic action of helium-neon laser (HNL):

- activates the enzyme system of the tooth pulp;
- inactivate Streptococcus mutans;
- improves the efficiency of remineralizing agents;
- actively influences the permeability of hard dental tissues.

HNL used in combination with drugs fluorine and drugs calcium-phosphate.

Principles of therapeutic and preventive effect of high ozone concentrations:

- elimination of all pathogens in the hearth of carious lesions;
- stimulation of non-specific response of the immune system - a synthesis of neutrophil granulocytes.

6. Protective sealants for open dentin (desensitizers). The composition protective **sealants** for open dentine usually includes nanofiller particles (7 nm); fluorine compounds; antiseptic (triclosan); methacrylate resin.

7. The method of treatment developed by the 3d Department of Therapeutic Dentistry (BSMU)

1. Initial root caries treatment with a mixture of sodium bicarbonate and water (1:1) using a rotating brush.
2. 0.01% miramistin solution.
3. The application of 2.5% calcium glycerophosphate solution.
4. Nanocrystalline hydroxyapatite gel is rubbed. Total of 3 treatments.
5. The fluorine-containing photocurable desensitizer.

Question 7. Features of preparation cavities at tooth root caries, the choice of filling materials.

1. Phase exception disclosure cavity (cavities as arranged on the surface of the tooth root, usually have no overhanging edges or undercuts).
2. Conducting necrectomy without prophylactic excision of tissue intact:
   2.1. because of the small thickness of hard tissue in the tooth root (1.5-2 mm from the root to the channel region in cutter lumen, 2.5 mm - molar region)
   2.2. because of the risk of cervical lesions pulp horns;

Figure 2. Safe depth of the formed carious cavity for the pulp.

3. Preventive expansion of carious cavity possible if: rapidly progressive flow caries, multiple root caries, poor oral hygiene, general pathology.
4. Formation oval cavity,
5. The creation of retention grooves in the dentin on the occlusal and gingival walls (if indicated)

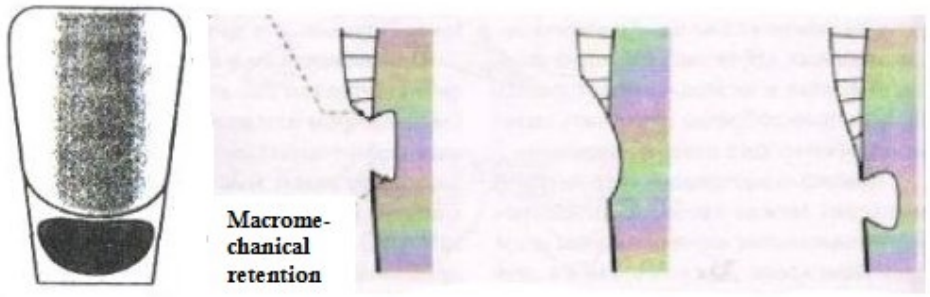


Figure 3. Optimization of conditions of fixing the seal with root caries (E. Hellwig, J. Klimek, T. Attigny, 1999)

6. The formation of additional sites on the oral surface of the tooth root to improve access to carious cavity on the contact surface of the root;

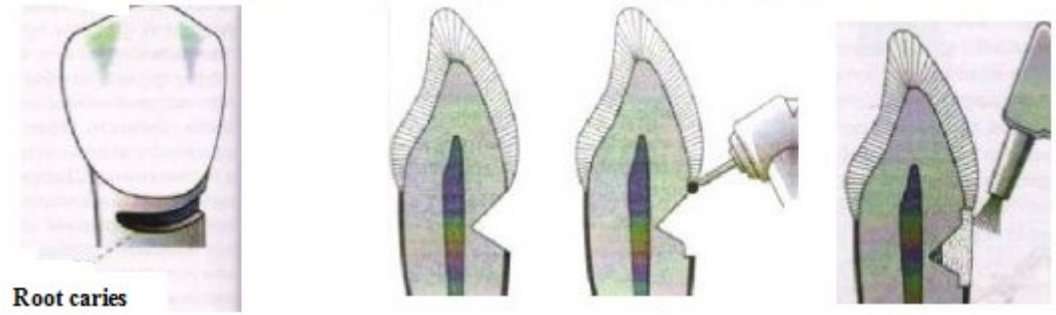


Figure 4. Additional platform for access to approximate cavities at the tooth root. Preparation with a rectangular protrusion

7. Preparation edge carious cavity with a rectangular ledge to prevent thinning of the edges of fillings when using the GIC.
8. Creating bevel enamel (2-5mm), if part of the cavity is located in the cement-enamel border, if you are using adhesive systems.

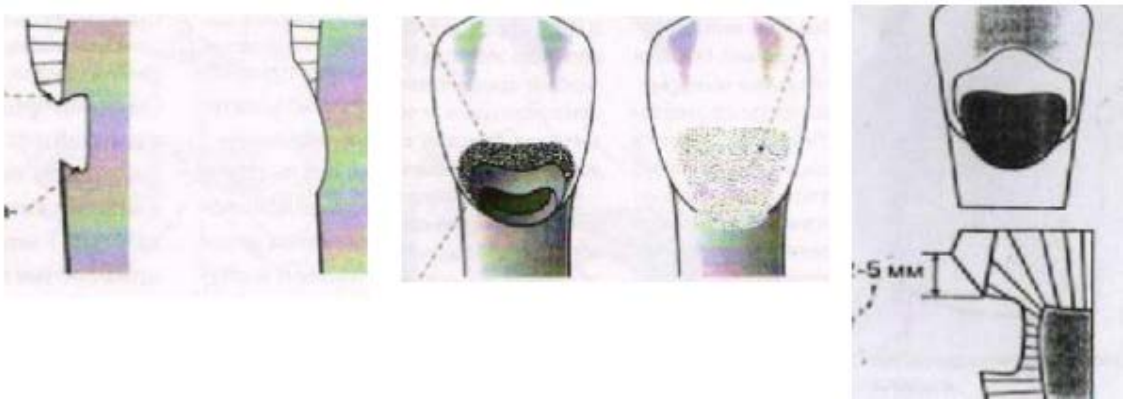


Figure 5. Enamel bevel when the cavity is in the area of the enamel-cement compound.

9. The urgency of the use of special cervical matrices when filling root caries is dictated by the close proximity to the gingival margin.

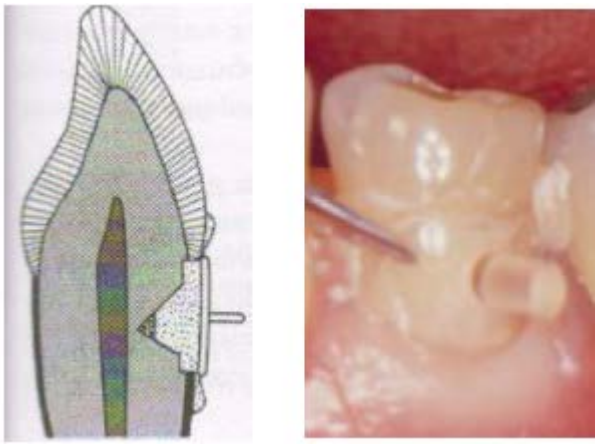


Figure 6. The use of cervical matrices.

The choice of filling materials

- glass ionomer cements,
- silver amalgam,
- compomers, composites pink colors.
- inlays (porcelain, stainless steel).

For the processing of seals in the area of the tooth root expedient use a low abrasion drills with rounded smooth finish, polishing discs, brushes.

Test control

1. Class V cavities by Black include:

a) cavity in the cervical third of the vestibular surfaces of the teeth
b) cavity in the cervical third of the lingual surfaces of the teeth
c) cavity in the cervical third of the interproximal surfaces of the teeth
d) cavity on vestibular root surface
e) cavity on lingual root surfaces
f) cavity on interproximal root surfaces

2. Which of these filling materials preferably used in the treatment cement caries?

a) glass ionomer cement
b) compomer
c) amalgam
d) composite

3. Indicate the indications in the restoration of the permanent teeth «Dyract» material:

a) restoration cavities V of class by Black
b) restoration cavities 1 of class by Black
c) restoration cavities 2 of class by Black
d) sealing wedge-shaped defects

4. Indicate the main method for preventing of postoperative sensitivity in the case of the use of photopolymers:

a) use of glass ionomer cement as an insulating lining
b) conduct polymerization through the tooth wall
c) an increase in the thickness of the adhesive layer (adhesive)
d) the use of layered composite application techniques
e) all of the above

5. For what purposes is recommended to use a caries detector?

a) for indicating carious dentin
b) to detect hidden caries
c) method for chemical treatment caries

6. Which drills are used for processing seals?

a) steel
b) diamond with blue, green stripe
c) diamond with red, yellow, white stripe
d) carbide

7. Indicate the stage of the photopolymer seal setup when it is necessary to choose the shade of the filling material (classical technique)?

a) before clean the tooth surface
b) after cleaning the tooth surface
c) prior to etching the enamel
d) after enamel etching
e) prior to the formation of carious cavity
f) after the formation of carious cavity

8. Cavities V of class by Black differentiate with:

a) hypoplasia;
b) fluorosis;
c) dental ankylosis
d) abrasion the teeth (wedge-shaped defects);
e) acid necrosis;
f) tooth erosion.

9. What is evaluated during the examination of affected tissues of the root surface?

a) color change,
b) density and relief of tissues,
c) defect area
d) the presence of pain during probing of the root.
e) all of the above

10. What should be remembered during the preparation and filling of carious lesions of the tooth root, located close to the gingival margin?

a) about gum protecting from mechanical and chemical damage;
b) about gum retraction to create access to the carious cavity;
c) about ensuring the dryness of the surgical field (protection from blood, gingival and oral fluids, about exudate from periodontal pockets).
d) all of the above

LESSON 2. INTERNAL TOOTH ROOT RESORPTION. CLINIC. DIAGNOSTICS. TREATMENT PLAN

The questions to be studied for the learning of the topic:

1. Internal tooth root resorption. Etiology and pathogenesis.
2. Internal tooth root resorption. The clinical picture.
3. The internal resorption of the tooth root. Diagnostics, differential diagnosis.
4. The internal resorption of the tooth root. Treatment plan.
5. Clinical cases.

Question 1. Internal tooth root resorption. Etiology and pathogenesis.

Studied many aspects of etiopathogenesis of internal resorption, but the real mechanism is not yet completely clear. In root canal creates favorable conditions for the activity of cells capable of resorption of hard tissue. Precipitating factor may be trauma as a result of the preparation of the crown or damage due to shock. On the cell metabolism is largely influenced by changes in the microcirculation. Root resorption occurs more frequently near the blood vessels. It was found that the active hyperemia with high oxygen partial pressure initiates and supports activities odontoclast. Resorption process may contribute electrical activity, such as piezoelectric potentials and the potentials of blood flow (the charges resulting from the blood stream through the vessels).

Cytological changes occur in the pulp when the reserve of undifferentiated connective tissue cells formed odontoclast. In teeth with internal resorption it has identified a number of histochemical changes. Multinucleated cells that are found in lacunar zones dentine resorption, had a similar structure with cells involved in bone resorption and the same enzyme composition (acid phosphatase and b-glucuronidase).

When scanning electron microscopy revealed another possible cause of internal resorption. According to Stanley (1972), along with the resorption often occurs deposition of mineralized tissue like bone or cement. The resulting fabric is unlike the normal, so called "metaplastic" cloth. It is believed that any resorption develops in the following sequence. Trauma causes tooth intrapulp bleeding. Inner resorption precedes the appearance disappearance of odontoblasts in the pulp cells, similar to macrophages. Hematoma is organized, that is replaced by granulation tissue. Proliferating granulation tissue puts pressure on dentin walls, forming predentin stops from the connective tissue differentiate odontoclast and resorption begins. When degradation becomes extensive, it may develop necrosis of the pulp. Complication of internal resorption is a perforation of the root surface of the crown or to the development of communication between pulp and oral cavity.

Heithersay (1985) believes that the cause of the internal resorption can be collateral blood flow through the shunt and large additional lateral channels. These features of blood supply can create sufficient conditions for the development of resorptive process. The author points out that in endodontic treatment often reveals a large additional channel.

Question 2. Internal tooth root resorption. Intrapulp cysts. The clinical picture.

Intrapulp cysts (cyst Sipovsky). According to P.V. Sipovsky who first described the processes, intrapulp cysts develop as a result of degenerative or necrobiotic processes taking place in the dental pulp. They may be located in the cavity of the crown and the root canal. Often there are multiple cysts.

When X-ray intrapulp cysts are found rarely. To this cyst could be detected on the radiograph, it should reach the known dimensions. Due to the increase in volume and an increase in cyst exerts pressure on the inside wall of the crown or root. As a result, at the appropriate places edge defects are formed (the pressure atrophy). These defects in the walls of the tooth crown to the X-ray can not be seen due to their large size and small thickness of the crown walls. At the root of the tooth growing cyst soon fills a narrow gap and root canal, putting pressure on its walls; it forms a semi-circular defects of various sizes, easily visible with X-ray diffraction study (figure 7).

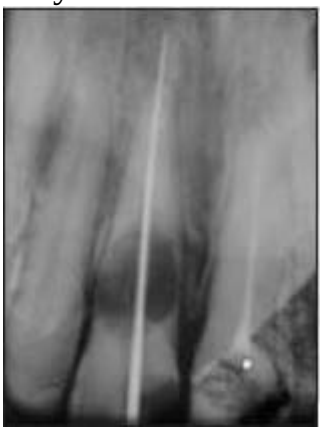


Figure 7. Defects from a cyst.

X-ray picture intrapulp cysts are quite characteristic. The tooth root at various levels, often in the middle section, is determined by the limited right-circular shape of the root canal expansion. The contours of this expansion, clear and smooth. More rarely found in the root of the tooth double or multiple cavities, sometimes slightly elongated internal resorption is usually asymptomatic and first revealed by X-ray examination. To determine the amount of loss of tooth tissue and create a treatment plan, you need to perform a few shots in a parallel technique from different angles. The pain may be caused by perforation of the crown when metaplastic tissue is exposed to corrosive irritating oral factors. Perforation of the root, leading to the development of pathological changes in periodontal, usually accompanied

by the patient's complaints. With extensive resorption crowns patient can observe the formation of spots.

Pulp tissue should be removed at the first detection of the pathological process. If the pulp is not removed, when the internal dynamic monitoring resorption can lead to complications. Spontaneous recovery is rare. Therefore, in all cases diagnosed internal resorption as possible must hold endodontic treatment faster.

Question 3. The internal resorption of the tooth root. Diagnostics.

Sometimes very difficult to determine the true etiology of this process, but, nevertheless, only a complete diagnostic study allows an accurate diagnosis and determine the correct treatment strategy.

Use the following diagnostic methods:

- ***subjective*** (collection of complaints, anamnesis)
- **objective**
  - Basic - inspection, sensing, percussion, palpation, thermodiagnostics (to assess the viability of the pulp).
  - Extra:

1. Periodontology examination uses a periodontal probe determination of the depth of the teeth-gingival pocket, the presence of subgingival dental plaque, root roughness
2. Electroodontodiagnosis (to assess viability pulp)
3. X-ray study to determine the size of periapical changes or presence of periodontal pathology marginal:
   - *intraoral X-ray*
   - *panoramic radiography*
   - *orthopantomography*
   - *cone-beam computed tomography (CBCT)*

Question 4. Internal tooth root resorption. Treatment plan.

There are three main areas of choice of tactics of treatment of the inside of the tooth root resorption.

- conservative endodontic treatment;
- recalcification or apexification using calcium hydroxide and hydroxyapatite;
- surgery.

The choice of approach is determined by the ability to comply with physician triad requirements for endodontic treatment (sterilization, cleaning and obturation of the root canal), and the prevalence and location of the defect.

If the requirements can be met and focus resorption not perforates the wall of the channel, used conservative endodontic treatment materials based

on mineral trioxide aggregate (MTA) "Trioxident" (Vladmiva, Russia), "Rootsil" (Belarus), «MTA ProRoot» (Dentsply, USA).

If three major endodontic principle can be met, but there is a perforation of the canal wall, not communicating with the oral cavity, it is necessary to use a method of temporarily filling channel (apexification) materials based on hydroxyapatite or calcium hydroxide.

Preparations based on calcium hydroxide in the treatment of tooth root resorption

Many authors recognize the need for these drugs as a temporary root fillings at different times with the purpose of prolonged antiseptic effect on the periapical tissues and the walls of the root canal, as well as to create conditions for the effective recovery of bone tissue in long-term period, which is especially important when resorption processes. Calcium-containing preparations are presented materials for temporary root canal obturation "Calasept» (Nordiska dental - Sweden (sterile pure calcium hydroxide)), "Calcicur" (Voco - Germany), "Metapasta" (Unident - South Korea), "Calasept" (Omega - Russian Federation), and for the constant «Vitapex» (Neo Dental Chemical-Japan), «Acroseal» (Septodont - France), "Akros" (Dentsply - Usa), "Mepasil" and «Ozomol-4» (Pierre Rolland Acteon Group - France) and are available in the form of paste or powder / liquid.

Preparations based on hydroxyapatite in the treatment of root resorption of teeth

The creation of a depot in a bone defect from osteoregenerating materials is of great importance for the stimulation of osteogenesis. In dental practice, the use of such drugs is widespread. The most popular received alloplastic (biocomposite) of calcium hydroxyapatite materials, physicochemical characteristics of which (crystal size, density, collagen content) determine osteoplastic properties. Materials based on hydroxyapatite are available in different forms: gel, beads, plates, membranes and also worked well: "Hydroxyapol", "Collapan", "Trapex-Gel", "Indost" and other production Research and Production Company "Polystom" (Russian Federation) and Belarusian counterpart - "Hydroxyapatite gel" production "Belmedpreparaty" (Republic of Belarus). After entering into the bone defect hydroxyapatite crystals are metabolized to calcium and phosphorus ions, keep the wound blood clot due to isomorphic substitution of calcium in the water, and a hydrogen ion. The action mechanism of hydroxyapatite in the wound: activates the differentiation of osteogenic cells; forms a strong chemical bond with bone, showing high bioinertness (inflammatory reaction, systemic and local toxicity are practically absent).

The hydroxyapatite resorption takes place without the formation of a fibrous capsule with the occurrence of "point" osteogenesis in the bone defect. Hydroxyapatite-based drugs are not included in the clinical protocols of endodontic treatment of dental diseases in the Republic of Belarus.

A surgical treatment is necessary in case of extensive destruction of the root, prolonged bleeding or perforation associated with the oral cavity. This method includes:

- ✓ Resection of the tooth root apex,
- ✓ Coronal radicular separation,
- ✓ Root amputation,
- ✓ In some cases, tooth removal.

Question 5. Clinical example (V.V. Zorina, magazine "Dental South", 2011, Russia)

30-year-old female-patient D. appealed to the clinic in order to restore the fillings in 21. A medium-sized carious cavity (within the dentin) with slightly softened pigmented dentin was found on the distal surface of the tooth during examination. Tooth sensitivity is saved; the bottom of the sensing carious cavity is painless. Diagnosis: chronic recurrent caries median of 21, Black's Class III. The drop of blood appeared after the first touch boron during the preparation. The painful reaction of sensing the perforation site is slightly. Spend infiltration anesthesia in the 21. A bright red pulp was detected after opening the tooth cavity. Pulp sensing was accompanied by severe bleeding that did not stop even after pulp extirpation. Remote pulp length matched 1/2 estimated lengths from the apex to the middle of the root canal. Bleeding from the root canal does not stop even after the application of effective hemostatic agents.

In view of the unusual symptoms had to clarify the status of somatic patients. She considers herself healthy and on the question of injury to the teeth said that as a child she had a bruised front teeth. The defect of hard tooth tissues of a rounded shape in the region of the upper third of the root (d= 5 mm), the remains of the tooth cement in the form of a thin line are found on the X-ray of the causative tooth (fig. 8). The diagnosis was made after diagnostic clarifications: internal resorption of 21 (intrapulp granuloma).

Treatment plan. The authors decided to remove the granulation tissue using thermocoagulation. The entrance to the tooth cavity was expanded. Thermocoagulation was carried out using a heated elongated plugger. Part of the granulation tissue was removed with a small excavator as possible. The cavity and root canal were washed with 3% sodium hypochlorite solution and 3% hydrogen peroxide solution. Bleeding stopped after drug treatment until "clean turunda". It was decided to apply the method of temporary filling of the cavity and channel using a paste based on calcium hydroxide due to the thin walls of the root in the area of the granuloma (fig. 9).

Within 3 months of observation there were no complaints. There are no visible pathological changes on the gingival mucosa in 21; percussion is painless. Temporary bandage (paste) removed in the third visit. The canal is

sealed with quick-hardening endodontic filling material with a metal pin (fig. 10). The carious defect is restored by the heliocomposite.

Within 12 months of observation there were no complaints. The gingival mucosa has a normal color without pathological changes. The seal is in satisfactory condition. Percussion is painless. On the radiograph: there are no changes in the apical periodontal region (fig. 11).

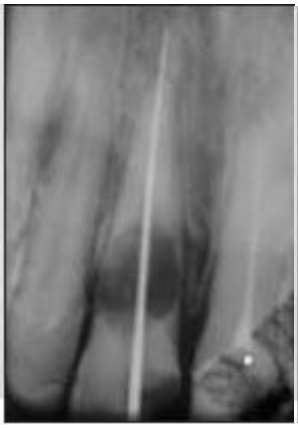


Figure 8. Rounded defect in tooth hard tissues and tooth cement residues in the form of a thin line

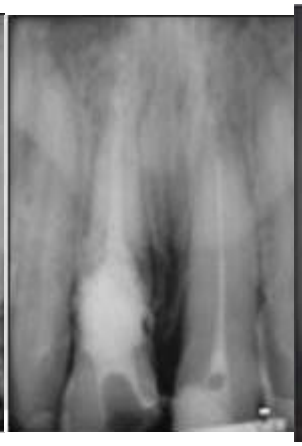


Figure 9. Temporary filling of the cavity and canal by a paste based on calcium hydroxide

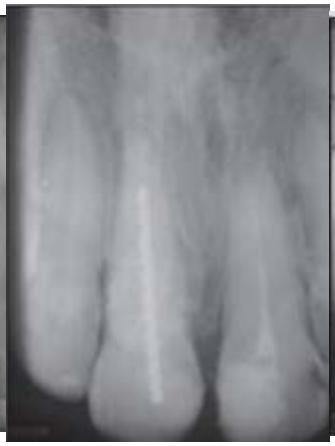


Figure 10. Canal filling with quick-hardening endodontic filling material with a metal pin

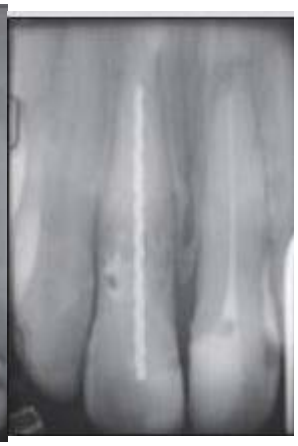


Figure 11. On the radiograph: there are no changes in the apical periodontal region

The root canal obturated. Disturbance of the filling attachment to the wall of the tooth is determined in the area of the root granuloma. This can be explained by the thin walls through which tissue fluid penetrates, eroding the filling material.

The treatment of internal resorption requires an individual approach and depends on the size and location of the intra-root granuloma.

The use of a paste based on calcium hydroxide with delayed permanent filling is justified in this clinical case. The anchor post in the case of this localization of the granuloma is not shown (there may be a break of the crown or a passive splintering of the root during its screwing). Therefore, a strong metal post is used. Localization was distributed as follows: 1/3 of the post is in the root canal after the granuloma, 1/3 is in the granuloma cavity, 1/3 is located above the granuloma.

Test control

1. The main types of bone resorption are:

a) Enamel resorption;
b) Physiological;
c) Resorption of dentine;
d) Pathological;

e) Cement resorption.

2. Root resorption of permanent teeth has the following type:

a) Species;
b) Pathological;
c) Population;
d) System;

3. Resorption is a pathological process arising when:

a) Mechanical damage of protective fabrics
b) Chemical damage of protective fabrics;
c) Irritated due to infection;
d) Stimulation due to compression;
e) For no apparent reason.

4. The dentin of the tooth root by root canal is protected:

a) Predentin;
b) Odontoclast layer;
c) Layer of odontoblasts
d) Cementoblasts layer;
e) Cement.

5. The dentin of the tooth root from the outer surface of the root is protected by:

a) Cement
b) Predentin;
c) Odontoclast layer;
d) A layer of odontoblasts;
e) Cementoblasts layer.

6. According to the ICD-10, tooth resorption is distinguished:

a) Abnormal external (outer)
b) The pulp;
c) The internal [internal granuloma] [pink spot];
d) Periodontal;
e) Unspecified.

7. From undifferentiated reserve cells of the connective tissue during internal root resorption in the pulp are formed:

a) Odontoblasts;
b) Odontoclast
c) Cementocytes;
d) Leukocytes;

e) Dentinal tubules.

8. Complication of internal resorption is:

a) Perforation of the root surface;
b) Increased tooth sensitivity;
c) Perforation of the crown surface;
d) Trigeminal neuralgia;
e) Crowding teeth.

9. The following treatments are applied at the internal root resorption:

a) Conservative endodontic treatment;
b) Antiviral treatment;
c) Recalcification with calcium hydroxide;
d) Treatment with antifungal drugs;
e) The surgical treatment.

10. As a filling material during endodontic treatment of tooth root resorption is used:

a) Materials based on mineral trioxide aggregate;
b) The paste of calcium hydroxide;
c) The paste based on zinc oxide eugenol;
d) The glass ionomer cements;
e) The paste of the epoxy resin.

LESSON 3. CLINICAL MANIFESTATIONS, DIAGNOSIS, METHODS OF TREATMENT OF DENTINE SENSITIVITY.

The questions to be studied for the learning of the topic:

1. Definition and classification of dentine sensitivity. Epidemiological data.
2. Etiology and pathogenesis. Theories of dentin sensitivity.
3. Methods of diagnosis of dentine sensitivity.
4. Methods of treatment of dentine sensitivity. Maintenance therapy and prevention dentin sensitivity.

Question 1. Definition and classification of dentine sensitivity. Epidemiological data.

The dentin sensitivity is a short sharp pain arising in response to temperature, tactile, chemical, osmotic stimuli to dentin, with the proviso that it cannot be attributed to a dental pathology.

The prevalence of dentine sensitivity among the world's population ranges from 8% to 57% and, according to some researchers, it is growing steadily.

I. Classification of dentin sensitivity according to the WHO - ICD-DA, (1994): К 03. «Dentin sensitive».

II. Dentin sensitivity Classification (L.N.Dedova, A.A.Solomevich (2006).

It displays a form, the topography of the tooth, prevalence, course and severity of the pathological process, which improves the diagnosis and treatment of dentin sensitivity, and also gives the ability to predict and differentially treat this disease.

Table 2. Dentin sensitivity Classification (L.N.Dedova, A.A.Solomevich (2006).

Dentin sensitivity	
The form	1. With the loss of dental hard tissues 2. Without loss of dental hard tissues
The topography of the tooth	1. Crown 2. Neck 3. Root
Prevalence	1. In one tooth 2. In the several teeth 3. In the region of the teeth
Course of a disease	1. Compensated 2. Subcompensated 3. Decompensated
The degree of severity	1. Mild 2. Moderate 3. Severe

Question 2. Etiology and pathogenesis. Theories of dentin sensitivity.

Etiology.

The literature describes the following factors predisposing to DS:

1) caries;
2) abrasion in the necks of the teeth;
3) erase the chewing surfaces of the teeth and cutting edges;
4) erosion of dental hard tissues,
5) vertical crack the enamel of teeth crowns;
6) gum recession,
7) liquid diet (red and white wine, fruity citrus juices, apple juice, yogurt) are able to remove the smear layer of dentin and open entrances to the dentinal tubules;
8) periodontal intervention;
9) abrasive toothpastes. Among these abrasives are calcium carbonate, dicalcium phosphate and aluminum oxide. Only silica is able to reduce dentin permeability and accumulate in the surface layer, blocking entrances to the dentinal tubules.
10) hard toothbrushes
11) number plaque. Patients with poor compliance with oral hygiene, are more likely to suffer from the black hole of the tooth root;
12) at night vital bleaching DS appears in 55-75% of cases.
13) postoperative sensitivity of dentin after restoration of teeth hard tissue in cases of incorrect operational activities.
14) psychosomatic disorders;
15) impaired function of the endocrine glands and mineral metabolism of the body. It is noted reduction of inorganic phosphorus and calcium in blood serum and oral fluid, calcium, phosphorus, magnesium - in gingival crevicular fluid and dental hard tissue biopsy material, inorganic calcium and phosphorus - in oral fluid;
16) change the pH of oral fluid promotes the emergence of DS. Oral liquid having an acid reaction in the range of 6,12-6,18 may cause generalized DS.

Theories dentine sensitivity

There are the following theories of dentin sensitivity: odontoblastic theory, receptor theory, the threshold theory, neuro-reflex theory. However, the most widespread hydrodynamic theory (M. Vgannstrom). The basis of this theory is based on the hypothesis of the hydrodynamic mechanism: dentin tubule author considered as a capillary tube containing a liquid or pulp transudate, which is easily displaced by exposure to air, heat, cold, the tip of the probe. This rapid, inward or outward motion of the fluid in the tubules leads to pressure changes in dentin and ultimately enhances the activity of nerve endings in the pulp or in the dentinal tubules. Recent studies show that

in DS responsible intradental myelinated A-fibers are activated by movement of fluid in the dentinal tubules.

Question 3. Methods of diagnosis of dentine sensitivity.

Survey Methods. Initially, patients for the study of pain sensations questionnaires were offered at DS (McGill Pain Questionnaire).

Recently wide application find pain scale. They are easier and allow us to solve the problem efficiently. Developed three types of processing such a scale.

1. The verbal rating scale type:
Verbal Rating Scale:

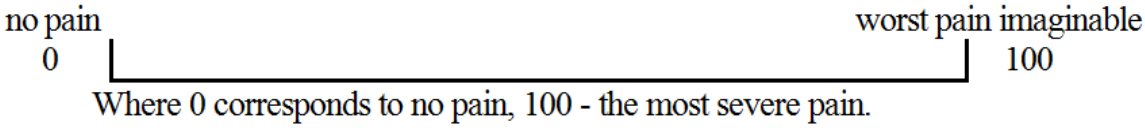
☐	☐	☐	☐	☐
none	slight	moderate	severe	intense

2. Schematic type of rating scale:
Numerical Rating Scale:

no pain — worst pain imaginable

0	1	2	3	4	5	6	7	8	9	10

3. Visual type rating scale:
Visual AnalogueScale (100 mm):



Where 0 corresponds to no pain, 100 - the most severe pain.

The scales are calculated in points. At present, more and more researchers are using a visual analogue scale (VAS) and a digital rating scale (NRS) is thus possible to determine the initial level of the black hole to a particular stimulus and its dynamics during treatment.

«Diagnosis and treatment planning of dentine sensitivity»

Disease history - INTERVIEW:

- Asking the patient to describe their pain;
- Asking the patient to indicate the stimuli that cause pain;
- Recognize the expected patient treatment;
- Study in detail the patient's food preferences (pay attention to the foods that contain acid diet: fruits and citrus juices, soft drinks, wines, ciders, etc.);
- To know the patient, whether he suffers GORD and often if he is vomiting, bruxism.

Diagnosis Dentin Sensitivity:

- Thermal and other tests (cold air, cold water, and others.);
- Survey of dental hard tissues;
- Periodontal disease;
- Ro-study (by prescription);
- Percussion of teeth;
- The study of occlusion;
- Selective local anesthesia;
- Transillumination.

DIFFERENTIAL DIAGNOSIS:

- A cracked tooth syndrome;
- Fracture restoration;
- Traumatic cleavage of dental hard tissues;
- Tooth decay;
- Gingivitis or periodontitis;
- Postoperative sensitivity of the dentin;
- Violation of fit of restorations;
- Pulpitis.

DIAGNOSIS - dentin sensitivity

INITIAL ACTIONS TO MITIGATE
Dentin sensitivity:

Patients are advised to:

- Avoid predisposing factors;
- Not to use the dietary acid;
- To carry out cleaning of the teeth long before a meal or immediately after a meal;
- Reduce the time of cleaning teeth up to a minute and use a soft toothbrush. If these measures do not bring relief, then carry out remedial measures.

Noninvasive:

- Special toothpaste;
- Topical application desensitizers.

INVASIVE:

- Mukogingival Surgery;
- Pulpectomy.

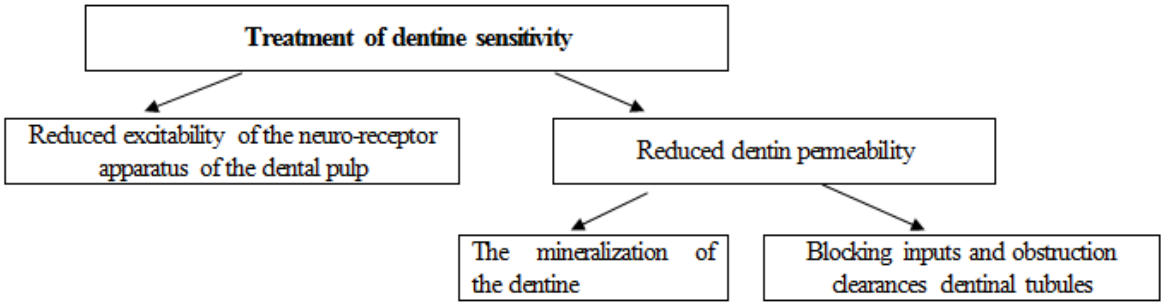
If dentin sensitivity is maintained, it is necessary to conduct additional tests to rule out periodontal pain of different nature, including chronic pain. The final treatment of the patient with the obligatory reminder to him of the need to avoid predisposing factors of the disease.

Question 4. Methods of treatment of dentine sensitivity. Maintenance therapy and prevention dentin sensitivity.

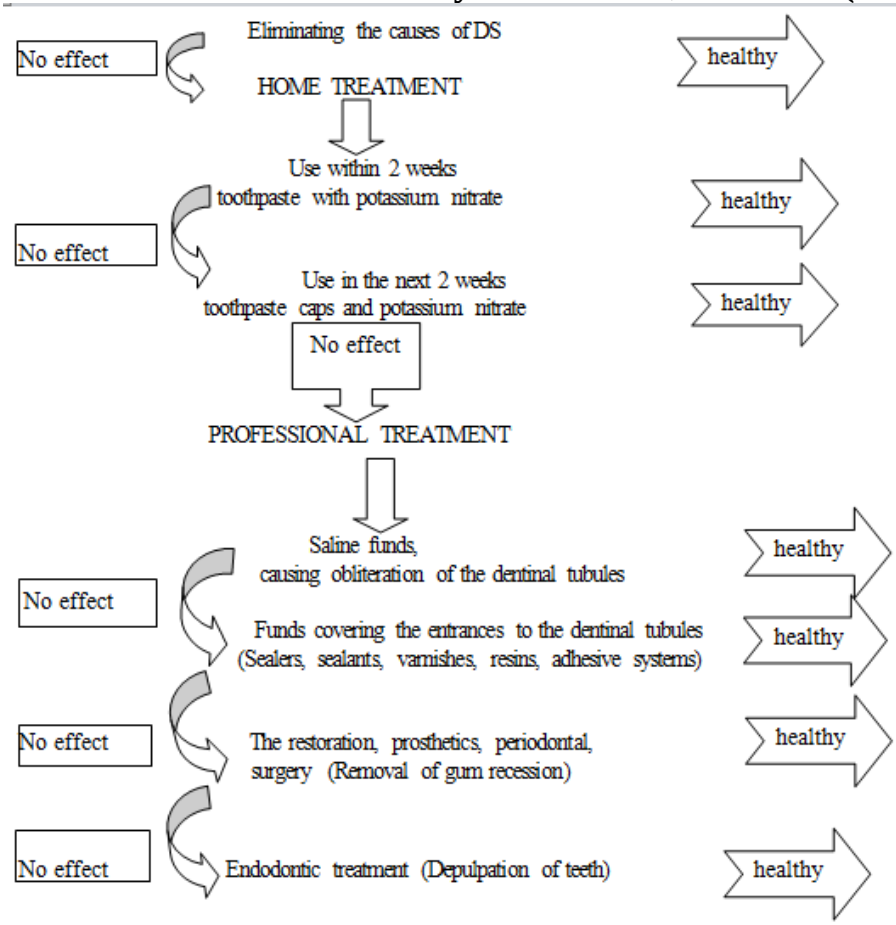
Treatment of dentin sensitivity are divided into:

1) Medication:
   - At home;
   - In the dental office;
2) Physical methods;
3) Combined;
4) Complex.

Modern main approaches in the treatment of dentine sensitivity



Treatment of dentine sensitivity P. Jacobson, G. Bruce (2001)



DRUG TREATMENT AT HOME

Potassium Drugs. It is assumed that the therapeutic effect of these drugs occur as a result of penetration into the dentin tubules potassium ions, where they accumulate surround sensory nerves in the tubular parts of the pulp and inhibit its activity, thus reducing the excitability of nerve cells. As a result, nerve impulses are transmitted or not, or they become much smaller. The optimum concentration of potassium nitrate is 5%. The use of potassium salts (nitrate and oxalate) allows to achieve the best effect. In toothpastes potassium salt is usually used together with fluorides - fluoride or sodium monofluorophosphate, tin fluoride. Aquafresh Sensitive Teeth, Oral-B Sensitive with Fluorid, Sensodyne Cool Gel, Sensodyne Fresh Mint Toothpaste, Sensodyne Fresh Mint, Sensodyne Original, Sensodyne F, Sensodyne Total Care, El-ce med Sensitive Plus, Colgate® Sensitive, Silca Sensitive.

Strontium salts. Strontium Salts occlusive open dentinal tubules and stimulate the formation of substitution dentin. Obturating ducts occurs through the binding of protein with strontium or calcium dentin matrix in replacement of hydroxyapatite crystal, resulting in reduced diameter of dentinal tubules. Strontium salts (chloride or acetate) are usually administered for therapeutic purposes in toothpastes. Pastes Sensodyne, Sensodyne Classic contain 10% strontium chloride. Sensitive contains Strontium acetate.

Phosphorus, calcium salts and hydroxyapatite. Calcium glycerophosphate stimulates natural dentin remineralization and thus contributes to its sclerosis lumen obturation of dentinal tubules.

Hydroxyapatite is synthesized drug calcium phosphate. It is almost "ideal" of calcium and phosphorus ratio - 1.67. Hydroxyapatite mechanically fills the open dentinal tubules in the exposed dentin. Pastes Novyy zhemchug, Novyy zhemchug kal'tsiy contain 1.5% calcium glycerophosphate. Oral-B Sensitive Original contains 17% hydroxyapatite. Parodontol contains hydroxyapatite.

Fluoride. Fluorine ions react with calcium ions in the liquid filling the dentinal tubules, thereby forming insoluble calcium fluoride. These precipitates are deposited in the tubules and gradually reduce their diameter.

Aminoftorid is organic fluoride. Due to the polarity of the molecule is oriented aminoftorid ftorpolyusom to hydroxyapatite. Aminoftorid slowly releases fluoride ions which react with calcium ions. Elmex Sensitive contains aminoftorid 1400 ppm of fluoride ions. Lakalut Sensitive contains aminoftorid, sodium fluoride, aluminum lactate.

Dentin Sensitivity disappears or decreases only in 20–40% of cases, regardless of the type of drugs, within 4–8 weeks in the case of home treatment. In other cases, treatment should be performed in the dental office.

DRUG TREATMENT IN THE DENTAL OFFICE

Classification desensitizers

I. Desensitizers containing resin
   1. Unfilled desensitizers containing HEMA:
      – Without glutaraldehyde;
      – With glutaraldehyde.
   2. Stuffed desensitizers containing HEMA.
   3. Dentin adhesive systems.

II. Desensitizers containing salt:
   – With oxalate;
   – Fluoride;
   – Strontium;
   – Calcium and hydroxyapatite;
   – Potassium;
   – Citrate;
   – Tin;
   – Combined.

III. Desensitizers containing resin and salt

IV. Other desensitizers.

I. Desensitayzers containing resin

1. Unfilled desensitizers containing HEMA. HEMA is a monomer which is the main component of the adhesive systems of earlier generations. It contains the active Hydrogen is capable of binding to collagen dentin closing or narrowing the lumen of the dentinal tubules. Drugs in this group usually contain an antiseptic or a fluoride, and water. Furthermore, this group of drugs restorations prepared dentine surface for best possible penetration of the adhesive into the dentinal tubules.

Unfilled desensitizers containing HEMA: *HurriSeal (Beutlich Pharmaceuticals L.P., USA), AquaPrep F (BISCO, USA), Hemaseal &Cide Desensitizer (Advantage Dental Pzoducts, Inc., USA), PzepEze Desensitizer (Jeneric / Pentzon, USA), MicroPrime Desensitizer (Danville Materials).*

Drugs in this group can be recommended for use in the prevention of postoperative dentin sensitivity when performing restorations.

Unfilled desensitizers containing HEMA and glutaraldehyde

Glutaraldehyde causes precipitation (coagulation) dentinal tubules proteins and HEMA contributes to its deeper penetration (50 to 200 microns). It is assumed that, when applied to the dentin means belonging to this group, there is collagen coagulation, and in their subsequent penetration into the dentin tubules are exposed dentinal fluid plasmoproteins precipitate which precipitate, which ultimately leads to the obstruction of the dentinal tubule lumen. For example: Gluma Desensitizer (Heraeus Kuizer, Germani).

Such desensitizers shown generally at elevated cervical sensitivity and sensitivity arising after preparation teeth under the crown dentine with sufficient thickness, as well as prior to cementation of crowns.

2. Stuffed desensitizers containing HEMA. The drugs in this group include nano-fillers with a particle size of about 7 nm on the basis of compomer (Sea1 & Rgotest, Dentsply) or Admira Rgotest ormoker ((VOCO, Germani) They are able to shallowly penetrate the dentinal tubules to form on the surface of the dentin airtight layer of resin addition, they contain fluoride and triclosan, which helps to slow down the formation of dental plaque. *This sealant is designed specifically for the protection and prevention of naked necks of the teeth from abrasion, caries and cement for the treatment of the DS.*

3. Dentin bonding agents. Components dentinal adhesive systems penetrate the dentinal tubules, occlusive their gaps, seal the entrances to them, create a barrier between bare dentin and the environment of the mouth.

The most promising in the treatment of DS adhesive systems 6-7-th generation (eg Rgomt L-Pop (Esre) iVond (Negaeus Kulzet), and others.) And self-etching primers (eg, SE Sleagfil Vond (Kugagu), and others.) preserving smear layer partially modifying it.

II. Desensitayzers containing salt.

1) Oxalates. It is assumed that the oxalic acid and its salts are capable of interacting with the surface of dentin, forming the precipitated crystals with calcium and phosphorus, and the gaps thus blocking the dentinal tubules. The best therapeutic effect gives consistent application of 3% hydro potassium oxalate solution and 30% solution of dipotassium oxalate.

Examples: Tenure Quick (Den Mat, USA.), Sensodin Sealant (Block Drug Co., USA), Butler Protect (J.O. Butler, USA), SuperSeal (Phoenix Dental, USA; Amalgadent, Australia).

2) Fluoride. Use lacquers, solutions, gels. Bifluorid 12 (VOCO) contains 6% sodium and calcium fluorides; Fluor Protector (Vivacare) and Fluocal (Septodont) contain 0.1% fluoride ions; Belak-F (Vladmiva) contains potassium fluoride.

Examples of lacquers are based on synthetic Servites and Dentin Protector (Ivoclar). The first contains the antimicrobial agents - chlorhexidine and thymol. It is believed that this component lacquer reduces the bacterial activity in dental plaque on the exposed root cementum and dentin, as it is known, that bacterial stimuli act as stimuli causing DS. Dentin Protector - one-component adhesive agent based on isocyanate polyurethane.

The technique of deep fluoridation is based on the fact that by using simple fluoride (eg sodium fluoride) on the enamel surface of the crystals of calcium fluoride formed, the amount of which is equal to about 1000 A. Interaction №1 liquid component containing magnesium fluoride silicate and

№2 liquid containing finely divided calcium hydroxide, applied in sequential them, causes the formation of crystals of calcium fluoride, magnesium and copper compounds as small as 50 A. The crystals included in the silica gel, which it creates a kind of plug length of 5-10 mm, completely covering the lumen of the dentinal tubules.

III. The combination of several salts

Table 3. The combination of several salts

D/Sense2 (Phoenix Dental USA)	The liquid used in the first step contains water, potassium phosphate, potassium carbonate and sodium methylparaben. The liquid used in the second stage contains water, calcium chloride, strontium and sodium benzoate	When applying the first liquid have a therapeutic effect mainly potassium ions. After application of the second liquid occurs rapidly as the reaction between the components of the first and second liquids, and calcium and phosphorus dentin. As a result, a layer of 3 m thick on the dentin surface and then crystallized and precipitated in the dentinal tubules macrocrystals occlusive their lumens.
Gipostez Ca/Sr (Raduga R)	Presented in the form of 2 fluids containing potassium phosphate and potassium carbonate (liquid №1); calcium chloride and strontium chloride (№2 liquid)	As a result sequential coating liquids №1 and 2 on the tooth surface reaction occurs, creating four crystalline layer of insoluble calcium and strontium salts which protect the dentinal tubules. Soluble potassium chloride salt penetrates deeply into the tubules, causing a therapeutic effect

A wide variety desensitayzery indicates that there is no means universal.

PHYSICAL METHODS OF TREATMENT

1. Electroanesthesia hard dental tissues

2. Audioanalgeziya
3. Hypnosis
4. Xenon laser leads to the melting of the dentine tubules and closing it.
5. The low-intensity gallium laser.
6. Electroacupuncture
7. Laser and magnetic reflexotherapy

COMBINATION METHODS OF TREATMENT:

1) Electrophoresis sodium fluoride solution,
2) Electrophoresis of 9% sodium fluoride and of gel strontium chloride
3) Tin fluoride or strontium chloride with ionizing toothbrush,
4) Drug vacuum electrophoresis with a 5% solution of calcium chloride and 10% solution of magnesium sulfate,
5) Fluorine-containing paint and laser based on yttrium-aluminum garnet activated by ions neodymium.

INTEGRATED TREATMENT

This is a general and local therapy sensitive dentin calcium and phosphorus. In particular calcium glycerophosphate. Local effects on the hard tooth tissue by means rubbing a paste with calcium glycerophosphate for 6-7 sessions to the sensitive dentin and 7-10 electrophoresis procedures with a solution of calcium glycerophosphate for the month.

A new way to treat sensitive dentin desensitizers, vacuum darsonvalization and calcium glycerophosphate (3rd department of therapeutic dentistry BSMU authors - LN Dedova, Solomevich AS).

Test control

1. Which of the hypotheses most fully explains the sensitivity of dentin?

a) hypothesis odontoblasts receptors;
b) hypothesis of direct stimulation of nerve endings;
c) hydrodynamic hypothesis.

2. How are predisposed to sensitivity of different groups of dentin of the teeth (ascending)?

a) molars - cutters - premolars - fangs;
b) cutters - premolars - teeth - molars;
c) premolars - cutters - molar - teeth.

3. Are gender influences the prevalence of dentine sensitivity?

a) men are affected more often;
b) women are affected more often;
c) gender does not matter.

4. The greatest frequency of dentin sensitivity symptoms is observed at age:

a) 15-30 years;
b) 30-40 years;
c) 40-50 years;
d) 50-60 years;
e) older than 60 years.

5. Indicate causes predisposing to dentin sensitivity:

a) excessive and improper cleaning of teeth;
b) excessive flossing;
c) gum recession;
d) tooth whitening;
e) excessive acidification of the mouth (drinks, juices and others.);
f) anatomical features;
g) all of the above.

6. Indicate methods for subjective assessment of pain in the diagnosis of dentin sensitivity

a) visual analogue scale;
b) numeric rating scale;
c) verbal scale;
d) all of the above.

7. What is the most effective in the professional treatment of dentin sensitivity?

a) the use of dentin adhesive system;
b) fluoride varnish application;
c) use a vacuum and darsonvalization desensitizers;
d) use of calcium glycerophosphate;
e) the use of vacuum darsonvalization, desensitizer and glycerophosphate calcium.

8. Most often for home treatment of dentin sensitivity is prescribed

a) rinse or toothpaste containing 5% KNO_3 solution;
b) mouthwash or toothpaste containing 1500 ppm F
c) rinse or toothpaste containing 5% KNO_3 solution and 1500 ppm F.

9. Indicate the point of application of drugs used for the treatment of dentin sensitivity:

a) the creation of the smear layer, sealing the entrances to the dentinal tubules, dentin sclerosis;

b) obturation of dentinal tubules lumen protein precipitation dentinal fluid;
c) persistent depolarization of the neuro-receptor apparatus pulp-dentin complex;
d) all of the above.

10. Indicate how to conduct the dentin sensitivity prevention during vital bleaching:

a) manufacturer reduces the concentration of active components in the bleaching unit;
b) manufacturer introduces into the bleaching agent 5% KNO_3 solution;
c) manufacturer introduces fluorides bleach composition.

LESSON 4. ERGONOMICS IN PERIODONTOLOGY

The questions to be studied for the learning of the topic:

1. Ergonomics: definition, objectives,
2. Requirements for the organization of the workplace dentist.
3. Basic provisions of the ergonomic system.
4. Optimization of the working doctor positions during the Periodontology procedures.
5. Ergonomics during dental plaque removal.
6. Rules for the assistant during the periodontology procedures.

Question 1.Ergonomics: definition, objectives,

International Ergonomic Association defines Ergonomics (or human factors) as “the scientific discipline concerned with the understanding of the interactions among humans and other elements of a system, and the profession that applies theoretical principles, data and methods to design, in order to optimize human well-being and overall system”.

Ergonomics is the science of matching working conditions and human capabilities. The goal is to allow people to perform work and other activities safely and efficiently.

The basic principle in ergonomics is to match tools, equipment, and work methods to the needs of the worker in order to enable him/her perform comfortably to his/her best. Thus, the need is to recognize conditions that lead to discomfort and implement changes to minimize or eliminate those conditions.

Modern ergonomics is an interdisciplinary applied science that studies the optimization possibilities of the man\machine system design by knowing the human’s physical and mental possibilities and limits, his/her capacity to learn, the factors generating errors, the work, the physiology, the human behavior as an individual and within a team, the managerial possibilities, the organizational culture (interdisciplinary study of anatomy, physiology, psychology, management), and the technical and designing possibilities (engineering, design).

Objectives of ergonomics:

1. Protection of the work of dentists;
2. Improving the efficiency and quality of their work;
3. Create for them a normal working environment;
4. Ensuring the safety and comfort of patients;
5. The development of the latest dental equipment.

Question 2. Requirements for the organization of the workplace dentist.

At present, the basic equipment for dental reception is considered to be:

1. Dental unit with recliner

2. Chair doctor.
3. Assistant Chair.
4. Furniture dental function.

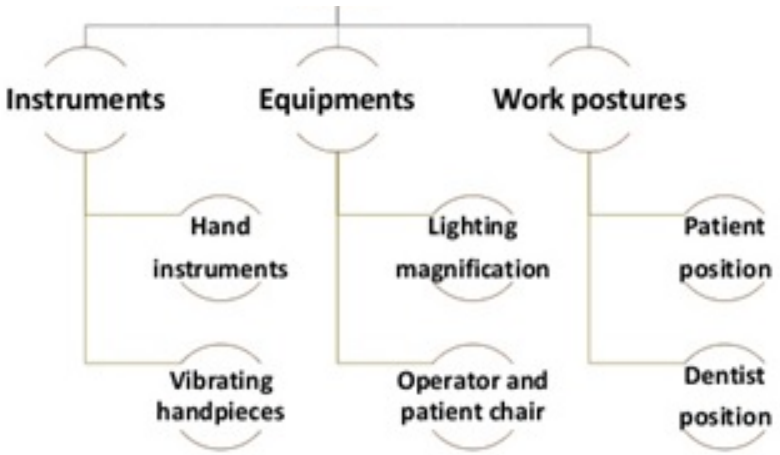


Figure 12. Application of ergonomics in dentistry

Patient chair ergonomic guidelines

Goal: Promote patient comfort, maximize patient access

Look for: 1. Stability; 2. Pivoting or drop-down arm rests (for patient ingress/egress); 3. Supplemental wrist/forearm support (for operator); 4. Articulating head rests; 5. Hands-free operation; 6. Small, thin headrest: Allows for greater leg room.

For Patient. If the clinician is to maintain a balanced posture, the patient must be placed in the supine position, parallel to the floor. The top of the patients head should be at the upper age of the head rest. Most dental chairs can be positioned so that the patient is well supported and reasonably comfortable. Once the patient in the supine position, the height of the patients chair should be set so that the patient's mouth is at the height of the clinicians optimal control point, approximately at the clinician's heart level.

Patient position: "Supine position"

– The patient's heels should be slightly higher than the tip of the nose. This position maintains good blood flow to the head. -An apprehensive patient is more likely to faint if positioned with the head higher than the heels.
– The chair back should be nearly parallel to the floor for maxillary treatment areas. (Chin up).
– The chair back may be raised slightly for mandibular treatment areas. (Chin down).

1) Contraindications for horizontal position.

1. Patients with left heart failure
2. Women in the last three months of pregnancy
3. Elderly people;
4. Patients who have problems with the spine;

5. Patients who have respiratory disease;

6. People, categorically do not want to be treated in this position.

For these patients, the backrest is set at an angle of 60 ° relative to the vertical plane.

Dental chair requirements

1) Sitting with an angle of 110° or a little higher between lower and upper legs.
2) The seat is divided in 2 parts:
   - A horizontal part at the rear for supporting the buttocks with a minimal length of 15 cm
   - An oblique front part declining 20° for an equal support of the thighs
   - Movable front part, an angle of more than 110° between lower and upper legs
3) The maximum depth of the seat shall be 40 cm and the width 40cm with a maximum of 43 cm.
4) A lumbar or pelvic support of 10 to 12 cm high that is adjustable vertically from 17-22 cm and for very tall dentists to 24 cm.
5) The pelvic support can rotate around a horizontal axis with an angle of 25° upwards and downwards.
6) The upholstery of the seat has to be sufficiently hard with a roughened surface. It has to be firm, depressing only slightly.
7) Support has to be given up to a point just before the elbow to maintain the agility of the underarm and hands.
8) Width of 10-12 cm and it being not too long.

Assistant chair. It should have a wide base, no less than five wheels for stability. Assistant to sit during work at 10-12 cm above the doctor for a better view. Therefore, over the wheels at a height of 10-12 cm is fixed footrest for the conservation the knee angle of 90 degrees.

Backrest assistant represented semicircular roller, which is movable relative to the base circle at 360 degrees for the assistant support body in any direction.

Question 3. Basic provisions of the ergonomic system.

General provisions for the dentist working posture in the sitting position

Parameters of the correct working postures (figure 13).

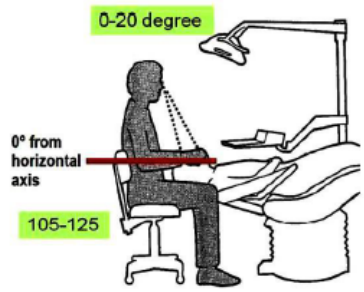


Figure 13: The upright and symmetrical sitting posture

1. The sitting posture is upright and symmetrical.
2. The shoulders hanging down relaxed with the upper arms beside the upper body.
3. The forearms have been lightly elevated.
4. The angle between lower and upper legs is approx5. The legs are slightly apart, making an angle of between 30-45°.
5. The patient‘s head is appropriately rotated in 3 directions.
6. The light beam of the dental operating light is as parallel as possible to the viewing.
7. The sitting location, between 09.00- 12.00 o‘clock, for left-handed people 03.00- 12.00.
8. The soles should he on the floor.
9. The patient‘s head is rotated and the sitting location adjusted.
10. Instruments held in 3 supporting points.
11. The upper part of the body should be perpendicular on the chair forward movements should be made without curving the spine.
12. The head could bend 20°-25°.
13. The arms should be close to the body.

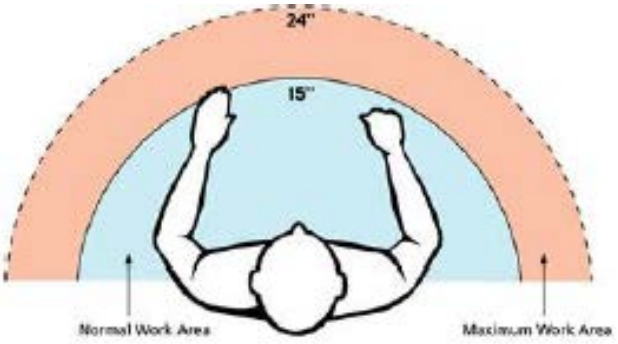


Figure 14. Equipment Layout.

Dental equipment should be located in a manner which allows you to maintain a neutral working posture. It should require minimum adjustment and effort to access so as to reduce postural deviation while working. Frequently used items should be kept within a comfortable distance (22–26 inches for most people) and not above shoulder height or below waist height. Frequently used items such as the syringe, hand piece, saliva ejector and high volume evacuator should be positioned so they are within a normal horizontal reach which is the arc created while sweeping the forearm when the upper arm is held at the side.

Items that are used less frequently used should be placed within the maximal horizontal reach which created when the arm is fully extended. The following image shows the difference between a normal and maximum work area.

Work in the sitting position. When working in sitting postures a chair is required to support the seat and back. In this situation one should alternate active and passive sitting postures. The active posture could be defined as the correct body posture that is maintained by the muscles of the back, the back being leaned forward. If viewed from the side, the back has a natural shape that looks like the letter S. This posture cannot be maintained for a very long time. The passive posture is he one in which the back is sustained by the dentists back of the chair.

Question 4. Optimization of the working doctor positions during the Periodontology procedures.

1) The main provisions of the concept of «p.d.» (proprioceptive derivation).

The concept of ergonomics was introduced into dentistry in order to improve the dental profession's working conditions; the work concepts included sit-down and four-handed dentistry. An American dentist, Dr. Daryl Beach, developed a new concept for dental practice. It focuses on the positions, movements, contacts and comfort that dentists can sense with their bodies. This concept is widely known as proprioceptive derivation (Pd). However, when the concept was first introduced, it was also identified as system or performance logic.

In the Pd concept the adjustable conventional dental equipment and the work process are causative factors behind the high prevalence of musculoskeletal discomfort in dentists. Therefore, instead of a tilted dental chair and an adjustable lamp, Pd introduces equipment with minimum adjustability. The patient lies horizontally during treatment, and the dentist consistently works in a full upright alert seated posture. The dentist's upright posture is considered to provide the best control of the fine stabilized finger movements required when operating in the mouth. By stabilizing the position of the mouth, the dentist and the assistant are able to easily reach necessary equipment and materials, they can work more accurately, more efficiently, and with less physical and mental wear and tear on both the patient and the dentist.

In this concept, proprioception means "a sense or perception, usually at a subconscious level, of the movements and position of the body and especially its limbs, independent of vision; this sense is gained primarily from input from sensory nerve terminals in muscles and tendons (muscle spindles) and the fibrous capsule of joints combined with input from the vestibular apparatus". In other words, the concept of Pd allows the dentist to use proprioceptive self-awareness to determine the most efficient, stress-free process of performing dental procedures.

When dentists learn Pd, starting with the training period, the dentist is neither told nor shown how to sit, how to position the patient, or how to

maintain the relations in the dental process (such as height of the supportive system, position of the dental instrument tray). Instead, dentists individually remember these setting via their proprioception. The determination of the dentist's posture, patient positioning, and the dental process are based on the five movements along with the ten-step protocol derived through the skilled practice of Pd.

1.1. Five Movements

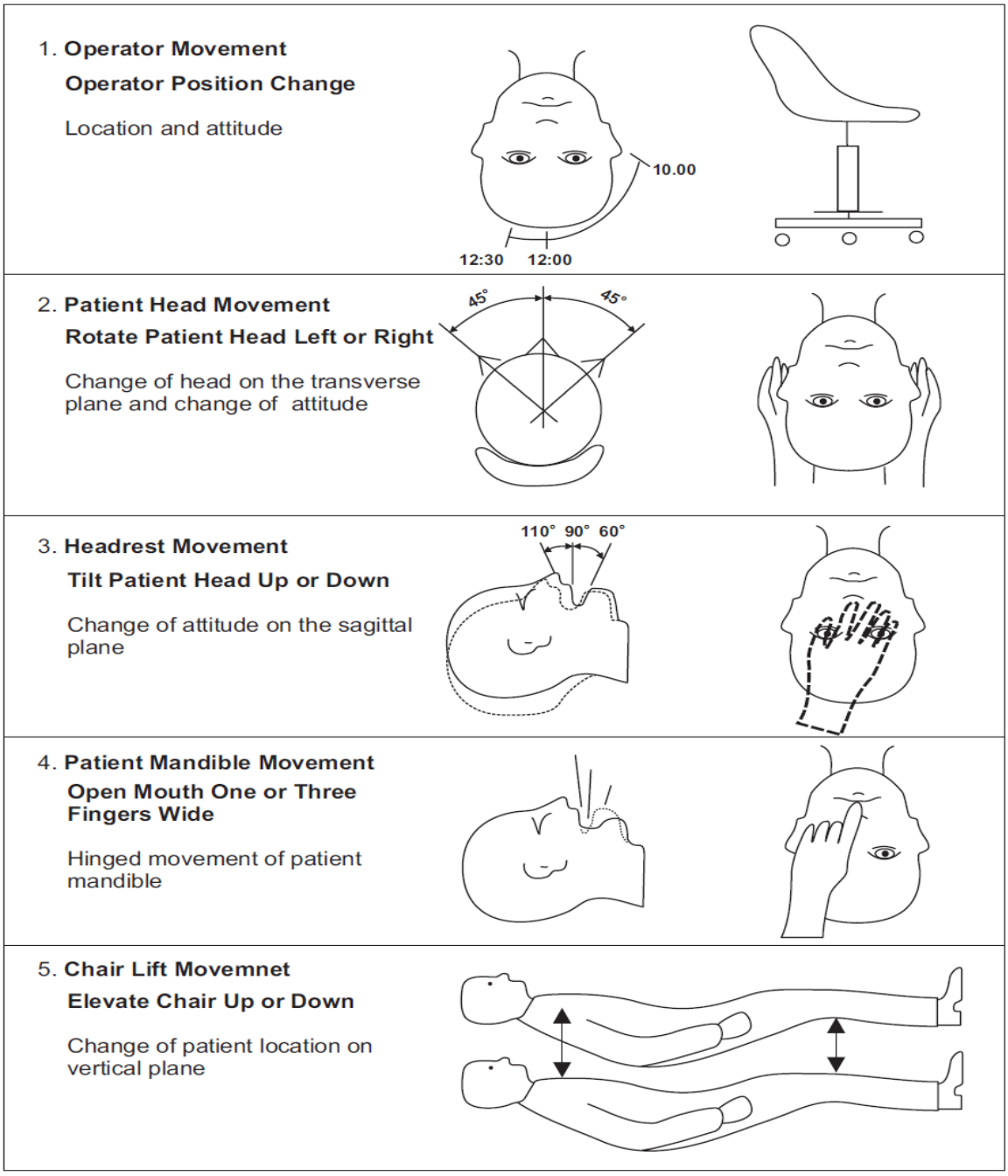


Figure 15. Five movements in Proprioceptive Derivation

During dental treatment based on Pd, dentists focus on five movements related to dentist performance, posture and patient's position (Figure 1):

1. Dentist movement around the patient's head in a clockwise or counter-clockwise direction;
2. Patient head movement by rotation to the left or right;
3. Patient head tilt upward or downward;
4. Patient mandible movement: minimising or maximising the mouth opening of patient;
5. Elevation of the patient's support by moving the supportive system upward or downward

1.2. Ten-Step Protocol

Dentists are additionally given a ten-step protocol guide to optimal perception and control of dentist performance:

1. Establish appropriate inter-maxillary opening;
2. Grasp instrument or item to be used with thumb and index finger;
3. Place instrument or item to place to task site;
4. Stabilize instrument or item with middle finger on task site or as proximal as possible;
5. Check posture to determine whether steps 1–4 have compromised (to cause impairment) posture. If not, then process directly to step 6. If posture is compromised, correct it by rotating patient's head right or left and by dentist's movement clockwise or counter-clockwise;
6. Check vector of force application (axis of instrument or item) to assure alignment with mid-sagittal plane. If adjustment is necessary, correct by rotating patient's head or dentist's movement;
7. Plan to move instrument or item from distant point to near point on task site;
8. Establish eye-to-task sight line with direct or indirect view as appropriate;
9. Stimulate performance to ensure optimal performance;
10. Perform act to achieve the planned outcome.

Pd is also combined with a training program called SATV (Skill, Acquisition, Training and Verification), which helps dentists in gaining self-derived experience. The SATV system is divided into skill acquisition, skill transfer, and skill verification phases.

In the skill acquisition phase, dentists use models for training. Body positions and setting requirements that are compatible with the highest imaginable level of clinical performance are recorded. These conditions are considered to minimize physical stress during dental treatment. The derivations are then used to adjust the SATV clinical setting to the dentist's unique body dimensions for optimal delivery of care.

The skill transfer phase emphasizes that the acquired basic skills may be applied to clinical procedures such as oral examination, extraction,

anaesthesia, tooth cavity restoration, root canal treatment and preparation for crowns and bridges.

Skill verification (by means of multimedia such as a camera, digital video recordings or data forms, and standardized simulated pathologies) of skill acquisition and transfer is used throughout the system

2) Working Posture and Techniques.

For right-handed clinicians, working in the range from 7 to 9 o'clock is commonly associated with twisting of the trunk and neck as well as working with an elevated elbow posture in order to gain access. The mirror image (3 to 5 o'clock) is equally problematic for left-handed clinicians. In an attempt to reduce such postural deviations a conservative range from 10 o'clock to approximately 12:30 is preferred and shown below (UBC, 2008).

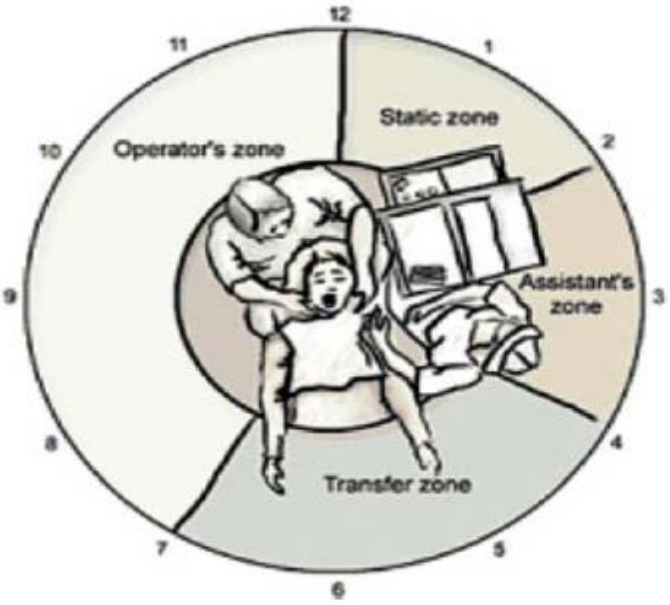


Figure 16. Position for right handed dentist

3) Ergonomic of tools. Instrumentation.

The design of dental instrumentation can play a key role in the prevention of negative health effects for its users. Dental clinicians are typically responsible for selecting and maintaining their own instruments and equipment. Although instrument design has come a long way since its beginning, dental professionals often select instruments based on familiarity rather than actual quality or specific properties (Sanders, 1997).

The goal of proper instrument selection should be to reduce force exertion while allowing for neutral joint positioning. The following table summarizes critical areas to consider when selecting new or evaluating existing instrumentation.

Table 4. Characteristics of tools

Handle shape and size	✓ Dental instrument diameter ranges from 5.6 to 11.5 mm. Larger handle diameters reduce hand muscle load and pinch force, although diameters greater than 10 mm (3/8 inch) have boon shown to offer no addition advantage (Dong. 2006). ✓ Alternating tools with different diameter sizes allows the user to reduce the duration of prolonged pinch gripping. Sleeves that fit over the handles of mirrors have been shown to reduce grip force (Simmer-Beck. 2006), but may not have the same effect on scaling instruments duo to the extra force required when scaling. ✓ "No. 4" handle lessens pinch gripping and can be purchased for most instruments. ✓ A round handle, compared to a hexagon handle will reduce muscle force and compression.
Weight	Lightweight instruments (15 g or less) help reduce muscle workload and pinch force (Dong. 2006).
Balance / Maneuverability	✓ The instrument should be equally balanced within the hand so that the tendency to deviate the wrist is reduced. ✓ Balancing an instrument is improved using a third digit rest compared a fourth digit rest since it does not engage the wrist as much while guiding and positioning the hand piece. The second digit (index finger) can detect very fine movements and should be placed close to the operating point. By not using the fourth digit as a stabilizer of the hand piece reduces the number of fingers in the oral cavity, improves the ability to position instruments, and involves as few joint segments as possible thereby improving the degree of control and providing enhanced tactile ability.
Ease of operation	The easier it is to operate a tool, the better. Less time is spent searching for buttons, thereby reducing the risk of error. Less time is also spent learning how to use the device. Simple activation is also important, such as using a foot pedal or handle turn to activate the tool as they do not require the operator to hold a button in a sustained pinch grip for extended periods of time.
Sharpness	As a tool becomes dull, additional force is required to perform tasks. As a result, it is important to maintain sharpness of the instruments.
Texture	Knurled handles such as diamond-shaped or crisscross patterns serve to reduce pinch grip force due to an increase in tactile sensation as a result of the knurl.

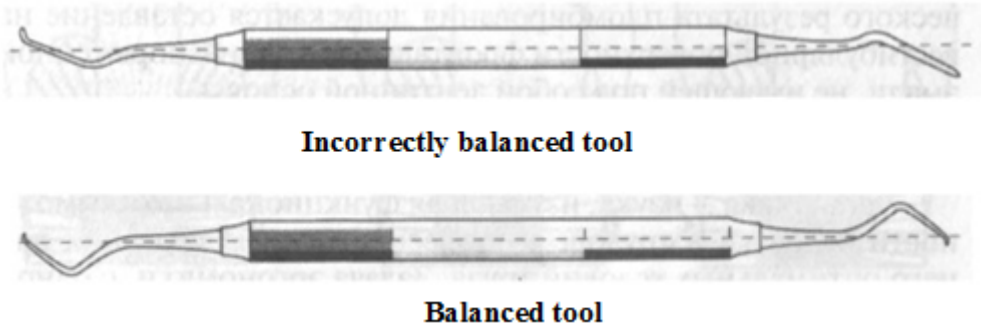


Figure 17. Balanced tool.

Dental Mirrors. Good quality mouth mirrors when used appropriately will be able to prevent awkward body positioning and maintain a neutral working posture. Use of dull, scratched mirror surface can cause strain to the eyes. So it is recommended to use scratch resistant, antifogging double sided mouth mirrors and compressed air to improve the clarity of operating field which enhances the ability to appreciate colour and to differentiate texture of tooth coloured restoration. Mirror to handle angle is set at 450 and held in a vertical manner. Handle of the mirror is made lighter with slight ribbing parallel to the long axis of handle to facilitate rotational movements.

Gloves. Gloves of proper size and fit should be selected for each dental healthcare worker, as it is a potential contributor to Carpal Tunnel syndrome

Magnification. Surgical magnification by using fixed microscopes mounted in ceiling or surgical loupes with lower magnification attached to a head band or mounted on operator’s glass can drastically augment the visual competence.

Delivery System. Practice of four handed dentistry maintains a position around the operating field with limited hand, arm and body movements. From an ergonomic viewpoint, over the head and over the patient delivery system better allow the dental assistant to access the hand pieces for bur changes and other operations.

Question 5. Ergonomics when dental plaque removal.

When working with the teeth of the upper jaw as much as possible omitted chair, legs are placed slightly above the patient's head.

When working with the teeth of the lower jaw of the rear seats can be lowered slightly and the patient's chin dropped to his chest.

Table 5. Recommendations Rights Institute (HPI, Japan) on the operator's position during the removal of dental plaque.

The position of the operator	*Work Area*
12 hours	Removal of dental plaque from anterior teeth UJ and LJ
11 hours – 1 hour	Removal of dental plaque with premolars UJ and LJ
10 hours	Removal of dental plaque with posterior teeth UJ and LJ

Rotate patient's head at an angle of 30-45 degrees to the right is recommended for removal of dental plaque from molars and premolars UJ (from the buccal surface - and left the palatal surface - right) and LJ (lingual surface - the right cheek - left).

Rotation angle of 30-45 degrees to the left of the vertical line is recommended for the removal of dental plaque from the posterior teeth: buccal surface with high and low - on the right, with the palatal and lingual surfaces - left

The position -15 'and -30' doctor and hygienist working with the teeth, which is located right on the lower jaw, using dental mirror - right side of the upper jaw.

The position -45 'and -60' doctor and hygienist working with teeth, is on the left in the lower jaw, as well as around the bottom row.

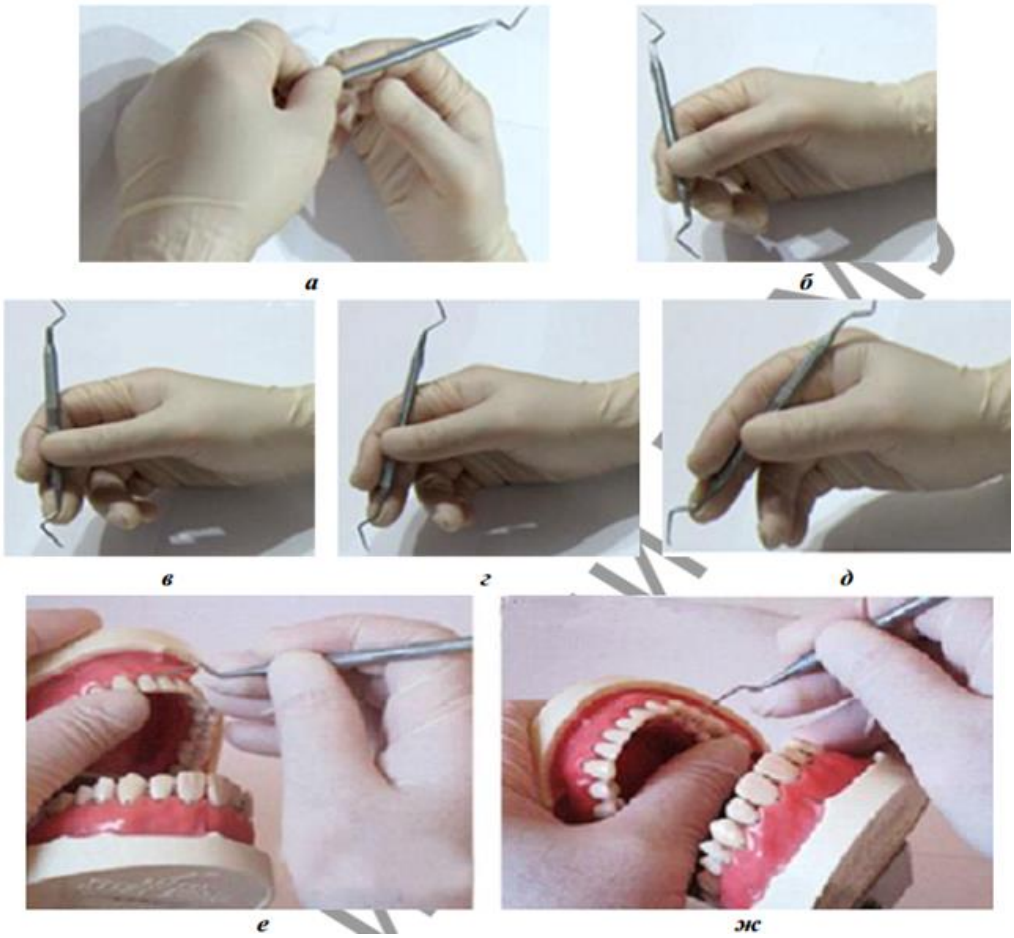


Figure 18. Methods of capturing and fixing the dental instruments in the hands of the operator, which allows to control the power of applications and accuracy of work.

Question 6. Rules for the assistant during the Periodontology procedures.

Dental Assistant will:

1. Conduct training workplace to receive a doctor.
2. Prepare the necessary tools and drugs for anesthesia applications, prepare the filling material.
3. Carry out the call of the patient.
4. providing a comfortable position of the patient in a chair for himself, and for the doctor.
5. Invite the dentist to the patient as it is ready.
6. Set up the dental light.
7. Serve tools to doctor.
8. To ensure the prevention transmitting infections of the patient and staff.
9. Work with the saliva ejector and a vacuum cleaner.
10. Independently conduct hygienic measures, and determine the index together with a doctor to carry out evaluation of the effectiveness of preventive measures.
11. Together with the manager (and in his absence - in consultation with a doctor) to appoint the patient on repeated visits.
12. To carry out the filling dental formula and the help to the doctor in the design of medical records.
13. Ensure the filling of forms, appointments as directed by a dentist.
14. To carry out health education to the patient at the time of admission.
15. To ensure the selection and viewing of television and video programs in the patient chair.

Test control

1. Dentist's position should be:

a) sitting;
b) the back straight;
c) of the foot flat on the floor;
d) the body slightly tilted forward;
e) the upper arms remain close to the body;
f) all of the above,

2. Indicate the head position on the lower jaw during working:

a) the maxillary position;
b) mandibular position.

3. The eyes of the interlocutors during a conversation with the patient should be located:

a) at the same level;
b) levels above the eye doctor the patient's eye;

c) the level of the patient's eye above the eye doctor.

4. In the supine position is not treated:

a) Pregnant women;
b) the elderly;
c) patients who have problems with the spine;
d) patients who have respiratory disease;
e) people who do not want to be treated in this position;
f) all the above.

5. At the 12 o'clock position more often work:

a) with the teeth in the lower dentition;
b) with teeth, to the right in the upper jaw;
c) with teeth, which is located in the left upper jaw.
d) Four front teeth maxilla and mandible

6. Dentist's elbows are:

a) at the height of the patient's head;
b) as close as possible to the body;
c) above the patient's head;
d) below the head of the patient.

7. The distance between the eyes of the dentist and the patient's face should be:

a) 40-50 cm;
b) 10-20 cm;
c) 80-90 cm.

8. The light incidence angle should:

a) coincide or be similar to the corner of our sight;
b) does not coincide with the angle of our view.

9. The angle of the horizontal position of the chair may not exceed:

1) $20\text{-}25^0$;
2) 5^0;
3) 10^0.

10. Indicate the angle of the horizontal position of the chair in the treatment of teeth of the lower jaw:

1) closer to 25^0;
2) closer to 45^0;
3) 90^0.

LESSON 5. PERIODONTAL TISSUE, TO DETERMINE THEIR STATUS. TERMINOLOGY IN PERIODONTOLOGY. FACTORS AFFECTING THE PERIODONTAL CONDITION. FEATURES OF THE BIOLOGICAL SYSTEM. THE CONCEPT OF DEVELOPMENT OF PERIODONTAL DISEASE.

The questions to be studied for the learning of the topic:

1. Periodontium or tooth's supporting structure
2. What is a periodontal diseases?
3. Types of periodontal disease
4. Risk factors
5. Etiology and pathogenesis of periodontal disease

Question 1. Periodontium or tooth's supporting structure

The periodontium (also known as marginal periodontium) is the supporting structure of a tooth, helping to attach the tooth to surrounding tissues and to allow sensations of touch and pressure.

The word comes from the Greek terms peri, meaning "around" and odons, meaning "tooth." Literally taken, it means that which is "around the tooth".

The periodontium consists of four principal components:

- gingiva or the gum
- cementum, covering the root of the tooth
- alveolar bone
- periodontal ligament

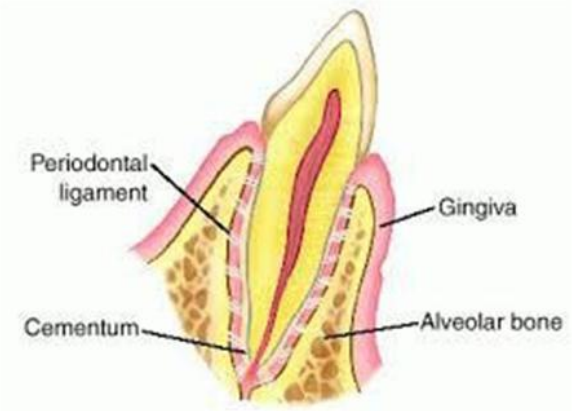


Figure 19. Structure of the periodontium.

Each of these components is distinct in its location, tissue architecture, biochemical and chemical composition. They have their own distinct functions and are capable of adaptation during the life of the structure.

1. Gingiva or the gum. Gingiva is a soft tissue that overlays the jaw bone and surrounds the teeth providing a seal around them. Gingival tissue is

tightly bound to the underlying bone creating an effective barrier (when healthy) for periodontal insults to deeper tissues.

Healthy gingiva is usually coral pink, but may contain melanin pigmentation. Healthy gingiva has a smooth "arcuate" appearance around each tooth, a firm texture that is resistant to movement and no reaction (such as bleeding) to normal disturbance such as brushing or periodontal probing.

Healthy gingiva

Gum infection, called gingivitis, occurs because of microbes in plaque placed on the tooth, if oral hygiene is poor.

Changes in color, particularly increased redness, together with edema or swelling and an increased tendency to bleed (for example, during teeth brushing) suggest an inflammation due to the accumulation of bacterial plaque.

In a more advanced stage of infection, a gingival retraction may occur; this leaves part of the roots naked.

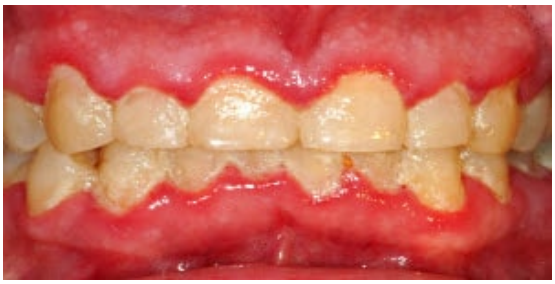


Figure 20. Gingivitis caused by the accumulation of bacterial plaque

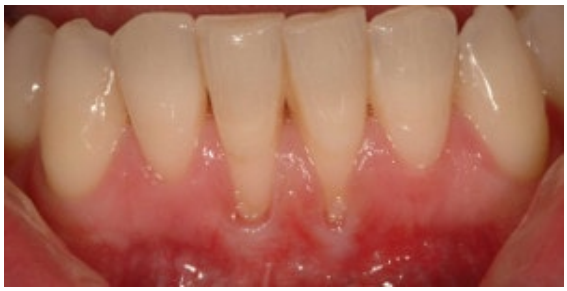


Figure 21. Gingival recession

Inflammation of the gums is the first stage of the periodontal disease. An early treatment is indicated, before the disease advances to a deeper tissue leading to a poorer prognosis.

2. Cementum. Cementum is a specialized calcified substance covering the root of a tooth. It is the part of the periodontium that attaches the teeth to the alveolar bone by anchoring the periodontal ligament.

Cementum is formed continuously throughout life because a new layer of cementum is deposited to keep the attachment intact as the superficial layer of cementum ages. It has a light yellow color and the highest fluoride content of all mineralized tissues.

3. Alveolar bone. The alveolar bone is the bone of the jaw that contains the tooth sockets (also known as dental alveoli oralveolar process) on bones that hold teeth.

The alveolar process contains a region of compact bone (called the lamina dura) which is attached to the cementum of the roots by the periodontal ligaments.

Like any other bone in the human body, alveolar bone is modified throughout life; under the effect of various external factors, it may suffer processes of bone resorption or bone formation.

4. Periodontal ligament. The periodontal ligament is a specialized connective tissue that attaches the cementum of a tooth to the alveolar bone. They are a network of elastic fibres that help support the tooth inside the alveolar bone socket.

Figure 22. Periodontal ligament.

The functions of the periodontal ligaments include attachment of the tooth to the bone, support for the tooth, formation and resorption of bone during tooth movement, sensation, and eruption.

The tooth moves slightly in its socket and puts tension on the periodontal ligaments when pressure is exerted on it (for example, during chewing or biting). This is called tooth physiologic mobility.

The periodontium exists for the purpose of supporting teeth during their function; a constant state of balance always exists between the periodontal structures and the external forces.

Most of the times, periodontal diseases are caused by bacteria from the dental plaque adhering to tooth surfaces.

The gum is the first barrier in case of bacterial infections. In the absence of treatment, the infection progresses to the periodontal ligaments and the alveolar bone involving the progressive loss of the alveolar bone around the teeth which can lead to the loosening and subsequent loss of teeth.

Figure 23. Progression of bacteria and periodontitis

Periodontology or Periodontics is the dental speciality that relates specifically to the care, maintenance and treatment of the periodontal tissues.

Question 2. What is a periodontal diseases?

Periodontal diseases are serious chronic infections that involve destruction of the tooth-supporting apparatus including the gingiva, the periodontal ligament and alveolar bone. These diseases are initiated by a local accumulation of bacteria, i.e., dental plaque, adjacent to the tooth. Periodontal diseases, including gingivitis and periodontitis, can affect one tooth or many teeth, and if left untreated, can lead to tooth loss particularly in adults. It is the most common dental condition in adults, and it is also one of the most common chronic inflammatory diseases, possibly affecting a majority of the population in the world. Although plaque is essential for the initiation of periodontal diseases, the majority of the destructive processes associated with these diseases are due to an excessive host response to the bacterial challenge. Therefore, periodontal disease is a multifactorial, complex disease. The purpose of this chapter is to provide a general overview of the types of periodontal disease, risk factors associated with the disease, and the etiology, pathogenesis and management of periodontal diseases.

Question 3. Types of periodontal disease

Periodontal diseases include two general categories based on whether there is attachment or bone loss: gingivitis and periodontitis. Gingivitis is considered a reversible form of the disease and generally involves inflammation of the gingival tissues without loss of connective tissue attachment. Periodontitis has been defined as the presence of gingival inflammation at sites where there has been a pathological detachment of collagen fibers from cementum, the junctional epithelium has migrated apically and bone loss can be detected radiographically. The inflammatory events associated with connective tissue attachment loss lead to the resorption of coronal portions of tooth supporting alveolar bone.

The concept of periodontal disease is continuously changing as new research evidence emerges. Therefore, the classification of periodontal disease has been changed since the system developed at the 1989 World Workshop in Clinical Periodontics. The classification presented in this chapter is based on the results developed at the 1999 International Workshop organized by the American Academy of Periodontology (AAP).

The classification of periodontal diseases now includes eight general types:

1. Gingivitis
2. Chronic periodontitis
3. Aggressive periodontitis
4. Periodontitis as a manifestation of systemic diseases

5. Necrotizing periodontal diseases
6. Abscesses of the periodontium
7. Periodontitis associated with endodontic lesions
8. Developmental or acquired deformities and conditions

The overall classification system is presented in the Table 1. In addition, the above classification is different from case types previously developed by the American Academy of Periodontology. The current case types for periodontal diseases include:

1. Gingivitis (Case Type I)
2. Mild periodontitis (Case Type II)
3. Moderate periodontitis (Case Type III)
4. Advanced periodontitis (Case Type IV)
5. Refractory periodontitis (Case Type V)

Periodontal Diseases (adapted from: Ann Periodontol 1999;4:1–6.3)

I Gingival Diseases
- Dental plaque-induced gingival diseases
- Nonplaque-induced gingival lesions

II Chronic Periodontitis
- Localized
- Generalized

III Aggressive Periodontitis
- Localized
- Generalized

IV Periodontitis as a Manifestation of IV. Systemic Diseases

V Necrotizing Periodontal Diseases
- Necrotizing ulcerative gingivitis
- Necrotizing ulcerative periodontitis

VI Abscesses of the Periodontium
- Gingival abscess
- Periodonal abscess
- Pericoronal abscess

VII Periodontitis Associated with Endodontic Lesions

VIII Developmental or Acquired Deformities and Conditions

INTERNATIONAL CLASSIFICATION OF DISEASES (ICD- 10)

K05 - Gingivitis and periodontal diseases
K05.0 - Acute gingivitis
K05.1 - Chronic gingivitis
K05.2 - Aggressive periodontitis
K05.3 - Chronic periodontitis
- K05.30 - Chronic periodontitis, unspecified
- K05.31 - Chronic periodontitis, localized

K05.32 - Chronic periodontitis, generalized
K05.4 - Periodontosis
K05.5 - Other periodontal diseases
K05.6 - Periodontal disease, unspecified

Question 4. Risk factors

There are a number of risk factors associated with periodontal diseases. Determining risk is helpful in developing recommendations for prevention and in determining strategies for the overall management of periodontitis. It has been recognized that the severity and progression of periodontal disease varies from individual to individual. Bacteria are essential for the initiation of the disease but it is the host response to the bacterial challenge that determines the severity and progression of the disease. Therefore, it is the host's immunologic reaction that determines susceptibility to the disease.

The general category for risk factors associated with each individual for developing periodontitis, includes genetic, environmental (e.g., tobacco use), and acquired risk factors (e.g., systemic disease). Risk factors (Table 2) and risk reduction strategies (Table 3) should be considered when assessing each patient. Some risk factors can be modified to reduce a patient's susceptibility. Environmental factors such as tobacco use, and stress can be managed with smoking cessation and stress management; for acquired factors such as systemic diseases, medications usually prescribed by the physician can be used to help in the management and control of chronic disorders (Table 3). The use of chemotherapeutic agents specifically designed to improve upon the clinical outcomes of mechanical treatments for periodontal diseases may be particularly useful in the management of those individuals with single or multiple risk factors. Risk assessment can help the practitioner to establish an accurate diagnosis, provide an optimal treatment plan, and determine appropriate maintenance programs. In patients with multiple risk factors, the practitioner may aggressively use pharmacologic adjuncts such as antimicrobials and host modulatory therapy in addition to mechanical therapy. It is also important to update and assess risk factors for each patient on a regular basis as some of these factors are subject to change throughout life.

Risk Assessment for Periodontitis

(Sources: J Periodontol 1994;65:260–267.29 J Periodontol 1995;66:23–29.30 J Periodontol 1999;70:711–723.31 J Periodontol 2000;71:1057–1066.32 J Periodontol 2000;71:1215–1223.33 J Periodontol 2000;71:1492–1498.34)

1. Heredity as determined by genetic testing and family history
2. Smoking, including frequency, current use, and history
3. Hormonal variations such as those seen in:

a) Pregnancy, in which there are increased levels of estradiol and progesterone that may change the environment and permit virulent organisms to become more destructive
b) Menopause, in which the reduction in estrogen levels leads to osteopenia and eventually osteoporosis

4. Systemic diseases such as:
   a) Diabetes (the duration and level of control are important)
   b) Osteoporosis
   c) Immune system disorders such as HIV
   d) Hematologic disorders such as neutropenias
   e) Connective tissue disorders such as Marfan's and Ehlers-Danlos syndromes
5. Stress as reported by the patient
6. Nutritional deficiencies and obesity that may require a dietary analysis
7. Medications such as:
   a) Calcium channel blockers
   b) Immunomodulatory agents
   c) Anticonvulsants
   d) Those known to cause dry mouth or xerostomia
8. Faulty dentistry such as overhangs and subgingival margins
9. Poor oral hygiene resulting in excessive plaque and calculus
10. History of periodontal disease

Risk Reduction Strategies

(source: Dent Clin North Am 2005;49:611–636.35)

1. More frequent visits for those with a genetic predisposition; use of pharmacotherapeutics for the management of periodontitis
2. Smoking cessation using one or more of the six approved regimens; these regimens are rarely successful as sole therapies (multiple forms of therapy often are used in combination with counseling to achieve success)
3. Hormonal variations such as those seen in:
   a) Pregnancy, which requires good oral care before conception to prevent complications during pregnancy; treatment during pregnancy may be necessary to prevent adverse pregnancy outcomes
   b) Menopause, which may require hormonal supplements, calcium, and other medications and supplements prescribed by the physician to prevent osteopenia
4. Systemic diseases that require consultation with the physician include:
   a) Diabetes (for improved glycemic control)
   b) Osteoporosis (requiring calcium supplements, bisphosphonates)
   c) Immune system and hematologic disorders
   d) Connective tissue disorders
5. Stress management; possible referral to a psychologist or psychiatrist

6. Nutritional supplementation and weight reduction; possible referral to a nutritionist
7. Medications can be changed in consultation with the physician
8. Corrective dentistry
9. Improved oral hygiene (brushing, flossing, use of antiseptics)
10. Occlusal adjustments.

Question 5. Etiology and pathogenesis of periodontal disease

Initially, periodontal disease was thought to be related to aging and was therefore uniformly distributed in the population, with the disease severity being directly correlated with plaque levels. Now as a result of extensive research, it has been shown that periodontal disease is initiated by plaque, but the severity and progression of the disease is determined by the host response to the bacterial biofilm. People with severe plaque and calculus accumulation will have gingivitis, but not necessarily periodontitis. On the other hand, certain individuals, despite maintaining adequate oral hygiene, find themselves susceptible to aggressive forms of periodontitis, with deep pocketing, tooth mobility, and early tooth loss. Clearly, the response of the periodontal tissues to plaque is different in these two different scenarios. Periodontal disease does not appear to behave as a classic infection, but more as an opportunistic infection. These observations led researchers to realize that the host response to the bacterial challenge presented by subgingival plaque is the important determinant of disease severity. Although plaque bacteria are capable of causing direct damage to the periodontal tissues, it is now recognized that the host immuno-inflammatory response to plaque bacteria produces destructive cytokines and enzymes resulting in periodontal tissue destruction. The host response is essentially protective by intent but can also result in tissue damage, including the breakdown of connective tissue fibers in the periodontal ligament and the resorption of alveolar bone. The host response to the plaque biofilm is modified by genetic factors (helping to explain why aggressive periodontitis tends to have a familial aggregation) and systemic and environmental factors (e.g., smoking, diabetes, stress).

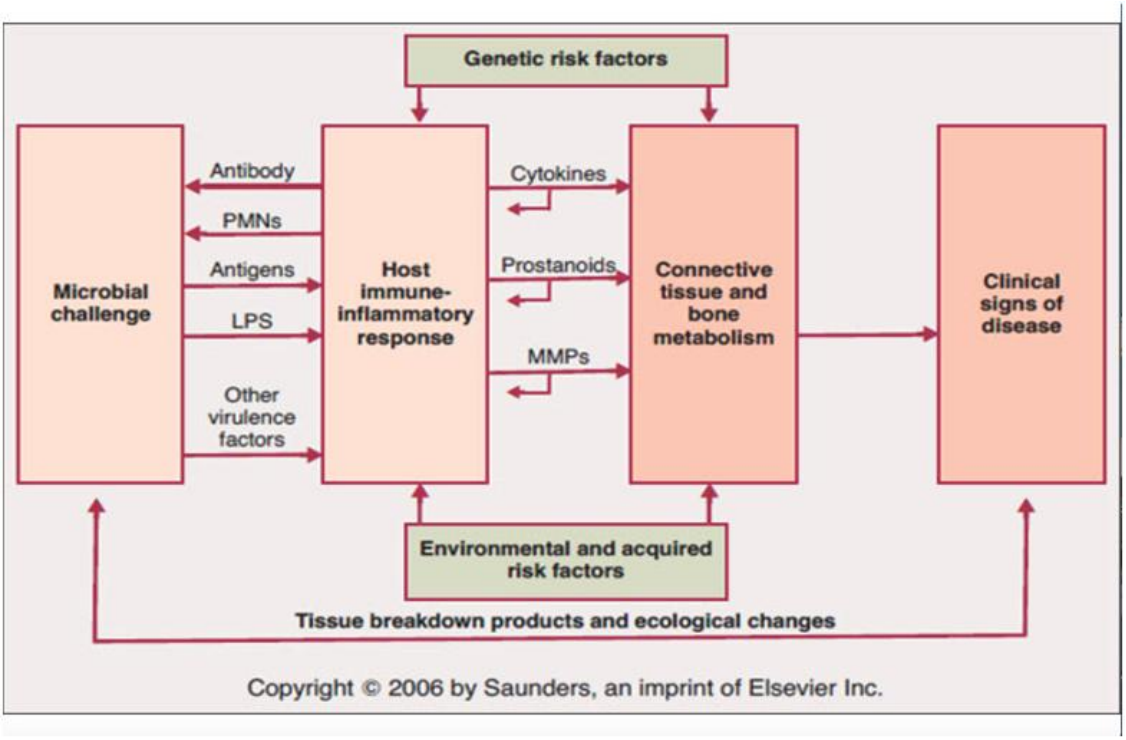


Figure 24. Schematic Illustration of the Pathogenesis of Periodontitis (Source: Carranza's Clinical Periodontology, 10th Ed. WB Saunders Company; 2006:275–282.36 Reproduced with permission.)

To better treat and manage periodontal diseases, we need a more detailed understanding of periodontal pathogenesis (Figure 1). The bacteria and their metabolic products (e.g., endotoxin) stimulate the junctional epithelium to proliferate, and to produce tissue-destructive proteinases. This infection also increases the permeability of the junctional epithelium which allows microbes and their products to gain access to the subepithelial connective tissue. Epithelial and connective tissue cells are thus stimulated to produce inflammatory mediators that result in an inflammatory response in the tissues. Microbial products also chemotactically attract a constant flux of pro-inflammatory cells migrating from the circulation to the gingival crevice. Neutrophils, or polymorphonuclear leukocytes (PMNs), are predominant in the early stages of gingival inflammation. Thus an immune response is generated in the periodontal tissues and pro-inflammatory cytokines such as IL-1β, TNF-α and matrix metalloproteinases (MMPs) are produced by inflammatory cells recruited to the lesion site. The functions of PMNs include phagocytosis and destruction of bacteria. Initially the clinical signs of gingivitis are evident. This response is essentially protective in nature to control the bacterial infection. In persons who are not susceptible to periodontitis, the primary defense mechanisms control the infection, and chronic inflammation (i.e., chronic gingivitis) may persist. However, in individuals susceptible to periodontitis, the above inflammatory process will eventually extend apically and laterally to involve deeper connective tissues

and alveolar bone, recruiting monocytes and lymphocytes to the site of infection at these later stages. These monocytes and macrophages are activated by the bacterial endotoxins such as LPS leading to the production of high levels of prostaglandins (e.g., prostaglandin E2, PGE2), interleukins (e.g., IL-1α, IL-1β, IL-6), tumor necrosis factor alpha (TNF-α), and MMPs by the host cells. The MMPs break down collagen fibers, disrupting the normal anatomy of the gingival tissues and resulting in destruction of the periodontal apparatus. If left untreated the inflammation continues to extend apically, and osteoclasts are stimulated to resorb alveolar bone triggered by the high levels of prostaglandins, interleukins, and TNF-α in the tissues. The elevated levels of pro-inflammatory mediators and MMPs are counterbalanced by a protective response in the host with elevations in anti-inflammatory mediators such as the cytokines IL-4 and IL-10, as well as other mediators such as IL-1ra (receptor antagonist) and tissue inhibitors of matrix metalloproteinases (TIMPs) (Figure2). Under normal healthy conditions, the anti-inflammatory mediators are balanced with inflammatory mediators, thereby controlling tissue destruction. If an imbalance is seen, with excessive levels of the pro-inflammatory mediators and upregulated MMP expression and activity and insufficient levels of protective anti-inflammatory mediators, the loss of periodontal connective tissue and bone will occur».

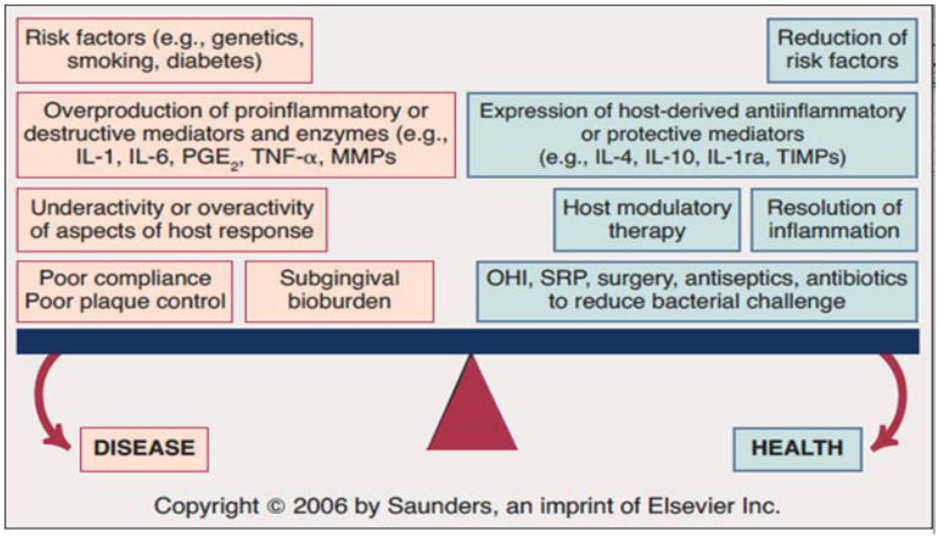


Figure 25. The Periodontal Balance Carranza's Clinical Periodontology, 10th Ed. WB Saunders Company; 2006:275–282.36 Reproduced with permission

Thus, plaque bacteria initiate the disease, inflammatory response developed by host produces excessive levels of pro-inflammatory mediators (prostaglandins, interleukins) and enzymes (MMPs) result in the destruction of periodontal tissue. If this inflammation continues and extends further apically, more bone is resorbed, and more periodontal tissue is broken down leading to deeper and deeper pockets and associated attachment and bone

loss revealed as the clinical and radiographic signs of periodontitis. In people with periodontitis these inflammatory mediators (e.g., prostanoids, and cytokines) and local oral bacteria will eventually enter into the circulation, stimulating the liver to produce acute phase proteins (notably CRP, but also fibrinogen, haptoglobin, etc.) which are "biomarkers" of a systemic inflammatory response. The ever expanding data supporting the fact that this systemic inflammatory response driven by the chronic infection and inflammation associated with periodontitis will eventually increase an individuals risk for developing a number of systemic diseases including cardiovascular diseases, adverse pregnancy outcomes, and diabetic complications.

Test control

1. Periodontium is a complex tissue, which comprises:

a) gum;
b) the periodontal ligament;
c) cement of the tooth root;
d) alveolar bone;
e) tooth pulp;
f) the blood vessels and nerves

2. Gum is divided into:

a) interdental;
b) free;
c) attached;
d) all of the above.

3. All epithelium covering the free gum is divided into:

a) oral;
b) crevice;
c) connector;
d) transition.

4. Functions of the periodontal ligament are:

a) the support, shock absorbing;
b) trophic;
c) touch;
d) plastic, construction;
e) all of the above.

5. Cellular cement is located:

a) in the apical part of the root;
b) in the furcation;

c) 3 covers the whole root;
d) all of the above.

6. Periodontium performs the following functions:

a) barrier;
b) bearing, shock absorbing;
c) trophic;
d) reflex regulation of chewing pressure;
e) plastic;
f) all of the above.

7. Periodontal examination includes:

a) the measurement of the depth of periodontal pockets;
b) the study of bifurcations of the teeth;
c) the definition of loose teeth;
d) the study of the bite with periodontal lesions;
e) indexes;
f) all of the above.

8. The criteria for assessing of the periodontal attachment condition are:

a) safety
b) localization
c) sounding depth
d) all of the above

9. The criteria for assessing of the gums condition are:

a) color
b) surface
c) contour
d) consistency
e) bleeding
f) all of the above

10. Indicate the international classification code "Gingivitis and periodontal disease":

a) J95
b) K05
c) H02
d) P10

LESSON 6. PLAN FOR EVALUATION OF PATIENTS WITH PERIODONTAL DISEASES

The questions to be studied for the learning of the topic:

1. Characteristics of the stages of survey of patients with periodontal disease.
2. The subjective examination: complaints, anamnesis.
3. Determination of the status of the oral cavity
4. The primary visual characteristics of periodontal tissues.
5. A detailed study of the periodontal tissues.

Question 1. Stages of survey design of patients with periodontal diseases.

Optimal diagnosis can be made in several stages.

The first stage

1. Introduction to patient.
2. The patient's medical history.
3. The patient's social history.
4. The patient's complaints.
5. Assessment of the patient's oral status.
    - 5.1. Hygiene.
    - 5.2. Breath.
    - 5.3. Saliva.
    - 5.4. Lips.
    - 5.5. Oral mucosa.
    - 5.6. Tongue.
    - 5.7. Palate.
    - 5.8. Pharyngeal area.
    - 5.9. Architectonics of the vestibule and the floor of the mouth.
    - 5.10. Examination of the teeth.
        - 5.10.1. Assessment of the number and causes of tooth loss.
        - 5.10.2. Sensitivity of dentine.
        - 5.10.3. Condition of the contact surfaces of the teeth.
        - 5.10.4. Tooth mobility.
        - 5.10.5. Percussion of the tooth.
        - 5.10.6. The presence of pathologic migration of the teeth.
        - 5.10.7. Examination of the occlusal contacts.
6. Primary visual characteristics of the periodontal tissues.
7. Preliminary diagnosis.
8. X-ray examination.

The second stage

1. A detailed examination of the periodontal tissues.
    - 1.1. The presence of the supra- and subgingival dental plaque.
    - 1.2. The level of the gingiva involvement in the pathological process.

1.3. The level of the periodontal tissues destruction, the presence of the periodontal pockets.
1.4. The presence of pus discharge from the periodontal pockets.
1.5. The level of the alveolar bone destruction.
1.6. Trauma from occlusion.
1.7. The state of periodontal microcirculation.

2. Additional methods of diagnosis.
2.1. Laboratory tests
2.1.1. microbiological examination;
2.1.2.biochemical studies;
2.1.3. immunobiological studies;
2.1.4. cytological examination
2.1.5. morphological studies
2.1.6. genetic studies.
2.2. Functional studies
2.2.1. definition of vacuum samples for resistance and permeability of capillaries of the gums
2.2.2. determination of the index of the peripheral circulation - IPC
2.2.3. laser-optical method for the diagnosis of microcirculation gums
2.2.4. dopplerography
2.2.5. biomicroscopy

Question 2. Subjective examination.

1) Complaints of the patient.

Main complaints:

- bleeding gums,
- loose teeth,
- spreading of the teeth with the appearance of spaces where none existed before,
- halitosis, itchy feeling in the gums,
- relieved by a digging with a toothpick,
- constant dull gnawing pain,
- dull rain after eating,
- deep radiating pains in the jaws,
- acute throbbing pain,
- sensitivity to percussion,
- sensitivity to heat and cold,
- burning sensation in the gums,
- extreme sensitivity to inhaled air.

2) History of the disease. When the disease was found first, communication with local irritating factors or common diseases, how was

developed the disease process, whether the aggravation and its possible causes, what and when was treated disease and its effectiveness, dispensary registration.

3) History of life. Patient's complaints of gastrointestinal diseases, liver disease, diabetes, rossibility of occupational disease, history of allergy, sensitivity to drugs and dental materials, rheumatic fever, heart disease, hypertension, angina pectoris, myocardial infarction, nephritis, abnormal bleeding tendencies, excessive ruising, infectious diseases.

Question 3. Determination of the status of the oral cavity. Objective examination.

1) *Extraoral (external) inspection*. Performed by palpation of the lymph nodes (submandibular, parotid, cervical, etc.), points of nerve endings, the masticatory muscles, temporomandibular joint.

2) *Intraoral inspection*: the definition of oral status.

It is carried out according to a survey plan.

Oral mucosa. Oral examination begins with visual color evaluation and relief of oral mucosa, there is a violation of integrity and availability lesion elements. Inspection of the oral mucosa is carried out by 2 mirrors on the anatomical and topographical areas with natural light. WHO (1997) recommends the following sequence of inspection:

1. Commissure mucosa of the lips;
2. The vestibule of the oral cavity
3. Mucous cheeks left and right;
4. Mucous hard and soft palate;
5. The back and side surface of the tongue;
6. The bottom surface of tongue and mouth floor.

Architectonics of the vestibule and the floor of the mouth.

- ✓ On examination the vestibule of the mouth need to mark its depth. The depth of the vestibule of the mouth - is the distance from the gingival margin to the transition fold. Determined with a graded probe or with dental mirror (diameter of mirror is equal to 20 mm). Vestibule of the mouth is shallow (small), if the depth is not more than 5 mm, medium - from 8 to 10 mm, deep - more than 10 mm.
- ✓ Shallow vestibule of mouth is a risk factor of periodontitis.
- ✓ Normal frenulum is a thin triangular fold of mucous membrane with a broad base on the lip, ending on the midline of the alveolar process by about 5 mm from the edge of the gum.
- ✓ There are medium frenulum (are attached at a distance of 1-5 mm from the interdental papilla), short (or strong) frenulum, weak frenulum.

Examination of the teeth:

1) hypersensitivity
2) proximal contact relations

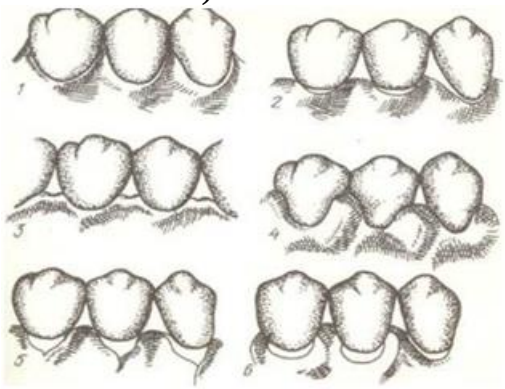
3) tooth mobility
4) sensitivity to percussion
5) pathologic migration of the teeth
6) pathological abrasion or delayed the natural abrasion of premolars and molars
7) presence of wedge-shaped defects
8) form of bite(occlusion)
9) the dentition with the jaws closed
10) examination of functional occlusal relationships
11) presence of traumatic occlusion

Question 4. Primary visual characteristic of periodontal tissues.

Table 6. Characteristic of periodontal tissues in normal and pathological conditions

	Normal appearance	*Pathological changes*
Surface texture	Free gingiva: smooth Attached gingiva: stippled	Acute condition: loss of stippling, with smooth, shiny gingiva. Chronic: hard, firm, with stippling, sometimes heavier than normal
Position	Fully erupted tooth: margin is 1-2 mm. Above CEJ, at or slightly below the enamel contour.	Enlarged gingiva: margin is higher on the tooth, above normal, pocket deepened. Recession: margin is more apical, root surface is exposed
Bleeding	No bleeding upon probing	Spontaneous bleeding. Bleeding on probing. Bleeding near margin in acute condition; bleeding deep in pocket in chronic condition.
Exudate	No exudate on pressure	White fluid, pus, visible on digital pressure. Amount not related to pocket depth
Position of junctional epithelium	In fully erupted tooth the JE is at the CEJ. During eruption: along the enamel surface	Position, determined by use of probe, is on the root surface.
Mucogingival lines	Makes clear demarcation between the pink, stipped, attached gingiva and the darker alveolar mucosa with smooth shiny surface	No attached gingival. Color changes may extend full height of the gingiva; mucogingival line obliterated

There are cleft gingiva (cleft Shtilman - localized narrow part gingival recession) and garlands (garlands McCall - thickening of the gums in the form of roller).



1 - the norm;
2 - atrophic;
3 - crateriform;
4 - spherical;
5 - dissected;
6 - scalloped

Figure 26. Changes in the gingival contour with periodontal diseases

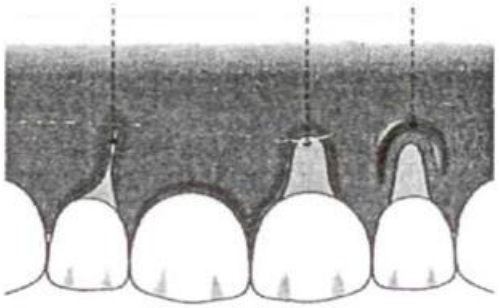


Figure 27. Changes in the gingival contour with periodontal diseases

Question 5. A detailed study of periodontal tissue

Two dental mirrors, probe, periodontal probe, tweezers detecting staining solution, dental floss, articulating carbon paper and gauze napkins are necessary to prepare for its examination.

1) Measure the depth of periodontal pockets.

Pockets, Loss of Attachment. Pocket formation without any loss of (connective tissue) attachment is seen in gingivitis in the form of the gingival pocket. Additional edema may create a pseudopocket. True pockets with loss of attachment are symptomatic of periodontitis.

One may differentiate between:

- Suprabony pockets -resulting from horizontal loss of bone
- Infrabony pockets -resulting from vertical, angular bone loss.

A true pocket will exhibit apical migration of the junctional epithelium down the root surface and transformation of the junctional epithelium (JE) into a pocket epithelium (Muller-Glauser & Schroeder 1982). If the most apical extent of the pocket is coronal to the alveolar crest, one speaks of a suprabony pocket. If the base of the pocket is apical to the alveolar crest, an infrabony pocket is present.

Types of pockets

A. Normal sulcus. Apical lermination of the JE is at the cementoenamel junction (arrow).

B. Suprabony pocket. Proliferating pocket epithelium. A remnant of junctional epithelium persists (pink).

C. Infrabony pocket. Extends beyond alveolar crest.

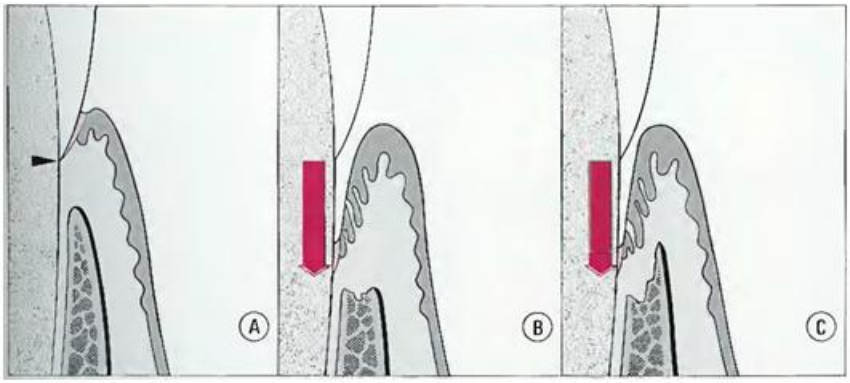


Figure 28. Types of pockets.

Infrabony Pockets. Vertical bone loss (infrabony pocketing) may exhibit various forms in relation to the affected tooth (Goldman & Cohen 1980).

Osseous defects are classified as follows:

3-wall bony pockets are bordered by one tooth surface and three osseous surfaces.

2-wall bony pockets (interdental crater) are bordered by two tooth surfaces and two osseous surfaces (one facial and one oral).

1-wall bony pockets are bordered by two tooth surfaces, one osseous surface (facial or oral) and a sort tissue border.

Crater ("cup") defects arc a combined form of pocket bordered by several surfaces of a tooth and several of bone. The defect surrounds the tooth.

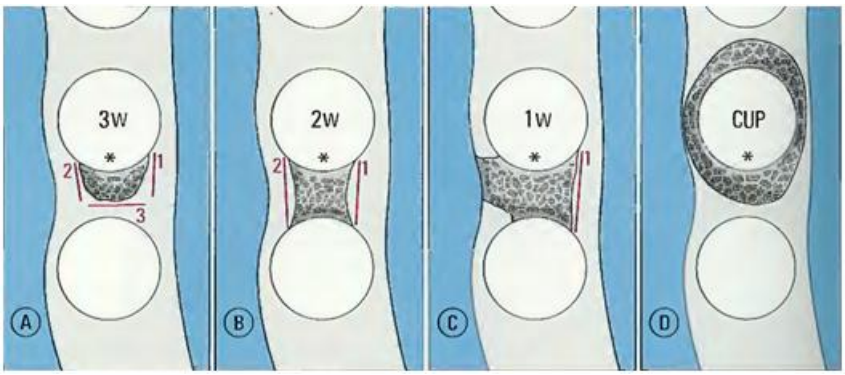


Figure 29. Osseous defects.

The causes for this wide variation in pocket morphology and resorption of bone are myriad and cannot always be wholly elucidated in each individual case. In the early stages of periodontitis, especially in the slowly progressing form (AP), bone loss is mostly in a horizontal direction. If

infrabony pockets then develop as the disease process advances, the following factors may account for the morphology of the resulting pocket:

- Local inadequate oral hygiene
- Local acute exacerbation elicited by specific bacteria in the pocket
- Crowding and tipping of teeth (plaque-retentive areas)
- Tooth morphology (root irregularities, furcations)
- Osseous morphology
- Improper loading due to functional disturbances.

The morphology of the bony pocket is of importance in both prognosis and treatment planning. The amount of bone remaining will affect the chances of osseous regeneration after treatment.

Pocket depth is determined by periodontal probe (bellied with Colour coded).

Types of special graded periodontal probes:

1. Probe Williams - marking 1-2-3-5-7-8-9-10 mm;
2. Marguis probe - marking 3-6-8-11;
3. WHO Probe - marking black band from 3.5 to 5.5 mm.

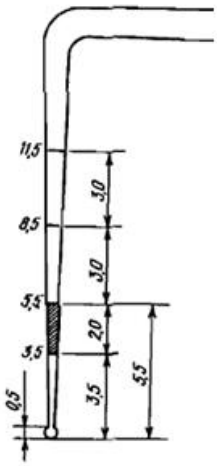


Figure 30. WHO's periodontal probe.

There are also electronic measuring system with automatic constant pressure. Examples of such systems are Interrrobe (Vaush & Lomb, USA), Florida-Rrobe (Florida-Rrobe Sorroration, USA), Rerirrobe (Ivoslar, Germany).

2) Research furcations of teeth.

The prevalence and severity of furcation lesions was determined using special probes with curved ends (Naber's and Wiliam's and in their absence) the usual universal probe or curette. The pathological process in the furcation also diagnosed radiographically.

Figure 31. The probe for the detection of furcation involvement in the pathological process

3) Furcation Involvement

Periodontal bone loss in multirooted teeth presents a special problem when bi- or trifurcations are involved. Partially or completely open furcations tend to accumulate plaque. Exacerbations, abscesses, progressive loss of attachment and rapid deepening of periodontal pockets occur frequently, especially with through-and-through furcation involvement. In addition, open furcations are particularly susceptible to caries.

Hamp et al. (1975) presented three degrees of furcation involvement (Fl-F3), and **Ramfjord and Ash (1970), Glickman** similarly classified furcation involvement into three degrees of severity, both measuring horizontally. In this, furcation involvement will be referred to according to horizontal measurements, as follows:

Class 1: The furcation can be probed to a depth of 3mm with the periodontal probe (F1).

Class 2: The furcation can be probed to a depth of more than 3 mm, but is not through-and-through (F2).

Class 3: The furcation is through-and-through and can be probed completely (F3).

This classification of furcation involvement is applicable both in the mandible and maxilla, where it can rarely be diagnosed radiographically. In such cases it is necessary to ascertain whether, in class F3 involvement, a through-and-through situation exists among all three roots of the maxillary molar or whether bone or attachment is still present on any two roots, e.g., the mesio-buccal and the palatal root. In order to ascertain furcation involvement in the maxillary molars, the probing must be performed from buccal, distobuccal and mesio-palatal aspects.

The vertical bone loss in an open furcation can also be classified (subclasses A-C, **Tarnow & Fletcher 1983**); it is measured from the roof of the furcation.

1 - vertical bone loss from 1 to 3 mm in furcations;

2 - vertical decrease in bone furcations of 4 to 6 mm;

3 - vertical bone loss in furcations of 7 mm and above.

Therapy: Class Fl and F2 furcation involvement may be successfully treated by root planing alone or by flap procedures (Ramfjord technique). A narrow furcation may be extended by odontoplasty. The class F3 involvement carries a less favorable prognosis, but can be treated the same as I and 2. Hemisection and resection of one root offer additional possibilities.

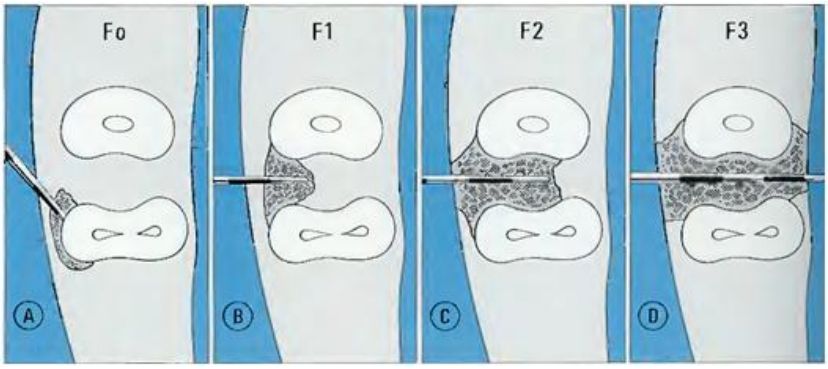


Figure 32 Furcation Involvement Degrees.

Classification of furcation involvement

A. F0: Pocket, but without furcation involvement.
B. F1: Furcation can be probed 3 mm in horizontal direction.
C. F2: Furcation can be probed deeper than 3 mm.
D. F3: Through-and-through furcation involvement.

Furcation involvement often occurs in conjunction with an infrabony defect.

4) Determination of tooth mobility. The most important causes of elevated tooth mobility are quantitative loss of tooth-supporting structure due to periodontitis, and qualitative changes in the periodontal ligament due to occlusal trauma.

It is important to differentiate between increased tooth mobility (stable increase; adaptation) and progressively increasing tooth mobility (unstable; pathologic). A "mini" functional analysis can determine clinically the more important functional findings such as premature contacts in retruded contact position, direction and extent of shifts during intercuspation, working side interferences, balancing side interferences, parafunctions (bruxism), TMJ changes, and habits such as lip biting, tongue thrust etc. Parafunctions related to the patient's occupation should also be recorded, e.g., holding nails between the teeth, pencil biting, or playing a musical instrument.

Degrees of Tooth Mobility (TM)

0 - normal (physiologic mobility)
1 - detectably increased mobility
2 - visible mobility up to 0.5 mm
3 - severe mobility up to 1 mm
4 - extreme mobility, vertical, mobility; tooth no longer functional.

Increased tooth mobility is determined by applying a labiolingual force of approximately 500g using two dental instruments.

5) Research of bite in case of periodontal lesion. This survey is conducted in two stages: the functional examination of the oral cavity and the study of diagnostic models.

Test control

1. What tools are used to detect tartar?

a) periodontal probes;
b) scalers;
c) curette;

2. The depth of the gingival sulcus in the normal condition is:

a) 0.5-1 mm;
b) up to 2 mm;
c) 3 mm.

3. Depth of periodontal pocket is measured

a) from the gingival margin to the base of the pocket;
b) from the enamel-cement border to the base of the pocket

4. Level of the epithelial attachment is measured

a) from the gingival margin to the base of the pocket;
b) from the enamel-cement border to the base of the pocket.

5. The inflamed gum has the following surface:

a) uneven, with elevations;
b) smooth.

6. What are the contours of the interdental papillae in the posterior region?

a) peaked;
b) rounded;
c) trapezoid.

7. Premature tooth contact can be detected by

a) using double-sided copy paper;
b) panoramic radiography;
c) intraoral radiography.

8. Indicate the examination sequence of the oral mucosa during the intraoral examination of the patient (WHO, 1997):

a) commissures, mucous membrane of the lips;
b) vestibule of the oral cavity
c) mucous cheeks on the left and on the right;

d) the mucosa of hard and soft palate;
e) the back and sides of the tongue;
f) the lower surface of the tongue and the bottom of the oral cavity;
g) all of the above

9. The main complaints of a patient with periodontal pathology are:

a) bleeding gums; swelling of the gums, discoloration,
b) bad breath;
c) pain while brushing or eating;
d) tooth mobility;
e) itching, burning, flying pains;
f) pain from cold or hot, fugitive pain;
g) discomfort during chewing.
h) aesthetic defects due to tooth displacement or the appearance of gaps between them.
i) difficulty in chewing food
j) all of the above

10. Identify the color of the gums in the normal condition:

a) bright red
b) coral pink
c) cyanotic

LESSON 7. METHODS FOR DIAGNOSIS OF PERIODONTAL DISEASE.

The questions to be studied for the learning of the topic:

1. Indices for evaluating of oral hygiene
2. The indices for evaluating the condition of the gums.
3. The indices for evaluating the condition periodontal tissues
4. Investigation of teeth (occlusal diagram, sensitivity).
5. Investigation of clinical pockets with periodontology probes.
6. Evaluation of suppuration from the periodontal pocket. Methods for detection of over- and subgingival tooth deposits

Question 1. Indices for evaluating oral hygiene

There are several groups of indices:

1) To assess the oral hygiene: OHI-S; Green, Vermillion, 1964; PHP, Podshadley, Haley, 1968; Fedorov-Volodkina 1968; plaque index Silness, Loe PLI (1964).

2) To assess the condition of gum: GI; Loe, Silness, 1963.

3) To assess the condition of periodontal tissues (periodontal codes): CPITN, WHO, 1982; CPI; P.A. Leus, 1988.

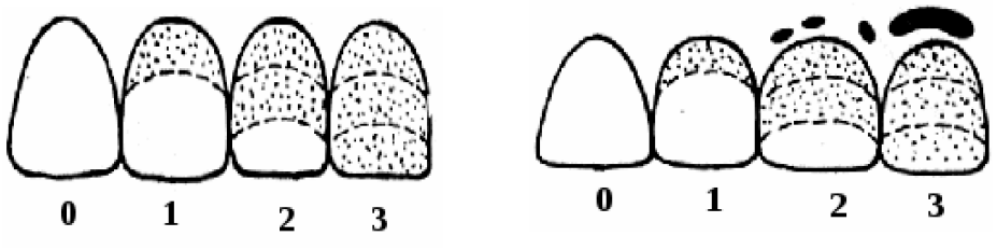
1) Oral Hygiene Index-Simplified (OHI -S) Green & Vermillion in 1964 simplified the OHI by including only six teeth surfaces rather than twelve that were representative of all anterior and posterior segments of the mouth. This modification was called Oral Hygiene Index-Simplified (OHI - S). The tooth used for the calculation must have the greatest area covered by either debris or calculus. The method for scoring calculus is the same as that applied to debris.

It has two components: debris index - simplified (DI-S) and calculus index – simplified (CI-S). The mouth mirror and shepherd’s crook or sickle type explorer are used to examine facial surfaces of teeth 11,16,26,31 and lingual surfaces of teeth 36, 46, by running the instrument from distal gingival crevice to mesial gingival crevice of a particular surface (S of tooth circumference) subgingivally. In the absence of selected molars, second or third molar and in absence of selected anterior teeth, the teeth 21 or 41 are substituted. At least two surfaces must have been examined for an individual score to be calculated.

The OHI-S score 0-1.2 of a person indicates good oral hygiene, 1.3-3.0 indicates fair oral hygiene and 3.1-6.0 indicates poor oral hygiene.

Table 7. Scoring Criteria

Scores	Debris	Calculus
0	No debris or stain present.	No calculus present.
1	Soft debris covering not more than one third of the tooth surface being examined or presence of extrinsic stains without debris regardless of surface area covered.	Supragingival calculus covering not more than one-third of the exposed tooth surface being examined.
2	Soft debris covering more than one third, but not more than two thirds, of the exposed tooth surface.	Supragingival calculus covering more than one-third but not more than two thirds of the exposed tooth surface and /or the presence of individual flecks of subgingival calculus around the cervical portion of the tooth.
3	Soft debris covering more than two thirds of the exposed tooth surface.	Supragingival calculus covering more than two-third of the exposed tooth surface or a continuous heavy band of subgingival calculus around the cervical portion of the tooth.



DI-S *CI-S*

Figure 33. Debris index DI-S and Calculus index CI-S

The index is calculated by the formula OHIS = DI-S + CI-S. While indicators of plaque DI-S and dental calculus CI-S divided on the number examined sextants

Interpretation

Meaning of OHI-S	Evaluation of OHI-S	Evaluation of oral hygiene
0,6	Low	Good
0,7-1,6	Middle	Satisfactory
1,7-2,5	High	Unsatisfactory
> 2,6	Very high	Bad

2) The index of the effectiveness of health PHP (Podshadley, Haley, 1968). The index is used for quality control of cleaning teeth during training. Record the presence of plaque (vestibular surfaces 16 and 26, 11 and 31, lingual - 36 and 46), but take into account under study the contamination of several sectors tooth crown surface.

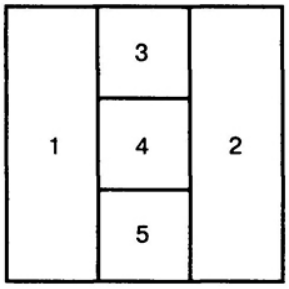


Figure 34. Dividing diagram of the vestibular surface of the tooth into sectors.

The presence of soft plaque is determined after rinsing the dye.
0 points - no staining;
1 point - staining in any sector.

Point's five sectors of the surface are summed to obtain PHP tooth.

PHP oral = (sum of PHP teeth) / (n teeth)

Evaluation of oral hygiene:
0 - excellent oral hygiene;
0.1-0.6 - good;
0.7-1.6 - satisfactory;
> 1.7 - unsatisfactory.

3) Plaque Index (PLI). It is unique among the indices because it ignores coronal extent of plaque and assesses only the thickness of plaque at the gingival area of the tooth using mouth mirror, and sickle type explorer or periodontal probe (Silness & Loe, 1964). It examines the same scoring units of the teeth (disto-facial/facial/ mesio-facial /lingual). Plaque index does not exclude or substitute a tooth with gingival restoration and crown.

Table 8. Scoring Criteria

0	no plaque at gingival area
1	a film of plaque on gingival margin and/or adjacent tooth surface, recognized only by running a probe across tooth surface
2	moderately soft deposits at margin and/or adjacent tooth surface that can be seen by naked eye
3	abundant soft matter at margin and adjoining surface

PLI of tooth calculated by the formula:

PLI = (total score of four surfaces) / 4

PLI of oral calculated as the average value of all examined teeth PLI.

The assessment of plaque thickness is so subjective that to obtain accurate data, highly trained and experienced examiners are required.

4) Fedorov–Volodkina's Hygienic index (1970)

To assess the hygiene of the oral cavity Fedorov and Volodkina (1970) proposed a hygienic index (quantitative and qualitative). The index is determined by the color intensity of the vestibular surface of the six lower frontal teeth (33, 32, 31, 41, 42, 43) by Schiller-Pisarev solution (1 g of crystalline iodine, 2 g of potassium iodide, 4 g of distilled water) or other iodine-containing solution. Defined plaque painted in dark-brown color.

Hygienic condition of oral cavity evaluated on a five-point system: coloring the entire surface of the crown of the tooth - 5 points,

3/4 surface - 4 points,

1/2 of the surface - 3 points,

1/4 of the surface - 2 points,

the absence of staining the surface of the crown of the tooth - 1 point.

The calculation is carried out according to the formula:

Hygienic index = sum of points / 6.

The index of hygiene of an oral cavity estimated as follows:

1.1-1.4 points - good care (optimal);

1.5-1.8 points - satisfactory;

1.9-2.5 points - unsatisfactory;

2.6-3.8 points - poor;

3.9-5.0 points - very bad.

Index of hygiene, reaching 2.6 and more points, indicates the absence of a regular oral care. Normal hygienic index does not exceed 1,5 points.

Question 2. The indices for evaluating the condition of the gums.

1) Gingival Index (GI) was developed to assess the severity and quality of gingival inflammation in individual or population **(Loe & Silness, 1963)**. Only gingival tissue is assessed by this index. Blunt periodontal probe is used to assess and palpate the bleeding tendency by running the probe along the soft tissue wall of the entrance of gingival sulcus.

Gingiva surrounding the tooth divided into 4 scoring units- Mesio-facial papilla, Facial marginal gingiva, Disto- facial papilla, Lingual marginal gingiva (to minimize examiners' variability in scoring, lingual gingiva were not subdivided).

All 4 scoring units are examined by visual examination (dental mirror) and periodontal probe and scored from 0-3 for each of them. Gingival index may be used for selected or all teeth.

Table 9. Scoring Criteria

Code	***Criteria***
0	Normal
1	Mild inflammation, slight color change, slight edema, no bleeding on palpation

2	Moderate inflammation, redness, edema, bleeding on probing
3	Severe inflammation, marked redness & edema, tendency to spontaneous bleeding

The GI score of 4 units are totaled and then divided by 4 (surfaces) to yield the GI score of a tooth. The GI score per person is obtained by totaling all of the tooth scores and dividing by the number of teeth examined (Table 2).

Table 2. Degree of gingivitis in relation to gingival index score

Gingival index score	*Degree of gingivitis*
0,1 - 1	Mild
1,1 - 2	Moderate
2,1 - 3	Severe

2) Papillary Marginal Attachment Index - PMA (M. Massler, J. Shour, C. Parma, 1960). The facial surface of gingiva around a tooth divided into three units: **Mesial interdental papilla (P), Marginal gingiva (M), and Attached gingiva (A).** Presence or absence of inflammation on each gingival unit recorded as 1 or 0 respectively.

Summation of these three units of a tooth is considered as PMA score of the tooth and summation of score of all teeth and divided by number of teeth; is considered as PMA score of the person.

It is used for epidemiological survey, in clinical trials and for patients' education.

Papillary-marginal-alveolar index gives an indication of the extent and severity of gingivitis. The index can be expressed in absolute terms or as a percentage.

A popular modification of the calculation of index PMA to the Parma is carried out using percent scheme:

$$\mathbf{PMA} = \frac{\sum \text{scores}}{3 \times n} \times 100\%$$

where, n - the number of examined teeth, 3 - maximum score of inflammation in the area of one tooth.

It is believed that the value of PMA from 1 to 33% of the patient there is a slight inflammation of periodontal, from 34 to 66% - moderately above 67% - heavy.

3) ***Index gingival recession (Stahl Morris, 1955)*** - evaluated in all groups of teeth and is defined as the distance from the gingival margin to the

enamel-cement border. To determine the gingival recession better to use special graded probes to determine recession or periodontal probe.

Recession index is calculated as:

$$\frac{\textbf{number of teeth with recession}}{\textbf{total number of teeth}} \times 100\%$$

Mild degree - up to 25%; Moderate -26-50%; Severe - 51-100%

For assessing the status of bone tissue use the index of exposed roots (the index of the recession). Measure the depth of the pocket, or, more precisely, the X-ray measure the distance between the top of the alveolar process and the cement-enamel boundary. These data are summed and divided by the number of examined teeth.

Question 3. The indices for evaluating the condition periodontal tissues

1) Community Periodontal Index of Treatment Needs CPITN (1982). The index that resulted after extensive field testing by the investigators from the World Health Organization (WHO) and the International Dental Fedaration (FDI) was called Community Periodontal Index of Treatment Needs (CPITN).

In epidemiological study, 10 index teeth are examined but only worst finding from index teeth is recorded per sextant resulting in six scores. It permits rapid examination to determine periodontal treatment needs.

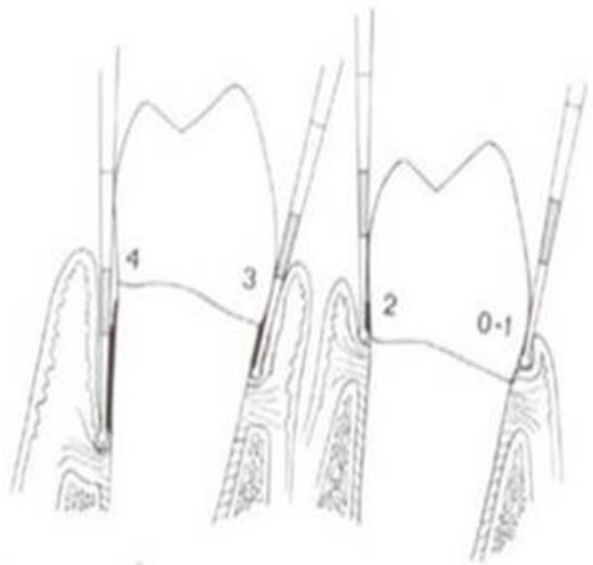


Figure 35. Criteria CPITN.

Methods. Persons older than 20 years investigating the periodontium in six groups of teeth (17/16, 11, 26/27, 37/36, 31, 46/47) on the upper and lower jaws with a special bellied WHO periodontal probe (i.e., 0.5mm ball tip and marking at 3.5mm, 5.5mm, 8.5mm and 11.5mm, black color coding between 3.5mm and 5.5mm). Young people under the age of 19 years

examined 16, 11, 26, 36, 31, 46. Teenagers and children under 15 years are only used codes 0.1 and 2, if in a dedicated inspection sextant less than 2 index teeth, while sextant excluded from the survey.

Table 10. Scoring Criteria

CPITN score	Periodontal status	Treatment need
0	Healthy periodontium	No treatment
1	Bleeding observed by probing/spontaneous	Improvement of Oral hygiene
2	Calculus felt by probe; entire black area is visible	1+ professional scaling
3	Pocket depth 4-3mm; Gingival margin on the black band	1+ professional scaling
4	Pocket depth >6mm; Entire black band is invisible	I+II+ complex surgery

2) Complex periodontal index, the CPI (P.A.Leus, 1988)

Methods. The condition of periodontal tissues is determined by conventional dental probe and dental mirrors. Dental tweezers can be used to determine mobility. 17/16, 11, 26/27, 37/36, 31, 46/47 examined in adults. A more serious condition is recorded in case several signs (higher score).

Criteria:

0 - healthy - plaque and periodontal lesions no signs;

1 - plaque - any number of plaque;

2 - bleeding - bleeding visible to the naked eye with gentle probing of periodontal groove;

3 - calculus - any amount of subgingival calculus in the region;

4 - pathological pocket - abnormal tooth-gingival pocket defined by the probe;

5 - tooth mobility - the mobility of 2-3 degrees.

CPI = Sum code / number of sextants (usually 6)

Interpretation. The values of the intensity level:

0.1 -1.0 - risk to the disease

1.1-2.0 - Light

2,1-3,5 - Average

3,6-5,0 - Heavy

3) Periodontal Index (A.L. Russel, 1956, 1967). It not only assesses all the gingival tissues encircling the tooth but also scores the supporting tissues. Mouth mirror, light source and explorer are used to assess tissue.

The scoring criteria are:

0 - (Absence of inflammation),

1 - (Mild inflammation, slight color change, change in texture only a portion of papillary or marginal gingiva but not in entire gingiva),

2 - (Mild inflammation involves entire papillary or marginal gingival unit),

4 - (when radiograph is advised, the disappearance of the closing of the cortical plates on the tops of the alveolar ridge.),

6 - (Moderate inflammation, redness, edema, and/or hypertrophy of marginal or papillary gingival unit with pocket formation),

8 - (Severe inflammation, redness, edema, spontaneous bleeding and ulceration with advanced destruction and impairment of function).

In doubtful condition, lower score should be considered. The periodontal index score per person is obtained by totaling all of the tooth scores and dividing by the number of teeth examined.

- 0.0-0.2 (Group PI score) – Clinically normal Reversible stage
- 0.3-0.9 (″) – Simple gingivitis (″)
- 0.7-1.9 - (″) – Beginning of periodontal destruction – (″)
- 1.6-5.0 - (″) – Established periodontal destruction - Irreversible stages
- 3.8-8.0 - (″) – Terminal disease (″)

Question 4. Investigation of teeth (occlusal diagram, sensitivity)

The degree of mobility of the teeth due to the severity and depth of the destruction of the tooth ligaments and character of destructive process in the periodontium. Mobility characterizes the direction and degree of deviation from the normal position of the tooth. It is determined by palpation, with tweezers or special equipment.

There are 4 degrees of pathological mobility by D.A Entin:

- I degree - the mobility in bucco-lingual (palatal) or vestibular-oral direction (no more than 1 mm);
- II degree - the mobility in bucco-lingual (palatal) or vestibular-oral direction (more than 1 mm) + the mobility in medio-distal direction;
- III degree - the tooth is mobile in all directions, including vertical;
- IV degree - the tooth is mobile in all directions, including vertical + rotational movements about the axis of the tooth.

Traumatic occlusion is the condition when a tooth or group of teeth feels traumatic stress during closing.

Traumatic occlusion is a constant symptom of generalized periodontitis and periodontal disease and is caused by disorders ligaments of the teeth and their displacement. The degree of severity of traumatic occlusion is associated with disease severity.

Occlusal-gram- getting prints of dental rows on the plastic material in the occlusal plane of the teeth (central occlusion). You can use wax plates, strips of foil, copy paper. After biting you can see imprints of teeth both jaws on the graph paper.

To determine the *density of occlusal contacts, supra-contacts* are used also a horseshoe-shaped copy paper or heated plate of wax. In the areas of early contact copy paper breaks, and the tissues of the tooth are colored. In the areas of early contact the wax has deeper sagging areas

Question 5. Investigation of Clinical pockets with Periodontology probes.

Periodontal probes are used to measure the depth of pockets and to determine their configuration. The typical probe is a tapered, rod-like instrument calibrated in millimeters, with a blunt, rounded tip. There are several designs with various millimeter calibratios.

When measuring a pocket, the probe is inserted with a firm, gentle pressure to the bottom of the pocket. The shank should be aligned with the long axis of the tooth surface to be probed. Several measurements are made to determine the level of attachment along the surface of the tooth.

Furcation areas can best be evaluated with the curved, blunt Nabers probe.

Explorers are used to locate subgingival deposits and carious areas and to check the smoothness of the root surfaces after root planing. Explorers are designed with different shapes and angles for a variety of uses.

Pocket depth was measured from each of the four sides of the tooth: the medial, buccal, distal, lingual.

Types of pockets:

a) gingival - no extended beyond the gums, is not associated with the destruction of periodontal tissue and with hypertrophy of the gums;

b) periodontal - extended beyond the gums, is associated with the destruction of the ligaments;

c) periosteal (supraosseous) - pocket, whose bottom is located above the alveolar bone;

d) intraosteal (intraosseous) – pocket, whose bottom is located between the alveolar bone and the root of the tooth + below the peaks of interdental septum (apical region of destructive changes is located inside the alveolar)

According to the location osteal pocket relative to the tooth (N.M. Goldman, 1949, 1958):

a) one-sided,

b) two-sided,

c) three-sided,

d) four-sided, or circular (combined).

This means that pocket has three, two or one bone walls. Pocket, which is located along only one surface of the tooth, called simply, covering two or more surfaces - integral (combined), and bend around it, or getting into a bifurcation - complex. The degree of bone loss does not always correspond to depth of pocket.

Loss of periodontal attachment - the clinical term that characterizes the destruction or loss of the tooth-epithelial attachment. It corresponds to the measurements conducted on the long axis of the tooth away from the cement-enamel connection to the apical border of periodontal destruction. Expressed in millimeters (mm).

Periodontal attachment saved (not lost), if:

- Exposure of the tooth root is absent + the vertical sounding of periodontal less than 3 mm - clinical gingival sulcus;
- Exposure of the tooth root is absent + gum hides the coronal part of the tooth due to hypertrophy + vertical sounding reveals clinical pocket, which depth - is the size hypertrophic gums - "false pocket."

Probes use to investigate the clinical periodontal pockets. With periodontal probes carried out:

1. Identification of tooth deposits.
2. Assessment of the surface condition of the tooth root.
3. Measure the depth of the periodontal pockets.
4. Determination of periodontal soft tissue bleeding by clinical probing pocket.
5. The measurement of the recession and gingival hypertrophy.
6. Measurement of furcational defects.

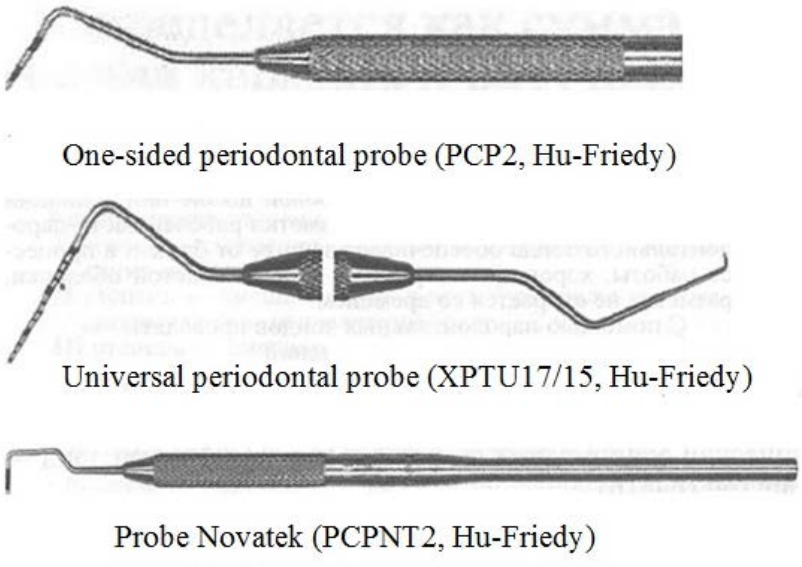


Figure 36. Types of probes.

Question 6. Evaluation of suppuration from the periodontal pocket. Methods for detection of over- and subgingival tooth deposits.

For a more exact identification of the pus of the periodontal pocket spend benzidine test (S.Sorrin, 1960).

For this purpose, 1 drop of a solution consisting of 0.5 ml of benzidine, 10 ml of polyethylene glycol and 15 ml of acetic acid (1:1000), mixed with 1 drop of 3% hydrogen peroxide solution and injected at turundae in the pocket. Turundae painted in green, blue-green, and blue. The color intensity indicates the amount of purulent discharge.

On examination the patient visually evaluate the presence, amount and nature of the exudate released from the pocket. To determine whether the contents of the periodontal pockets in the clinical setting with your finger (or a cotton swab) easily presses on the gum in the projection of the corresponding apex of the tooth and gradually move it to the gingival margin. In the presence of purulent exudate evaluate its intensity.

The intensity of the pyorrhea can be measured in points:

0 points - no pus;

1 point - little pus (pus in a periodontal pocket);

2 points - moderate pyorrhea (pus swept over the edge of the pocket);

3 points - pus strong (in the marginal gingival traces of pus, abscesses).

The final results are calculated by determining the average of the number of surveyed areas of the gums.

Methods for detection of over- and subgingival tooth deposits.

To assess the hygiene of the oral cavity use indexes based on staining of dental plaque. For this purpose use Lugol's solution (1 part of iodine, 2 parts of potassium iodide and 17 parts of water), the standard tincture of iodine, 2% aqueous solution of methylene blue, 2% solution of fuchsin, etc.

Dental plaque becomes dark brown if iodine preparations are applied to the tooth surface. Fuchsin stains plaque in a bright pink color, methylene blue – blue.

For this purpose, it is widely used special tablet that dissolves in the saliva, are stained tooth deposits.

Test control

1. Indicate oral hygiene indices:

a) OHI-S
b) GI
c) PMA
d) CPITN

2. Indicate indices of the gum condition:

a) OHI-S
b) GI
c) CPITN

3. Indicate indices of the periodontal tissues condition:

a) CPITN
b) OHI-S
c) PMA

4. How many degrees of pathological tooth mobility exist?

a) 2
b) 6
c) 4
d) 1

5. List the types of pockets:

a) gingival;
b) periodontal;
c) periosteal (supraosseous);
d) intraosteal (intraosseous)
e) all of the above

6. According to the location osteal pocket relative to the tooth (N.M. Goldman, 1949, 1958) are:

a) one-sided,
b) two-sided,
c) three-sided,
d) four-sided, or circular (combined).
e) all of the above

7. With periodontal probes carried out:

a) identification of tooth deposits.
b) assessment of the surface condition of the tooth root.
c) measure the depth of the periodontal pockets.
d) determination of periodontal soft tissue bleeding by clinical probing pocket.
e) the measurement of the recession and gingival hypertrophy.
f) measurement of furcational defects.
g) all of the above

8. What color will the tooth surface be when painted with iodine?

a) blue
b) dark brown
c) bright pink

9. What color will the tooth surface be when painted with methylene blue?

a) blue

b) dark brown
c) bright pink

10. What color will the tooth surface be when painted with fuchsin?

a) blue
b) dark brown
c) bright pink

LESSON 8. ADDITIONAL METHODS OF RESEARCH IN CLINICAL PERIODONTOLOGY

The questions to be studied for the learning of the topic:

1. Examination of patient with periodontal diseases
2. X-ray examination of periodontal tissues
3. Laboratory, microbiological, immunological, morphological, biochemical and cytological research
4. Functional methods

Question 1. Examination of patient with periodontal diseases

Evaluation of the patient's periodontal status requires obtaining a relevant medical and dental history and conducting a thorough clinical and radiographic examination with evaluation of extraoral and intraoral structures. All relevant findings should be documented. When an examination is performed for limited purposes, such as for a specifically focused problem or an emergency, records appropriate for the condition should be made and retained.

Optimal diagnosis can be made in several stages.

The first stage

1. Introduction to patient.
2. The patient's medical history.
3. The patient's social history.
4. The patient's complaints.
5. Assessment of the patient's oral status.
    - 5.1. Hygiene.
    - 5.2. Breath.
    - 5.3. Saliva.
    - 5.4. Lips.
    - 5.5. Oral mucosa.
    - 5.6. Tongue.
    - 5.7. Palate.
    - 5.8. Pharyngeal area.
    - 5.9. Architectonics of the vestibule and the floor of the mouth.
    - 5.10. Examination of the teeth.
        - 5.10.1. Assessment of the number and causes of tooth loss.
        - 5.10.2. Sensitivity of dentine.
        - 5.10.3. Condition of the contact surfaces of the teeth.
        - 5.10.4. Tooth mobility.
        - 5.10.5. Percussion of the tooth.
        - 5.10.6. The presence of pathologic migration of the teeth.
        - 5.10.7. Examination of the occlusal contacts.
6. Primary visual characteristics of the periodontal tissues.

7. Preliminary diagnosis.
8. X-ray examination.

The second stage

9. A detailed examination of the periodontal tissues.
    9.1. The presence of the supra- and subgingival dental plaque.
    9.2. The level of the gingiva involvement in the pathological process.
    9.3. The level of the periodontal tissues destruction, the presence of the periodontal pockets.
    9.4. The presence of pus discharge from the periodontal pockets.
    9.5. The level of the alveolar bone destruction.
    9.6. Trauma from occlusion.
    9.7. The state of periodontal microcirculation.
10. Laboratory diagnosis.

The survey consists of several stages of the patient's visits. Usually it is carried out within two visits in order to perform the targeted treatment.

Question 2. X-ray examination of periodontal tissues

In general, radiographs should supplement the clinical examination. The periodontal condition is assessed by considering the overall level of the alveolar bone, the relation between root length and bone level, the presence of infrabony lesions, the presence of furcation involvement, the width of the periodontal ligament, the presence and appearance of a lamina dura, etc. An orthopantomogram can provide only an overview and does not allow definite conclusions about extent and severity of periodontal disease. It furthermore provides valuable information about any abnormal/pathological processed in the jaws, the surrounding tissues, and the neighboring areas of the oral cavity as well as caries and present restorations. An orthopantomogram is usually sufficient in cases of mild periodontitis. In cases of moderate or advanced periodontitis and for comprehensive dental treatment planning, a full-mouth survey consisting of intraoral radiographs is indispensable.

Radiographic Changes seen in Periodontal Disease

Periodontal disease causes inflammatory lesions in the marginal bone. Both osteoblastic and osteoclastic activity is seen. Osteoclastic activity will cause changes in the morphology of the crestal bone. Initial response is destruction of bone. Chronic lesions will show some osteosclerosis.

Mild Marginal Periodontitis

- ✓ Localized erosions of the marginal bone
- ✓ Thinning of crestal lamina dura
- ✓ Loss of sharp border with the lamina dura of the adjacent teeth
- ✓ Loss of spiking in the anterior
- ✓ Slight loss of bone height (<1/3)

Moderate Marginal Periodontitis

- ✓ Generalized form demonstrates horizontal bone loss
- ✓ Localized defects include vertical bone loss and loss of buccal and lingual cortices
- ✓ Loss of buccal or lingual cortex is difficult to view radiographically. It may be seen as decreased density over the root surface
- ✓ Horizontal bone loss refers to the loss in height of the crestal bone around the teeth. Horizontal bone loss may be: mild, moderate, severe.
- ✓ Crest remains generally horizontal

Severe Marginal Periodontitis

- ✓ Patient may have horizontal or vertical bone loss, or a combination of generalized horizontal bone loss with localized vertical defects
- ✓ Bone level is in the apical 1/3 of the root
- ✓ Clinically, the teeth may be shifting, tipping, or drifting
- ✓ Bone loss may be more extensive than is apparent on the radiographs
- ✓ Vertical Bone Loss usually localized to one or two teeth. May be several areas of vertical bone loss throughout the mouth

Question 3. Laboratory, microbiological, immunological, morphological, biochemical and cytological research.

Laboratory diagnostic methods. Laboratory diagnostic methods are used when there is difficulty in assessing periodontal status or in determining the treatment effectiveness properly. The goal of laboratory diagnosis is to conduct a cytological examination of the periodontal pocket with the aim of revealing microorganisms. Along with it, microbiological, biochemical and immunological examinations are used for diagnosis, selection of medicines and treatment. If the clinician has difficulties in diagnosing periodontal diseases, the deformed areas of affected tissue should be subjected to the histological analysis.

Microbiological examination. Microbiological examination reveals the composition of microflora in periodontal pockets determines its sensitivity to antibiotics or other drugs and controls the treatment effectiveness. The content of periodontal pockets, oral liquid, as well as material, obtained by curettage, are used for examination. It is usually carried out before and after treatment to monitor its quality. Microbiological diagnosis has a great importance, especially in progressive periodontitis, because periodontal disease is caused by periopathogenic microorganisms. Due to identification of bacterial enzyme activity, you can define such pathogens, as Porphyromonas gingivalis, Bacteroid forsythus, Treponema denticole.

Immunological examination allows us to estimate the nonspecific and specific protective mechanisms of the body. Local immunity is determined by the lysozyme concentration in the mixed saliva as a nonspecific protection factor, by the concentration of immunoglobulins sIgA, IgA, IgG in oral

liquid, by the vitality of cells in the periodontal pocket and by the proteolysis activity. Identification of the immunoglobulins level allows not only to identify their secretion violations, but also to apply the quality control of the treatment. Proteolytic activity (the activity of enzymes, that destroy proteins) is analyzed in the periodontal tissues, gingiva liquid and in the saliva. The state of the cellular nonspecific protection (state of T- and B-systems of immunity, the functional activity of polymorphonucleocytes), as well as specific protective factors (antibodies to periodontopathogenic bacteria) are evaluated by the immunological examination of the peripheral blood.

Cytological examination. Cytological examination is based on the structural features study of the cellular elements and cell conglomerates. The qualitative and quantitative composition of cells, particularly their color qualities, is evaluated. Dentists pay attention to the color of cytoplasm (basophilia), dystrophic and necrotic changes, phagocytosis, the presence of microbes, cells aggregation and other. The quantitative and qualitative changes in cells can testify the inflammation development, activity and direction of the pathological process growth, the level of tissues destruction. The method is simple, safe for the patient, effective and reliable. It allows you to get the results quickly, and to repeat examination if necessary. Cytological method is used to determine the treatment effectiveness. Impression smears, re-impression smears, scraping smear from the mucosa surface, periodontal pockets, erosion, ulcer, fistula, as well as punctate from the area, located in the deep-lying tissues, can be used as material for the cytological study.

Biochemical examination. Biochemical blood analysis is conducted in order to identify inflammation markers, changes in metabolic processes, especially in the metabolism of lipids, responsible for the blood vessels condition. The metabolism of the alveolar ridge bone tissue depends on the hormonal changes in the body, the level of calcium in the blood, as well as other external and internal causes, influencing the bone system. In this regard, the identification of hormone levels and biochemical markers of bone remodeling (parathormone, thyrocalcitonin, osteocalcin, C-terminal telopeptide of type I collagen and ß-Cross-laps, alkaline phosphatase, D3 vitamin, calcium, glycohemoglobin, and others) levels are required for patients with chronic forms of periodontitis. It allows to detect osteopenia and osteoporosis, as well as it is used for timely correction of metabolic disorders of the bone tissue in the whole body.

Question 4. Functional methods.

Functional methods: vacuum test to determine the gingival capillary resistance, vacuum test to determine the gingival capillary permeability, peripheral blood circulation index, biomicroscopy, dopplerography, laser-optical diagnosis of the periodontal tissues microcirculation.

Laser and ultrasound Doppler flowmetry. These methods were developed by the Austrian physicist Christian Andreas Doppler, who put forward the hypothesis of the frequency of the electromagnetic or acoustic waves, reflected from a moving object. The presence of the reflected signal indicates the presence of blood flow in the area of the ultrasonic examination. The ultrasonic waves diffusion and reflection are the two main processes, which underline the basis of the diagnostic action of ultrasonic equipment. Doppler flowmetry is used to examine the microcirculation in normal state and in case of pathology. It allows to determine the volumetric and linear rates of blood flow in the gums and other parameters.

The identification of the periodontal capillary pressure. The identification of the periodontal capillary pressure (J.L. Denisova, L.A. Denisov, 2012) is one of the most important factors, that determines the disorder of capillary blood flow. A special device with pneumatic compressor, which forms a pressure in the range of 20–70 mm Hg, is used to determine the capillary pressure in the periodontium. In normal condition the capillary pressure in the periodontium is about 20 mm Hg (J. L. Denisova, 2012). It is decreased to 18.3 mm Hg in case of gingival recession and increased to 28,3–35,0 mm Hg in case of chronic periodontitis. The identification of the capillary pressure in the periodontium in the normal and pathological states is used to determine the periodontal tissues status and to plan individual treatment dosage (orthodontic, physical therapy and other procedures).

Laser-optical diagnosis of the periodontal tissues microcirculation. Laser-optical diagnosis of digital speckle photography (LODdsp) (S.P. Rubnikovich) has several advantages over other optical functional diagnostic methods:

1) real-time monitors the microcirculation state in the periodontal tissues and the stress-strained level of the teeth and dental prosthesis due to accelerated computer processing;

2) provides lack of compression effect on the oral cavity soft tissues due to non-contact laser application;

3) allows to conduct the cross-sectional examination of all topographical areas of the gum and the oral cavity (20×30 mm and more);

4) gives the possibility of differentiated preventive therapy for the treatment the microcirculatory disorders in the periodontal tissues;

5) is painless for the patient and safe for the doctor;

6) does not require additional protections;

7) is available for wide practical use.

The speckle area, which consists of tiny 1–2 micron granules, is formed during the dispersal of the coherent radiation in a diffuse object. The similar speckle area is formed by the laser radiation dispersal by biological tissues. Visible coherent radiation, penetrating to the depth of 1–2 mm into the

mucous membrane and skin of a person, is dispersed by the red blood cells, flowing in the smallest tissue capillaries. As a result of interference in the dispersed light, dynamic speckle area is formed, varying in space and in time as a result of the red blood cells movement. The analysis of the laser speckle areas dynamics allows the doctor to form an instant map of the speed of the red blood cells movement in the examined oral tissues. The device for laser-optical diagnosis consists of a laser, that generates optical radiation; a light guide, pointed to the examination object, i. e. periodontal tissue, dental tissue, denture; the recording optical system is a digital CCD camera, which is connected to the computer with special software. After recording the resulting image is transferred to a personal computer (7), where it is converted in a special program with the aim of obtaining numerical characteristics of the microcirculation intensity in the tissues. Methodology of the laser-optical diagnosis based on digital speckle photography. The device for laser-optical diagnosis and treatment of periodontal tissues on the basis of digital speckle photography consists of the laser, that generates optical radiation, with a fiber optic cable, focusing this radiation on the examination object (periodontal tissue, dental tissue, denture, orthodontic equipment); a recording optical system for reproducing the reflected image on the camera. This camera is fixed to the table, using metal rails and the holder with a moving base by means of external screw. After recording the resulting image is transferred to a personal computer, where it is adapted by a special program with the aim of obtaining numerical characteristics of the microcirculation intensity in the periodontal tissues. The patient sits on a chair, his head is fixed motionlessly in the ophthalmological retainer, the access to the oral cavity is achieved using a dental dam retractor. The focus of lighting and receiving optical systems are pointed to the examined area. The distance between the focus and the gum should be 1 cm, so that there is no contact between them. The focus is oriented at obtaining the most clear image of the selected area with the subsequent registration of the microcirculation dynamics using a digital camera. After recording, the image is transferred to a personal computer, where they are processed by special software in order to obtain numerical microcirculation characteristics. Microcirculatory status of periodontal tissues is determined by the intensity of the gum microcirculation and is calculated in conventional units with the help of computer programs, by comparing with normal indicators. The use of laser-optical diagnosis will allow you to perform early diagnosis of latent manifestations of the disease, to identify indications for pathogenetic therapy and to assess the effectiveness of treatment during primary and follow-up examinations.

Test control

1. The first stage of patient examination with periodontal diseases consists of

a) introduction to patient
b) the patient's medical history, social history, complaints
c) assessment of the patient's oral status
d) primary visual characteristics of the periodontal tissues
e) preliminary diagnosis
f) X-ray examination
g) all of above

2. The detailed examination of the periodontal tissues consists of

a) the presence of the supra- and subgingival dental plaque
b) the level of the gingiva involvement in the pathological process
c) the level of the periodontal tissues destruction, the presence of the periodontal pockets
d) the presence of pus discharge from the periodontal pockets
e) the level of the alveolar bone destruction
f) trauma from occlusion
g) the state of periodontal microcirculation
h) all of above

3. Assessment of the patient's oral status includes

a) hygiene assessment
b) breath assessment
c) saliva assessment
d) oral mucosa assessment
e) lips and tongue assessment
f) palate assessment
g) pharyngeal area assessment
h) architectonics of the vestibule and the floor of the mouth
i) all of above

4. Teeth examination of patient with periodontal diseases includes

a) assessment of the number and causes of tooth loss.
b) sensitivity of dentine.
c) condition of the contact surfaces of the teeth.
d) tooth mobility.
e) percussion of the tooth.
f) the presence of pathologic migration of the teeth.
g) examination of the occlusal contacts.
h) all of above

5. Additional examination methods include:

a) study of occlusion in case of periodontal disease;
b) X-ray examination;
c) determination of tooth mobility;
d) determination of durability of gingival capillaries;
e) study of saliva pH.

6. Laboratory research methods include:

a) study of occlusion in case of periodontal disease;
b) bacteriological examination;
c) determination of resistance of gingival capillaries;
d) biochemical studies;
e) genetic research.

7. X-ray examination of the periodontium shows:

a) the condition of the roots, the ratio of the root-crown, the presence of a pocket, periapical changes;
b) the presence of tartar;
c) expansion of the periodontal ligament;
d) destructive processes of the alveolar bone;
e) root resorption;
f) the involvement of furcation in the pathological process;
g) anatomical malformations;
h) alveolar bone changes.
i) all of above

8. Mild Marginal Periodontitis includes

a) Localized erosions of the marginal bone
b) Thinning of crestal lamina dura
c) Loss of sharp border with the lamina dura of the adjacent teeth
d) Loss of spiking in the anterior
e) Slight loss of bone height (<1/3)
f) all of above

9. Moderate Marginal Periodontitis includes

a) generalized form demonstrates horizontal bone loss
b) localized defects include vertical bone loss and loss of buccal and lingual cortices
c) loss of buccal or lingual cortex is difficult to view radiographically. it may be seen as decreased density over the root surface
d) horizontal bone loss refers to the loss in height of the crestal bone around the teeth. horizontal bone loss may be: mild, moderate, severe.
e) crest remains generally horizontal

f) all of above

10. Severe Marginal Periodontitis includes

a) patient may have horizontal or vertical bone loss, or a combination of generalized horizontal bone loss with localized vertical defects
b) bone level is in the apical 1/3 of the root
c) clinically, the teeth may be shifting, tipping, or drifting
d) bone loss may be more extensive than is apparent on the radiographs
e) vertical bone loss usually localized to one or two teeth. may be several areas of vertical bone loss throughout the mouth
f) all of above

LESSON 9. GINGIVITIS: CHRONIC SIMPLE MARGINAL GINGIVITIS. CHRONIC GINGIVITIS: HYPERPLASTIC, ULCERATIVE, DESQUAMATIVE.

The questions to be studied for the learning of the topic:

1. The mechanism of developing chronic simple marginal gingivitis.
2. Classification of gingivitis and periodontal diseases.
3. Clinical manifestations and diagnosis of chronic simple marginal gingivitis.
4. Chronic hyperplastic gingivitis.
5. Chronic ulcerative gingivitis.
6. Chronic desquamative (symptomatic) gingivitis.
7. Differential diagnosis of gingivitis.

Question 1. The mechanism of developing chronic simple marginal gingivitis.

Figure 37. Development scheme of periodontal diseases.

Gingivitis and periodontitis are inflammatory diseases of bacterial origin.

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

Table 11. Bacterial attack and host response

Bacteria ←————————————————→Host response	
Bacterial products - Chemotactic substances - Antigens, mitogens - Enzymes - Toxins *Bacterial invasion / Infection* *Amount and composition of plaque* - Plaque amount (quantity, oral hygiene) - Plaque composition (quality, pathogenicity) - Plaque retention (natural and iatrogenic)	*Positive host response* - Exudation - Leukocytic infiltration (phagocytosis) - Immune response - [Normal function] *Defective or weak host response* - Leukocyte defect - Immune defect - Systemic diseases - [Functional disturbances, occlusal trauma]

The strength of bacterial attack depends on amount and composition of plaque, ability of the organisms to invade tissues, and metabolic products.

The effectiveness of the host response to bacterial challenge will determine the severity of an ensuing gingivitis, the initiation of periodontitis, and the rapidity with -which destruction of periodontal tissues proceeds.

An absolutely plaque-free condition in the oral cavity is unachieveable, an illusion, and probably even unphysiologic. Nevertheless, gingival and periodontal health can be maintained if the plaque contains few virulent organisms and if an effective host response is mounted.

The most important bacterial products in terms of inflammation and tissue destruction are antigens, mitogens and chemotactic substances. If bacteria invade the tissue directly, one may speak of a true infection. Bacterial enzymes and various toxins can probably cause tissue injury and destruction directly, without an immediate host response. Bacterial products including hyaluronidase, chondroitin sulfatase, proteolytic enzymes as well as eytotoxins in the form of organic acids, ammonia, hydrogen sulfide and bacterial endotoxins (lipopolysaccharide, LPS) can be demonstrated in tissues.

Accumulated Debris - Microbial Plaque

Food debris clings only lightly to the teeth and oral mucosa, and can be easily rinsed away with water.

Food impaction may occur in interdental spaces when fibrous foodstuff become trapped, but can be removed mechanically.

Microbial plaque is a structured, resilient, yellow- grayish substance that adheres tenaciously to teeth. It is comprised of bacteria in a matrix of salivary -glycoproteins and extracellular polysaccharides like glucans(e.g., dcxtrans, mulans) and fructans (e.g., levan). This matrix makes it impossible to rinse plaque away with -water; it must be removed mechanically by means of hand instruments, the toothbrush or other oral hygiene aids. Supragingival plaque and subgingival plaque are two distinct morphological and microbiological entities.

Further, one can distinguish between adherent plaque and nonadherent subgingival plaque. The pathogenicity of bacterial strains within plaque varies -considerably. Plaque adhering to the tooth surface can become calcified.

Supragingival Plaque

Accumulation - Alteration of composition. Within minutes to hours, an absolutely clean tooth is covered by a 0.1-0.8 µm thick pellicle composed of salivary glycoproteins. Upon this pellicle, primarily gram- positive colony-forming bacteria (Streptococcus and Actinomyces species) become established within 24 hr. During the course of the next few days, the plaque increases in quantity as gram-negative cocci as well as gram-positive and -

negative rods and filaments gain a foothold. After 3 weeks, there is a significant increase in filamentous organisms, especially at the gingival margin (Listgarten et al. 1975; Listgarten 1976a). The metabolic products of the plaque microorganisms provoke an elevated level of PMN migration and sulcus fluid flow in the host tissues. This is the host's attempt to wall off invading bacteria. As gingivitis increases in severity, the junctional epithelium loses some of its resiliency, permitting the ingress of bacteria between the tooth and the epithelium. A gingival pocket develops.

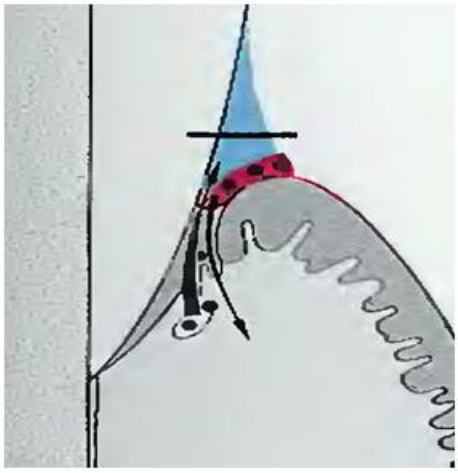


Figure 38. 1-week-old plaque - Metabolic interactions

Initial host response to plaque microorganisms (blue, gram-positive; red, gram-negative). Increased migration of PMNs (thick arrow), with formation of a wall of leukocytes. Chemotactic substances from plaque (thin arrow). The black horizontal line indicates the level from which the section of plaque was taken.

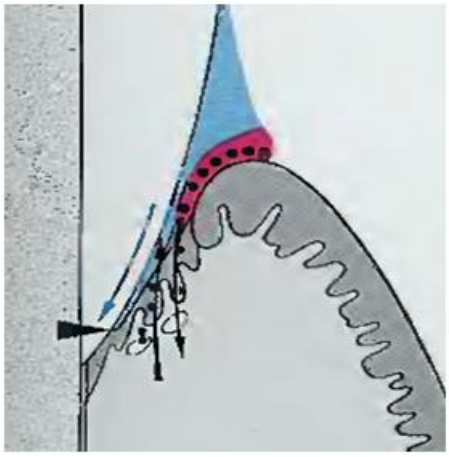


Figure 39. Expansion of supragingival plaque - Gingival pocket

The weakening of the attachment between tooth and JE permits apical immigration of gram-positive plaque bacteria (blue) in a thin layer, forming a gingival pocket. Gram-negative anaerobic bacteria (red) are observed mainly on the soft tissue -aspect of the sulcus, and elicit an enhanced host response.

Subgingival Plaque

Adherent flora - Nonadherent flora. In the subgingival region it is possible to differentiate between adherent and nonadherent plaque. A dense plaque layer of varying thickness adheres to the tooth (root) surface. The composition of this adherent layer resembles the supragingival plaque associated with gingivitis: some gram-positive cocci but primarily filaments - and Actinomyces species. The adherent plaque can become mineralized to form subgingival calculus. Near the soft tissue surface are observed freely moving bacterial accumulations ("swimmers") comprised almost exclusively of gram-negative anaerobes: cocci, spirochetes and rods (Bacteroides species, especially B. gingivalis). These nonadherent, partially motile, gram-negative,

pathogenic anaerobes increase sharply in number in acute inflammatory lesions. They appear to play an important role in the progression of periodontitis (Listgartcn 1976a; Slots 1979; Page & Schroe- der 1982; Lindhe 1983).

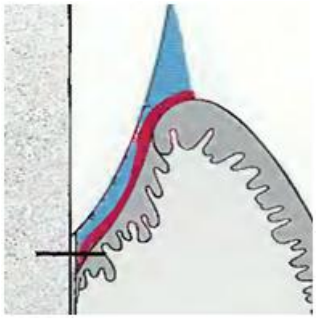


Figure 40. Subgingival flora. Diagram: Adherent plaque shown as blue: nonadherent bacteria (swimmers) shown as red. Black bar represents level from which the depicted plaque was taken.

Natural Factors Favoring Plaque Retention

The most important naturally occurring plaque-retentive factors or "nitches" include:

- Supra- and subgingival calculus
- Crowding of teeth in the arch
- Enamel projections, pearls, CEJ
- Mouth breathing.

Calculus is dead, calcified plaque. By itself it is not pathogenic, but due to its rough surface it is an ideal substrate for retention of pathogenic microorganisms.

Crowding leads to increased accumulation of plaque because self-cleansing mechanisms are foiled and because oral hygiene is more difficult.

Rough areas at the cementoenamel junction enhance plaque retention. On molars one often observes enamel projections or enamel "pearls" that may extend into the furcation.

Mouth breathing leads to dehydration of the oral cavity, rendering the plaque tougher and stickier. The protective function of saliva is reduced.

Iatrogenic Factors Favoring Plaque Accumulation

Restorative dentistry - from a simple restoration to a full-mouth reconstruction - if performed improperly can do more harm than good to the patient's oral health.

Fillings that appear to be perfect clinically and macro- scopically almost always exhibit deficiencies at the margins when viewed microscopically. Thus, when filling -margins are located subgingivally they are always an irritation for the marginal periodontium (Renggli 1974; Hammer & Hotz I979).

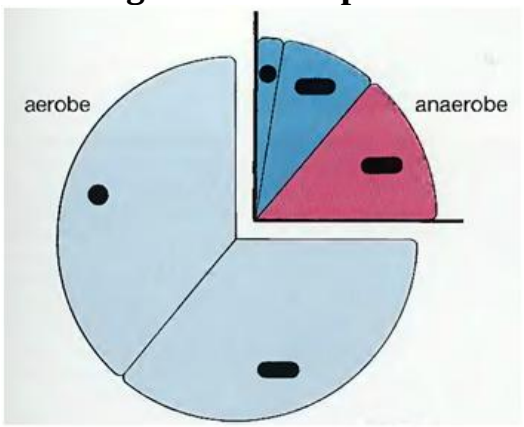
Overhanging margins of restorations and crowns accumulate additional plaque. Gingivitis ensues. The composition of the plaque changes. The number of gram- -negative anaerobes, those mainly responsible for the initiation and progression of periodontitis, increases rapidly (Langetal. 1983).

Gross iatrogenic irritants such as poorly designed clasps and prosthesis saddles may exert a direct traumatic influence on periodontal tissues.

Bacterial Flora and Types of Disease

Healthy gingiva. Clinically healthy gingiva can persist even in the presence of a thin (60 µm), adherent layer of plaque that is a few cell layers thick. The pie slices depicted below do not represent plaque quantity; rather, they depict the qualitative relationships -between aerobes/anaerobes and gram +/gram -.

Figure 41. Composition of dental plaque with healthy gingiva

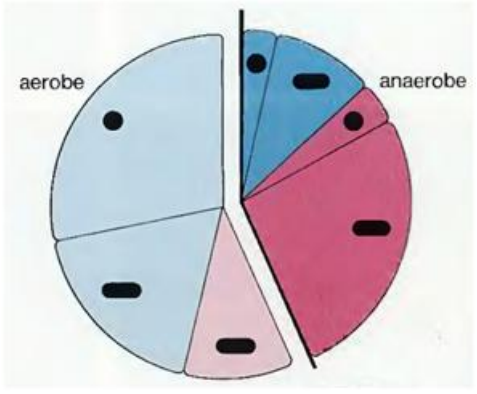


Light blue: gram + aerobes
Dark blue gram + anaerobes
Light red: gram - aerobes
Dark red: gram- anaerobes

The size of the pie slices represents the percentage composition of classifiable cocci and rods. In the extremely thin plaque, gram-positive aerobic cocci and rods -predominate (75%). These appear to be relatively nonpathogenic to the periodontal tissues.

Gingivitis - Adult periodontitis (AP). When gingivitis is manifest clinically, one generally observes plaque layers 400 µm and more thick. This quantitative increase in plaque plays an important role in the development of gingivitis. Alterations in the qualitative composition of plaque occur simultaneously. The gingivitis plaque (present supragingivally) is similar to that of quiescent, slowly progressing adult periodontitis (AP).

Figure 42. Composition of plaque in gingivitis (and AP)



Especially in sites where plaque accumulates, gram-negative anaerobic organisms begin to predominate at the cost of gram- positive aerobic cocci and rods. In particularly black pigmented types of Bacteroides and various forms of Spirochetes begin to predominate.

Table 12. Classified organisms of dental plaque

	GRAM POSITIVE		GRAM NEGATIVE	
	Facultative Anaerobes	Anaerobes	Facultative anaerobes	Anaerobes
Cocci	STREPTOCOCCUS S. mutans, S. mitis STAPHYLOCOCCUS MICROCOCCUS	STREPTOCOCCUS S. intermedius PEPTOSTREPTOCOCCUS PEPTOCOCCUS	NEISSERIA BRANHAMELLA	VEILLONELLA ACIDAMINOCOCCUS
Rods	ACTINOMYCES A. naeslundii A. viscosus BACTERIONEMA ROTHIA LACTOBACILLUS	ACTINOMYCES A israelii ARACHINA EUBACTERIUM BIFIDOBACTERIUM PROPIONIBACTERIUM CLOSTRIDIUM	ACTINOBACILLUS A. actinomycetemcomitans CAPNOCYTOPHAGA C. gingivalis C. ochracea C. spuligena EIKENELLA E. corrodens CAMPYLOBACTER HAEMOPHILUS	BACTEROIDES B. gingivalis B. melaninogenicus B. ss Inlermedius B. ss. melaninogenlcus FUSOBACTERIUM F. naviforme F. nucleatum LEPTOTRICHIA SELENOMONAS WOLINELLA
Spirochetes				TREPONEMA T. denticola T. vincentii T. macrodentium T. oralis

Cofactors in Etiology and Pathogenesis

The bacterial insult and the host response to it may be modified by various cofactors that can alte the clinical course of gingivitis and periodontitis.

Local:

- ✓ Saliva quantity and composition
- ✓ Mouth breathing
- ✓ Mechanical, chemical, thermal, allergic and actinic irritation
- ✓ Functional disturbances, occlusal trauma, orofacial muscular parafunctions (clenching, bruxism), parafunctions related to occupation.

General:

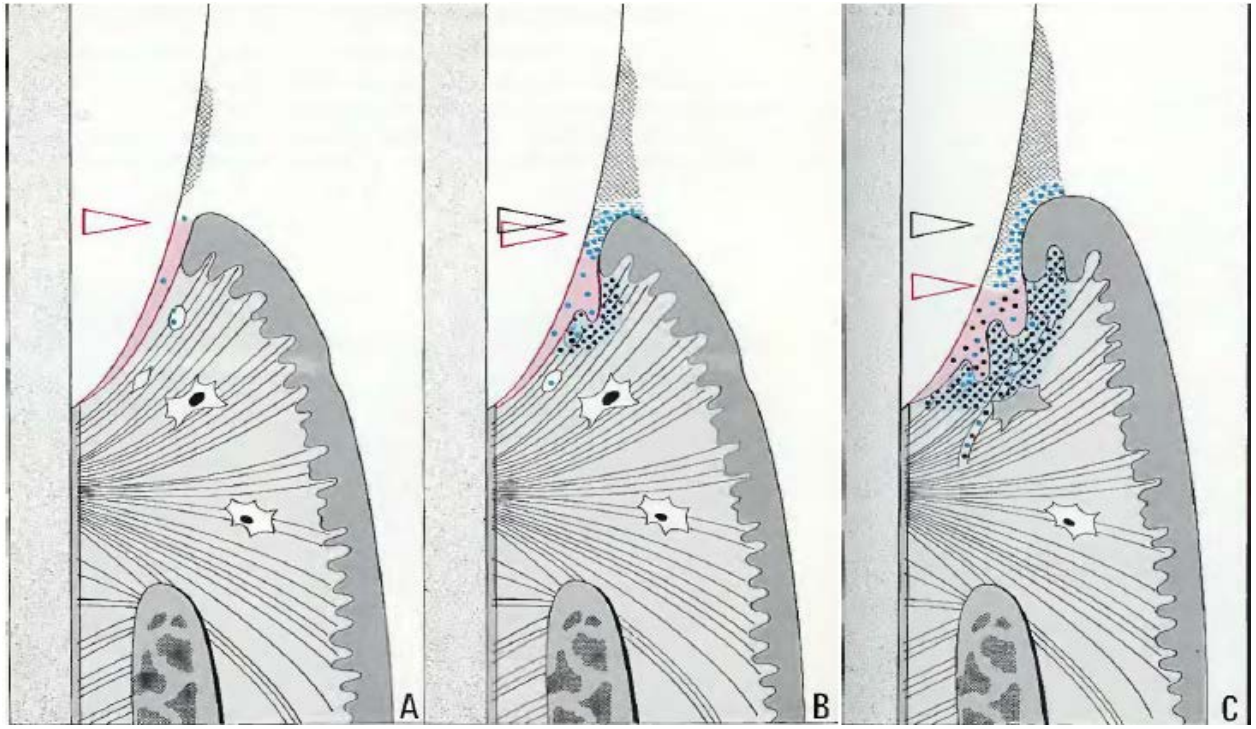
- ✓ Severe systemic disease
- ✓ Endocrine disturbances
- ✓ Stress
- ✓ Medicaments
- ✓ Nutrition
- ✓ Age.

The saliva has a protective function. Salivary mucin (a glycoprotein) lines the oral mucosa as a protective film. Depending on flow rate and viscosity, saliva may exert a greater or lesser cleansing effect. The content of bicarbonate, phosphate and minerals will determine saliva's buffering capacity and its remineralizing potential. -Secretory immunoglobulins (slgA) as well as lysozyme, catalase, lactoperoxidasc and additional enzymes help to determine the antimicrobial activity of saliva.

Mouth breathing leads to dehydration of the oral mucosa, cancelling the protective functions of saliva. Dryness allows plaque to cling even more tenaciously to the tooth surface.

Age does not necessarily predispose one to periodontal diseases, although the histologically detectabl ebiochemical alterations in connective tissue may be age -related. Clinically, one may observe a certain involution of the healthy periodontium. This is, however, likely to be less an age-related phenomenon than the result of years of exposure to exogenous factors such as intensive oral hygiene, chronic inflammation, or iatrogenic irritants (Gorman 1967; Hansen 1973; Sauerwein 1983).

Figure 43. Histopathogenesis of Gingivitis



	(A). Healthy gingiva	(B). Initial/Early gingivitis	(C). Established lesion
Plaque	Little primarily gram+, aerobic	Primarily gram + aerobic	Gram+ and gram- (gingival pocket)
Junctional epithelium/ pocket epithelium	Normal junctional epithelium without rete ridges	Initial alteration and lateral proliferation of the junctional epithelium in coronal region	Lateral proliferation of JE. deepening of sulcus with formation of gingival pocket or pseudopocket

Vessels Inflammatory cells, infiltrate, exudate	Few PMNs from subepithelial vascuiature in junctional epithelium very minimal exudate from the sulcus	Vasculitis exudation of serum proteins. PM N migration, accumulation of lymphoid cells, very few plasma cells, appearance of immunogloblins and complement	Acute inflammatory alterations; predominance of plasma cells; immunoglobulins in connective tissue, JE and gingival sulcus; increased sulcus exudate
Fibroblasts, connective tissue, collagen	Normal	Cytopathic alterations of fibroblasts; collagen loss in infiltrated connective issue areas	Severe fibroblast injury, further loss of collagen continued infiltration
Alveolar bone	Normal	Normal	Normal
Course of disease	-	Initial lesion 2-4 days after plaque accumulation early lesion 4-7 days	Manifest I week after plaque accumulation, can persist for years without progressing

Healthy gingiva (A). Absence of plaque or very little accumulation; normal junctional epithelium (pink); minimal sulcus depth (red arrow). A few PMNs (blue dots) transmigrate the JE in the direction of the sulcus bottom. Dense collagenous fiber system: intact fibroblasts.

Initial/Early gingivitis (B). Early plaque accumulation. In the initial lesion, increased transmigration of PMNs (blue dots) within the JE. As the early lesion develops, the PMNs create within the slightly deepened sulcus (red arrow) a wall against the plaque bacteria. A -lymphocytic infiltrate (black dots) occurs in the subepithelial tissues.

Established lesion (C). The gingiva responds to a massive accumulation of plaque. All of the -characteristics of gingivitis are manifest but may be more or less pronounced both clinically and histologically. The junctional pithelium, i.e., the epithelial -attachment, may actually be displaced somewhat apically as a consequence of the advancing front of accumulating plaque, resulting in the formation of a gingival pocket (distance between red and black arrows). Nevertheless, at this stage there is no loss of connective tissue attachment. The differentiated inflammatory infiltrate - protects the deeper structures of the periodontium

Question 2. Classification of gingivitis and periodontal diseases.

Classification of periodontal diseases (XVI Plenum of the All-Union Society of dentists, November, Yerevan, 1983):

Gingivitis is an inflammation of the gums caused by adverse impact of local and general factors and the flowing without destroying the integrity of dentogingival connection.

Form: catarrhal, ulcerative, hyperplastic.

Severity: mild, moderate, severe.

Current: acute, chronic, aggravated.

Prevalence: localized, generalized.

ICD-DA 1994 WHO

K05. Gingivitis and periodontal diseases

Includes: disease of edentulous alveolar ridge

K05.0 Acute gingivitis

Excludes: acute pericoronitis (K05.22)

acute necrotizing ulcerative gingivitis [fusospirochaetal gingivitis] [Vincent's gingivitis] (A69.10)

herpesviral gingivostomatitis (B00.2X)

K05.00 Acute streptococcal gingivostomatitis

K05.08 Other specified acute gingivitis

K05.09 Acute gingivitis, unspecified

K05.1 Chronic gingivitis

K05.10 Simple marginal

K05.11 Hyperplastic
K05.12 Ulcerative
Excludes: necrotizing ulcerative gingivitis (A69.10)
K05.13 Desquamative
K05.18 Other specified chronic gingivitis
K05.19 Chronic gingivitis, unspecified
K05.2 Acute periodontitis
K05.20 Periodontal abscess [parodontal abscess] of gingival origin without sinus
Periodontal abscess of gingival origin with no reference to sinus
Excludes: acute apical periodontitis of pulpal origin (K04.4)
acute periapical abscess of pulpal origin (K04.6, K04.7)
K05.21 Periodontal abscess [parodontal abscess] of gingival origin with sinus
Excludes: acute apical periodontitis of pulpal origin (K04.4)
acute periapical abscess of pulpal origin (K04.6, K04.7)
K05.22 Acute pericoronitis
K05.28 Other specified acute periodontitis
K05.29 Acute periodontitis, unspecified
K05.3 Chronic periodontitis
K05.30 Simplex
K05.31 Complex
K05.32 Chronic pericoronitis
K05.33 Thickened follicle
K05.38 Other specified chronic periodontitis
K05.39 Chronic periodontitis, unspecified
K05.4 Periodontosis
Juvenile periodontosis
K05.5 Other periodontal diseases

Question 3. Clinical manifestations and diagnosis of chronic simple marginal gingivitis.

Simple marginal gingivitis (05.10 K) corresponds to a chronic catarrhal gingivitis. Simple marginal gingivitis is exudative inflammation of the gums that develops in response to the pathogenic effects on their of dental plaque microorganisms.

Patient complaints: minor itching in the gums, bleeding them by mechanical stimulation (for example, when brushing teeth, taking hard food), an unpleasant taste in the mouth, bad breath.

Anamnesis. The disease begins gradually. It is almost asymptomatic for a long time. Most commonly occurs in children and young adults (25-30 years).

Non-mineralized supragingival dental deposits, chronic mild inflammation of the gums (swelling, bleeding, hyperemia) are detected during an ***objective examination***.

The consistency of the gums is a loose, edematous. Gingival surface is smooth, shiny during edema; the typical "orange peel" appearance disappears.

The contour the gums: increase in the volume of gingival papillae due to edema, loss of pointed shape.

The feature of gingivitis is absence of clinical pockets because the integrity of the periodontal attachment does not occur. Teeth are motionless, not displaced. Changes in the bone tissue of the interdental septa and other parts of the jaw bones are not determined during an X-ray examination.

Question 4. Chronic Hyperplastic gingivitis.

Chronic Hyperplastic gingivitis (K.05.11) is chronic inflammatory process of gum tissue, accompanied by their proliferation.

According to the medical literature, there are five different ***types of gingival hyperplasia***:

1. Gingival enlargement associated with inflammation
2. Gingival enlargement induced by medication
3. Gingival enlargement associated with different medical disorders (systemic involvement)
4. Gingival enlargement associated with cancer
5. False gingival enlargement.

Determining the type of gingival enlargement is essential, because each type has its own set of causes. The moment the type has been identified, so will be the causes. Once the cause is identified, the proper treatment can be administered. Because, when it comes to gingival hyperplasia, the treatment depends on the cause.

These are the most common causes of gingival hyperplasia:

1. Medication:
    - Gingival enlargement is encountered in people who are diagnosed with epilepsy and treated with phenytoin
    - The treatment with phenobarbital can also lead to this problem
    - Other drugs that can induce such problems are:
    - Cyclosporine (immunosuppressant)
    - Calcium-channel blocking agents (nifedipine, amlodipine)
2. Systemic causes
3. Pregnancy
4. Teenage (reaching puberty)
5. Vitamin deficiency (especially vitamin C)
6. Pyogenic granuloma
7. Leukemia

8. Granulomatosis
9. Sarcoidosis
10. Cancerous growths (benign or malign).

Local factors: pathological occlusion (crowding of the teeth), inefficient prosthetics, overhanging fillings, Cervical caries, poor hygiene, etc.

Clinical manifestations. Chronic hypertrophic gingivitis seen an increase in the volume of gingival papillae with the formation of false periodontal pockets. Gingival epithelial attachment in chronic hypertrophic gingivitis is not broken. Pathological changes in bone alveoli in chronic hypertrophic gingivitis because it is not.

Forms of chronic hypertrophic gingivitis. There is *edematous* and *fibrous* forms of CHG according to clinical and morphological changes. Morphologically edematous form of hypertrophic gingivitis manifests edema of the connective elements papilla, vasodilatation, swelling of collagen fibers, lymphoplasmacytic infiltration of tissues.

The clinical picture of edematous forms of hypertrophic gingivitis: complaints appear on aesthetic defect because of the unusual form of gum, on pain when brushing teeth, and during the meal.

Gingival papillae are enlarged, swollen, hyperemic or cyanotic, bleed during probing. Papillae have a glossy surface. The recess remains after pressing on the papilla surface by the blunt part of the instrument. Can be detected of tooth deposits.

The clinical picture is the fibrous form of hypertrophic gingivitis manifests patients' complaints to the unusual form of gum disease and the associated aesthetic defect. *Objectively*:

- ✓ Pale pink, dense, enlarged gingival papillae;
- ✓ Soreness and bleeding are absent;
- ✓ False gingival pocket.

Can detect the hard and soft of tooth subgingival deposits.

Diagnosis of hyperplastic gingivitis is not difficult, as a rule. To assess the patient's dental status, questioning, examination, palpation of the gums, probing of clinical pockets, Schiller-Pisarev test (with edematous form) are enough. In doubtful cases showed X-ray investigation.

A general blood test should be performed to exclude blood disease for all patients. Patients with hypertrophic gingivitis should consult medical specialists appropriate profile (gynecologist, endocrinologist, hematologist, etc.), in some cases require in-depth study of the hormonal status of the patient.

There are three degrees of severity:

I - mild hyperplastic gingivitis - gums hyperplasia reaches no more than 1/3 of the tooth crown,

II - moderate degree - no more than 1/2 of the tooth crown,

III - severe form - the gum covers 2/3 or full crown.

Question 5. Chronic Ulcerative gingivitis.

Chronic Ulcerative gingivitis (K.05.12) is inflammation of the gums, accompanied by necrosis and ulceration of her tissues.

The predominance of tissue alteration underlies these processes. The leading role in the genesis of ulcerative gingivitis belongs to a decrease in the resistance of the gingival mucosa to the fusospirochetal microflora of the oral cavity. Often is a consequence of acute ulcerative gingivitis

Clinical picture: pains in the gums, putrid breath, bleeding gums. During examination of the oral cavity: the gingival margin covered by gray fetid deposit. Bleeding sharply painful surface and ulceration are exposed after its removal. The apexes of the gingival papillae as if cut off, the contours of the gingival margin are disturbed. There are abundant non-mineralized dental plaque, dental calculus. Regional lymph nodes are enlarged, little painful during palpation.

Differential diagnosis - AIDS manifestation, blood diseases.

Question 6. Chronic Desquamative gingivitis

It's polyetiology disease. There are *independent* and *symptomatic* gingivitis. Symptomatic gingivitis does not refer to specific periodontal diseases. At the present time, it is considered a reflection of various systemic diseases. Such as:

- Dermatoses (lichen planus, pemphigoid);
- Endocrine disruption (diabetes, thyroid disorders, metabolic disorders);
- Some infectious diseases;
- Drug reactions.

Many of these begin with the DG, the changes appear only later in other areas of the oral mucosa or the skin. The vestibular surface of the gum is affected in the first place, then the palatine surface is involved in the process, but the changes here are less pronounced. DG is more common in women.

Local: redness and desquamation of surface epithelium. The process is localized in the area of attached gingiva is usually marginal gingiva is not affected. It is prolonged.

Species: atrophic (erythematous), erosive (desquamative), bullous form.

There are three forms:

1. Mild: erythema marginal and attached gingiva with desquamation of surface epithelium, painless on palpation;
2. Moderate: gingival surface is red, shiny, spotted. The epithelium of the gingival "erased", revealing the bleeding surface. There is a painful gum.

3. Severe: gums surface is bright red, epithelium can tear by pieces. There is sharp pain during palpation. Patients complain on pain, inability to eating, dryness, burning sensation in the mouth.

Question 7. Differential diagnosis

Differential diagnosis of chronic simple marginal gingivitis - hyperplastic (edematous form) gingivitis, chronic simplex periodontitis (mild).

Differential diagnosis of localized hyperplastic gingivitis and epulis. Specific features epulis - form tumors (in the form of a sheet or mushroom), its coloring (red meat, with brownish or a cyanotic shade), change in bone - bone loss in space epulis legs.

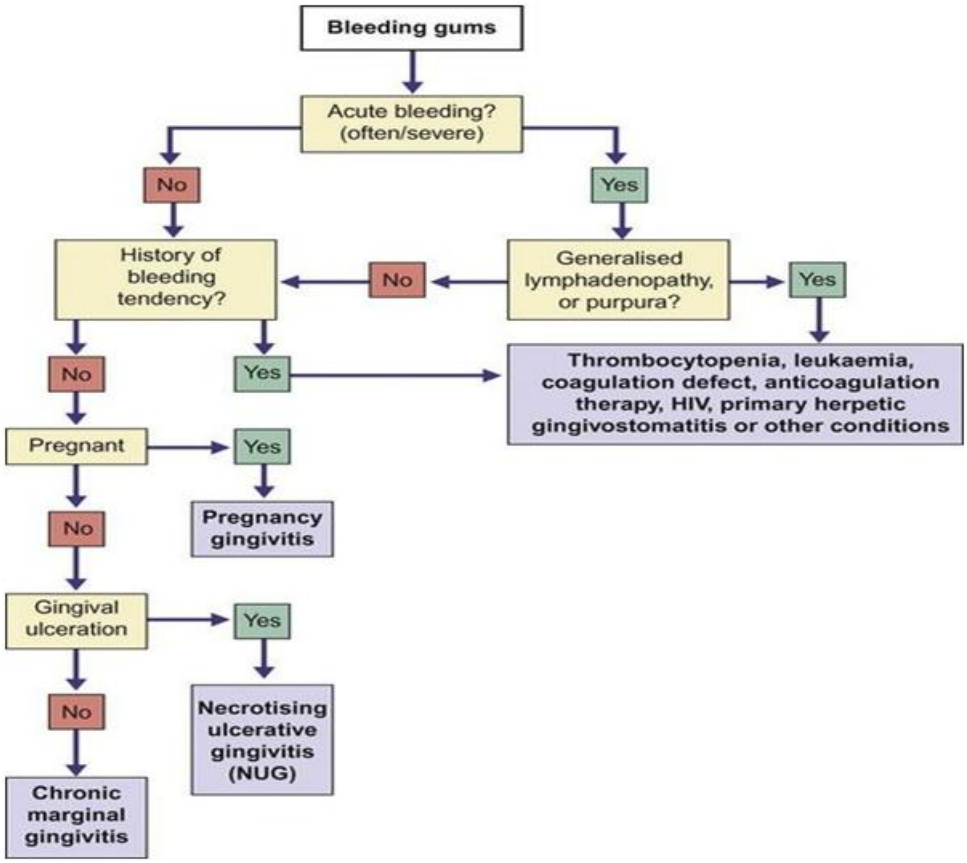


Figure 44. Algorithm for bleeding gums diagnosis

Table 13. Acute necrotizing ulcerative gingivitis/desquamative gingivitis and chronic destructive periodontal diseases

Acute necrotizing ulcerative gingivitis	*Desquamative gingivitis*	*Chronic destructive periodontal disease*
Bacterial smear shows fusospirochetal complex	Bacterial smears reveals numerous epithelial cells, a few bacterial forms	Bacterial smears are variable
Painfulness	May or may not be painful	Painless
Acute onset	Chronic onset	Chronic onset

Marginal gingival affected	Other areas of oral mucosa	Marginal gingival affected
Characteristic fetid odor	None	Some odor present but not fetid
Papillary and marginal necrotic lesions	Papilloma does not undergo necrosis	Papillae do not undergo notable necrosis

Test control

1. Specify the complaints of the patient with a simple marginal gingivitis:

a) pain and bleeding gums when brushing teeth and eating;
b) periodically there are bleeding gums during brushing teeth and taking solid food, bad breath;
c) pain and bleeding gums, aggravated by eating and brushing teeth, gingival overgrowth;
d) pain during biting, tooth mobility, bleeding gums during brushing, bad taste in the mouth.

2. Clinical symptoms of the simple marginal gingivitis are:

a) bleeding from the sulcus;
b) gum color change from pale to cyanotic;
c) increased gingival papillae, pale pink in color, firm to the touch;
d) the consistency of gum is loose, edematous.

3. Diagnostics simple marginal gingivitis is based on the following symptoms:

a) bleeding;
b) edema;
c) pain;
d) gum color change.
e) presence of periodontal pockets.

4. Gingival pseudopocket is present in the case of:

a) chronic simple marginal gingivitis;
b) chronic hyperplastic gingivitis (swollen form);
c) chronic hyperplastic gingivitis (fibrous form);
d) acute herpetic stomatitis;
e) all of the above.

5. The complaints of the patient in the case of edematous form of hyperplastic gingivitis are:

a) paresthesia and ulceration of the gums;
b) aesthetic defect due to the unusual form of the gums;
c) sore gums during brushing teeth and while eating;
d) bad breath, pain, poor general condition;
e) severe aching pain in the gums.

6. The complaints of the patient in the case of fibrous form of hyperplastic gingivitis are:

a) paresthesia and ulceration of the gums;
b) aesthetic defect due to the unusual form of the gums;
c) sore gums during brushing teeth and while eating;
d) bad breath, pain, poor general condition;
e) severe aching pain in the gums.

7. The complaints of the patient with chronic ulcerative gingivitis are:

a) paresthesia of the gums;
b) aesthetic defect due to overgrowth of the gums;
c) bleeding gums;
d) putrid breath;
e) pain in the gums.

8. Describe the gingival margin condition during chronic ulcerative gingivitis:

a) swelling, bleeding, pain of individual gingival papillae;
b) atrophied papilla, gingival margin with symptoms of congestive hyperemia;
c) gingival margin covered with gray malodorous bloom, after the removal of which is exposed acutely painful bleeding surface;
d) the top of the gingival papillae, as it were cut off, the contours of the gingival margin violated.

9. Desquamative gingivitis occurs during the following diseases:

a) dermatoses (lichen planus, pemphigoid);
b) endocrine disruption (diabetes, thyroid disorders, metabolic disorders);
c) some infectious diseases;
d) drug reactions.
e) all of above

10. Indicate the code of desquamative gingivitis according to ICD-10:

a) K05.12
b) K05.10
c) K05.13
d) K05.18

LESSON 10. CHRONIC PERIODONTITIS. CLINICAL FEATURES, DIAGNOSIS.

The questions to be studied for the learning of the topic:

1. The role of local and systemic factors in the development of chronic periodontitis.
2. Clinical features of chronic simple and complex periodontitis.
3. Periodontal pocket: definition, mechanism of formation, types, methods of diagnosis.
4. Furcation involvement into the pathological process: diagnosis, classification.

Question 1. The role of local and systemic factors in the development of chronic periodontitis.

Risk factors in the development of chronic periodontitis:

1) bacterial flora of the mouth;
2) age;
3) smoking;
4) systemic diseases:
   - reduced number and function of polymorphonucleocytes;
   - hormonal disorders;
   - blood diseases (including leukemia);
   - genetic predisposition;
5) stress.

Question 2. Clinical features of chronic simple and complex periodontitis.

In case of simple periodontitis the dentist clinically determines chronic inflammation of periodontal tissues: hyperemia, swollen gingiva, bleeding on probing, destruction of the periodontal ligament, tooth loosening. The dentist reveals radiographically chronic inflammation of periodontal tissues: suprabony periodontal pocket, horizontal resorption of the alveolar bone. In case of complex periodontitis the dentist clinically determines chronic inflammation of periodontal tissues: hyperemia, swollen gingiva, bleeding on probing, destruction of the periodontal ligament, tooth loosening, degenerate and necrotic changes of the blood vessels. The dentist detects radiographically chronic inflammation of periodontal tissues: infrabony periodontal pocket, vertical and angular resorption of the alveolar bone.

Question 3. Periodontal pocket: definition, mechanism of formation, types, methods of diagnosis.

Periodontal pocket is a pocket outside of the gingiva, penetrating deeper into the destroyed periodontal tissue.

The mechanism of the periodontal pockets formation:

1. Inflammation

2. Destruction of the interdental fibers (Sharpey's fibers), proliferation of the epithelium along the root, displacement of the epithelial attachment. Local stimulus primarily lies in the mechanism of the periodontal pockets formation, which causes inflammation of the gingiva. Then there is a destruction of interdental fibers (Sharpey's fibers) again, which contributes to the proliferation of the epithelial attachment along the root and apical migration of the epithelial attachment. Once formed, the periodontal pocket is a focus of chronic inflammatory process.

There are different types of periodontal pockets, depending on the topography, localization, prevalence, depth of the lesion, bone tissue involvement:

- ✓ an infrabony periodontal pocket is a periodontal pocket in which the bottom is apical to the level of the adjacent alveolar bone;
- ✓ a suprabony periodontal pocket is a periodontal pocket in which the bottom is coronal to the underlying bone. This condition is associated with horizontal bone resorption;
- ✓ a simple periodontal pocket is a pocket, which is located at one surface of the tooth;
- ✓ a compound periodontal pocket is a pocket, which is detected at two or more surfaces of the tooth. The basis of the periodontal pocket is at the level of marginal gingiva;
- ✓ complex periodontal pocket is a pocket, that covers the tooth from all its sides.

Dentists evaluate the presence of periodontal pockets using the periodontal probe. While determining the depth of the periodontal pocket, it is necessary to take into account the presence of dental plaque, bleeding on probing and painful reaction on probing.

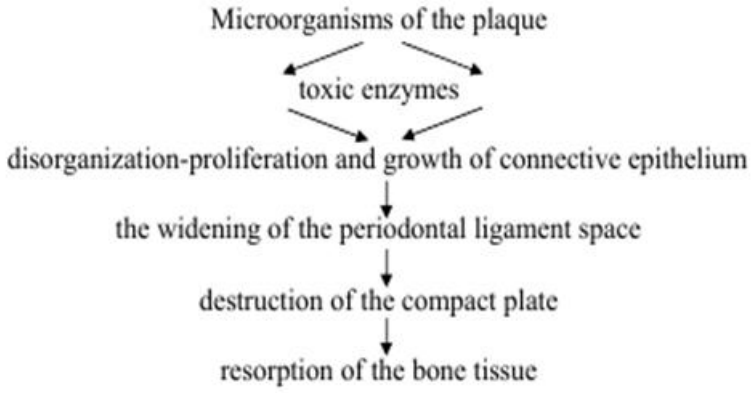


Figure 45. The mechanism of the periodontal pocket formation.

Question 4. Furcation involvement into the pathological process: diagnosis, classification.

Furcation is usually involved into the pathological process in case of periodontitis. This is a region of the alveolar bone in the area of the root furcation. The presence of the furcation involvement is diagnosed using the furcation probe and the X-ray. Researchers have suggested several classifications of furcation involvement into the pathological process.

Table 14. Classifications of furcation involvement into the pathological process.

Class A	Class B	Class C
1-3 mm	4-6 mm	7-9 mm
Class I	Class II	Class III
Initial involvement	Partial involvement	Complete or perforating involvement
F1	F2	F3

Test control

1. What is the characteristic feature of chronic simple periodontitis?

a) dentine sensitivity;
b) presence of periodontal pockets;
c) integrity of the epithelial attachment; d) gingival hyperplasia.

2. What is diagnosed in case of chronic simple periodontitis?

a) one osseous wall periodontal pocket;
b) two osseous walls periodontal pocket;
c) three osseous walls periodontal pocket;
d) suprabony periodontal pocket.

3. What is the characteristic of the chronic simple periodontitis?

a) quick course with a rapid teeth loss at a young age;
b) long course;
c) quick progression.

4. What kind of the bone resorption is typical for chronic simple periodontitis?

a) horizontal;
b) vertical;
c) complex;
d) combined.

5. What are the characteristics of the chronic simple periodontitis?

a) destruction of the dento-gingival attachment with the formation of periodontal pockets;
b) preservation of the dento-gingival attachments with the depth of the gingival sulcus more than 3 mm on probing;
c) halitosis;
d) localized gingival recession.

6. What causes chronic complex periodontitis?

a) local irritants;
b) occlusal trauma;
c) local irritants and occlusal trauma or disharmony;
d) only general factors.

7. What are the characteristics of the chronic simple mild severity periodontitis?

a) 1/3–1/2 bone tissue resorption of interdental septum, degree I or degree II tooth pathological mobility, Class I and Class II furcation involvement, periodontal pocket depth up to 5 mm;
b) more than 1/2 bone tissue resorption of interdental septum, degree II or degree III tooth pathological mobility, Class II and Class III furcation involvement, periodontal pocket depth more than 5–6 mm;
c) less than 1/3 bone tissue resorption of interdental septum, absence of tooth pathological mobility, Class I furcation involvement, periodontal pocket depth up to 4 mm.

8. What are the characteristics of the chronic simple moderate severity periodontitis?

a) 1/3–1/2 bone tissue resorption of interdental septum, degree I or degree II tooth pathological mobility, Class I and Class II furcation involvement, periodontal pocket depth up to 5 mm;
b) more than 1/2 bone tissue resorption of interdental septum, degree II or degree III tooth pathological mobility, Class II and Class III furcation involvement, periodontal pocket depth more than 5–6 mm;
c) less than 1/3 bone tissue resorption of interdental septum, absence of tooth pathological mobility, Class I furcation involvement, periodontal pocket depth up to 4 mm.

9. What are the characteristics of the chronic simple severe periodontitis?

a) 1/3–1/2 bone tissue resorption of interdental septum, degree I or degree II tooth pathological mobility, Class I and Class II furcation involvement, periodontal pocket depth up to 5 mm;

b) more than 1/2 bone tissue resorption of interdental septum, degree II or degree III tooth pathological mobility, Class II and Class III furcation involvement, periodontal pocket depth more than 5–6 mm;
c) less than 1/3 bone tissue resorption of interdental septum, absence of tooth pathological mobility, Class I furcation involvement, periodontal pocket depth up to 4 mm.

10. What is the initial radiographic sign of chronic periodontitis?

a) desintegration of the cortical plates of interdental septa tops, widening of the periodontal ligament space and osteoporosis;
b) widening of the periodontal ligament space in the apical area of the tooth root;
c) bone resorption of the interdental septa up to 1/3 of the root length;
d) resorption of the bone tissue in the apical area of the tooth root.

LESSON 11. PROGNOSIS OF PERIODONTAL DISEASES. TREATMENT PLANNING OF PERIODONTAL DISEASES.

The questions to be studied for the learning of the topic:

1. Methods of examining dental patients for making diagnosis, determining the prognosis and planning the treatment.
2. General and local factors determining the prognosis of periodontal diseases.
3. Stages of treatment planning.

Question 1.Methods of examining dental patients for making diagnosis, determining the prognosis and planning the treatment.

Prognosis of the disease is the conclusion about the disease forthcoming development and outcome on the basis of objective data. Medical prognosis is a prediction of the illness or disease outcome, based on the knowledge of the pathological processes development. The prognosis for patients with periodontal diseases exists in two aspects: overall prognosis (the prognosis of the disease) and individual prognosis (the prognosis of the patient). Sometimes the prognosis of the disease and the prognosis of the same patient may not be the same. There are good (favorable) and poor (unfavorable) prognoses. If the disease is well known to be completely curable (most gingivitis cases), we speak about favorable prognosis (prognosis bona). A poor or unfavorable prognosis (prognosis mala) is made when the regular rational treatment courses do not allow to achieve a stable remission, and the tooth loss is inevitable. Prognosis should be determined after the diagnosis and before making up a treatment plan. It is necessary to take into account the following:

1. The pathological process duration.
2. Each tooth involvement into the pathological process.
3. The patient’s age.
4. The number of teeth in the patient’s oral cavity.
5. If the treatment strategy can be changed in case of loosing the «key» tooth during treatment. How long the «key» tooth can be preserved.
6. If it is possible to restore the defect of dentition by the denture.
7. Status of the tooth supporting structures.
8. If the patient has received any treatment before and what the reaction of the periodontium to the previous treatment was.
9. What is the oral cavity status and how can it be improved.
10. If there are parafunctions and if they can be eliminated.
11. If the patient can control the dental plaque.
12. If there are enough opportunities and the periodontologist’s experience to help the patient.

13. If the doctor has enough professionalism level and qualification to guarantee the optimal treatment. The prognosis of the disease depends on the development of new clinical medicines at the moment. At the present stage of the Periodontology development (with new technologies both pharmacological and operational application), even in case of diagnosing aggressive periodontitis and applying a timely and rational treatment, the prognosis is far from being hopeless.

Question 2. General and local factors determining the prognosis of periodontal diseases. The following factors influence the patient's prognosis:

1. Gender.
2. Age.
3. The patient's organism resistance (reactivity). Some patients have a marked positive effect shortly after the treatment beginning, other patients have little effect regardless of the treatment intensity. Therefore, you can accurately determine the prognosis after initial pre-treatment when the doctor determines the response of the body.
4. Constitutional and personality traits (hypochondria, anxiety, a tendency to focus on fixing the painful sensations contribute to a more severe and prolonged disease course).
5. Suffered and accompanying diseases, especially diabetes, the gastrointestinal tract, cardiovascular system, blood, and thyroid diseases.
6. Susceptibility to drugs, especially such drugs as antibiotics, Trichopolum, chlorhexidine and others.
7. Professional risks.
8. Bad habits.
9. Socio-economic conditions (many high-tech methods - flap surgery, orthodontic methods are not available for certain patients).

Early detection of the disease and the patient's age affect the disease prognosis. If the patient has a continuos pathological process, it is difficult to control it. Young people have a better prognosis than adults and elderly people. If the patient is young, the pathological process is faster and more «malignant». At the same time, reparative processes in young people are more complete, compared to older generations. Nevertheless manifestations of periodontal tissues destruction and the individual prognosis are always worse in young patients, than in older ones.

The prognosis for the individual teeth depends on the following: the teeth mobility, occlusal trauma, the bone loss level, periodontal pockets (not their depth). The individual teeth prognosis depends on the periodontal pockets base, the level of frenulum attachment, degenerative changes in the pulp and the amount of roots in the tooth. The presence of occlusal trauma destroys the periodontal tissues aggressively, resulting in the vertical

resorption and intraosseous pockets formation. The dentist should take into account the amount of remaining bone: the prognosis is good, when there is a slight loss of the bone tissue. Clinicians make the prognosis on the basis of intraosseous pockets presence. The pocket depth is not the decisive factor for the prognosis, localization of the pocket base is much more important. If the periodontal pocket base is located near the root apex, then there is a high probability of bacterial products penetration through the apical foramina, which significantly worsens the prognosis. However, if you perform a combined treatment, it is possible to achieve the pathological process stabilization. The probability of intraosseous pockets treatment depends on the bone defect contour and the magnitude of the remaining bone wall. It should be noted that the prognosis of endodontically treated teeth is little different from the vital teeth prognosis. The periodontal re-attachment is likely to occur in vital than non-vital teeth because the vital tooth root recovers the attachment better. The furcation involvement in multirooted teeth is a rather unfavorable situation; nevertheless these teeth have the advantage over single-rooted teeth, as they have additional support. However, maintaining hygienic measures with such teeth is very difficult. If there is a possibility of prosthodontic treatment application, reconstruction of the dentition integrity, then the prognosis is good. You should take into account the «key» teeth, their compensatory ability: if they can stand the extra load as supporting elements. To optimize the prognosis in patients with aggressive periodontitis, you should take into account the following clinical-laboratory features: gingiva morphofunctional condition, the level of the periodontal tissues destruction, neutrophils functional activity, elastase-inhibitory activity of the blood serum, α1 PI (proteinases inhibitor) activity, and α2 — MG (macroglobulin).

The computer program «Prognosis of Periodontal Diseases» was developed at the 3rd Department of Therapeutic Dentistry, BSMU for identification and analysis of prognostic signs of the disease by means of construction a programmed graph (J. L. Denisova, 2012). The following prognostic criteria for the disease development were included into the program «Prognosis of Periodontal Diseases»: impaired microcirculation according to the laser and optical diagnosis (from 0 to 60 conventional units), the percentage of the alveolar bone loss, depending on the patient's age (from 0 to 100 %), the proportion of segments with bleeding on probing with the total number of segments in % (0 to 100 %), the number of segments with periodontal pockets of 5 mm depth or more (from 0 to 100 %), the number of missing teeth (from 1 to 32), the presence of dentofacial deformities (tooth migration, tooth crowding, and others) («Yes», «No»), systemic diseases (osteopenia or osteoporosis, type I, type II diabetes or IL-1 polymorphism, stress, etc.) («Yes», «No»), environmental factors such as cigarette smoking («does not smoke», «former smoker» - has not smoked over the past 5 years

and over, «smokes sometimes», «smokes up to 20 cigarettes a day», «smokes more than 20 cigarettes a day», «smokes more than 40 cigarettes per day»). Each parameter (vector) in programmed chart has its own measurement scale. All prognostic criteria are interpreted depending on the risk level of possible disease development or progression (low, medium, high). There is a lower risk level in the central part, while there is a high risk level at the periphery. There is a zone of moderate risk level of periodontal diseases progression between them. A comprehensive evaluation of prognostic criteria determines the individual risk level of possible disease progression, treatment, preventive measures and the frequency of visits. The program «Prognosis of Periodontal Diseases» calculates the size of the filled up part of the functional diagram in percent. A low risk of periodontal diseases development is determined when all the parameters are within low limits of the chart or one parameter was in the category of medium risk. An average risk level of the periodontal diseases development is determined if two parameters are in the middle sections of the chart, but not more than one parameter is in the category of high risk. There is a high risk of the disease development when the patient has the most predictive characteristics at high limits. When the dentist calculates the parameters of the diagram area, he identifies three colors depending on the risk level. The low level corresponds to the green color, medium to yellow and high to red. The developed method of analyzing the prognostic signs of periodontal diseases development and progression allows to determine in regard to both quality and quantity the risk level of periodontal diseases development and progression, the necessary diagnostic and treatment activities, and the dynamic monitoring frequency. It allows the dentist to inform the patient visually about the risk level of the periodontal diseases development, to conduct the monitoring of the ongoing prognostic signs and to increase the treatment effectiveness.

Question 3. Stages of treatment planning.

Currently, periodontists have come to the same opinion that in 92 % of cases gum inflammation occurs only from the local irritating factors which lead to conduct the main activities of their elimination. The treatment planning of periodontal diseases is a planned integrated system of manipulations, involving the order, sequence and timing of the implementation, aimed at restoring and maintaining oral health.

I. Initial therapy.

Hygienic measures:

- dental plaque removal;
- oral hygiene motivation and training;
- the control of the dental plaque growth.

Elimination of iatrogenic factors in the oral cavity.

Temporary splinting.

Elimination of parafunctions (bruxism).

Events on the gingival wall (curettage).

II. Re-evaluation of periodontal tissues.

III. Orthodontic treatment.

IV. Prosthodontic treatment.

V. Surgical treatment.

VI. Supportive periodontal therapy.

The initial therapy consists of the elimination of etiological factors and particular irritating factors, such as dental plaque, iatrogenic factors and potentially harmful habits. Nevertheless, this stage is called «initial», it remains the most important and effective method of periodontal diseases treatment. Hygiene measures consist of the dental plaque removal up to OHI-S = 0,5–0,6 together with the patient motivation about the need for these activities; teaching oral care, control of the plaque presence and growth. These three procedures are very important in the treatment as it is possible to make great efforts to eliminate periodontal pockets during follow-up, but it will all be in vain if the primary success of these procedures is not achieved at the beginning. Initial therapy includes manipulations on the gingival wall or elimination of proliferative epithelial lining of the gum by the curettage method. This method has always been considered formal because it gives short-term effects. However, periodontists don't refuse this method and some international periodontists use it at the preparatory treatment stage.

During the elimination of iatrogenic factors you need to detect poor Class II, Class V, Class IV dental fillings with bad contact points and dentures with long crowns and traumatic intermediate parts. The next step is re-evaluation, which evaluates the response of periodontal tissues to the activities of the preparatory phase and recording of the objective data into the patient's medical history. At this stage you should estimate the patient's oral hygiene, gingiva condition, periodontal pockets depth and the microcirculation status of the periodontium.

At the next stage, dentists carry out orthodontic treatment in case of dental anomalies and deformities. The position of this treatment phase is controversial in the treatment plan. Some dentists believe that it should be carried out in the early stages, other dentists believe this procedure is complicated, as it requires a lot of time (1–2 years). However, it is impossible to obtain a favorable periodontal disease prognosis and to ensure rational prosthodontic treatment, implantation, permanent teeth splinting and some surgical interventions without timely removing dental anomalies and deformities. Orthodontic treatment provides functional stimulation necessary to maintain the periodontal tissue health besides getting the occlusal adjustment. Dentists apply prosthodontic treatment in case of teeth and dentition defects. This step is performed after implantation.

Surgical treatment with regular professional teeth cleaning within 3 months is aimed at the removal of the inflammation in periodontal pockets and the factors causing it, as well as restoration of the gingival contour and mucogingival relationships. Radical therapy, aimed at reducing the pocket depth, has been questioned for many years. This type of surgical intervention was believed to lead to the gingival recession, dentine sensitivity and root caries.

Researchers from many universities have conducted a series of clinical trials to assess the need of such interventions and their effectiveness. These studies were firstly carried on in 1968 by a group of Michigan scientists headed by Professor Ramord. The scientists compared closed curettage and modified flap surgery according to the Widmann–Neumann method. It was determined that flap surgery resulted in the attachment regeneration even when the pockets depth was 7–12 mm. Closed curettage did not allow to achieve it, especially in the molar region. If the pocket depth was more than 4 mm, the subgingival calculus was not possible to remove without full viewing. Swedish researchers have studied the same problem. The swedish scientists have proved that only a proper and careful post-operative care determines the treatment outcome. If there is no perfect oral hygiene, the prognosis is unfavorable. In order to plan the surgical treatment, you need to assess the condition of the periodontal pocket, the reparative ability of the oral mucosa, the possibility of continuous professional oral care within 3 months and the patient's readiness for a long-term treatment. Supportive therapy is the final stage in periodontal disease treatment. This therapy is focused on regular oral hygiene monitoring with its constant correction and detection of dental caries, fillings, occlusion and monitoring the microcirculation status in the periodontium. Scientists have found that first disturbances occur in the microvasculature in case of periodontal diseases. Therefore, it is a sign of the periodontal tissue state, depending on the level of the pathological process, and its normalization after the whole medical treatment. This period is the most critical for full stabilization of the periodontal regeneration process. Physiotherapy is one of the most effective methods of microcirculation normalization in the periodontal tissues.

Test control

1. The prognosis for patients with periodontal diseases exists

a) overall prognosis;
b) individual prognosis;
c) mixed prognosis.

2. Indicate types of prognosis:

a) good (favorable)
b) poor (unfavorable)

c) guarded

3. Does the general prognosis influence the prognosis of individual teeth with periodontal disease?

a) yes;
b) no;
c) yes, but only the prognosis of molars with furcation involvement.

4. What factors are most important in determining the prognosis of periodontal diseases?

a) the amount of dental plaque and the degree of periodontal tissue destruction;
b) the concentration of Streptococcus mutans in saliva and saliva buffer capacity;
c) the severity of caries and the level of dental care;
d) all of the above.

5. How does the speed of disease progression affect the general prognosis of periodontal diseases?

a) does not influence patients aged 18–20 years;
b) periodontal diseases progressing both rapidly and slowly have the same prognosis;
c) prognosis is poor in case of a rapidly progressive periodontitis.

6. Does the prognosis of periodontal diseases depend on the patient's trust and desire to cooperate with the doctor?

a) depends only at the age of 18–20 years;
b) depends;
c) does not depend;
d) does not depend only at the age of 18–20 years.

7. What prognosis is made at the first stage?

a) the prognosis for individual teeth;
b) the general prognosis;
c) the general prognosis and prognosis for individual teeth are carried out simultaneously.

8. What factors are taken into account in determining the prognosis of individual teeth?

a) tooth mobility and periodontal pocket depth;
b) furcation involvement and tooth morphology;
c) bone destruction degree on each surface of the tooth, caries and its complications;

d) all of the above.

9. What factors determine a good prognosis of periodontal diseases?

a) loss of sufficient amount of bone, surrounding the teeth, tooth mobility, Class I furcation involvement, acceptable communication with the patient;
b) sufficient amount of bone, surrounding the teeth, sufficient control of etiological factors (plaque), adequate communication with the patient;
c) absence of bone loss, excellent state of the gingiva, the patient's full confidence in the doctor.

10. Motivation of patients with periodontal diseases should be carried out:

a) once a year.
b) 2 times per year.
c) during the patient's first visit.
d) during each visit to the patient.

LESSON 12. INITIAL THERAPY OF PATIENTS WITH PERIODONTAL DISEASES: HYGIENIC PROCEDURES, ESPECIALLY OF RESTORATION.

The questions to be studied for the learning of the topic:

1. Stages of hygienic procedures as a part of the initial therapy of periodontal diseases.
2. Methods and tools, applied for professional oral hygiene.
3. The main features of carious cavities preparation for dental tissues restoration in patients with periodontal diseases.
4. The sequence of actions for the restoration of the contact point and the shape of the tooth crown in patients with periodontal diseases.

Question 1. Stages of hygienic procedures as a part of the initial therapy of periodontal diseases.

Hygienic measures in patients with periodontal diseases are carried out in a certain sequence.

They include:

- ✓ motivation of the patient;
- ✓ selection of the appropriate home hygiene tools and the training to use
- ✓ them;
- ✓ control of dental plaque growth;
- ✓ consistent and thorough removal of dental plaque.

During the first visit, the doctor examines the oral cavity, evaluates the condition of the teeth, periodontium and oral hygiene with the use of OHI-S (Green–Vermillion), PHP (index of the oral hygiene effectiveness) hygiene indices. The doctor shows the patient dental plaque on his teeth with the help of dental instruments and dental plaque indicators. During the same visit, the doctor conducts a conversation with the patient about the role of dental plaque in the development of dental caries and periodontal diseases, conducts selection of individual oral hygiene appliances, gives recommendations for the oral care and conducts professional teeth cleaning.

During the second visit the patient comes to the doctor with the recommended hygiene appliances. The doctor determines the state of the oral hygiene, if needed, the doctor conducts the correction of toothbrushing technique on the model and directly in the oral cavity, demonstrates the technique of using interdental appliances. The doctor removes the remaining dental plaque. During the following visit, the doctor controls the toothbrushing improvement, if it’s necessary, corrects the oral hygiene. During each visit, the dentist conducts a thorough removal of plaque and remaining dental tartar (supragingival dental plaque), especially in unaccessible dental surfaces.

Question 2. Methods and tools, applied for professional oral hygiene.

Stage I. Dental plaque assessment (dental plaque localization and quantity). Dentists use the additional lighting for it. Determination of periodontal pocket depth and subgingival dental calculus, furcation involvement is performed using a periodontal probe.

Classification of dental plaque (WHO, 1995)

K03.6 Deposits [accretions] on teeth
Includes: staining of teeth NOS
KO3.60 Pigmented film
Black
Green
Orange
K03.61 Due to tobacco habit
KO3.62 Due to betel-chewing habit
K03.63 Other gross soft deposits
Materia alba
K03.64 Supragingival calculus
KO3.65 Subgingival calculus
KO3.66 Dental plaque
K03.68 Other specified deposits on teeth
K03.69 Deposits on teeth, unspecified

Orange pigmented dental plaque is localized on the oral and vestibular surfaces of the anterior teeth. It is created by special chromogenic microbes (Sentra marcescens, etc). Green pigmented dental plaque is located on the vestibular surface of the upper incisors. It is found mostly on the milk teeth. The occurrence of this kind of dental plaque on the teeth is associated with specific bacteria and fungi - Licren clentalis. Black pigmented dental plaque is located on the lingual surfaces of the teeth closer to the gingival margin, firmly attached to the tooth surface.

It is often found in women even with good hygiene. It is formed by Bacteroides melaninogenicus and other chromogenic bacteria. Accretions on the teeth due to using tobacco look like dark brown accretions, products of tobacco smoke, firmly attached to the enamel cuticle and are difficult to remove. Accretions due to chewing betel look like bright black pigmented accretions. Other extensive soft accretions (soft dental plaque) are accumulations microbes, their metabolic products, epithelium, proteins, lipids, saliva and etc. Supragingival calculus is a soft substance, calcified by saliva minerals. Subgingival calculus is a consequence of long-term dental plaque accumulation, resulting in gum hyperplasia or recession.

Stage II. Anesthesia. Dentists apply local or sometimes general anesthesia for removing dental plaque.

Stage III. Preoperative treatment. Antiseptic debridment of the gingival margin and the precervical area of the teeth with non-irritating medicines using an injector or sprayer.

Stage IV. Scaling and root planing. Mechanical dental plaque removal. Scaling is a scraping of the dental plaque, its waste products from the surface of the dental crown and root of the tooth. Root planing is a smoothing of the root surface in the area of dental cementum and dentine. Physical method of dental plaque removal. Dental plaque can be removed with ultrasonic devices. A chemical method of dental plaque removal. The chemical method allows to remove the pigmented dental plaque. For this purpose, the dentists use low concentrations of sulfuric, trichloroacetic, lactic, ascorbic acids, etc.

Stage V. Polishing of the tooth surfaces (crowns and roots). After removal of calculus (dental scale), the dentist should polish hard dental tissues and fillings with abrasive polishing pastes.

Stage VI. Fluoridation and covering the teeth with medicines to prevent sensitivity. Polished surfaces of the crown, cervical part of the tooth and root of the tooth are to be coated with fluoride varnishes or special solutions. The effectiveness criteria of the preliminary stage activities. After the preparatory activities the dentist must assess the state of oral hygiene, periodontal pockets and the microcirculation of the periodontal tissues.

Question 3. The main features of carious cavities preparation for dental tissues restoration in patients with periodontal diseases.

The choice of dental filling materials for dental restoration in patients with periodontal diseases. The choice of the type of dental filling materials in patients with periodontal diseases (especially when the dental filling material is in contact with the gingiva) should be carried out taking into account the oral hygiene level, the microflora nature and the severity of the disease. All dental filling materials are acceptable in case of chronic gingivitis and chronic periodontitis of mild severity, however, the preference should be given to dental amalgam and composite materials. The use of microfilling and hybrid composite filling materials is justified in case of moderate and severe chronic periodontitis. Characteristics of the stages of carious cavities preparation in patients with periodontal diseases. Class II and Class V caries usually occurs with the damage of the tooth crown and root areas in patients with periodontal diseases. The stages of cavity preparation in these cases conform to the common principles of preparation of dental tissues carious defects. Classical preparation of proximal defects using the elongated bur implicates the opening of the decay cavity (opening of the proximal contact), removal of overhanging margins, necrectomy. The weakened surfaces of the tooth should be removed not less than 2 mm high. Enamel margins are to be beveled and smoothed. Medical treatment of cavities should be undertaken

after the stages of preparation. It is recommended to use 0.5–2 % aqueous chlorhexidine solution (for example «Consepsis») for this purpose. Features of the operative area isolation during the teeth restoration in patients with periodontal diseases. Rubber Dam should be used to isolate the dental operative area in patients with periodontal diseases. It is necessary to place a clamp on the tooth, which retracts its gum and allows the dentist to visualize clearly the borders of the carious lesion. The dentist can use the retraction cord for this purpose as well, if you work without Rubber Dam. The retraction cord is to be inserted under the gum, using a special tool (a pack) before the preparation of the cavity.

Question 4. The sequence of actions for the restoration of the contact point and the shape of the tooth crown in patients with periodontal diseases.

The restoration of the contact point and the shape of the tooth crown in patients with periodontal diseases. Dentists recommend to use hybrid, microhybrid (traditional and condensible), nanofilled composite materials, as well as organically modified ceramics for restoration of proximal cavities, which extend to a contact point and occlusal surface. The restoration of proximal defects in patients with periodontal diseases requires application of extra long contouring metal, plastic or combined matrix, matrix holders, rings, fixators and wedges. However, it should be remembered that none of the matrix systems provide a tight adaptation over the tooth surface and prevent excessive insertion of filling material. The filling of the cavity, located near the gingiva, is conducted using active or passive methods. Dentists use special tools (for example, «Optra Contact») or prepolymerized composite inlays to restore the contact points of large decay cavities. Finishing dental restorations in patients with periodontal diseases. The stage of grinding and polishing of the restorations is extremely important, as well-finished restorations cannot create conditions for accumulation of food debris and adhesion of pathogenic microorganisms. A smooth surface of restorations is comfortable for the patient’s self-care. For these purposes the following instruments should be applied: the finishing knives, plastic discs, diamond burs, 12–30-edged tungsten carbide burs, ceramic composite tools of aluminium abrasive grain, rubbers and silicone polishers, brushes, felt polishers and polishing pastes of different abrasivity. You should choose burs with small working parts and secure end for the removal of overhanging margins of restoration while working in the subgingival area. Dentists apply metal and plastic strips with different dispersion, special oscillating tools (for example, «Profin Lamineer», «Intensive» files) for shaping, grinding and polishing the proximal surfaces of the restoration. It is important to polish occlusal and proximal surfaces, contact points, as well as to create the correct boundary of buccal and oral surfaces when finishing the restoration. If the

bending of the surface is insufficient, the food bolus will cause injury to the marginal gingiva. If the bending of the surface is excessive, there is a difficulty in the self-purification of the tooth. The distance between the maximum point of bending of the vestibule-oral surfaces and the epithelial attachment point is 0.75 mm from the lingual surface and 0.50 mm from the vestibular surface in the lower premolars and 1.00 mm in the lower molars.

Efficiency criteria of the tooth restoration quality in patients with periodontal diseases. Evaluation of the restoration quality effectiveness in patients with periodontal diseases is carried out directly after the treatment, shortly after the treatment and when the patient is followed-up. Quality indicators of dental restorations are: absence of patient's complaints and the highest «A» grades, according to the USPHS criteria: anatomical form, marginal fit, marginal staining, surface and colors matching. Dentists determine the presence/absence of a contact point, the overhanging margins of the filling on the proximal surfaces and in the subgingival area by means of probing, using dental floss and the radiographic method. Absence of inflammation of the interdental gingiva in the area of the restored teeth contact surfaces is determined clinically.

Test control

1. What is the value of OHI-S index (J. C. Green, J. R. Vermillion, 1964) that proves the effectiveness of hygienic measures?

a) 0,7–1,6;
b) 0,8–1,1;
c) 0–0,6;
d) 0,1–2,1.

2. What is the value of GI index (H. Loe, J. Silness, 1963) that proves the effectiveness of the initial treatment stage?

a) 1,1–2,0;
b) 0,2–0,8;
c) 1,3–1,4;
d) 2,1–3,0.

3. Indicate the contraindication for ultrasonic removal of dental calculus:

a) gastrointestinal tract disease;
b) locomotor system disease;
c) blood disorders;
d) hormonal disease.

4. What instrument is used for root planing?

a) hand dental instruments;

b) piezoelectric scaler;
c) magnetostrictive scaler;
d) sound scaler.

5. What is the advantage of dental calculus ultrasonic removing in comparison with the manual method?

a) quick removal of dental calculus, minimal discomfort for the patient, minimal soft tissue trauma;
b) high quality, smooth surface of the tooth;
c) effect of tooth whitening;
d) abscence of bleeding.

6. When is the ultrasonic scaler used?

a) for scaling;
b) for curettage;
c) for gingival plastic surgery;
d) for correction of filling overhanging margins.

7. Indicate the correct sequence of events at the initial stage of periodontal diseases complex treatment:

a) iatrogenic factors elimination in the oral cavity;
b) dental plaque removal up to the OHI-S = 0.5–0.6 with a thorough motivation of the patient about the need for these activities;
c) control of dental plaque growth;
d) oral hygiene training.

8. What does the initial therapy of patients with periodontal diseases include?

a) emergency care;
b) carrying out professional oral hygiene;
c) correction of fillings and dentures;
d) the final restoration of the dentition integrity.

9. What is conducted at the preliminary stage of the periodontal diseases treatment?

a) periodontal abscess treatment;
b) motivation;
c) professional hygiene;
d) gingival plastic surgery.

10. What are the characteristics of the temporary dental splint?

a) it does not injure the soft tissues;
b) it does not create areas for dental plaque accumulation;

c) it is firmly fixed to teeth;
d) it finally restores the dentition integrity.

LESSON 13. COMPLEX TREATMENT OF PERIODONTAL DISEASE: DRUG THERAPY AND PERIODONTAL PLASTIC ISSUES INCORPORATED. ORGANIZATION OF ACTIVITIES FOR THE PREVENTION OF PERIODONTAL DISEASE.

The questions to be studied for the learning of the topic:

1. Management of periodontal diseases
2. The antimicrobial approach
3. Mouthrinses and dentifrices
4. Locally applied antimicrobials
5. Systemic antimicrobials
6. Periodontal maintenance therapy

Question 1. Management of periodontal diseases

Periodontal management includes a complete assessment of each individual patient. Medical and dental history, clinical and radiographic examination, as well as assessment of risk factors, are all important to making an accurate diagnosis, prognosis and developing an optimal treatment plan. There are many treatment options available for the management of periodontal diseases, and review of treatment outcomes or reevaluation is key to successful management and long-term maintenance. In the past, treatments that focused on reduction of the microbial load were basically the sole consideration for all periodontal therapy. Currently, due to increased knowledge of the host response, host modulation therapies have been used as adjunctive approaches to both non-surgical and surgical treatments to aid in reducing probing depths, increasing clinical attachment levels and in regeneration of the lost attachment apparatus. It is likely in the future that more effective therapeutic approaches will include multiple, synergistic host modulation therapies combined with treatments that target the microbial etiology.

In addition to reducing the bacterial challenge and modulating the host response, reduction of risk is also a key treatment strategy when managing periodontitis. For example, it is known that smoking can contribute to the development of periodontal disease and make the management of the disease more difficult therefore smoking cessation would benefit all patients with periodontitis. Smoking cessation can be undertaken in the dental office (if staff is appropriately trained) or in a medical setting. There are a variety of medications to aid with smoking cessation, counseling is important as well and alternative medicine such as acupuncture may be used. Systemic diseases such as diabetes will increase patients risk for periodontitis when poorly controlled. When treating people with diabetes knowing the patients level of diabetic control is important to assessing risk and collaborating with medical colleagues to improve on control of diabetes is essential to assure successful

periodontal treatment. Periodontitis is also prevalent in CVD patients and periodontal therapy may have a positive impact on the overall health status of these individuals.

The management of patients with periodontitis can therefore involve the following complementary treatment strategies:

1. Patient education, including oral hygiene instruction and explanation of the rationale for any adjunctive treatments.
2. Risk factor modification and risk reduction.
3. Reduction of the bacterial burden by traditional S&RP
4. Intensive periodontal treatment with local delivery systems or general antimicrobial therapy with oral administration of antibiotics.
5. Host modulation therapy
6. Periodontal surgery.

It is the responsibility of the dentist to provide appropriate treatments on an individual basis. A combination of treatment approaches (discussed below) for each patient as listed in Figure 3 will provide the ultimate periodontal treatment and result in a better prognosis.

Question 2. The antimicrobial approach

Traditional periodontal therapy based on the antimicrobial approach consists of mechanical non-surgical and surgical therapies which may or may not be supplemented by local antiseptics and/or local or systemic antibiotics.

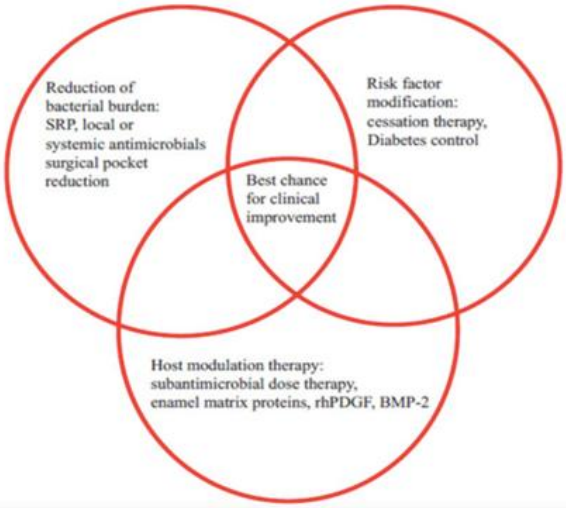


Figure 46. Complementary treatment strategies in periodontitis
Adapted from: Carranza's Clinical Periodontology, 10th Ed. WB Saunders Company; 2006:813–827.41 Reproduced with permission.

Question 3. Mouthrinses and dentifrices

Antiseptic mouthrinses have been used to reduce plaque levels and gingivitis. Two clinically proven ADA-accepted antiseptic mouthrinses are Peridex (chlorhexidine gluconate) and the four-essential oils in Listerine. An

association between oral conditions such as periodontal disease and several respiratory conditions such as pneumonia and chronic obstructive pulmonary disease has been noted. The plaque surrounding the teeth is an excellent harbor for respiratory pathogens. Studies have shown that using a chlorhexidine oral rinse can reduce the risk of pneumonia in institutionalized patients with poor oral hygiene.

Locally Applied Antiseptics. Periochip contains the active ingredient of chlorhexidine gluconate (2.5 mg) that is released into the pocket over a period of 7 to 10 days. It has been found to suppress the bacteria in the pocket for up to 11 weeks post application.46 Periochip is the only FDA approved locally applied antimicrobial can be used as an adjunct to SRP procedures aid in the reduction of pocket depths. Other locally applied antimicrobials are antibiotics.

Dentifrices. Major improvements in the oral health of populations in developed countries have been seen over the last 50 years. Most of this resulted from the reduction in the caries rate of about 50%, and the principle reason for this is thought to be the addition of fluoride to dentifrices.

Modern, commercially available dentifrices, in addition to providing anti-caries effects of fluoride, also contribute to the reduction of plaque, gingivitis, calculus formation, relief of dentin hypersenitivity, and tooth stain. They also reduce halitosis and result in a clean, fresh mouth feel. Two dentifrices available in the US that are approved by the FDA for their effects on reduction of gingivitis include a stannous fluoride/sodium hexametaphosphate dentifrice and a triclosan/copolymer/sodium fluoride dentifrice.

There is a large amount of literature on these and other dentifrices containing chlor -hexidine and other agents in the control of gingivitis. A review of the clinical efficacy and safety of a triclosan/copolymer/sodium fluoride dentifrice was carried out by Blinkhorn and colleagues. They found about 200 articles dating from 1998 to 2008 relating to this dentifrice and concluded that twice daily use of this dentifrice will result in clinically significant improvement in plaque control and gingivitis and slower progression of periodontal disease. Further long-term studies extending over several years with these dentifrices are needed to establish whether or not short-term effects seen will be sustained over the long term, and indeed result in preventing the initiation of periodontitis and slowing the progression of already existing periodontitis.

It should be noted that the antiplaque and antigingivitis effects of dentifrices during a tooth brushing regimen are mainly on the occlusal and smooth surfaces of the teeth, and that interproximal plaque and gingivitis control is not optimally reduced with tooth brushing alone, with or without a dentifrice. Interproximal aids such as flossing, interproximal brushing, and to some extent, flushing with effective mouthrinses is often needed for full

plaque control on interproximal surfaces of the teeth. As periodontal disease is often initiated and progresses more rapidly in interproximal spaces, it is clear that interproximal cleansing is an important adjunct to toothbrushing with dentifrices.

Question 4. Locally applied antimicrobials

Atridox is a FDA-approved locally delivered tetracycline system. It comes with a 10% formulation of doxycycline in a bioabsorbable, ‘‘flowable’’ poly-DLlactide and N-methyl-2-pyrrolidone mixture delivery system that allows for controlled release over 7 days. This system is applied subgingivally to the base of the pocket through a cannula. Atridox is a resorbable site-specific locally applied antibiotic proven to promote clinical attachment gains and reduce pocket depths, bleeding on probing, and levels of pathogenic bacteria for up to 6 months post- placement.35 Peridontal disease has been linked to systemic diseases such as diabetes. Research has shown that periodontal treatment with topically delivered doxycycline 10 mg in periodontal pockets produced favorable clinical results in diabetic patients.

Arestin is a FDA approved minocycline microsphere system that is bioadhesive and bioresorbable, allowing for sustained release of 1 mg of minocycline. Arestin can be used as an adjunct to SRP procedures for reduction of pocket depth in patients with adult periodontitis. Arestin is delivered to sites of 5 mm or greater. Periodontitis has been associated with increased systemic inflammation, which is directly linked to diabetes and cardiovascular diseases. Recent research has shown that periodontal therapy with local arestin administration resulted in decreased HbA1c levels in diabetic subjects48 and significant reductions in systemic inflammatory biomarkers which are risk factors for CVD.

Question 5. Systemic antimicrobials

Systemic antimicrobial therapy is usually reserved for advanced cases of periodontitis:

1) for sites that have not responded to treatment, so-called “refractory periodontitis”

2) for patients demonstrating progressive periodontal destruction.

Systemic antibiotics can be used as adjuncts to conventional mechanical therapy, but strong evidence for their use as a monotherapy has not been developed. For these special situations, randomized double-blinded clinical trials and longitudinal assessments of patients indicate that systemic antimicrobials may be useful in slowing disease progression. Metronidazole can be used to cure acute necrotizing ulcerative gingivitis, and metronidazole amoxicillin combination therapy can be used to treat aggressive adolescent periodontitis associated with Aggregatibacter actinomycetemcomitans. Systemic antibiotic therapy has the advantage of simple, easy administration

of drugs to multiple periodontal sites. However, patient compliance needs to be considered, inability to achieve adequate concentrations at the site of infection, adverse drug reactions, and the development of antibiotic-resistant can be issues. Common antibiotic therapies for the treatment of periodontitis include metronidazole, clindamycin, doxycycline or minocycline, ciprofloxacin, azithromycin, metronidazole and amoxicillin, and metronidazole and ciprofloxacin. For adult patients with acute periodontal abscesses, amoxicillin is used as an adjunct to incision and drainage. For patients with allergies to β-lactam drugs, azithromycin or clindamycin would be the choice.

Question 6. Periodontal maintenance therapy

Upon completion of active periodontal therapy, periodontal maintenance visits should include:

1. Update of medical and dental histories.
2. Evaluation of current extra- and intraoral periodontal and peri-implant soft tissues as well as dental hard tissues and referral when indicated (e.g., for treatment of carious lesions, pulpal pathoses, or other conditions) and diagnostic-quality radiographs when appropriate.
3. Assessment of the oral hygiene status with reinstruction when indicated.
4. Mechanical tooth cleaning to disrupt/remove dental plaque, biofilms, stain, and calculus. Local delivery or systemic chemotherapeutic agents may be used as adjunctive treatment for recurrent or refractory disease.
5. Ongoing assessment of risk factors to identify an individual who may be more highly susceptible to ongoing breakdown of the periodontal or peri-implant tissues, with elimination or mitigation of new or persistent risk and etiologic factors with appropriate treatment.
6. Identification and treatment of new, recurrent, or refractory areas of periodontal and peri-implant pathoses.
7. Establishment of an appropriate interval for periodontal maintenance. The patient should be kept informed of:
   - ✓ Areas of persistent, recurrent, refractory, or newly occurring periodontal or peri-implant disease.
   - ✓ Changes in the periodontal prognosis and risk factors associated with periodontal diseases.
   - ✓ Advisability of further periodontal treatment or retreatment of indicated sites.
   - ✓ Status of dental implants.
   - ✓ Other oral health problems that may include caries, defective restorations, and non-periodontal mucosal diseases or conditions.
   - ✓ Changes that would warrant referral to, or consultation with, other dental or medical specialists.

Test control

1. The objectives of the complex therapy of periodontal disease are:

a) increase in the therapeutic effect of the various methods, techniques, manipulation, effects of drugs;
b) increase the probability of the therapeutic effect of incomplete etiological and pathogenetic diagnosis
c) all of the above.

2. Which drug is most effective mouthwash in the treatment of periodontal disease?

a) potassium permanganate solution 1 : 5000;
b) 1% hydrogen peroxide -s;
c) chlorhexidine 2%;
d) iodinol.

3. The concentration of fluorine salt in the food should be:

a) at least 250 mg / kg;
b) at least 150 mg / kg;
c) at least 50 mg / kg.

4. Adult recommended paste containing the active fluorine concentration:

a) 500-1500 ppm (0,05-0,15%);
b) 500 ppm (0,05%);
c) 2000 ppm (0,2%).

5. In the human diet should be the products of the following major groups:

a) bread, milk, meat, fruit and vegetables;
b) milk, meat, starchy, fruit and vegetables;
c) carbohydrates, dairy, meat, fruit and vegetables.

6. How many times a day is recommended to eat:

a) not more than five times;
b) no more than 3 times;
c) no more than 7 times.

7. The complex methods of prevention include:

a) oral hygiene;
b) the use of fluoride;
c) a balanced diet;
d) all of the above is true.

8. The objectives of primary prevention are:

a) maintaining health, prevent impacts on human factors, natural and social environment
b) detection of diseases, the prevention of the disease process and its complications;
c) identification of a severe form of the disease, prevention of complications, decrease disability, mortality.

9. The main purpose of professional oral hygiene is

a) eliminate the influence on the periodontal tissue of microbial factors local measures;
b) removal of tooth deposits;
c) training of oral hygiene.

10. Assessment of the effectiveness of preventive measures based on the following principles:

a) survey at the beginning and end of the program the same age groups;
b) examination should be carried out a good team of experts;
c) must be used the same codes;
d) all of the above.

8 SEMESTER

LESSON 1. EXTERNAL RESORPTION OF TOOTH ROOT: CLINIC, DIAGNOSIS AND TREATMENT PLAN

The questions to be studied for the learning of the topic:

1. The external resorption of the tooth root. Etiology and pathogenesis.
2. The external resorption of the tooth root. Classification.
3. The external resorption of the tooth root. The clinical picture.
4. The external resorption of the tooth root. Diagnostics, differential diagnostics.
5. External resorption of the tooth root. The treatment plan.

Introduction. Resorption (Latin resorbeo -. Absorb) means absorption. Root resorption is a process during which there is decrease of root dentin, cement and bone surrounding the tooth. Resorption may be physiological and pathological (Fig. 47).

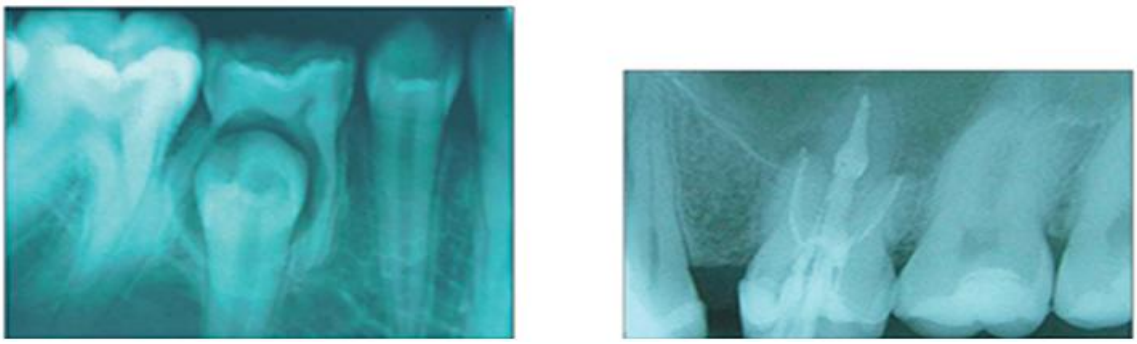


Figure 47. Physiological and pathological resorption

Question 1. External resorption of the tooth root. Etiology and pathogenesis.

The physiological root resorption of deciduous teeth is observed between the change of teeth. Root resorption of permanent teeth is pathological, because normally mineralized tissue formed teeth should not be subject to resorption. To understand the mechanism of bone resorption is necessary to know that the root of the tooth dentin from the root canal and protected predentin layer of odontoblasts, and from the outer surface of the root - cement and cementoblasts. In cases of violation of predentin and cement or as a result of mechanical damage to the exposed dentine surface accumulations occur osteoclasts, which cause bone resorption. Resorption of the basic substance of bone and cartilage calcification occurs by allocating osteoclasts hydrolytic enzymes. However, to activate the bone resorption and maintain the phagocytosis process, processes the cells require constant stimulation and if the stimulation of the part of the exposed dentin or cement

is not strong enough for long-term resorption, the root resorption will be of a transitory character, at the same time regeneration of tissue to form cement-tissue will be observed both the inner and the outer surface of the root. Resorption in this case is called transient.

External tooth resorption always starts with the roots and comes from periodontal. Such resorption may be due to iatrogenic cause (such as orthodontic treatment, tooth overload carrying bridge). External resorption occurs as after injuries (for example, when a tooth subluxation) and because of the pressure properly erupting adjacent teeth. Resorption is often observed in ankylosis teeth. Root resorption are also a consequence of chronic periapical long process. Biological mediators (metalloproteinase antibody concentration IG> 5 times, cytokines, prostaglandins, endotoxins, neutrophils). They activate autoimmune reaction dentin odontoclast inhibit calcitonin and support the inflammatory process. Statistically, 87.3% in the external inflammatory resorption occurs around the apical foramen.

If the collection of history you can not specify the reason resorption, talk about the pathological resorption. In these cases, we should not forget about the existence of hereditary syndromes hypoplastic dentin by type Kapdepona syndrome. Picture radicular dentin hypoplasia also occurs as a consequence of insufficient education dentin.

Resorption may occur in case of damage the protective tissue (mechanical or chemical) and irritation due to infection or compression. Damage (eg, 30% hydrogen peroxide, used for whitening teeth) affects non-mineralized tissue, are to cement and dentin. To the naked mineralized tissue attached multinucleated cells that initiate the resorption. Without further stimulation of osteoclasts process gradually subsides and within two or three weeks, there is a restoration cement-tissue.

Question 2. External resorption of the tooth root. Classification.

Currently, there are several classifications to indicate the root resorption.

<u>According to ICD-10 are distinguished:</u>

K03.3. Pathological resorption of teeth

K 03.30 - the outer (external);

K 03.31 - inside [the inner granuloma] [pink spot];

K 03.39 - pathological resorption of teeth, unspecified;

K 03.4 – hypercementosis;

K 03.5 - ankylosis.

Zwi Fus, Igor Cesis, Shaul Lin, 2009 (Department of Endodontics Dentistry Faculty. Maurice and Gabriela Goldshleger Tel Aviv University, Israel) proposed a ***clinical classification of root resorption***, which is widely used by practicing dentists. According to them, the etiology of root resorption

has two components: the damage and irritation. Based on this, root resorption caused by:

1) pulp infection,
2) periodontal infection,
3) notphysiological squeezing during orthodontic treatment,
4) pressure teeth or tumors,
5) ankylosis (still occurring as a result of the formation of bone, cartilage or fibrous adhesions).

The same damage may lead to the development of various forms of root resorption.

Classification Robert J. Kanas and Scott J. Kanas (2012) is based on etiology and morphological characteristics of root resorption. There are odontogenic and notodontogenic resorption, depending on the localization process - external and internal. The authors identify the following types of root resorption:

Transient resorption occurs after a trauma, orthodontic and periodontal treatment. This type of resorption is reversible, since after elimination of etiological factor resorptive process is terminated.

Apical inflammatory resorption can be caused by pulpitis or trauma (intrusion or avulsion), resulting in the pathological process develops in the periapical region. The process usually takes a long time and are asymptomatic. Sometimes there is a positive percussion.

Cervical inflammatory resorption. Lesions of the tooth root in the cervical area can develop both above and below the level of the bone tissue of the alveolar bone. Among the causes of isolated apical periodontitis, trauma, teeth whitening, orthodontic treatment, bruxism, dental hard tissue disease or idiopathic processes. Asymptomatic, discovered by accident during a radiographic examination. Pulp vitality tests is positive.

Resorption as a result of tumors or maxillary cysts. It may be caused by odontogenic keratcyst, ameloblastoma, giant cell reparative granuloma, cementoblastoma, osteoblastoma, osteosarcoma and chondrosarcoma.

Induced systemic disorders. Among the reasons for such release system deviation as endocrine pathology (hyperparathyroidism, diabetes mellitus), genetic disorders and hereditary diseases (Gaucher's disease, hypophosphatasia, Papillon-Lefevre syndrome, Gorlin-Goltz syndrome), kidney and liver diseases, disorders of bone formation processes. For the diagnosis it is necessary to analyze the data of biochemical blood tests, urine analysis, data and other genetic studies.

External root. The appearance of external root resorption usually occurs at the site of the periodontal ligament. Osteoclasts calcify root surface and phagocytose its component parts. As a result of resorption lacunae formed, which can then be replenished secondary cement. Through this mechanism, the presence of small-sized gaps possible to restore the original

anatomical shape of the tooth. Radiological gaps root surface are not detected. In inflammatory diseases of the periodontal gap can become infected, leading to a recurrence of inflammation. External resorption can be detected radiologically only when it reaches a certain size: a diameter of at least 2 mm and a depth of at least 1 mm.

There are four types of external resorption (Andreassen (1988)):

1. Surface;
2. The flat on the lateral and / or apical root surface;
3. Deep replacement form with anchylosis;
4. External granuloma.

Question 3. External resorption of the tooth root. Clinical presentations.

The ones of the fairly common outer tooth resorption are apical, lateral and cervical resorption.

Apical resorption occurs in the form of periapical inflammatory processes, the implementation of measures and orthodontic treatment as idiopathic process (mainly in multi-rooted teeth).

The reasons are often rapidly progressive resorption cup shape, caused by inflammation are severe sprains, replantation and transplantation, followed by inflammation in the periapical region, as well as primary periapical inflammation (fig. 48).

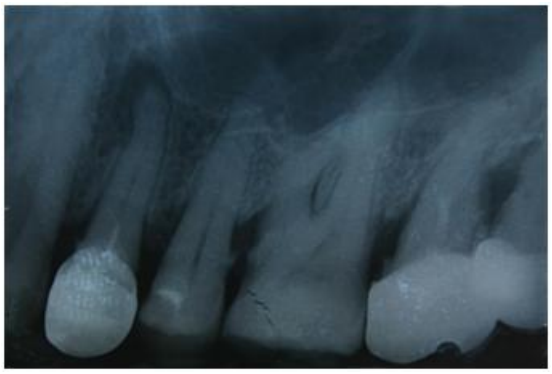


Figure 48. Periapical inflammation.

Thus there is also an intensification of resorption processes. Often resorption lacunae and bone defects filled with granulation tissue. After a few months the complete dissolution of the tooth root can happen.

External granuloma refers to infrequent peripheral circumpulpar resorption process of unknown etiology.

Cervical resorption is resorption in the cervical part of the root, may also occur after orthodontic tooth movement, operational impacts on the jaws and alveolar process, teeth whitening and many other traumatic manipulations. As if under the influence of damaging factors include developing a local necrosis of the periodontal ligament, cervical resorption may take the form of replacement resorption or anchylosis. As the name

implies, cervical resorption begins in the cervical portion of the periodontal epithelial attachment below. In this zone of damage to the root surface, which begins resorption can be very small. A small area of exposed tissue penetration occurs resorbing cells in the root dentin. First resorption center does not reach the pulp chamber and extends along the root surface due to the pronounced protective predentin properties. Due to the similar nature of the spread of root resorption inside wall cervical resorption is also often referred to as external-internal or invasive.

Eventually resorption hearth reaches the root canal. Furthermore, cervical resorption can capture alveolar bone near the resorption lacunae. In connection with this X-ray pattern may resemble a periodontal pathology with the formation of periodontal pockets (fig. 49).

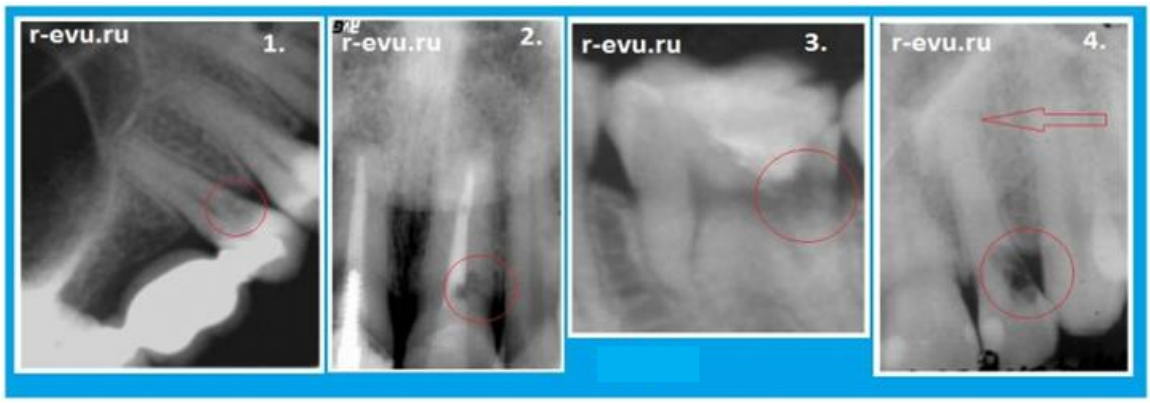


Figure 49. Resorption on the radiograph.

Lateral form of resorption is partially or completely reversible. It is common among patients with acute periodontal inflammation, combined with a rapid deterioration of bone tissue. Call lateral resorption may also be injuries (sprains) and replantation of teeth.

Question 4. External resorption of the tooth root. Diagnostics.

Sometimes very difficult to determine the true etiology of this process, but, nevertheless, only a complete diagnostic study allows an accurate diagnosis and determine the correct treatment strategy.

Use the following diagnostic methods:

- ***subjective*** (collection of complaints, anamnesis)
- **objective**
  - Basic - inspection, sensing, percussion, palpation, thermodiagnostics (to assess the viability of the pulp).
  - Extra:

1. Periodontology examination uses a periodontal probe determination of the depth of the teeth-gingival pocket, the presence of subgingival dental plaque, root roughness
2. Electroodontodiagnosis (to assess viability pulp)

3. X-ray study to determine the size of periapical changes or presence of periodontal pathology marginal:

- *intraoral X-ray*
- *panoramic radiography*
- *orthopantomography*
- *cone-beam computed tomography (CBCT)*

Question 5. External resorption of the tooth root. Plan of treatment.

There are three main areas of choice of tactics of treatment of the inside of the tooth root resorption.

- conservative endodontic treatment;
- recalcification or apexification using calcium hydroxide and hydroxyapatite;
- surgery.

The choice of approach is determined by the ability to comply with physician triad requirements for endodontic treatment (sterilization, cleaning and obturation of the root canal), and the prevalence and location of the defect.

If the requirements can be met and focus resorption not perforates the wall of the channel, used conservative endodontic treatment materials based on mineral trioxide aggregate (MTA) "Trioxident" (Vladmiva, Russia), "Rootsil" (Belarus), «MTA ProRoot» (Dentsply, USA).

If three major endodontic principle can be met, but there is a perforation of the canal wall, not communicating with the oral cavity, it is necessary to use a method of temporarily filling channel (apexification) materials based on hydroxyapatite or calcium hydroxide.

Preparations based on calcium hydroxide in the treatment of tooth root resorption

Many authors recognize the need for these drugs as a temporary root fillings at different times with the purpose of prolonged antiseptic effect on the periapical tissues and the walls of the root canal, as well as to create conditions for the effective recovery of bone tissue in long-term period, which is especially important when resorption processes. Calcium-containing preparations are presented materials for temporary root canal obturation "Calasept» (Nordiska dental - Sweden (sterile pure calcium hydroxide)), "Calcicur" (Voco - Germany), "Metapasta" (Unident - South Korea), "Calasept" (Omega - Russian Federation), and for the constant «Vitapex» (Neo Dental Chemical-Japan), «Acroseal» (Septodont - France), "Akros" (Dentsply - Usa), "Mepasil" and «Ozomol-4» (Pierre Rolland Acteon Group - France) and are available in the form of paste or powder / liquid.

Preparations based on hydroxyapatite
in the treatment of root resorption of teeth

The creation of a depot in a bone defect from osteoregenerating materials is of great importance for the stimulation of osteogenesis. In dental practice, the use of such drugs is widespread. The most popular received alloplastic (biocomposite) of calcium hydroxyapatite materials, physicochemical characteristics of which (crystal size, density, collagen content) determine osteoplastic properties. Materials based on hydroxyapatite are available in different forms: gel, beads, plates, membranes and also worked well: "Hydroxyapol", "Collapan", "Trapex-Gel", "Indost" and other production Research and Production Company "Polystom" (Russian Federation) and Belarusian counterpart - "Hydroxyapatite gel" production "Belmedpreparaty" (Republic of Belarus). After entering into the bone defect hydroxyapatite crystals are metabolized to calcium and phosphorus ions, keep the wound blood clot due to isomorphic substitution of calcium in the water, and a hydrogen ion. The action mechanism of hydroxyapatite in the wound: activates the differentiation of osteogenic cells; forms a strong chemical bond with bone, showing high bioinertness (inflammatory reaction, systemic and local toxicity are practically absent).

The hydroxyapatite resorption takes place without the formation of a fibrous capsule with the occurrence of "point" osteogenesis in the bone defect. Hydroxyapatite-based drugs are not included in the clinical protocols of endodontic treatment of dental diseases in the Republic of Belarus.

A surgical treatment is necessary in case of extensive destruction of the root, prolonged bleeding or perforation associated with the oral cavity. This method includes:

- ✓ Resection of the tooth root apex,
- ✓ Coronal radicular separation,
- ✓ Root amputation,
- ✓ In some cases, tooth removal.

Test control

1. Resorption is a pathological process arising when:

a) Mechanical damage of protective fabrics
b) Chemical damage of protective fabrics;
c) Irritated due to infection;
d) Stimulation due to compression;
e) For no apparent reason.

2. The main types of bone resorption are:

a) Enamel resorption;
b) Physiological;
c) Resorption of dentine;

d) Pathological;
e) Cement resorption.

3. From undifferentiated reserve cells of the connective tissue during internal root resorption in the pulp are formed:

a) Odontoblasts;
b) Odontoclast
c) Cementocytes;
d) Leukocytes;
e) Dentinal tubules.

4. Root resorption of permanent teeth has the following type:

a) Species;
b) Pathological;
c) Population;
d) System;

5. As a filling material during endodontic treatment of tooth root resorption is used:

a) Materials based on mineral trioxide aggregate;
b) The paste of calcium hydroxide;
c) The paste based on zinc oxide eugenol;
d) The glass ionomer cements;
e) The paste of the epoxy resin.

6. The dentin of the tooth root by root canal is protected:

a) Predentin;
b) Odontoclast layer;
c) Layer of odontoblasts
d) Cementoblasts layer;
e) Cement.

7. The dentin of the tooth root from the outer surface of the root is protected by:

a) Cement
b) Predentin;
c) Odontoclast layer;
d) A layer of odontoblasts;
e) Cementoblasts layer.

8. The following treatments are applied at the internal root resorption:

a) Conservative endodontic treatment;
b) Antiviral treatment;

c) Recalcification with calcium hydroxide;
d) Treatment with antifungal drugs;
e) The surgical treatment.

9. According to the ICD-10, tooth resorption is distinguished:

a) Abnormal external (outer)
b) The pulp;
c) The internal [internal granuloma] [pink spot];
d) Periodontal;
e) Unspecified.

10. Complication of internal resorption is:

a) Perforation of the root surface;
b) Increased tooth sensitivity;
c) Perforation of the crown surface;
d) Trigeminal neuralgia;
e) Crowding teeth.

LESSON 2. ENDOPERIODONTITIS: CLASSIFICATION CHARACTERISTIC, CLINIC, DIAGNOSIS AND TREATMENT METHODS.

The questions to be studied for the learning of the topic:

1. Etiology and pathogenesis endoperiodontal lesions.
2. Endoperiodontitis. Classification.
3. The clinical manifestations endoperiodontal lesions.
4. Endoperiodontitis. Diagnostics.
5. The treatment of patients with lesions endoperiodontal.
6. Evaluation of the effectiveness of treatment endoperiodontal pathology.

Question 1. Etiology and pathogenesis endoperiodontal lesion.

Endoperiodontology is learning processes within and surrounding tissues of the tooth. In this connection it is necessary to take into account the state of the pulp and its influence on periodontal ligament and alveolar bone, as well as the status of periodontal tissue and their effects on tooth pulp and pathways dental pulp relationship with periodontitis: apical foramen, lateral canals, the dentinal tubules.

Causes of endo-periodontal disease:

1) 1.anatomo-topographical and functional integrity of the periodontal tissues,
2) anatomical structural features of the root canal,
3) integrity t mechanisms for violations of the microbial biocenosis
4) pathogenetic particular microflora in endodontic pathology.

Clinically this can be caused by:

1) necrosis drainage through the pulp with periodontal ligament (usually with accompanying periodontal disease);
2) toxins from entering through the side and additional tubules, especially in furcation;
3) perforation tool or endodontic root pin.

Adverse factors leading to endoperiodontal lesions include endodontic side channels; poor quality endodontic treatment; a significant loss of the periodontal apparatus; the presence of periodontal pockets; bad oral hygiene; occlusal trauma; furcation involvement and acute periodontal abscess. At the same root resorption and incorrect procedure «root planning» during the removal of dental plaque increases the adverse effect of conductive paths through the dentinal tubules. In addition, the pathological process in periodontal tissues with deep periodontal pockets, suppuration, abnormal mobility of the tooth and alveolar bone resorption Angular may adversely affect the state of the dental pulp.

Cases falling infection in the pulp retrograde way through lateral tooth root canals with deep bone pockets. Important it is the fact of the adverse

effects of chronic apical periodontitis the infection on periodontal tissue microcirculation.

Pathogenesis. With the death of the pulp bacteria spread through the system of root-canal: makrokanalu, lateral and dentinal tubules penetrate into periodontal tissue, causing inflammatory changes in it. Since periodontal tissues are morphological and functional periodontal unit system, it is possible the penetration of microorganisms in the root canal system retrograde through the periodontal pocket. However, it is known that the spectrum of endodontic mixed anaerobic flora coincides with periodontal microbial landscape, which is mainly represented by the following microorganisms: Fusobacterium, Prevotella, Lac-tobacillus, Porphyromonas, Pep-tostreptococcus, Fubacterium, Capnocytophasa, Spirochetes, as well as mushrooms. In acute and chronic inflammatory process of the qualitative composition of microflora influences the nature and course of the pathological process. However, it should take into account not only the qualitative and quantitative composition of microflora, but also the body's ability to resist bacterial elements that migrate from endodont in periodontal, or neutralize them. This process can be long-term; periodontal usually responds with the initial destruction of the periodontal ligament and alveolar bone. There is a balance of processes, "endodontist-periodontal" progression-free or completed form cysts. Thus mixed microflora detected histologically, as immunokomplemental dentridov cells, macrophages, T and B-lymphocytes.

Question 2. CLASSIFICATION OF ENDOPERIODONTITIS

Table 15. Classification of endoperiodontitis (L.N. Dedova 2012).

The flowing	*The form*	*The prevalence*	*Degree of severity*
Acute	Simple	Localized	Mild
Chronic	Complex		Moderate
Exacerbation of chronic, including abscess	Symptomatic		Severe
Remission			

Classification of endoperiodontal lesions (Guldener and Langeland (1982)).

Today abroad use the classification of the endo-periodontal lesions proposed Guldener and Langeland (1982). According to this classification, there are three classes of endo-periodontal lesions.

Class I - mostly endodontic problem (a).

Class II - mostly periodontology problem (b).

Class III - combined periodontology and endodontic lesion (c).

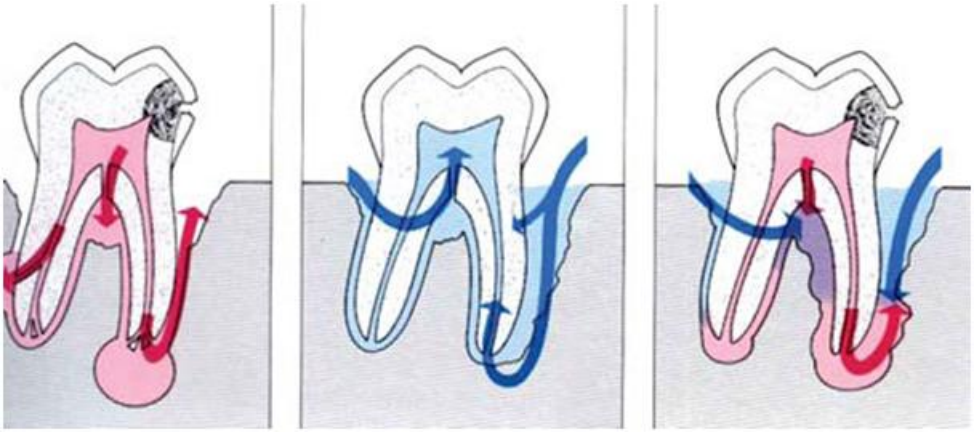


Figure 50. Endo-periodontal lesions.

Question 3. Clinical manifestations of endo-periodontal lesions.

From the etiological point of view, endoperiodontal processes develop on the basis of endodontic or periodontological pathology or their combination.

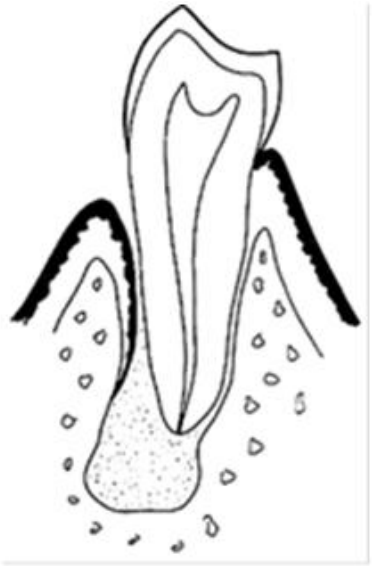


Figure 51 shows a tooth with apical periodontitis and fistulous course, opening in the periodontal pocket. The existence of fistulous led to the exposure of the root surface and the formation of periodontal pockets. In this case, the patient will need a root canal, and the treatment of periodontology.

In the second case, Pulp-periodontal lesion develops as a result of periodontal disease marginal, where the periodontal pocket extends in the direction of the root tip and causes the development of retrograde pulpitis. In these cases, you should also conduct combined edodontical and periodontical treatment.

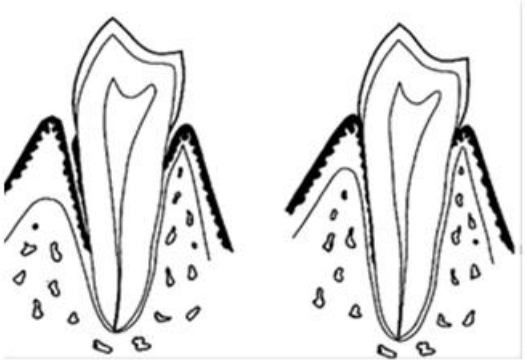


Figure 52 shows a tooth with periodontal pocket reaching apical foramen. In the case of retrograde pulpitis may need endodontic treatment. In addition to the true endo-periodontal lesions in this disease often include defects of the clinical and / or radiological picture of the combined lesions.

An example of such a situation: periapical center with fistulous course that runs along the root surface and causing radiographically visible bone resorption, similar in appearance to the bony pocket of periodontitis. However, exposure of the root and the formation of the pocket may not occur or occur only after a considerable time.

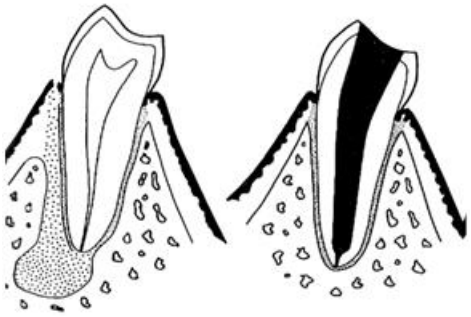


Figure 53 shows a tooth with apical periodontitis and fistulous course that runs along the root surface and the periodontal pocket through the opening. However, the root surface is still covered with cement (left). In this case, an endodontic treatment lead to full recovery with regeneration of periodontal tissue (right).

In cases where the cement on the root surface is maintained at least a very thin layer of cement and periodontal ligament, the combined loss of apical and marginal periodontium will only require endodontic treatment. Periodontology treatment thus contraindicated because curettage can destroy the protective layer on the surface of the root, which will inevitably lead to the formation of a periodontal pocket. Similarly periodontal pathology may have a clinical picture similar to endodonto-periodontal lesions due to a combination of atrophy of bone tissue in the apical and marginal periodontium. In this case, to maintain the vitality of the pulp will indicate tooth sensitivity. Endodontic treatment is not required because of its inefficiency.

Thus, the clinical symptoms of **acute endoperiodontitis** mark throbbing pain in the jaw area causal topographic, tenderness to palpation and

percussion, increased tooth mobility, swelling of the marginal gingiva. In addition, the pus periapical abscess may penetrate the periodontology pocket. In one case, purulent process penetrates into the upper jaw, sinus or nasal cavity via the periodontal ligament. Usually, this is results of the narrow passage in periodontal pocket, which can be formed from different sides of the root. Such a passage may be defined in the usual probing along the tooth root to its apex. The passage in the area of multi-rooted teeth is, as a rule, in the furcation. The destructive zone in the area of furcation (similar to destruction during periodontal disease) is detected during an X-ray examination. In another case, the contents of the periapical abscess may penetrate the cortical bone plate in the apex and find the course through the gingival sulcus without the presence of osteoporosis.

Chronic endoperiodontitis often determined by X-ray data that reflects the destructive process in the periapical and marginal parts of the alveolar bone. Form endoperiodontitis diagnose the presence of clinical symptoms and X-ray data, which mark the presence of side channels, the quality of root canal filling, the state of the root apex, the presence of furcation involvement, the level of destruction interalveolar partitions.

Simple endoperiodontitis has primary endodontic origin and complex occurs in the periodontology patients with moderate and severe degrees of severity and presence of occlusal trauma in which there is a violation of intact tooth cementum.

Symptomatic endoperiodontitis is destructive process of periodontal and endodontic patients with perforation of the tooth root, a vertical fracture of the tooth root, resorption of the tooth root and other local etiological factors. The severity of endoperiodontitis depends on the severity of clinical symptoms and the level of destruction of alveolar bone.

Question 4. Methods of diagnosis endoperiodontal lesions.

Sometimes very difficult to determine the true etiology of this process, but, nevertheless, only a complete diagnostic study allows an accurate diagnosis and determine the correct treatment strategy.

Use the following diagnostic methods:

- ***subjective*** (collection of complaints, anamnesis)
- **objective**
  - Basic - inspection, sensing, percussion, palpation, thermodiagnostics (to assess the viability of the pulp).
  - Extra:

1. Periodontology examination uses a periodontal probe determination of the depth of the teeth-gingival pocket, the presence of subgingival dental plaque, root roughness
2. Electroodontodiagnosis (to assess viability pulp)

3. X-ray study to determine the size of periapical changes or presence of periodontal pathology marginal:

- *intraoral X-ray*
- *panoramic radiography*
- *orthopantomography*
- *cone-beam computed tomography (CBCT)*

Question 5. Interview tactics of treatment of patients with endoperiodontal lesion.

It should be emphasized that the success of treatment endoperiodontal damage depends primarily on the quality of endodontic treatment, and it should be a priority. At the same time at this stage of the treatment of the pathological focus only endodontical methods impossible. It is necessary to conduct the Periodontology treatment, which leads to the elimination of the defect due to the formation reattachment periodontal tissues.

The effectiveness of treatment depends on properly selected therapeutic measures. Periodontology treatment should include professional oral hygiene, removal of subgingival dental plaque, smoothing the surface of the tooth root. Local anti-inflammatory therapy and conservative treatment of periodontal disease is aimed at eliminating periodonto pathogenic oral microflora. Thus, in planning the treatment is given priority endodontotherapy along with the control of dental plaque growth using c F, Ca, P-containing toothpastes.

Periodontal destruction in sealed root canals is considered as a result of the low quality of root fillings. It is known that in the blank spaces of the channel there is a growth of the bacterial flora pathogenicity, resulting in the infectious process by conductive paths extend in periodontium. Quality reobturation leads to stabilization and regression of pathological process Endodontic treatment should include instrumental and pharmacological treatment of root canal system. For the impact on periapical inflammatory focus, you can use a sterile suspension of hydroxyapatite or calcium hydroxide followed by permanent occlusion under fluoroscopic guidance.

Results successful treatment depends not only on the obturation channel quality, but also on the degree of pathogenicity of a bacterial infection in it. In the presence of perforations in emergency medical activities based on use of calcium hydroxide materials bactericidal treated gypsum and mineral trioxide aggregate complex MTA. If urgent measures have proved ineffective, then spend hemysection or removed (possibly implanted) tooth. It should be noted that the closer the perforation is to the root of the gingival margin, the greater the probability of proliferation of epithelial furrow to the site of perforation. Reparation after isolation of the tooth root perforation is unpredictable, so in such cases, to predict difficult. All patients it is advisable to carry out a combined endodontical and periodontological treatment.

It is advisable to renounce the use of biological method of pulpitis treatment in patients Periodontology, and in the course of complex treatment of periodontitis expand the indications for depulpation teeth with moderate and especially severe periodontal disease. Under indications of surgical treatment of periodontal disease.

Question 6. Evaluation of the treatment of pathology endoperiodontitis.

The effectiveness of the treatment is assessed by clinical and radiological data with the analysis of the following indicators:

1) the elimination of pain;
2) the absence of pain when biting (percussion);
3) the absence of changes in the transitional fold;
4) the absence of necrotic odor from the root canal;
5) the absence of suppuration from the periodontal pockets;
6) the reduction in the depth of periodontal pockets;
7) the decrease in mobility;
8) according radiovisiography:
   - degree of increase or decrease in the hearth of destruction of periapical region,
   - change the depth of the periodontal bone pockets.
   - absence of active processes and vertical lacunar resorption,
   - seal cortical plate,
   - increase in height of the alveolar bone.

Weather favorable, if the main reason for the endo-periodontal lesion is penetration of infectious agents through the root canal system is the possibility of endodontic treatment and sterilization of the root canal system. But the lesion of the combined outcome of the disease depends on the prevalence of endodontic or periodontological cause of the process. With the predominance of horizontal type of bone resorption and the possibility of adequate endodontic treatment of a tooth can function as a complete unit of the teeth-jaw system. If there is a combination of bone resorption, the success of treatment will depend on the regenerative capacity of the bone tissue.

When planning treatment of endo- periodontological l disease in the first place must take into account the patient's willingness to cooperate, to explain the benefits and possible complications that may arise during the course of treatment. Only a comprehensive approach, including endodontic, periodontological and surgical treatment aimed at eliminating the infection, will provide a stable positive result.

Test control

1. Class 2 endo-periodontal lesions according to Guldener and Langeland is characterized as:

a) endodontic problem;

b) combined periodontological and endodontic lesion;
c) periodontological problem;
d) soft tissue pathology of the maxillofacial region;
e) all of the above.

2. Causes of the endo-periodontal lesions are:

a) pulp necrosis with drainage through the periodontal ligament in the presence of concomitant diseases periodontal;
b) perforation of the root;
c) dental plaque;
d) chronic pulpitis;
e) blood disease.

3. What type of percussion is more painful in case of periodontal abscess?

a) horizontal;
b) vertical;
c) comparative;
d) all of these;
e) percussion painless.

4. Tooth pulp in case of periapical abscess is

1. non-vital;
2. vital in the crown part;
3. vital in the apical part;
4. vital of individual sites;
5. all vital.

5. Is the development of periodontal abscess possible without disorder the integrity of the crown?

1. yes
2. no

6. Select the appropriate features that characterize the clinical picture with periodontal abscess:

a) there is loss of bone tissue of the interalveolar septum
b) attached gingiva has fistula;
c) horizontal percussion is painful;
d) there are periodontal pockets;
e) all of the above

7. Select the appropriate features that characterize the clinical picture when periapical abscess:

a) there is bone destruction in the periapical region;
b) there is a loss of bone tissue of the interalveolar septum;
c) may occur in case of the intact tooth;
d) there is a deep carious cavity or filling;
e) there are always pockets;
f) the pockets are often absent.

8. The effective treatment of the endoperiodontal pathology on the radiograph can be assessed as:

a) the absence of active processes and vertical lacunar resorption;
b) sealing cortical plate;
c) the increase in the height of the alveolar bone;
d) the absence of fistula;
e) all of the above.

9. The primary factor in the treatment of endoperiodontal lesions is

a) periodontological treatment;
b) correction of immunity;
c) the use of antibiotics;
d) endodontic treatment;
e) the surgical treatment.

10. Patient's treatment plan with endoperiodontal lesions includes the following methods:

a) surgical
b) orthodontic;
c) endodontic treatment;
d) the regular examination and qualitative carrying out individual and preventive health;
e) all of the above.

LESSON 3. ACUTE INFLAMMATORY DISEASE OF PERIODONTAL TISSUES. CLINICAL MANIFESTATIONS, DIAGNOSIS AND TREATMENT OF ACUTE ULCERATIVE GINGIVITIS AND ACUTE PERIODONTAL ABSCESS.

The questions to be studied for the learning of the topic:

1. Acute periodontal abscess: diagnosis, clinical manifestations.
2. Acute periodontal abscess: differential diagnosis.
3. Methods and means of emergency care for acute periodontal abscess.
4. Acute ulcerative gingivitis: etiology, pathogenesis.
5. Diagnosis and clinical manifestations of acute ulcerative gingivitis
6. Differential diagnosis of acute ulcerative gingivitis.
7. Methods and means of emergency care for acute ulcerative gingivitis.

Question 1. Acute periodontal abscess: diagnostics, clinical manifestations.

Periodontal abscess is a local inflammatory process in periodontium, characterized by pronounced formation and a limited accumulation of purulent exudate within the periodontal pocket due to a violation of exudate outflow.

Causes: improper and incomplete removal of subgingival dental deposits, penetration of a foreign body into the dentogingival pocket, trauma, the spread of bacterial infection from the root canal through the lateral tubules to the periodontium, perforation of the root wall or its fracture.

Table 16. Diagnostics of APA

1) Complaints:	➢ Pain localized, aching (throbbing), painful bite on the tooth, a feeling of "grown up" tooth; ➢ Mobility of the causative tooth (teeth); ➢ Discomfort in the oral cavity, unpleasant aftertaste, hilatosis; ➢ Painful edema (swelling) of the gums in the area of the causative tooth.
2) Anamnesis:	➢ Disease of periodontal tissues, periodontal abscesses in the past, teeth mobility. ➢ Diabetes mellitus, bruxism ➢ Closure of drainage from the pocket.
3) Local status:	➢ There are all the classic signs of acute inflammation; Gingiva is hyperemic, sometimes with a cyanotic shade. ➢ Adenopathy; ➢ In the area of the causative tooth: the obturation of the pocket with the formation of swelling

	(abscess), with drainage of which - purulent exudate; ➢ Mobility of the causative tooth. In patients with generalized periodontitis: tooth mobility, fan-shaped migration of teeth, high values of GI and CPI indices.
Diagnostic tests:	- Percussion is painful (more horizontal); - Palpation of the alveolar process in the area of the causative tooth is sharply painful; when pressing a finger in the area of the bottom of the pocket - there may be a purulent exudate from under the gingival margin. - Loss of the causative tooth (2-3 degrees); - A test for the viability of pulp - a normal response to temperature stimuli and normal indications of electrodontometry; - Radiography: ✓ destruction of periodontal tissues - horizontal (vertical) ✓ pathological changes are possible in the periapical area/ ✓ a V-shaped defect of the bone tissue of the septum is possible, surrounded by an intense and significant zone of osteoporosis. ✓ if there is a fistulous course, the combination of an abscess with periapical tissues allows the introduction of a gutta-percha pin into the fistula. ✓ control of occlusal contacts: it is possible to identify premature contact with the antagonists.
Key symptoms:	- adenopathy; - increased body temperature (not always). - mobility of the tooth (teeth); - vertical migration of the tooth; - sensitivity to slight percussion

Question 2. Acute periodontal abcesses: differential diagnostics.

Table 17. Differential diagnostics of APA

Differential sign	**Acute periodontal abscess**	**Acute periapical abscess**	**Abscess at endoperiodontite**
Course of disease	**Acute**	**Acute**	**Acute**
Pain	+	+	+

Regional lymphadenitis	+	-	+
Presence of periodontal pocket	+	+/-	+
Probing of the carious cavity	+/-	-	-
Percussion	+ +	+	++
Presence of a sinus tract	+/-	+/-	+/-
Tooth mobility	+ +	-	+/-
Palpation	Painful focus of tissue infiltration, closer to the marginal gingiva	Painful focus of tissue infiltration, in the projection of the apex of the root	Painful focus of tissue infiltration, located throughout the length of periodontium of the causative tooth
Thermometry	+/-	-	-
EOM (Reaction to an electrical stimulus)	+/-	-	-
Rg-diagnostics: Destruction in the periapical area	-	+	+

Question 3. Methods and means of emergency assistance in acute periodontal abscesses.

1st visit

1. Anesthesia
2. Drainage of the hearth. Two ways:
   a) through the periodontal pocket (find a hole and gently stretch the aperture of the pocket, get exudate), curettage;
   b) a traditional incision (gingivotomy), curettage.
3. Recommendations: antiseptic treatment (rinse with a 1.5% solution of hydrogen peroxide at home, at least 3 times a day).

Antibiotics are prescribed only with an increase in body temperature ("Amoxiclav").

2nd visit

1. Revision of the focus, antiseptic treatment.
2. Professional hygiene.
3. Reversion of individual hygiene and re-motivation.
4. Planning further complex treatment of periodontal disease.

Question 4. Acute ulcerative gingivitis: etiology, pathogenesis.

Acute ulcerative gingivitis (AUG) is the acute, painful, necrotizing, rapidly progressive inflammation of the gingiva, which may enter a subacute or chronic stage. The disease seldom occurs as a generalized process throughout the entire dentition, nor are its clinical manifestations of identical severity. It may be quite advanced in individual anterior teeth, while the adjacent premolars or molars are not affected at all or only mildly so. The reverse may also be the case. There is no all-encompassing reason for this irregular appearance, but poor oral hygiene, locally predominating pathogenic bacteria and the presence of plaque retentive areas are likely participants. Without treatment, AUG may progress to Periodontitis ulcerosa.

Epidemiology: Next to Gingivitis simplex, AUG is one of the most common diseases of the gingiva. Incidence figures ranging from 0.1-10% of the adolescent population have been reported.

Microbiology: Predominance of Spirochetes, Bacteroides and fusiform bacteria in plaque.

The etiology of AUG is not completely unterstood. In addition to plaque and a previously existing gingivitis, the following factors are suspected:

Local factors:

- Poor oral hygiene
- Predominance of Spirochetes, Bacteroides and fusiform bacteria in plaque
- Plaque-retentive areas (crowded teeth, overhanging restorations etc.)
- Smoking (local irritation from tar substances)

Systemic factors

- Poor general health
- Fatigue or psychic stress
- Smoking (nicotine and chemotaxin)
- Age (Young men 18-30 years)
- Season of the year (September/October and December/January)

Patients with AUG are often young, heavy smokers, who exercise poor oral hygiene and are indifferent to their oral disease, becoming interested in treatment only during painful exacerbations.

Histopathology. The clinical and histopathological pictures in AUG are correlated. The histopathology of AUG is significantly different from that of simple gingivitis.

As a consequence of the acute reaction, an enormous number of PMNs transmigrate the junctional epithelium in the direction of the sulcus and the col. In contrast to the situation in simple gingivitis, PMNs also migrate toward the oral epithelium and the tips of papillae, which undergo necrotic destruction. The ulcerated wound is covered by a clinically visible whitish pseudomembrane that consists of bacteria, dead leukocytes and epithelial

cells, as well as fibrin. The tissue subjacent to the ulcerated areas is edematous, hyperemic and heavily infiltrated by PMNs. Spirochetes often penetrate into the tissue. An inflammatory infiltrate is observed in deeper tissue zones. Plasma cells dominate the histologic picture of long-standing AUG.

Question 5. Diagnosis and clinical manifestations of acute ulcerative gyngivitis.

Table 18. Diagnosis and clinical manifestations of AUG

Complaints	1) The lesions are extremely sensitive. 2) Complains of a constant radiating, gnawing pain that is intensified by eating spicy or hot foods and chewing. 3) There is a metallic foul taste, and the patient is conscious of an excessive amount of "pasty saliva". 4) There are complaints related to the general intoxication of the body (headache, weakness, insomnia, loss of appetite, fever).
Anamnesis	Smoking (worsens microcirculation in periodontal tissues, retention factor of plaque); alcohol abuse, stress, infections of upper respiratory tract.
External inspection	In rare (severe) cases, symptoms of general intoxication are possible: lymphadenopathy, fever, general malaise.
Intraoral Signs and Symptoms	1. Lesions are characterized by punched out, crater-like depressions at the crest of the interdental papillae, subsequently involving marginal gingiva and rarely attached gingiva. 2. These craters are covered by grayish pseudomembranous slough, which is demarcated from the remaining of the mucosa by a pronounced linear erythema. 3. Fetid odor and increased salivation 4. Other signs include gingival hemorrhage or pronounced bleeding on the slightest stimulation. 5. Abundant dental plaque (both plaque and tartar), which indicates poor hygiene of the oral cavity. The indexes of the OHI- S are unsatisfactory.
Additional diagnostic tests	✓ microbiological studies indicate a predominance of Gram-negative microflora; ✓ indicators of general and biochemical blood analysis reflect the state of the pathological process.
Key features:	✓ ulceration and necrosis of the interdental papillae; ✓ pain; ✓ smell from the mouth; ✓ increase in body temperature by 1°C compared with the norm.

Question 6. Differential diagnosis of acute ulcerative gingivitis.

Table 19. Differential diagnosis of AUG

Symptoms	***Acute ulcerative gingivitis***	***Herpetic gnigovostomatitis***	***Diphtheria***	***Secondary period of syphilis (ulcer syphilid)***	***HIV-necrotizing gingivitis***
Morphological element	Ulcer in the area of the interdental papillae with a gray-white necrotic plaque	Small multiple ulcers with gray plaque	Ulcer with uneven edges, with tightly fused fibrinous white-gray membrane	Ulcers are grayish-white	Ulcer of interdental papillae of gray-yellow color
Localization	Interdental gum, marginal gingiva	Gums, cheeks, lips	Retromolar region, soft palate, arches, amygdala	Gums, tongue, soft palate, arms, tonsils	The interdental, marginal gingiva.
Course of disease	Acute	Acute	Acute	Acute or chronic	Acute, with frequent relapses
Cause of the disease	Complex of factors	Virus	Bacillus diphtheria	Treponema pallidum	Bacterial infection in the background of immune suppression
Investigation of a smear from destruction elements	Mixed anaerobic gram-negative flora	Multinucleated giant epithelial cells	Bacillus diphtheria	Treponema pallidum	Fusospirochetes bacteria
Contagiousness	-	+	+	+	+
Contingent of patients	Young men 18-30 years old	Children	Adults, children	Adults	HIV-infected patients
Transmission paths	-	Airborne-droplet	Airborne-droplet	Contactual, sexual	Hematogenous, sexual
The reaction of the lymph nodes	Lymph nodes are enlarged and painful on palpation	Lymph nodes are enlarged and painful on palpation	Lymph nodes are enlarged and painful on palpation	Lymph nodes are enlarged, dense, painless	Lymph nodes are enlarged and can be painful on palpation
Halitosis	+	-	-	-	+
Serological tests	-	-	-	+	+

Question 7. Methods and means emergency care with acute ulcerative gingivitis.

Emergency treatment is carried out for a minimum of 3 visits. The patient is observed for 2 weeks. Not earlier than a month after the acute period, carry out radical measures (surgical treatment).

1st visit:

1. ***Anesthesia***. Application anesthesia, especially in the form of aerosols (lidocaine 10%, Peril-spray) or gel (Anaestho Gel, Xylonor) based on lidocaine.
2. ***Necrotized tissues*** are removed. As much as the patient's condition allows, foci of necrosis, dental deposits (preferably with sharp curettes, excavators) are removed to OHI-S <0.6 and irrigated with a 3% solution of hydrogen peroxide.
3. ***Enzymes*** are also used in the form of applications: trypsin, chymotrypsin.
4. ***Provide training in oral hygiene, motivation*** to thoroughly care for the oral cavity. It is recommended to avoid smoking and drinking alcohol.
5. ***Home treatment by medication***.

Local:

- ✓ Rinse the mouth with a 1.5- 3% hydrogen peroxide solution every hour.
- ✓ Use of antibiotics in the form of applications, pastes, ointments and medical dressings: Periodonton (Septodont), gramicidin C, Metrogil-denta.

General:

- ✓ Treatment with metronidazole (trichopolum, flagel, clion): inside by 0.25 g 3 times a day for 5-7 days.
- ✓ Purpose of analgesics.
- ✓ Desensitizing and vitamin therapy.

2nd visit (after 24 hours)

The patient's condition improves considerably by the second day. The pain disappears completely or is present slightly. The gums are hyperemic, but without necrotic plaque. The procedures of the 1st day are repeated. The patient follows all the instructions of the 1st day.

The dentist conducts:

- ➢ Professional oral hygiene, removal of foci of necrosis;
- ➢ Irrigation with a 3% solution of hydrogen peroxide.
- ➢ Correction of individual hygiene of the oral cavity, repeated motivation, including habits and lifestyle.

Drugs that *improve epithelization* are used in case of improvement in general condition and local condition:

1) keratoplastic agents (fitodont, polymineral, Kalanchoe ointment, Dogrose and sea-buckthorn oil, karatolin, inhalipt, aloe vera, solcoseryl, l0% methyluracil),

2) ointment with glucocorticoids (Lokokarten, Sinalar, Deperzolone, prednisolone, etc.)
3) medicinal plant preparations (chamomile, sage leaf, serpentine root, oak bark), etc.

Third visit: scaling and root planing are repeated, plaque control instructions are given. Hydrogen peroxide rinses are discontinued.

Fourth visit: oral hygiene instructions are reinforced and thorough scaling and root planing are performed.

Fifth visit: appointments are fixed for treatment of chronic gingivitis, periodontal puncture and pericoronal flaps, and for the elimination of all local irritants.

Test control

1. Indicate the local factors contributing to the development of acute periodontal abscess:

a) incorrect and inadequate removal of subgingival dental deposits,
b) penetration of a foreign body in pocket,
c) injury,
d) perforation of the root wall or its fracture.
e) all of the above.

2. Antibiotics in the treatment of acute periodontal abscess are used in case of:

a) fever;
b) painful percussion of neighboring teeth
c) in any case during the all treatment;
d) are not used at all.

3. Indicate the not mandatory symptoms of acute periodontal abscess:

a) mobility of the tooth (teeth);
b) sensitivity to weak percussion;
c) increase in body temperature.
d) the gum is hyperemic, edematous, painful.

4. What complaints the patient does not present with an acute periodontal abscess:

a) pain localized, aching (throbbing), with nipping,
b) the feeling of a "grown up" tooth;
c) mobility of the causative tooth (teeth);
d) painful swelling (swelling) of the gums in the area of the causative tooth.
e) spontaneous aching pain, worse from cold.

5. List the complaints characteristic of acute apical periodontitis (with serous exudate):

a) acute pain;
b) paroxysmal pain;
c) constant aching pain;
d) pain intensifies from cold;
e) pain increases with nibbling;
f) pain calms down from the cold.

6. List the complaints characteristic of acute apical periodontitis (with purulent exudate):

a) acute unsufferable pain;
b) paroxysmal pain;
c) constant aching pain;
d) pain intensifies from cold;
e) the feeling of a "grown up" tooth;
f) tooth touching is painful.

7. Indicate the characteristic of the marginal gingival in acute ulcerative gingivitis:

a) the marginal gingiva is covered with a dirty gray coating, after removal of which an erosive, bleeding surface is formed;
b) the gingival papillae are enlarged, compacted, cover part of the crown;
c) marginal gingiva and separate gingival papillae are hyperemic, swollen, bleed easily.

8. At what age is most commonly diagnosed acute ulcerative gingivitis?

a) 15-30;
b) 30-45;
c) 0-70.

9. Indicate the local factors that contribute to the development of acute ulcerative gingivitis:

a) poor oral hygiene;
b) smoking;
c) anomalies of the location of the teeth;
d) all of the above.

10. What emergency interventions are carried out in case of acute gingivitis on the first visit?

a) anesthesia;
b) removal of plaque and dental deposits;

c) recommendations: rinsing with a 1.5% solution of hydrogen peroxide at home at least 3 times a day;
d) motivation, training in oral hygiene;
e) all of the above.

LESSON 4. CLINICAL MANIFESTATIONS, DIAGNOSIS, PLANNING OF GINGIVAL RECESSION AND PERIODONTAL ATROPHY TREATMENT.

The questions to be studied for the learning of the topic:

1) Gum recession: definition, epidemiology, etiology, pathogenesis, risk factors.
2) Classification of gingival recession.
3) Types and clinical manifestations of gum recession.
4) Diagnosis of gum recession.
5) Planning treatment for recession.
6) Periodontal atrophy: definition, etiology, pathogenesis.
7) Clinical manifestations with periodontal atrophy.
8) Diagnosis and differential diagnostics with periodontal atrophy.
9) Planning treatment for periodontal atrophy.

Question 1. Gum recession: definition, epidemiology, etiology, pathogenesis, risk factors.

Gingival recession accounts for 5-10% of all attachment loss.

Recession is progressive displacement of gums towards the apex, leads to its denudation.

- is defined as a seemingly inflammation-free clinical condition characterized by the retreat in an apical direction of the facial and less often of the oral (palatal, lingual) periodontium.

Despite recession of the gingival margin, the interdental papillae usually fill the entire embrasure areas in younger patients. Recession is usually localized to one or several teeth; generalized gingival recession is seldom observed. Teeth exhibiting gingival recession do not display increased mobility. The periodontal supporting structures are generally good.

Teeth are never lost due to gingival recession alone!

If the patient's oral hygiene is inadequate, or if the recession reaches the movable oral mucosa, secondary inflammation may ensue and eventually pocket formation (periodontitis) may occur.

Etiology:

1) Anatomico-physiological features of the structure of the alveolar process.

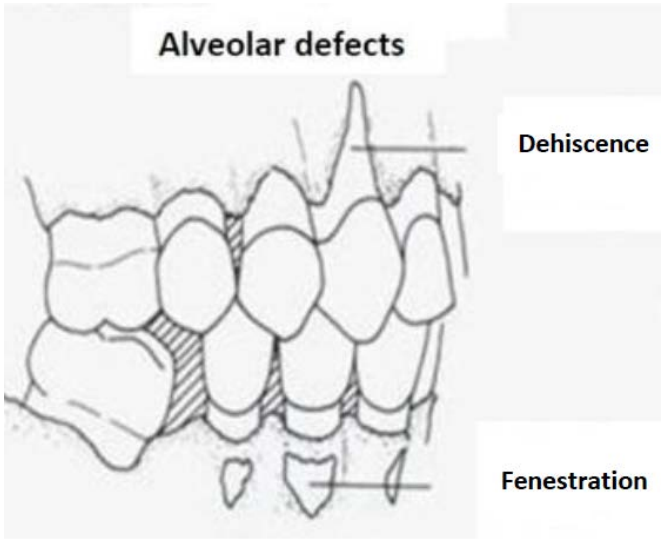


Figure 54. Anatomico-physiological features of the structure of the alveolar process.

The causes of recession have not been completely elucidated. It is probable, however, that a primary factor is purely the morphology and anatomy of the situation. The facial plate of bone overlying the root is usually very thin. Not infrequently the root surface is completely denuded of alveolar bone or exhibits fenestrations in the thin osseous lamella. The total lack of bone over the facial root surface is referred to as a dehiscence. This situation is most frequently observed on cuspids and incisors, less often on premolars and seldom on molar teeth (except mesiobuccal root of 1st maxillary molars!). The situation is also frequently associated with tooth positional anomalies such as buccoversion, supereruption etc. Despite the lack of a buccal plate of bone over the root, the gingival margin may maintain its normal position just coronal to the CEJ.

2) Mukingingival anomalies and deformations.

- ✓ A shallow vestibule of mouth (< 5 mm.).
- ✓ Frenum pulls, especially when fibers of the frenum attach near the gingival margin
- ✓ Orthodontic treatment (tooth movement labially; arch expansion).
- ✓ Some possible etiologic factors in gingival recession include:
- ✓ Improper, traumatic toothbrushing, e.g., horizontal scrubbing
- ✓ Mild chronic inflammation that may be only slightly visible clinically
- ✓ Iatrogenic factors

Question 2. Classification of gingival recession.

1) International Classification of Diseases(1995):

K06 Other disorders of gingiva and edentulous alveolar ridge

Excludes: atrophy of edentulous alveolar ridge (K08.2), gingivitis (K05.0, K05.1)

K06.0 Gingival recession

Includes: postinfective

postoperative

K06.00 Localized

K06.01 Generalized

K06.09 Gingival recession, unspecified

2) Leus P.A. and Kazeko L.A. (Republic of Belarus)

Three groups of recession of a gum with the account of a leading etiological condition and a clinical picture are revealed:

I. Traumatic: recession of the gum is a consequence of a long-acting injury

II. Symptomatic: recession of the gum is one of the symptoms of gingivitis, periodontitis.

III. Physiological: recession of the gum is a sign of aging of the body and periodontal tissues.

By localization:

I. Localized recession of the gum is observed in several teeth, more often one or two.

II. Generalized: a group or several groups of teeth.

III. Systemic recession of the gum is observed in all teeth and is caused by systemic factors

By severity:

I. Mild degree: the size of the gum recession is up to 3 mm.

II. Moderate: the size of the gum recession is 3-5 mm.

III. Severe degree: the size of the gingival recession is > 5 mm.

Traumatic recession is localized and generalized. Symptomatic recession is localized, generalized and systemic. Physiological recession is systemic.

3) It is also recommended to distinguish between two types of recession: ***visible and hidden***. The visible recession can be determined by the doctor as the distance from the enamel-cement border to the gingival edge (recession height) and the distance between the vertical edges of the recession at the level of the enamel-cement border (recession width). A hidden recession is detected only when probing.

4) The Sullivan and Atkins Classification of recession is a descriptive system first presented in 1968. It was thought at the time that it was often not possible to achieve complete soft tissue coverage for the denuded root surface. The classification system described the type of recession and expected amount of root coverage utilizing the free gingival graft procedure. Deep, wide defects were thought not to be able to be covered, although

almost complete coverage was possible with narrow defects. The four classes of recession are:

1. Shallow, narrow
2. Deep, narrow
3. Shallow, wide
4. Deep, wide

5) ***Miller presented a system of classifying recession*** based both on the apical extent of the recession and the adjacent interproximal bone height. The system also predicts the amount of root overage possible. With no interproximal bone loss, most roots may be covered with new soft tissue to the cementoenamel junction.

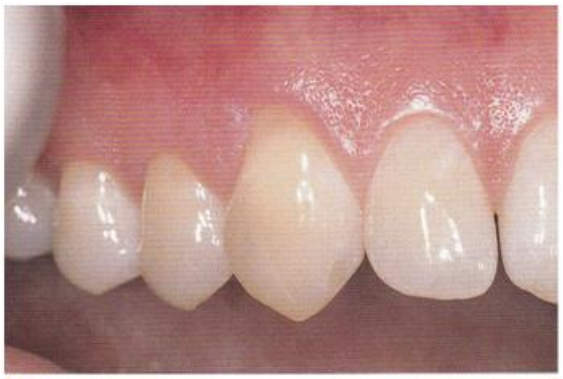


Figure 55. Miller Recession Classitication. Class 2 recession
Class 2. Recession apical to the mucogingival junction with no adjacent interproximal bone loss. 100 % root coverage to the CEJ is possible.

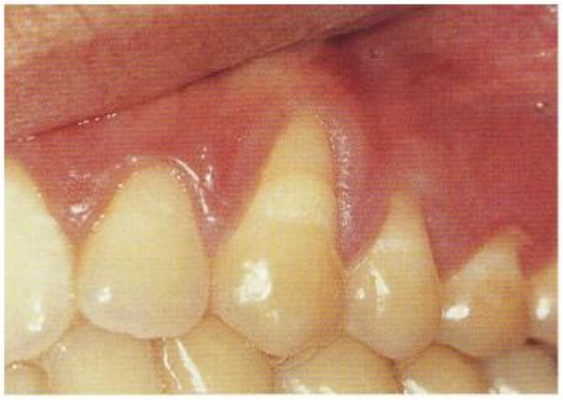


Figure 56. Miller Recession classitication. Class 1 recession
Class l. Recession coronal to the mucogingival junction with no interproximal bone loss. 100% root coverage to the cementoenamel junction is possible.

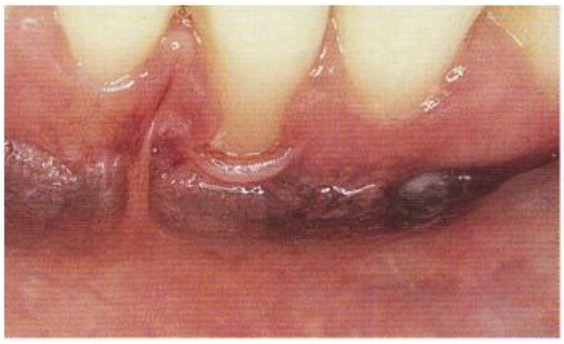


Figure 57. Miller Recession classification. class 3 recession
Class 3. Recession apical to the mucogingival junction with mild to moderate adjacent interproximal bone loss. Some root coverage is possible, based on the level of the interproximal bone.

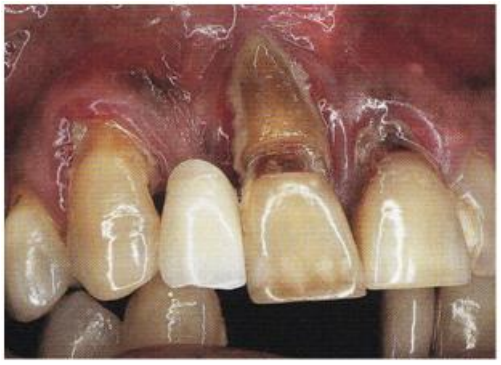


Figure 58. Miller Recession classification. class 4 recession
Class 4. Severe recession with accompanying severe bone loss. No root coverage can be expected.

Question 3. Types and clinical manifestations of gum recession.

Clinical Symptoms. The clinical manifestations of recession are numerous. Gingival recession usually begins with a gradual apical migration of the entire facial aspect of the gingiva, re-vealing the CEJ. Less frequently the first sign of recession is the relatively rapid formation of a small groove in the gingiva, a so-called Stillman's cleft. This can expand into a pronounced recession. As a consequence of recession, the remaining attached gingiva may become somewhat thickened and rolled, a non-inflammatory fibrotic response known as McCall's festoons.

If the recession proceeds as far as the mucogingival line, secondary inflammation of the marginal gingiva may ensue. Though rare, recession may also be observed on the palatal roots of maxillary molars and on the lingual aspect of mandibular incisors.

Esthetic considerations may prompt the patient to seek professional care if recession becomes pronounced in the maxillary anterior segment. As root surfaces are exposed, cervical sensitivity may also become a problem.

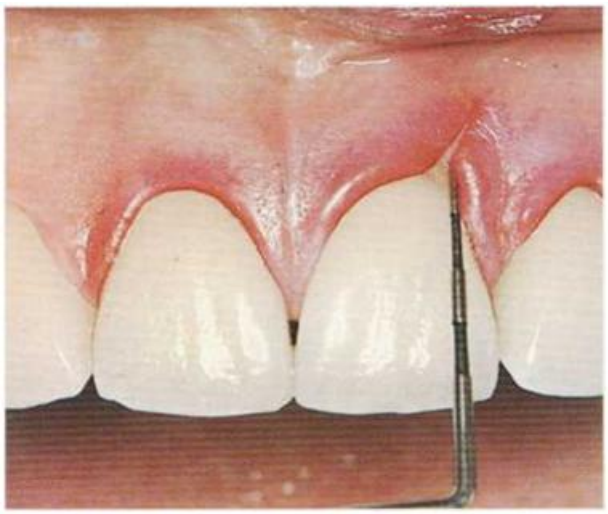


Figure 59. Stillman's cleft
The periodontal probe reveals the presence of a cleft, which may expand laterally and develop into general recession. The exposed root surface is often very sensitive, and is usually covered with plaque, which leads to secondary inflammation.

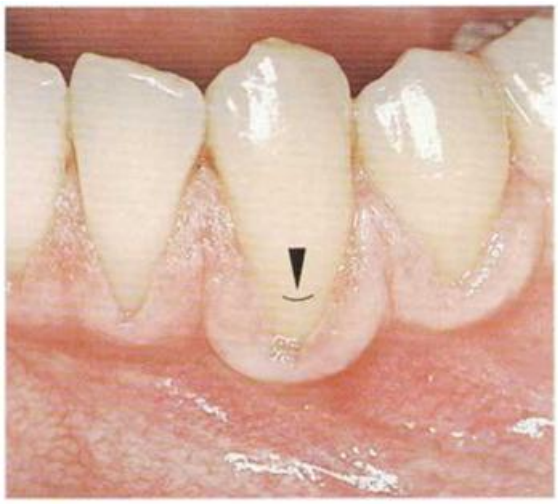


Figure 60. **McCall's festoons**
The attached gingiva in this case consists of nothing more than a collar-like, fibrous thickening. The arrow indicates the CEJ. This may be a tissue response to further recession beyond the mucogingival line. It is not an indication for mucogingival surgery!

In addition to the classical recession, there are other forms of clinically observable attachment loss:

- Recession of the entire periodontium in old persons. This condition is not the rule. It may be caused by a mild chronic inflammation and shrinkage; it can be enhanced by improper tooth brushing technique or other iatrogenic irritation.
- Recession with a superimposed secondary periodontitis. This combination occurs seldom, because patients who practice proper oral hygiene usually exhibit neither plaque accumulation nor inflammation.
- Destruction or shrinkage of the gingiva as a conse-quence of untreated periodontitis.
- Status following periodontal therapy. "Long teeth," open interdental spaces and cervical sensitivity are often the uncomfortable consequences of treatment for advanced periodontal disease.

Types

Traumatic localized recession

- Common among young people.
- It arises more often on the vestibular surface of the upper canine, resection or premolar.
- The size of the recession is insignificant (1-2 mm.).
- Ablation of the root of the tooth is accompanied by aesthetic deficiency and hyperesthesia.
- The presence of MC Colls Festoons.

Etiology: incorrect traumatic tooth cleaning + anatomical predisposition (protruding position of individual teeth, small anterior cavity of the mouth, lateral cords of the mucosa in the area of these teeth).

Traumatic generalized recession

- Young people more often.
- Vestibular surface, usually premolars and canines.
- Small size.

- The presence of MC Colls Festoons in causal teeth
- Complaints about aesthetic deficiency, short-term soreness from thermal, chemical irritants.
- There are no signs of an inflammatory process, and oral hygiene is satisfactory

Symptomatic localized recession

- Young people more often.
- Any surface of several teeth, more often one or two.
- In case of poor hygiene there is localized recession of the gums in combination with all the signs of gingivitis: an abundance of dental deposits (dental calculus in particular), bleeding, swelling and gum flushes.
- Possible iatrogenic injuries - surgical interventions on the gums, overhanging seals in the cervical region, traumatic caries treatment.

Symptomatic generalized recession

- Can occur at any age.
- One of the symptoms of chronic periodontitis (less often - chronic gingivitis).
- More often - in several groups of teeth, both on the vestibular and on the oral surfaces, and in persons older than 35 years of recession, the interdental gum may also be affected.
- More often begins with a lingual or palatine surface, the lower frontal teeth and the upper first molars are affected.
- Characteristic of the presence of Stillman's Clefts.
- Uneven recession (different sizes in different parts of the jaw).

Symptomatic system recession.

- With periodontitis (at any age), when it acts as the leading clinical sign of the disease.
- Practically at all surfaces of all teeth
- is due to systemic (somatic) factors.
- Uniformity - practically the same value in different parts of the dentition.
- can be significant (mostly 3-5 mm) and leads to pronounced denudation of the root of the tooth.
- Often combined with wedge-shaped defects.

Physiological system recession

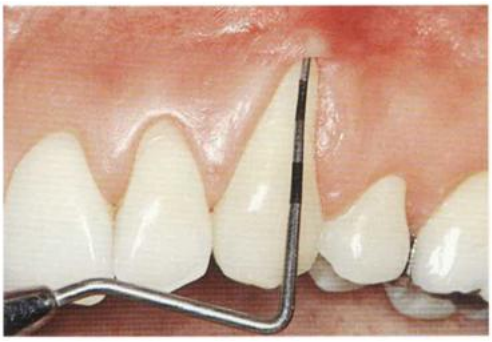
- typical for people aged 60 years or more.
- It is a consequence of aging periodontal disease.
- on the surface of each tooth, the value is insignificant.
- Even with strong denudation of the roots, the teeth remain firm.
- There are no inflammatory changes in the periodontal tissues.

Question 4. Diagnosis of gum recession.

Recession of the gingiva may be the primary reason a patient seeks dental care. Recession is easy to recognize during an examination of the oral cavity, but a more objective assessment is required for recording recession in the patient's records. The extent of gingival recession is measured with the periodontal probe as the mm distance from the CEJ to the gingival margin. It also must be determined whether a normal sulcus or a pathological pocket is present at the recession site. The width of the remaining attached gingiva between the free gingival margin and the mucogingival line is of little consequence as long as no inflammation is present (Wennstrom 1982; Wennstrom & Lindhe 1983).

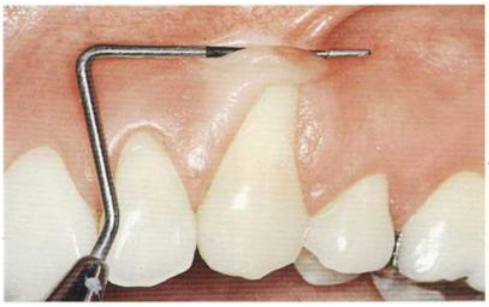
However, a recession site where/70 attached gingiva remains is a significant observation, particularly when the movable mucosa or frena extend directly into the gingival margin. The dentist also must clarify whether or not the recession is an esthetic problem for the patient.

Figure 61. Attachment loss through recession



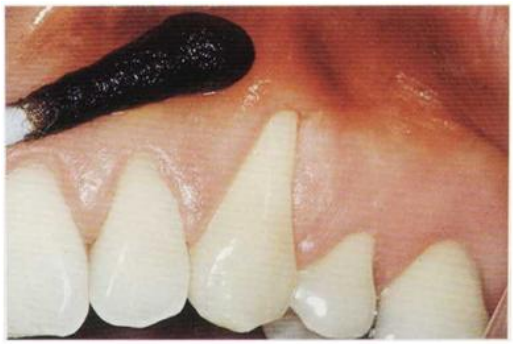
Recession on the facial surface of the cuspid is measured at 5 mm between the CEJ and the gingival margin. The probe extends an additional 2 mm into the gingival sulcus. It appears that there is absolutely no attached gingiva remaining in this area. Also absent is a McCall's festoon, which, if present, could be considered as a reparative attempt on the part of the host tissues to the minimal gingival width.

Figure 62. Roll test



Using a finger or a periodontal probe, the movable mucosa is pushed toward the recession site. This permits verification of presence or absence of attached gingiva, which will offer resistance. In this case, the movable mucosa extends directly to the gingival margin.

Figure 63. Iodine test



Gingiva and oral mucosa are painted with Schiller or Lugol solution (a solution of iodine and potassium iodide). The mucosa takes on a brown color owing to its glycogen content, while the glycogen-free attached gingiva remains unstained. The iodine test depicted here reveals that no attached gingiva remains on the facial surface of tooth 23.

The index of gum recession (1955) Stahl, Morris, which allows you to determine the prevalence of pathology in the subject/

Severity	**Index Value**
Mild	up to 25%
Moderate	26-50%
Severe	51-100%

$$\textbf{The index of gum recession} = \frac{\text{number of teeth with recession}}{\text{total number of teeth}} \times 100\%$$

Question 5. Planning treatment for recession.

In many cases, recession can be halted by changing the patients brushing method to improve plaque control. After re-evaluation, surgery mayn't be indicated in such cases. The following steps may spare the patient with recession a surgical procedure:

1. Thorough oral prophylaxis: Scaling, root planing, and polishing.
2. Modification of the patient’s home care technique. The vertical-rotatory method (modified Stillman) in which the toothbrush moves from the gingiva onto the tooth surface has been shown to be beneficial in patients with recession.
3. Photographs or study models for later comparison (progressive recession).
4. Initially, short-interval recall.

If recession continues to progress after this “observation phase’’, mucogingival surgery is indicated.

Exceptions to this rule include frena that radiate directly into the marginal gingiva and elicit pronounced localized recession. In these cases, surgery should be performed immediately after initial therapy.

Some surgical procedures that were common in the past are performed only infrequently today, e.g., extension operation without a FGG, sliding flap procedure, and direct coverage of a denuded area with a free gingival graft.

The indications for mucogingival surgery as a treatment modality for recession have been sharply reduced in scope in recent years. Numerous studies have demonstrated that periodontal health (freedom from inflammation) can be maintained regardless of the finite width of attached gingiva.

This realization has reduced the indications for mucogingival surgery to attempting 1) to halt progressive recession or 2) to achieve a creeping attachment.

If such mucogingival surgery is performed, an additional benefit may be a widened band of attached gingiva and a slight deepening of the buccal vestibule, both of which may enhance plaque control (Lang & Loc 1972). In rare instances, mucogingival surgery may be employed in an attempt to cover a denuded area for esthetic reasons.

The term mucogingival surgery includes the following:

- Gingival extension without free gingival graft (FGG)
- Gingival extension using a FGG
- Frenotomy/frenectomy
- Extension operation of Edlan-Mejchar (1963)

Covering a denuded root surface by means of:

- Sliding flap (Grupe & Warren 1956)
- Direct free gingival graft (one-step procedure)
- Coronal flap repositioning after FGG (two-step procedure).

Question 6. Periodontal atrophy: definition, etiology, pathogenesis.

Periodontal atrophy is a dystrophic, non-inflammatory lesion of periodontal tissues, accompanied by a generalized evenly decrease their volume.

In clinical practice, periodontal atrophy is relatively rare, no more than 3-4% of individuals. With this form, there are no inflammatory and periodontal pockets.

The exact cause is unclear. Periodontal atrophy is considered as premature development of involutive processes, or as a manifestation of general metabolic disturbances of the organism in periodontal tissues. Also in the new WHO taxonomy, this form of periodontal disease is treated as a "generalized gum recession".

Pathogenesis. Morphological examination of periodontal tissues reveals various neurotrophic changes depending on the form of periodontal atrophy.

Physiological periodontal atrophy: in all periodontal tissues the amount and mitotic activity of fibroblasts, as well as the synthesis of collagen, are reduced. There is decrease cornification in the epithelium of the gums, as well as atrophic changes in the prickle cell layer. In the periodontal ligament, the collagen is distributed normally, but the fiber bundles become thicker and

stiffer. The volume of the organic matrix and the number of Malassé islands are decreasing. Forms of hyaline, they sometimes lead to calcification of connective tissue or its cartilaginous change. The periodontal cleft is contracted. Blood supply is loosened, in the periodontal vessels there are atrophic changes.

Symptomatic periodontal atrophy: there are microcirculatory disorders, metabolic disorders with a sharp delay in the processes of protein synthesis in all periodontal tissues, as well as violations of innervation of tissues. There is proteinaceous dystrophy of cells, atrophy of epithelial with a decrease in the amount of glycogen in the epithelium of the gums. There is mucoid swelling, fibrinoid changes, a decrease in the activity of oxidation-reduction processes in the connective tissue of the periodontal ligament. In bone tissue, there is a delay in the renewal of bone structures with thickening of the bone trabeculae up to the expressed osteosclerosis and loss of spongy substance. Changes in bone tissue are combined with microvasculature changes (hyalinosis and arteriosclerosis of vessels, contracting of their lumen and obliteration). Nerve fibers of periodontal tissues are changed, with the progression of periodontal atrophy their dystrophy is intensified.

Question 7. The clinical picture of periodontal atrophy.

Classification of periodontal diseases (LN Dedova, 2002-2012):

Periodontal atrophy:

1. Form	1.1. Physiological 1.2. Symptomatic
2. Prevalence	2.1. Generalized
3. Degree of severity	3.1. Mild 3.2. Moderate 3.3. Severe

<u>The clinical picture of periodontal atrophy:</u>

- Generalized gum recession
- Decrease the height of the interalveolar bone with conservation of the compact plate
- Absence of inflammation and periodontal pocket

Physiological periodontal atrophy is a physiological process that is characteristic for almost all healthy patients of adult age, and it is the result of age-related changes.

Symptomatic periodontal atrophy is a pathological process that is noted at any age as a symptom of systemic pathology. Complaints of patients depend on the stage of the disease and the severity of the pathological process.

At light disease degree of severity, complaints are usually absent. Medium and heavy degrees of severity are accompanied by itching, a feeling of aches, numbness in the gums and jaws, the appearance of dentine sensitivity to chemical and thermal stimuli. Patients complain about "gum subsidence and lengthening of teeth". Patients also note complaints about aesthetic disorders, violations of diction. In the anamnesis, there are cardiovascular diseases, disturbance of metabolic processes, neurogenic and endocrine pathology, etc.

Clinical picture: the presence of combined non-carious lesions (enamel erosion, wedge-shaped defects) with good oral hygiene. The gums are pale, dense, painless, does not bleed when probing, there is no mobility of the teeth and dentogingival pockets. There is also a generalized recession of the gums of different severity.

With the further development of the disease, the necks and roots of the teeth are exposed, the fan-shaped divergence of the teeth occurs, their displacement, interstices appear between the teeth (diastema, trema), the function of speech is disrupted, while the teeth remain relatively stable, the mobility does not exceed the first degree.

Question 8. Diagnostics of periodontal atrophy

Diagnosis of periodontal atrophy is based on the clinical and radiological and morphological characteristics of periodontal tissues.

Table 20. Differential diagnosis of periodontal atrophy

Nosological form		Periodontal atrophy	Gum recession	Periodontitis in remission
Symptom		-	-	±
Dental deposits		-	-	±
Bleeding gums		-	-	±
Dental pocket		-	-	±
Rg-diagnosis of the alveolar process	Cortical plate	+	+	-
	Reduction in the height of the alveolar process	+	-	+
	The direction of reduction in the height	Horizontal	-	Horizontal or vertical

Clinical examination: carry out a detailed study of periodontal tissues and the definition of indices:

- oral hygiene;
- inflammation in the gums;
- recession of the gum;
- sensitivity of dentin - Complex index of differential sensitivity of teeth.

X-ray picture of periodontal atrophy is characterized by the following changes: bone pattern of alveolar process slightly differentiated, small-celled, cortical plate retained. Cortical plate looks less contrasted against the background of sclerized spongy substance. The height of the alveolar process is reduced, its peak has a distinct shape. Decrease in the height of the interdental septum: for mild degree of severity - up to 1/3 of the length of the root of the tooth, for moderate degree of severity - from 1/3 to 1/2 the length of the root, for severe degree of severity - more than 1/2 the length of the root.

When diagnosing periodontal atrophy, an important role is played by the methods of functional diagnostics for studying the state of microcirculation of periodontal tissues. These methods determine the condition and function of periodontal vessels, identify signs of hypoxia of periodontal tissues: determination of capillary pressure of periodontium (Y.A. Denisova, 2012), laser-optical method (LOM) (S.P. Rubnikovich, 2011),

A novel laser-optical method (LOM) for blood microcirculation diagnostics in vivo is described. The method is based on laser probing of biotissues, on digital dynamic speckle photography, and on PC assisted correlation analysis of images obtained in the real-time operation mode. Microcirculation intensity has been determined in patients under normal conditions and with chronic periodontitis by LOM. LOM allows determining microcirculation defects in periodontal tissues. It is proved that LOM can be used for earlier diagnostics of periodontitis.

Question 9. The treatment plan of periodontal atrophy

The treatment plan of periodontal atrophy can include the same phases and stages as when drawing up a treatment plan for other periodontal disease.

1) motivation for hygiene of the oral cavity
2) control of the plaque and control of the diet
3) correction of seals and dentures
4) correction of occlusal contacts, reassessment of gum condition, evaluation of oral hygiene
5) selective grinding, sanding of unbroken mounds
6) periodontal surgery
7) rational prosthetics (to restore the integrity of the dentition and to normalize the occlusal load), splinting.
8) Supportive treatment (periodic visits, oral hygiene control, assessment of tooth mobility, assessment of inflammation, correction of occlusion).

Treatment of periodontal atrophy. Due to the fact that the development of periodontal atrophy is associated with dystrophic changes in periodontal tissues, the treatment is mostly symptomatic.

Treatment directed:

1) on the normalization of tissue metabolism,
2) improvement of oxidation-reduction processes,
3) cessation of microcirculatory disorders
4) decreased sensitivity of the necks and roots of the teeth.

For these purposes, prescribe vitamin C, electrophoresis of vitamin B1 with novocaine. All types of massage, darsonvalization of gums, exposure to microwaves, novocain blockades, submucosal injections of angiotrophin (water extract of bovine pancreas released from insulin), oxygen and Chonsuridum (chondroitinsulfuric acid participates in the construction of the basic substance of connective tissue), vacuum therapy are shown.

Local treatment should be performed against the background of active vitamin therapy (vitamin-mineral complexes for 30 days 2 times a year), prescribing calcium preparations.

The method of treatment of periodontal diseases with the help of vacuum-hematomas (Kulazhenko). Vacuum-hematomas promote the normalization of tissue metabolism, stimulate the ability of tissues to regenerate. Hematomas are made along the mucogingival fold in the area of the teeth; Course - from 6-8 to 12-14 procedures.

Vacuum-hematomas promote the normalization of tissue metabolism, stimulate the ability of tissues to regenerate. Hematomas are made along the mucogingival line in the area of the teeth; Course - from 6-8 to 12-14 procedures.

The introduction of oxygen under the mucous membrane of the mucogingival fold increases oxidation-reduction processes, promotes the elimination of hypoxia in periodontal tissues and has a stimulating effect. Oxygen is injected through a thin needle into the region of the first premolar, 5-7 ml per side; Course 10-15 injections every other day.

Hyperbaric oxygenation (If there is equipment), a course of 10-15 injections, is also used.

The introduction of calcium and fluoride ions into the bone tissue of the alveolar process can be carried out by electrophoresis and phonophoresis; the course of treatment is 10 procedures.

Calcium glycerophosphate, fluoride preparations and other desensitizers are used, when hypersensitivity of hard tissue of the teeth in the region of cervical line.

Test control

1. Indicate the color of the gum in the case of periodontal atrophy:

a) bright red, edematous;
b) pale, dense;
c) cyanotic, loose;

2. Indicate the presence or absence of pathological pockets in the case of periodontal atrophy:

a) there are only in the incisor area;
b) there are only in the molar area;
c) are determined in the area of all teeth;
d) are absent

3. In the case of periodontal atrophy is diagnosed:

a) more often non-carious lesions of hard tissues of the teeth;
b) more often carious lesions of hard tissues of the teeth;
c) non-carious and non-carious lesions of the hard tissues of the teeth equally;
d) very rarely non-carious and carious lesions of the hard tissues of the teeth

4. What treatment methods are possible in the treatment of periodontal atrophy?

a) use of non-steroidal anti-inflammatory drugs
b) selective sanding
c) antibiotic therapy
d) local application of sclerosing agents

5. What types of pockets are typical for periodontal atrophy?

a) absence of pocket
b) false
c) intraosseous

6. Causes the recession of the gums are:

a) cleaning of teeth
b) flossing
c) para-functional habits
d) periodontal disease
e) orthodontic treatment
f) all of the above

7. How to identify a hidden gum recession

a) visually.
b) using air jets.
c) probing.
d) palpation

8. Clinical methods for diagnosing gum recession are:

a) CPITN.

b) PI no Rassel, PDI.
c) Stahl-Morris Index.

9. Where is the recession of the gums more often?

a) more on the upper jaw.
b) more on the lower jaw.
c) equally on both jaws

10. The size of the gum recession is:

a) the distance from the enamel-cement border to the level of the edge of the gum from the vestibular and oral tooth.
b) distance from cutting edge of tooth to edge level
c) the distance from the enamel-cement border to the level of the edge of the gum from all surfaces of the tooth.

LESSON 5. CLINICAL MANIFESTATIONS, DIAGNOSIS, DIFFERENTIAL DIAGNOSIS, PLANNING OF PERIODONTAL DISEASE TREATMENT WITH RAPIDLY PROGRESSIVE COURSE.

The questions to be studied for the learning of the topic:

1. Definition of rapidly progressive forms of periodontitis. Classification.
2. Risk factors for aggressive forms of periodontitis
3. The main clinical features of prepubertal periodontitis.
4. Main clinical features, pathogenesis of LAP
5. Main clinical features, pathogenesis of GAP
6. Treatment of patients with rapidly progressive periodontitis.

Question 1. Definition of rapidly progressive forms of periodontitis. Classification.

Aggressive periodontitis (rapidly progressive periodontitis) is characterized by the rapid loss of attachment and bone loss occurring in an otherwise clinically healthy patient with the amount of microbial deposits inconsistent with disease severity and familial aggregation of diseased individuals.

1) The 1989 World Workshop in Clinical Periodontics recommended the following categories of periodontitis. This system is the one most familiar to a majority of clinicians.

I. Adult periodontitis
II. Early-onset periodontitis
    A. Prepubertal periodontitis
        1. Generalized
        2. Localized
    B. Juvenile periodontitis
        1. Generalized
        2. Localized
    C. Rapidly progressive periodontitis (postjuvenile)
III. Periodontitis associated with systemic disease
IV. Necrotizing ulcerative periodontitis
V. Refractory periodontitis

2) The American Academy of Periodontology convened the 1999 International Workshop for a Classification of Periodontal Diseases and Conditions to reassess the disease classification system that was developed by the 1989 World Workshop in Clinical Periodontics(partly).

II. Chronic Periodontitis.
    A. Localized
    B. Generalized
III. Aggressive Periodontitis.

A. Localized
B. Generalized

Aggressive periodontitis was formerly classified as early onset periodontitis, i.e. localized juvenile periodontitis (LJP) has been changed to localized aggressive periodontitis; generalized aggressive periodontitis was previously classified as generalized juvenile periodontitis (GJP) and rapidly progressive periodontitis (RPP).

Aggressive Periodontitis (Juvenile Periodontitis). Definition was given by Baer, who described it, "as a disease of the periodontium occurring in an otherwise healthy adolescents, which is characterized by a rapid loss of alveolar bone around more than one tooth of the permanent dentition".

A more recent definition by Genco et al in 1986 describes localized juvenile periodontitis as a disease occurring in otherwise healthy individuals under the age of 30 years with destructive periodontitis localized to the first permanent molars and incisors not involving more than two other teeth.

Generalized juvenile periodontitis is defined as destructiveperiodontitis in individuals under the age of 30 years affecting more than fourteen teeth, i.e. generalized to an arch or an entire dentition.

The amount of destruction is not commensurate with the amount of local irritants. It exists in two forms: a. Localized and b. Generalized.

1. Previously classified as generalized juvenile periodontitis (GJP) and rapidly progressive periodontitis (RPP).
2. Usually affects individuals under the age of 30, but older patients may also be affected.
3. They usually produce a poor antibody response to the pathogens present.
4. Two types of gingival responses may be seen, one is a severe form, which is characterized by acute inflammatory changes in the gingival tissue. Other cases, the gingival tissue may appear pink, free of inflammation but in the presence of deep pockets.

Question 2. Risk factors for aggressive forms of periodontitis

Microbiologic Factors. A. actinomycetemcomitans has been implicated as the primary pathogen associated with this disease. Microscopically, the lesions of localized aggressive periodontitis have revealed bacterial invasion of connective tissue that reaches the bone surface. These invading bacteria have been identified as A. actinomycetemcomitans, Capnocytophaga sputigena, Mycoplasma subspecies and Spirochetes.

Immunologic Factors. Some of the immune defects that have been implicated in the pathogenesis of localized aggressive periodontitis are:

1. Approximately 75 percent of patients with localized aggressive periodontitis (LAP) have dysfunctional neutrophils, which are seen as decreased in the chemotactic response to several chemotactic agents,

including the complement component C5a, N-formylmethionyl leucylphenylalanine (FMLP) and leukotriene B4. The defect is also associated with a 40 percent deficiency in glycoprotein, GP110, on the neutrophil surface.

2. Patients with LAP demonstrate a strong antibody response to A. actinomycetemcomitans which explains the limitation of the infection. In LAP the dominant serum antibody is IgG2 type which is specific to antigens of A. actinomycetemcomitans.
3. In generalized form of aggressive periodontitis diverse microbial patterns including organisms associated with chronic periodontitis have been implicated. Host response is often characterized by defects in either neutrophils or monocytes.

Genetic Factors. Some of the above mentioned immunologic defects seen in aggressive periodontitis may have a genetic basis, i.e. familial clustering of neutrophil abnormalities may be seen. It has been suggested by some authors that, a major gene plays a role in aggressive periodontal disease, which could be transmitted through an autosomal dominant mode of inheritance.

Environmental Factors. Smoking is one of the factors that can influence the extentof destruction seen in young patients. Especially, smokers with generalized aggressive periodontitis exhibit more number of teeth affected by loss of clinical attachment than non-smokers with generalized aggressive periodontitis.

Question 3. The main clinical features of prepubertal periodontitis.

Prepubertal periodontitis occurs in localized and generalized forms.

1. Commonly seen in children below the age of 11 years.
2. Generally seen affecting the deciduous and mixed dentition.
3. Associated with underlying systemic disorders like Papillon -le Fevre syndrome, Down syndrome and Neutropenia.
4. Extensive destruction of bone both in the anterior and posterior teeth.

Localized Prepubertal Periodontitis:	✓ The age of onset is approximately 4 years. ✓ Plaque levels are usually low. ✓ Alveolar bone loss is rapid. ✓ Defect in neutrophil or monocyte functions has been reported.

Generalized Prepubertal Periodontitis:	✓ Entire width of attached gingiva appears to be fieryred. ✓ Gingival hyperplasia, cleft formation and recession. ✓ Rapid destruction of the alveolar bone. ✓ Systemic involvement like recurrent bacterial infections. ✓ Defects in polymorphonuclear leukocytes and monocytes.

Question 4. Main clinical features, pathogenesis of localized aggressive periodontitis (localized juvenile periodontitis).

Age and Sex Distribution. Affects both the sexes and is seen mostly between puberty and 20 years of age. Some studies show predilection to female patients.

Distribution of Lesions. Three areas of localization of bone loss have been described:

1. First molar and/or incisors.
2. First molar and/or incisors + additional teeth (not exceeding 14 teeth).
3. Generalized involvement.

For localized juvenile periodontitis, classic distribution is in the first molars and incisors with least destruction in the cuspid, premolar area.

Limitations of destruction to certain teeth could be for the following reasons:

- ✓ Production of opsonizing antibodies against A. actinomycetemcomitans called burn-out phenomenon.
- ✓ Bacteria antagonistic to A. actinomycetemcomitans may develop, thereby decreasing the number of colonization sites.
- ✓ actinomycetemcomitans may loose its leukotoxin producing ability for unknown reasons.
- ✓ Localization of the lesions could also be due to the defect in cementum formation (hypoplastic/aplastic cementum).

Clinical Findings.

1. The most striking feature is lack of clinical inflammation despite the presence of deep periodontal pockets.

2. There is a small amount of plaque, which forms a thin film on the tooth and rarely mineralizes to become calculus.

3. Most common initial symptoms are mobility and migration of first molars and incisors. Classically, a distolabial migration of the maxillary incisors with diastema formation occurs, lower incisors rarely migrate compared to upper incisors, all changes followed by sequelae of migration are seen.

4. As the disease progresses, other symptoms like root surface sensitivity, deep dull radiating pain, periodontal abscess formation and regional lymph node enlargement may occur.

Radiographic findings:

1. Vertical/angular bone loss around, the first molars and incisors in an otherwise healthy teenagers is a diagnostic sign of classic Juvenile periodontitis (J.P.) "Arc-shaped" loss of alveolar bone extending from the distal surface of the 2nd premolar to the mesial surface of the 2nd molar is seen.

2. Frequently, bilaterally symmetrical patterns of bone loss occurs, called as "mirror image pattern". Pathogenesis of aggressive periodontitis is due to an interplay of several factors, these include the specific microbiology of subgingival plaque, defects in cementum, hereditary factors, impaired PMNs function and disorders of the immune system.

Pathogenesis of LJP is related to the interplay of several factors. These include the specific microbiology of subgingival plaque, defects in cementum, hereditary factors, impaired PMN functions and disorders of the immune system.

Histopathology/Microscopic Features

These are the same as those seen during pocket formation.

- Like ulcerated pocket epithelium.
- Accumulation of various inflammatory cells in the connective tissue mainly leukocytes, plasma cells and small number of lymphocytes and macrophages.
- Electron microscopic studies of juvenile periodontitis revealed bacterial invasion of connective tissue that reaches the bone surface.
- Two types of bacteria are considered to be pathogens in localized aggressive periodontitis - A. actinomycetemcomitans and Capnocytophaga. A. actinomycetemcomitans is a short, facultatively anaerobic, non-motile Gram-negative rod.

Virulence factors produced by A. actinomycetemcomitans are as follows:

a. Leukotoxin - destroys polymorphonuclear leukocytes (PMNs) and macrophages.
b. Endotoxin - activates host cells to secrete inflammatory mediators (PG's, IL1b, TNFa).
c. Bacteriocin - may inhibit the growth of beneficial species.
d. Immunosuppressive factors - may inhibit IgG and IgM production.
e. Collagenase - causes degradation of collagen.
f. Chemotactic inhibition factors - may inhibit neutrophil chemotaxis.

Immunologic findings: Immune defects that have been implicated in the pathogenesis of localized aggressive periodontitis are functional defects of polymorphonuclear leukocytes/monocytes, thereby it impairs the

chemotactic attraction of PMNLs (polymorphonuclear leukocytes) to the site of infection.

Question 5. Main clinical features, pathogenesis of generalized aggressive periodontitis

Previously classified as Generalized Juvenile Periodontitis (GJP) and Rapidly Progressive Periodontitis (RPP) (21-35 years).

Generalized aggressive periodontitis is usually characterized by 'generalized interproximal attachment loss affecting atleast three permanent teeth other than first molars and incisors.' Patients with generalized aggressive form may exhibit minimal amounts of microbial plaque associated with the affected teeth, i.e. quantitatively, the amount of plaque seems to be inconsistent with the amount of periodontal destruction; qualitatively, most pathogenic organisms may be associated, e.g. Porphyromonas gingivalis, A. actinomycetemcomitans, and Bacteroids forsythus.

Clinical Characteristics

a. Age and sex distribution: It affects persons between puberty and 35 years (but may be older). No sex discrimination is seen.
b. Distribution of lesion: No specific pattern is observed, all or most of the teeth are affected.
c. Two types of gingival responses, may be seen in generalized aggressive periodontitis. One is severe, acutely inflamed tissue which is often proliferating, ulcerated and fiery red, spontaneous bleeding and suppuration are commonly seen. In the other cases, the gingival tissue may appear pink and free of inflammation but deep pockets can be demonstrated by probing.
d. Some of the patients may have systemic manifestations such as weight loss, mental depression and general malaise.

Radiographic Findings. No definite pattern of distribution occurs but the radiographic picture can range from severe bone loss associated with the minimal number of teeth, to advanced bone loss affecting the majority of teeth in the dentition.

Question 6. Treatment of patients with rapidly progressive periodontitis.

1. Treatment LAP. Prognosis is no more considered as poor for patients with aggressive periodontitis. The following treatment has been tried in the past with varying results:

1. Extraction: Extraction of involved teeth especially first molars results in uneventful healing. Transplantation of developing third molars into the sockets of previously extracted 1st molars has been tried but with limited success.

2. Standard periodontal therapy: Includes scaling, root planing, curettage, flap surgery with/without bone grafts, root amputation,

hemisection, occlusal adjustment and strict plaque control has been tried. However, response is unpredictable and frequent maintenance visits are a must.

3. Antibiotic therapy: Several authors reported successful results using antibiotics as adjuncts to standard therapy.

- Genco and coworkers reported scaling and root planing and tetracycline 250 mg q.i.d for 14 days every 8 weeks.
- Several other investigations have also noticed excellent bone fill in cases of localized juvenile periodontitis treated with tetracycline, flap surgery and placement of grafts.

Local application of antibacterial drugs in the treatment of rapidly progressive periodontitis has a good therapeutic effect. Controlling the increase in plaque from the patient with antiseptic antibacterial agents reduces the activity of the microbial factor in the rapidly progressive periodonet. One of the most popular and common local antimicrobial agents are gels with antibacterial drugs.

The penetration of rinsers into deep periodontal pockets is limited and, consequently, ineffective. Delivery of chemotherapeutic substances in the periodontal pocket with the use of a variety of delivery systems: threads, ointments, chips, gels, collagen fiber matrices. As an active substance in these systems, antimicrobials are used.

Current Approach to Therapy

- In almost all cases, systemic tetracycline hydrochloride 250 mg qid for atleast a week should be given in conjunction with local mechanical therapy. If surgery is indicated, systemic antibiotics are advised with patient instructed to begin taking the antibiotic approximately 1 hour before surgery.
- Doxycycline 100 mg/day may also be used.
- Chlorhexidine rinses should be prescribed.
- In refractory cases, tetracycline resistant Actinobacillus species have been suspected. In such cases, a combination of amoxicillin and metronidazole has been suggested.

It also includes full mouth disinfection which has been proposed by Quirynen et al. Since it was observed that A. actinomycetemcomitans has the ability to translocate from one person to another and from site to site, full mouth disinfection was implemented. <u>It includes the following steps</u>:

a. Full mouth scaling and root planing (in 2 sessions within 24 hours).
b. Brushing the dorsum of the tongue with an antimicrobial agent (1% chlorhexidine gel) for one minute.
c. Mouthrinsing with antimicrobial agents.
d. Home irrigation systems.

2. Treatment of generalized aggressive periodontitis.

Treatment: in the past, the prognosis for juvenile periodontitis was considered to be poor.

Current therapy: systemic tetracycline hydrochloride 250 mg q.i.d. for at least 1 week should be given in conjunctionт with the local mechanical therapy. Several reports have shown excellent bone fill in cases of LJP treated with tetracycline, flap surgery and placement of grafts.

In refractory cases, tetracycline resistant A.a. has been suspected. In such cases a combination of amoxicillin and metronidazole has been suggested.

Test control

1. What microorganisms have the leading role in the etiopathogenesis of the rapidly progressive forms of periodontitis?

a) Gram-negative aerobes
b) Gram-positive aerobam
c) Gram-negative anaerobes
d) Gram-positive anaerobes

2. Does cellular and humoral immunity disorders play an important role in the pathogenesis of RPP?

a) Yes
b) No

3. Is it true that RPP is autoimmune?

a) Yes
b) No

4. Indicate the age when prepubertal JP occurs:

a) Until 11 years old
b) 11-13 years
c) 21-35 years old

5. Indicate the age when post-juvenile periodontitis occur:

a) Until 11 years old
b) 11-13 years
c) 21-35 years old

6. Select the appropriate signs that characterize the clinical picture in prepubertal JP:

a) Develops in a temporary or mixed bite
b) Can be localized and generalized

c) The disease is accompanied by otitis media of the middle ear, skin manifestations
d) boys and girls get sick the same
e) All of the above

7. Select the appropriate signs that characterize the clinical picture with a localized form of JP:

a) Lesions in the area of 1-2 teeth
b) Lesions of all groups of teeth
c) Women are sick more often than men
d) Men are sick more often than women
e) On Rg: arcuate defect of the bone tissue of the alveolar process in the region of the first molar

8. Select the appropriate signs that characterize to the clinical picture in the generalized form of JP:

a) Periodontitis affects all groups of teeth
b) Absence of clinical inflammation in the presence of deep pockets
c) Migrating the upper incisors to form a diastema
d) All of the above

9. Select the appropriate signs that characterize the clinical picture in post-juvenile periodontitis:

a) Clinically frequent exacerbations
b) Exacerbations are accompanied by high fever, weight loss, depressive conditions
c) The disease occurs with different degrees of severity of concomitant gingivitis
d) All of the above

10. The treatment plan for patients with RPP includes the following methods:

a) Surgical
b) Antibiotic therapy with a preliminary study of microflora
c) Clinical examination of patients and their relatives
d) Regular examination and quality of individual and preventive hygiene
e) All of the above

LESSON 6. RELATIONSHIP OF PERIODONTAL DISEASE WITH SYSTEMIC DISEASES. SYMPTOMATIC PERIODONTITIS AS MANIFESTATION OF SYSTEMIC DISORDERS: MINERAL METABOLISM AT OSTEOPOROSIS, BLOOD DISORDER, IMMUNODEFICIENCY, ENDOCRINE DISCORRELATIONS IN DIABETES.

The questions to be studied for the learning of the topic:

1. The relationship of periodontal disease with systemic diseases.
2. The symptomatic periodontitis as a manifestation of disorders of mineral metabolism osteoporosis
3. The symptomatic periodontitis as a manifestation of disorders in blood diseases
4. The symptomatic periodontitis as a manifestation of irregularities in immunodeficite condition.
5. The symptomatic periodontitis as a manifestation of disorders in endocrine discorrelation diabetes.

Question 1. The relationship of periodontal disease with systemic diseases.

The influence of periodontitis on the development and course of systemic pathology can be noted as caused by:

- bacterial flora of the oral cavity, in response to the action of which develops the inflammatory reaction;
- local clinical symptoms of periodontitis - bleeding, decrease in attachment level, periodontal pockets and suppuration of them, loss alveolar- bones.

There are the following basic pathogenetic mechanisms each of which to a greater or lesser extent, explain the role of periodontitis as a risk factor in the development of systemic diseases:

1. The reflex mechanism. The leading role is withdrawn and the infectious agents (microbes and toxins) which creates the focus of the receptive fields of irritation. Last by reflex through the centers of the brain cause dysfunction of the autonomic nervous system and regulation of work of internal organs and body systems with the subsequent development of functional and degenerative changes.

2. The suppuration. Constant contact with the digestive tract and upper respiratory tract purulent exudate, eye-catching and periodontal pockets, can have adverse influence on the development and clinical picture of diseases of digestive and respiratory disorders systems.

3. The low level of immunological reactivity of human body, in which the possible penetration of microorganisms from the periodontal foci of infection in the blood stream (sepsis).

4. The toxemia. The flow of toxins into the blood of microorganisms present in the hearth chronical infection leads to a prolonged low-grade fever, changes in blood picture and other clinical symptoms characteristic of a chronic intoxication.

5. The allergic mechanisms. Microorganisms in the foci of infection, causing the sensitization of the human body and the formation of specific antibodies. In secondary infection of the body the same kind of germs there are more symptoms of the inflammatory response in periodontal tissues and other organs.

6. The autoimmune mechanism. Blood appear antigens tissue origins, which are formed as a result of tissue damage and protein breakdown. The cytotoxins as autoantigens also change the sensitivity of the human body and cause sensitization. In periodontal tissues in response to the impact of microflora transpire synthesis of cytokines (prostaglandins, interleukin-new (IL-1, IL-6), tumor necrosis factor (TNF), gamma interferon) that affect the development of inflammation both locally and systemically.

7. The endotoxins. Inflammatory processes localized in the maxillofacial region (including the periodontium) are accompanied by endogenous intoxication. This is due to a factor in blood fractions of endotoxins lysosomal origin, products of tissue decay, bacterial toxins and other toxic products.

The periodontal disease can affect pregnancy, diabetes, cardiovascular, pulmonary and other diseases.

Question 2. The symptomatic periodontitis as a manifestation of disorders of mineral metabolism, osteoporosis.

According to the classification of diseases of the periodontium (L. N. Dedova, 2002-2012), periodontitis associated with systemic pathology, represented a form of symptomatic periodontitis.

The symptomatic periodontitis is a group of diseases of the periodontium in which pathologists-clinical changes of periodontal tissue are a symptom of systemic diseases.

The symptomatic periodontitis as a manifestation of systemic diseases compared with other pathological processes in periodontal tissues is accompanied by more complex and deep disturbances in the biological system of the periodontium. Symptomatic periodontitis is characterized by a feature of the course, active involvement of the whole complex of periodontal tissue, inadequate reaction to the traditional treatment, the need to actively support this therapy.

Osteoporosis is a common systemic disease of the skeleton characterized by low bone mass and impaired bone and blood microcirculation, leading to increased bone fragility and fracture risk. In recent decades, this problem has acquired special significance due to two

closely related demographic processes: a rapid increase in the population of elderly and old people, particularly women in the postmenopausal period of life. It is known that estrogen deficiency characteristic of postmenopausal osteoporosis, accelerates the processes of remodeling, contributes to the imbalance between bone resorption and bone formation, accelerated bone loss, development of osteoporosis and its complications.

In the first 5-10 years after the menopause osteoporosis is a major clinical manifestation of the periodontal diseases. Bone mass the human skeleton is reduced by 2-5% per year, and by the age of 60-70 is reduced by 25-30%. Gingivostomatit this period resembles desquamation, and postradiation after irradiation of the gonads. The mucous membrane of the gums and other parts of the mouth dry, shiny, has different shades ("marbling" pattern), easily injured and bleeding. Hypersensitivity and trauma do not allow patients to use dentures. Decrease in bone mass of the skeleton contributes to reducing the height of the interdental bone septum, reduction of attachment of the gums.

Question 3. The symptomatic periodontitis as a manifestation of disorders in the blood diseases

The symptomatic periodontitis on the background of leukemia. In patients with leukemia symptom practical periodontal disease accompanied by bleeding, ulceration, hyperplasia of the gums or formation of periodontal pockets.

Bleeding gums is often found in patients even in the absence of clinical manifestations of gingivitis. Bleeding may be an early manifestation of leukemia develops due to thrombocytopenia. The tendency for bleeding and hemorrhages can occur on the skin and mucous membranes. These symptoms can also appear after chemo-therapy.

The hyperplasia of the gums develops as a result of chronic inflammation due to the action of microorganisms of dental plaque or infiltration by leukemic cells. Hyperplasia of the gums starts with interdental papillae, level of marginal and attached gums, pocket formation. They accumulate bacterial plaque, which causes the relapse of the inflammatory process. This contributes to the further development of hyperplasia of gums.

The ulceration of the gums. Symptomatic periodontitis leukemia is accompanied by ulceration gums. Along with localization on the gums ulcers precaut deep submucosal layers to spread in the mouth.

The symptomatic periodontitis on the background of agranulocytosis. On the background of acute onset, characterized by fever, weakness, lesions of the throat, ulceration in the oral cavity and oropharynx, involving the tissues of the periodontium. Local inflammatory process affects the periodontium in the form of hemorrhages and ulcerations, necrosis of the

marginal and attached gingiva. This is accompanied by a generalized subalveolar bone destruction.

Question 4. The symptomatic periodontitis as a manifestation of irregularities in immunodeficiency condition.

The symptomatic periodontitis in HIV-infected patients is expressed Toms hemophilia, congestive hyperemia, acute and chronic ulcerative electrictions of the gums, irregular generalized bone resorption.

The symptomatic periodontitis in HIV-infected patients has a comprehensive necrosis of the gums, rapid destruction of periodontal ligament and bone with a tendency to the formation of deep intraosseous pockets, which can lead to exposure and sequestration subalveolar bones.

In AIDS patients with microbiological examination of the contents of periodontal pockets can be detected by atypical microorganisms (C. albicans, enteric pathogens).

Along with symptomatic periodontitis in the oral cavity of HIV-infected patients may have the following manifestations: hairy leukoplakia, candidiasis (including which restores cheilitis), viral infections (herpes, papillomavirus, Epstein - Barr, cytomegalovirus), sarcoma Caposhes, non-Hodgkin's lymphoma, recurrent aphthae, atypical ulceration, diseases of the salivary glands.

Question 5. The symptomatic periodontitis as a manifestation of disorders in endocrine discorrelation diabetes.

The development of the changes in the periodontium of diabetic contribute to angiopathy and increase vascular permeability, changes in the peripheral nerve endings and metabolic disorders in the tissues (Belyakov Y. A., 1983). For insulin-dependent diabetes in the oral cavity has a variety of nonspecific clinical symptoms; dryness of mucous membranes, burning sensation, cracked lips, language, the imputation of hard tissues of teeth (violation of terms of eruption, hypersensitivity, pain when chewing and percussion, enamel hypoplasia). In type II diabetes these changes can appear only in severe course.

Very quickly catarrhal gingivitis goes into a deep destructive lesions of the periodontal. It is also mentioned vertical type resorption of interdental partitions. The formation of a periodontal pocket is accompanied by abundant growth of granulation tissue, purulent secretions, mobility, displacement, and rapid loss of teeth. The defeat of the periodontium becomes generalized. Develop often exacerbated periodontal abscesses.

Speaking about the possible mechanisms of increased risk of development of periodontal disease in diabetes, it should be remembered that:

1) vascular disorders due to increased thickness of basal membrane microvessels, their obliteration, leading to tissue ischemia with disorders of reparative Regenertion and protective mechanisms, and of particular importance in the development of degenerative degenerative disorders;
2) bacterial colonization and invasion are likely to worsen, although pathogenic microflora in DM is the same as without it;
3) chemotaxis and phagocytic capacity of neutrophils, reflecting the dysfunction of neutrophils, can be suppressed, especially when a poor metabolic control of the disease;
4) collagen synthesis is suppressed in diabetes, and its disintegration under the action of collagenase strengthened suppressed the growth and proliferation of fibroblasts, formation of their matrix of bone tissue. This leads to disruption of normal reparative processes;
5) the degree of increase in gingival fluid levels of markers of activation of polymorphonuclear leukocytes (elastase, glucuronidase) was significantly higher than in patients without diabetes.

Perhaps there are other mechanisms of the system (e.g., other endocrine disorders) and is of local nature, associated with diabetes mellitus and is involved in the expression of the periodontal complex.

Test control

1. The influence of periodontitis on the development and course of systemic pathology may be determined:

a) bacterial microflora of the oral cavity, in response to the action of which develops the inflammatory reaction;
b) local clinical manifestations of periodontitis;
c) the climatic conditions of the place of residence.

2. The main pathogenetic mechanisms of the role of periodontitis as a risk factor in the development of systematic diseases are:

a) age;
b) toxaemia;
c) allergic mechanisms;
d) autoimmune mechanism.

3. The dentist's role in the treatment of periodontitis associated with systemic diseases is as follows

a) treatment of the underlying systemic disease;
b) normalization of individual oral hygiene;
c) treatment of periodontal lesions in cooperation with relevant specialists.

4. Gingivostomatitis during menopause with osteoporosis looks like:

a) desquamative;
b) postradiation after irradiation of the gonads;
c) ulcerative necrotic.

5. Common signs of patient's treatment with periodontitis pathology associated with systemic diseases include:

a) mandatory treatment planning;
b) a radical position regarding dental treatment with a controversial prognosis;
c) high level of motivation of the patient and his manual skills;
d) cooperation with the dentist, including carers for patients with disabilities;
e) all of the above.

6. Indicate the factors that are taken into account during the planning of patient's treatment with periodontitis, associated systemic diseases.

a) the age of the patient;
b) the condition of the oral cavity;
c) previous experience of treatment.
d) all of the above

7. Features of treatment of periodontitis symptomatic patients with blood diseases (acute and chronic leukemia) are:

a) the conducting of periodontal treatment 1 day before chemotherapy and radiation therapy;
b) planning reconstructive (surgical) methods of treatment;
c) treatment dental measures taking into account the recommendations of the doctor-hematologist with the treatment of underlying disease;
d) the application of periodontal dressings only after a full stop of bleeding;
e) carrying out physiotherapeutic treatment.

8. Indicate the features of treatment of symptomatic periodontitis in patients with HIV-infection and AIDS:

a) cooperation with an immunologist, infectious disease specialist, etc.;
b) planning reconstructive (surgical) methods of treatment;
c) treatment dental measures taking into account the recommendations of an infectious disease specialist with the treatment of the underlying disease;
d) sparing and atraumatic carrying out of medical manipulations;
e) mandatory conducting of physiotherapy treatment.

9. Indicate the clinical symptom characteristic of the periodontal lesions associated with diabetes:

a) dental plaque without tendency to mineralization;
b) the absence of gingivitis;
c) the frequent occurrence of periodontal abscesses.

10. Treatment features of the symptomatic periodontitis in patients with insulin-dependent uncontrolled diabetes mellitus are as follows

a) conducting therapeutic interventions in the morning after breakfast;
b) use anaesthetics containing adrenaline
c) planning reconstructive (surgical) methods of treatment;
d) treatment dental measures taking into account the recommendations of an endocrinologist with the treatment of the underlying disease;
e) mandatory conducting of physiotherapy treatment.

LESSON 7. OCCLUSIVE TRAUMA IN PERIODONTOLOGY.

The questions to be studied for the learning of the topic:

1. Trauma from occlusion. Definition and terminology.
2. Etiology and pathogenesis primary occlusal trauma and secondary occlusal trauma.
3. Role of the trauma from occlusion in the progression of periodontal disease.
4. Trauma from occlusion. Histologic changes.
5. Clinical signs and symptoms of occlusal traumatism.
6. Trauma from occlusion: treatment plan.
7. Occlusal adjustment and occlusal appliance therapy.
8. Splints in periodontal therapy.

Question 1. Trauma from occlusion. Definition and terminology

Occlusion: It is defined as the functional relationship between the components of the masticatory system including the teeth, supporting tissues, neuromuscular system, temporomandibular joints and craniofacial skeleton.

Intercuspal position (ICP): The position of the mandible when there is maximal intercuspation between the maxillary and mandibular teeth.

Excursive movement: Any movement of the mandible away from ICP.

Protrusion: Movement of the mandible anteriorly from ICP.

Retrusion: Movement of the mandible posteriorly from ICP.

A physiologic occlusion: It is when no signs of dysfunction or disease are present and no treatment is indicated.

A non-physiologic (or traumatic) occlusion: It is associated with dysfunction or disease due to tissue injury, and treatment may be indicated.

A therapeutic occlusion: It is the result of specificint erventions designed to treat dysfunction or disease.

According to Orban and Glickman et al (1968): Trauma from occlusion is defined as, when occlusal forces exceed the adaptive capacity of periodontal tissues, the tissue injury results. This resultant injury is termed as trauma from occlusion.

WHO in 1978 defined trauma from occlusion as “damage in the periodontium caused by, stress on the teeth produced directly or indirectly by the teeth of the opposing jaw”.

Other terms often used are, traumatizing occlusion, occlusal trauma, occlusal overload, periodontal traumatism, occlusal disharmony, functional imbalance and occlusal dystrophy. One must note that trauma from occlusion refers to the tissue injury, not the occlusal force. An occlusion that produces such an injury is called as traumatic occlusion.

In occlusal traumatism, the etiologic factor is any force in excess of the adaptive capacity of the periodontium, the morbid pathobiologic event is

injury within the periodontal ligament and alveolar bone, and the signs and symptoms are pain, mobility and/or fremitus, pathologic migration of teeth, excessive occlusal wear, and widening of the periodontal ligament space in radiographs.

As defined in the Glossary of Periodontal Terms, occlusal traumatism is the "functional loading of teeth" (force is primary etiologic factor), usually off - axis, that is of sufficient magnitude (excess of the adaptive capacity) to induce changes to the teeth (eg, fractures, occlusal wear) or supporting structures (inflammation in the periodontal ligament and alveolar bone, also known as the "lesion of trauma from occlusion"). The changes may be temporary (reversible) or permanent (irreversible).

Different Types of Occlusal Forces

a. Normal or physiological occlusal forces which rarely exceeds 5N which is required to provide positive stimulus to maintain the periodontium.
b. Continuous forces/very low forces (e.g. orthodontic forces).
c. Impact forces-which are mainly high forces but of short duration, if it is beyond the adaptive capacity, may result in tooth fracture.
d. Jiggling forces are considered to be most traumatic, e.g. high fillings, premature contacts on crowns and fillings.

Adaptive capacity is the ability of the teeth and tissues of the periodontium to sustain the effects of, or adapt to, forces acting on the periodontium without injury. The adaptive capacity is affected quantitatively and qualitatively by local and systemic contributing factors. When it is exceeded, occlusal traumatism occurs.

The etiologic forces that produce occlusal traumatism may not always be occlusal in nature, but they may be generated by orthodontic or prosthodontic appliances and/or habits of compulsion, such as pipe smoking or fingernail biting. The more inclusive designation, periodontal traumatism, is preferred by some over occlusal traumatism because it allows for nonocclusal forces and occlusal foroes as etiologic factors.

Types:

1. Depending on the onset and duration.
2. Depending on the cause:
    a. Due to the alterations in the occlusal forces.
    b. Reduced capacity of the periodontium.
3. Depending on the onset and duration:
    a. Acute trauma from occlusion (TFO).
    b. Chronic trauma from occlusion (TFO).

Acute trauma from occlusion: Results from the abrupt changes in the occlusal forces, such as that produced by biting on a hard object, in addition, could also be due to iatrogenic factors (faulty restorations/prosthetic appliance).

Chronic trauma from occlusion: As a result of the gradual changes produced in the periodontium due to the tooth wear, drifting movement, extrusion of the teeth combined with parafunctional habits such as bruxism and clenching.

4. Depending on the cause

Table 21. Types of TFO: Depending on the cause

Primary trauma from occlusion	*Secondary trauma from occlusion*
It is a tissue injury, which is elicited around a tooth with normal height of periodontium. For example: Insertion of high fillings, insertion of the prosthetic replacement, orthodontic movement in functionally-unacceptable positions.	It is related to situations in which occlusal forces cause injury in a periodontium of reduced height. For example: Periodontitis.

In summary,

1. The criterion that determines whether an occlusion is traumatic is whether it produces periodontal injury, nothow the teeth occlude.
2. Any occlusion that produces periodontal injury is considered traumatic.
3. Malocclusion is not necessary to produce trauma.

Question 2. Etiology and pathogenesis primary occlusal trauma and secondary occlusal trauma.

The injury to the periodontium caused by forces acting on teeth is called the lesion of trauma from occlusion or more simply, occlusal trauma.

The lesion of trauma from occlusion is located within the periodontal ligament in areas where the ligamentis either under pressure (crushed) or under tension (torn) (figure 64).

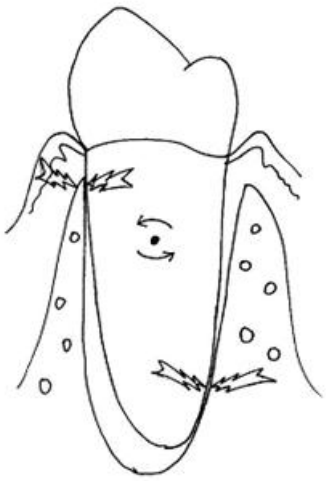


Figure 64. A line drawing showing the reciprocal areas of crush and tear (lightning bolts) of a periodontal ligament when excessive non-axial forces are applied to a tooth. The tooth rotates within the alveolus around a point center of rotation located within the root.

The crush or tear produces a physical injury resulting in local necrosis of the periodontal ligament and a typical inflammatory response. The histologic appearance of the periodontal ligament is described as "hyalinization", or a decrease in the cellular component of the tissue.

Resorption of nearby alveolar bone also occurs as an outcome of the inflammation. The resorption will occur on the periodontal ligament side of the alveolar bone proper with mild injury (frontal resorption) and/or on the marrow surfaces of the supporting alveolar bone (rear resorption). The degree of necrosis, inflammation, and resorption will depend upon the amount of force acting on the teeth and the adaptive capacity of the periodontium

Primary occlusal trauma is the injury resulting in tissue changes (injury to the attachment apparatus) from excessive (in excess of the normal adaptive capacity of the periodontium) occlusal (and other) forces to a tooth or teeth with a healthy, anatomically normal, periodontium in a systemically well patient. Primary occlusal trauma is usually reversible once the forces that produced it are controlled (figure 65).

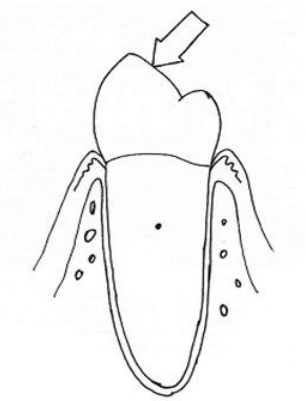


Figure 65. A line drawing showing the morphological requirements for primary occlusal trauma. There are excessive non-axial forces acting on a tooth with a normal, healthy periodontal attachment apparatus. The crown to root ratio is 1:2 and the point center of rotation is in the coronal 1/3 of the root.

Secondary occlusal trauma is the injury resulting in tissue changes (injury to the attachment apparatus) from normal or excessive (in excess of the reduced adaptive capacity of the periodontium) occlusal (and other) forces to a tooth or teeth with reduced support (figure 66). Because these same contributing factors that "reduced" the adaptive capacity of the periodontium may be difficult to control or change, secondary occlusal trauma is difficult to reverse following force control.

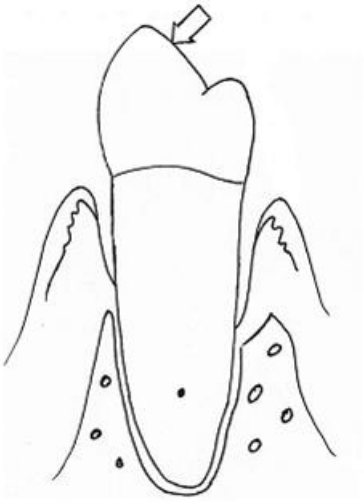


Figure 66. A line drawing showing the morphological requirements for secondary occlusal trauma. There are normal nonaxial forces acting on a tooth with a periodontal attachment apparatus that has been reduced by periodontiis. The crown to root ratio is 2:1 and the point center of rotation is in the apical 1/3 of the root.

Question 3. Role of the trauma from occlusion in the progression of periodontal disease.

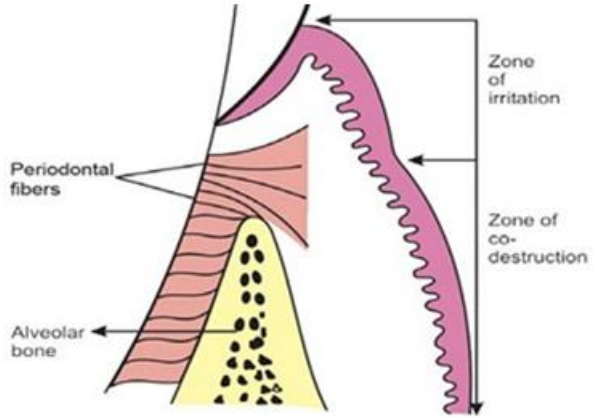


Figure 67. Zone of irritation and zone of co-destruction

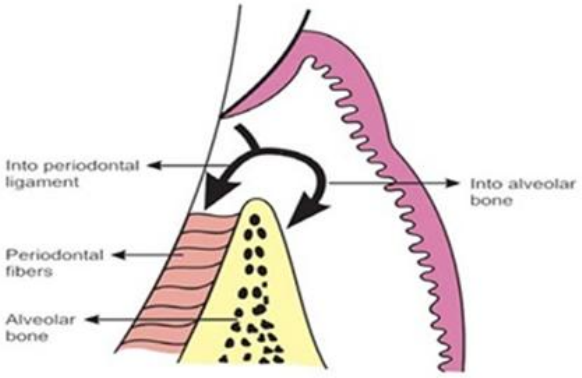


Figure 68. Pathway of inflammatory process
The inflammatory lesion in the zone of irritation can in tooth not subjected to trauma, propagate into the alveolar bone, while in tooth also subjected to trauma from occlusion, the inflammatory infiltrate spreads directly into periodontal ligament

Role of trauma from occlusion in the progression of periodontal disease is explained by two concepts, (a) Glickman's concept supports trauma from occlusion that is a co-destructive factor of importance, especially in situations where angular defects combined with infrabony pockets are found in one or several teeth.

Where as Waerhaug's concept refutes this hypothesis and proposes that, angular defects and infrabony pockets occur often at sites not affected by trauma from occlusion. He proceeds to explain, that, angular defects occur, when the subgingival plaque of one tooth reaches a more apical level than the plaque on the neighboring tooth and also when the volume of the alveolar bone surrounding the roots is comparatively large.

The summary of this concept is that trauma from occlusion is a co-destructive factor of importance especially in situations where angular defects combined with infrabony pockets are found in one or several teeth.

In conclusion, four possibilities can occur when a tooth with gingival inflammation is exposed to trauma.

1. Trauma from occlusion may alter the pathway of extension of gingival inflammation to the underlying tissues. Inflammation may proceed

to the periodontal ligament rather than to the alveolar bone and the resulting boneloss would be angular with infrabony pockets.

2. It may favor the environment for the formation and attachment of plaque and calculus and may be responsible for development of deeper lesions.

3. Supragingival plaque can become subgingival if the tooth is tilted orthodontically or migrates into an edentulous area, resulting in the transformation of a suprabony pocket into an infrabony pocket.

4. Increased tooth mobility associated with trauma to the periodontium may have a pumping effect on plaque metabolites increasing their diffusion.

Other Properties.

Effect of Insufficient Occlusal Force. It may also be injurious to periodontal tissues which results in the thinning of the periodontal, ligament, atrophy of the fibers, osteoporosis of alveolar bone and reduction in alveolar bone height. Hypofunction can result from an openbite relationship, absence of functional antagonists, or unilateral chewing habits.

Reversibility of Traumatic Lesion. Trauma from occlusion is reversible. When the injurious force is removed, the repair occurs. The presence of inflammation in the periodontium as a result of plaque accumulation may impair the reversibility of traumatic lesions.

Question 4. Trauma from occlusion. Histologic changes.

The response of tissues to increased occlusal forces is explained under three stages.

- Stage 1: Injury
- Stage 2: Repair
- Stage 3: Adaptive remodeling of the periodontium.

Stage 1: Injury. When a tooth is exposed to excessive occlusal forces, the distribute, while maintaining the stability of the tooth. This may lead to certain well-defined reactions in the periodontal ligament and alveolar bone, eventually resulting in adaptation of the periodontal structures to altered functional demand. When the tooth is subjected to horizontal forces the tooth rotates or tilts in the direction of force. This tilting results in the pressure and tension zones, within the marginal and apical parts of the periodontium. Depending on the types of forces there can be many histologic changes

Stage 2: Repair. TFO stimulates increased reparative activity.

When bone is resorbed by excessive occlusal forces, the body attempts to reinforce the thinned-bony trabeculae with new bone. This attempt to compensate for lost bone is called buttressing bone formation, which is an important feature of reparative process associated with trauma from occlusion (also occurs during inflammation or tumors).

Buttressing bone formation can occur within the jaw, called central buttressing and on the bone surface, called as peripheral buttressing. It

usually occurs on the facial and lingual plates of the alveolar bone, if it produces a shelf-like thickening of alveolar bone it is referred as lipping.

Stage 3: Adaptive Remodeling of the Periodontium. If the repair process cannot keep pace with the destruction caused by occlusion, the periodontium may get remodeled in order to maintain the structural relationship. This may result in thickened periodontal ligament, angular defects in the bone with no pocket formation, loose teeth and increased vascularization.

In summary, histometric changes shown during these three stages are:

1. The injury phase shows an increase in areas of resorption and a decrease in bone formation.

2. The repair phase shows increase in areas of bone formation and decreased

3. Remodeling phase shows return of normal resorption and formation.

In the response to increased occlusal forces, the periodontal ligament gradually increases in width on both the sides of the tooth. This is associated with, (a) Inflammatory changes in the ligament tissue, (b) Active bone resorption. (c) Progressive mobility of the teeth. When the effect of the forces applied has been compensated by the increased width of the periodontal ligament space, the ligament no longer shows the above mentioned signs, except that the tooth remains hypermobile, but the mobility is no longer of the progressive type.

Question 5. Clinical signs and symptoms of occlusal traumatism.

1) In acute situations: Excessive tooth pain, tenderness on percussion, increased tooth mobility (hypermobility) is seen. In severe cases, periodontal abscess formation and cemental tears can be seen. Others such as presence of infrabony pockets, furcation involvement, attrition, pathologic migration may also be present.

2) Clinical Indicators for Trauma from Occlusion. The clinical signs and symptoms of injury in the periodontal ligament are commonly:

1. Pain or discomfort around one or more teeth on percussion, function, and/or parafunction. Pain is one of the four cardinal signs of inflammation (dolor/pain, calor/heat, rubor/redness, and tumor/swelling). Pain then is a sign of inflammation in the periodontal ligament.

2. Tooth mobility. Tooth mobility may be physiologic (ie, horizontal movement limited to the width of the periodontal ligament), or pathologic (ie, horizontal and/or vertical movement beyond the expected boundaries of the periodontal ligament). Mobility occurs when fibers of the periodontal ligament are injured or destroyed by inflammation resulting from excessive forces acting on teeth. It will also occur when the adaptive capacity of the periodontium has been altered by marginal inflammation or systemic disease.

Mobility is commonly observed when a tooth has reduced periodontal attachment.

The Miller classification scheme for tooth mobility is as follows:

- Grade (degree) I. The slightest distinguishable movement in a horizontal direction. Tooth mobility is classified as physiologic mobility.
- Grade (degree) II. Movement in a horizontal direction of a tooth within 1 mm of its normal position.
- Grade (degree) III. Movement of a tooth in a horizontal direction greater than 1 mm from its normal position. Grade III mobility also includes teeth that are depressible and/or can be rotated in their periodontal support.

3. Fremitus as determined by palpable or visible movement of teeth under vertical (axial) or horizontal (nonaxial) occlusal forces. Fremitus is detected using fingertips placed on the crowns of teeth while the patient occludes. Fremitus is "functional mobility."

4. Pathologic migration of teeth. Pathologic migration of teeth usually occurs when teeth have lost their normal periodontal support due to periodontitis and subsequently migrate from their normal position in the dentition in response to occlusal and non-occlusal.

Pathologic migration occurs most frequently in the anterior region, but posterior teeth may also be affected. The teeth may move in any direction and the migration is usually accompanied by mobility and rotation. Pathologic migration in occlusal or incisal direction is called as “extrusion”.

Pathogenesis. Two major factors play a role in maintaining the normal position of the teeth.

1. The health and normal height of the periodontium.
2. The forces exerted on the teeth.

The Health and Normal Height of the Periodontium. A tooth with weakened periodontal support is unable to withstand the forces and moves away from the opposing force. It is important to understand that the abnormality in pathologic migration rests with the weakened periodontium. The force itself need not be abnormal. Forces that are acceptable to an intact periodontium become injurious when periodontal support is reduced. Pathologic migration may continue even after a tooth no longer contacts its antagonist.

Changes in the Forces Exerted on the Teeth. Changes in the forces may occur as a result of (a) unreplaced missing teeth, (b) Failure to replace first molars, or (c) other causes. These forces do not have to be abnormal to cause pathologic migration, if the periodontium is sufficiently weakened.

a. Unreplaced missing teeth: This leads to drifting of teeth into the spaces created by unreplaced missing teeth. Drifting differs from pathologic migration, in that it does not result from destruction of the periodontal tissues. However, it usually creates conditions that leads to periodontal

diseases and thus the initial tooth movement is aggrevated by loss of periodontal support.

b. Failure to replace first molars: it consists of the following:

1. The second and third molars tilt resulting in decrease in vertical dimension.
2. The premolars move distally and the mandibular incisors tilt or drift lingually.
3. Anterior overbite is increased.
4. The maxillary incisors are pushed labially and laterally.
5. The anterior teeth extrude due to disappearance of incisal apposition.
6. Diastema is created by the separation of the anterior teeth.

Other causes:

1. Pressure from the tongue: It may either have a direct effect, that is, it may cause drifting of teeth in the absence of periodontal disease or may contribute to pathologic migration of the teeth with reduced periodontal support.
2. Pressure from the granulation tissue of periodontal pocket: It has also been listed as a contributing factor to pathologic migration. Usually tooth may return to their original position after pockets are treated, but if the destruction of the periodontium is more severe on one side of a tooth rather than the other, the healing tissue may pull the tooth in the direction of lesser destruction.
3. Tooth loss
4. Malocclusion.
5. Wear facets.
6. Posterior bite collapse. Posterior bite collapse is the product of tooth loss and pathologic migration.
7. Widened periodontal ligament spaces around affected teeth in radiographs. Widened periodontal ligament (PDL) spaces usually indicate that an adaptive response has occurred either to excessive force on a normal periodontium or to normal or excessive forces on a reduced periodontium. Widened PDL spaces together with an intact lamina dura suggest that repair occurred following injury (figure 69).

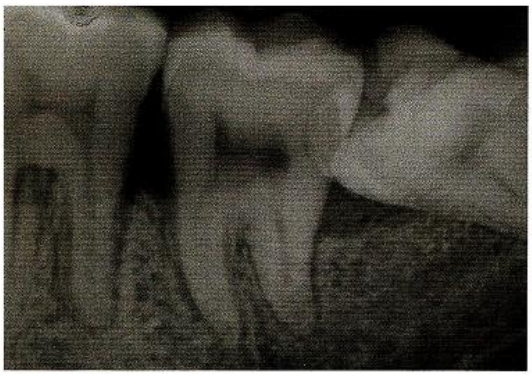


Figure 69. A radiograph of mandibular molars with severe bone loss. Widened periodontal ligament spaces in radiographs are signs of occlusal traumatism. The retained third molar is probably a contributing factor to the degree of periodontal attachment loss.

8. Fractured tooth/teeth.
9. Thermal sensitivity.
10. Muscle hypertonicity.

3) Radiographic changes

1. Increase in the width of the periodontal ligament space often with thickening of the lamina dura along the lateral borders of the root, apical and bifurcation areas.
2. “Vertical” rather than horizontal destruction of the interdental septum.
3. Radiolucency and condensation of the alveolar bone.
4. Root resorption.

Question 6. Trauma from occlusion: treatment plan.

When an adequate quantity of periodontal support is present to withstand the normal forces of occlusion, yet excessive parafunctional forces exceed the adaptive capacity of the attachment apparatus, the disease process is referred to as primary occlusal trauma. When the quantity of there maining normal attachment apparatus has been compromised by periodontal disease and cannot with stand the normal forces of occlusion, the disease process is referred to as secondary occlusal trauma.

A comprehensive plan of occlusal therapy will include any or all of the following items:

1. Occlusal adjustment by selective grinding of occlusal surface
2. Occlusal appliances such as "night guards"
3. Orthodontics
4. Restorative dentistry
5. Tooth removal (as a last resort)
    a. In cases of primary occlusal trauma with gingivitis or periodontitis. The treatment is simple and conservative. First, periodontal therapy is done which include plaque control, scaling and root planing. If there is progressive mobility then occlusal therapy in the form of selective grinding and the use of night guard may be justified.
    b. In cases of secondary occlusal trauma and advanced periodontitis, the treatment is often complicated. It often requires advanced periodontal therapy, including root resection, antimicrobial therapy and regenerative procedures along with adjunctive orthodontics, occlusal adjustment by selective grinding and splinting for periodontal stabilization is advocated.

A typical force control treatment plan for a patient with the diagnosis of generalized moderate chronic periodontitis with occlusal traumatism would be:

Re-evaluation of inflammatory disease control. After a minimum of 4-6 weeks (the time for repair of the dento-gingival junction), the patient is re-examined and the results of that examination are compared with those

recorded at the initial examination. This is a critical stage in treatment as decisions about the working diagnosis and continuing active therapy are made depending upon the answers to the following questions:

1. Are there any persistent signs and symptoms of gingival inflammation or debris present?
2. If so, is there anything short of periodontal surgery that can be done to improve the conditions?
3. Is there any residual tooth mobility/fremitus?

If the answer to the first and second question is "no" and the answer to the third question is "yes", the patient' sworking diagnosis of generalized moderate chronic periodontitis with occlusal traumatism is supported, and treatment should proceed to the occlusal therapy phase.

If, however, the answer to the first and second question is "yes", then considerations for improving the patient's oral hygiene, refining scaling and root planing, instituting antimicrobial therapy, additional correction of plaque retentive factors, and discussions concerning progress made in smoking cessation might be appropriate.

Assuming that the answers to the first and second questions are "no" and the answer to the third question is "yes", the next step in treatment is to determine what is responsible for the mobility and fremitus.

Assessment of parafunctional occlusal habits of compulsion. Parafunction is "abnormal function, as in bruxism". Bruxism(tooth grinding, occlusal neurosis) is "a habit of grinding, clenching, or clamping the teeth. The force generated may damage both tooth and attachment apparatus." It should be emphasized that bruxism occurs during vertical (clenching)and horizontal (grinding) nonfunctional movements of the mandible. It may occur during sleep (nocturnal bruxism) or during waking hours (diurnal bruxism). Parafunctional tooth contacts tend to be repetitive and of greater force and duration than the more random and “fleeting" functional tooth contacts.

Occlusal analysis. Ideally, the occlusal analysis should be carried out on an adjustable articulator with the maxillary cast mounted on the hinge axis on an anatomical articulator, and the mandibular cast should be mounted in centric relation. Alternatively, the analysis may be performed in the mouth. In either case, occlusal contacts should be recorded along the operator-guided, patient-generated, movements of the mandible that originate in centric relation.

Question 7. Occlusal adjustment and occlusal appliance therapy.

Occlusal adjustment is the reshaping of occlusal surfaces of teeth by grinding to create harmonious contact relationships between upper and lower teeth. This process is also known as occlusal equilibration or selective grinding. The term odontoplasty is commonly used to describe the act of occlusal adjustment/occlusal equilibration/selective grinding. The guiding

principals of occlusal adjustment are the preservation and refinement of supporting cusp tips in centric occlusion and the elimination of lateral interfering centric and excursive contacts. Wear facets are also eliminated by vertical grooving and rounding of edges to reduce the tooth contact surface area during parafunction.

1. Occlusal adjustment. It is axiomatic that the muscles of mastication be free of myospasm or other functional disorders before occlusal adjustment is performed. Spastic, splinted muscles occur in response to noxious or injurious tooth contacts and result in neuromuscular patterns of jaw movement that "avoid" the offending tooth contact(s). These same contacts may be the target contacts of occlusal adjustment, and it is pointless to adjust an occlusion when target contacts cannot be recorded. Stressful life-styles or events commonly provide a psychic background for myospasm (figure 70).

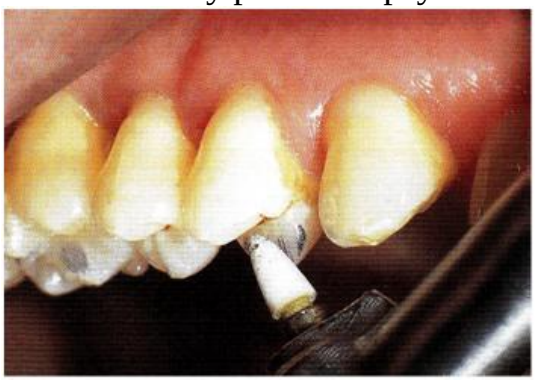


Figure 70. A clinical photograph showing occlusal adjustment of acentric interference by coronoplasty. Flame shaped or conical highspeed rotary instruments are ideal for the reduction of inclines and facets.

Pain or tenderness in the masseter, temporalis, or lateral pterygoid muscles, hypertrophy of the masseter muscles, limitation in opening the jaws, and/or subconscious resistance to movement of the mandible in hinge-axis and border movements are signs of myospasm. The application of heat to the affected muscles, and the use of an anterior bite-plane appliance for 1-2 weeks is usually all that is required to "deprogram" avoidance patterns and ultimately relieve the myospasm. Patients who are refractory to this noninvasive therapy may have a more serious form of myofascial pain dysfunction (MPD) syndrome or other temporomandibular joint (TMJ) disease. Patients with refractory pain and dysfunction will require care by a specialist and should be referred for treatment as soon as possible.

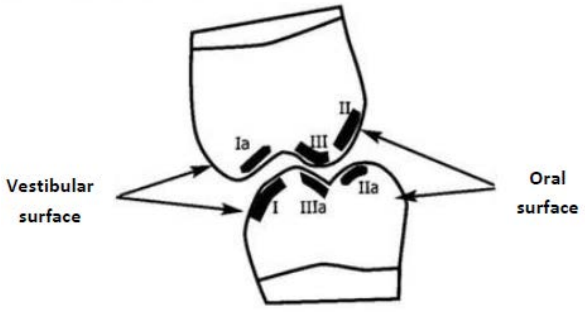


Figure 71. Classification of premature occlusal contacts (supra-contacts) of the teeth according to Jankelson

Table 22. Technique of functional occlusal polishing

Visit	*Class of premature occlusal contacts*	*Occlusion*	*Intervals between visit dates in days*
1	III	Distal	
2	I	Central	3–5
3	II	-	7–10
4	III	-	3
5	Control of all classes; Polishing of teeth	-	10–14

2. Occlusal appliance therapy.

1. The primary function of appliance therapy in periodontal patients is force control in secondary occlusal trauma. Additional benefits from occlusal appliances in periodontitis patients include splinting of teeth in the appliance, control of super-eruption, and anchorage for minor tooth movement. Typically, the force control appliance will provide full occlusal coverage of either the maxillary or mandibular teeth. The decision over which arch will be chosen for appliance therapy will be affected by Angle's classification, the location of teeth that might require splinting, and the location of teeth that need to be controlled for super-eruption. Wrought wire clasps and the anterior Hawley appliance wire are unnecessary unless the appliance is modified to accomplish anchorage for orthodontics. The occlusal scheme for full occlusal force control appliances is maximum cuspal contact on the flat occlusal surfaces of the guard in centric relation and all excursions. Because there are no indentations for cusp inter digitation, full occlusal appliances do not have a centric occlusion. As such, they have been proven to be effective in the long-term maintenance of muscle "deprogramming." They are not recommended for initial deprogramming of splinted muscles of mastication (Figures 72 and 73).

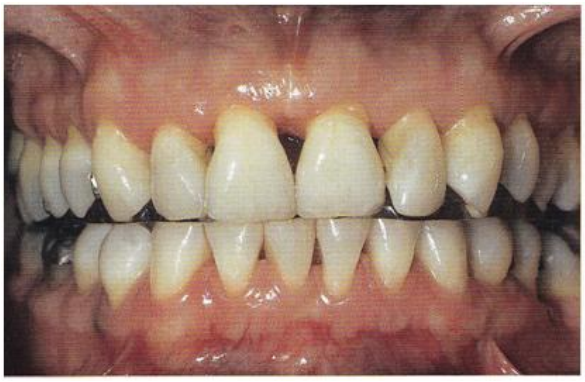


Figure 72. A clinical photograph of a full occlusal maxillary appliance for force control.
The appliance is designed to produce a minimal increase in the vertical dimension of occlusion.

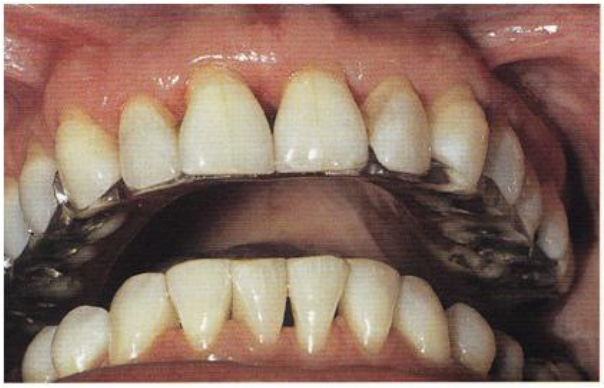


Figure 73. The appliance will provide maximum occlusal contact in all excursions. It will also provide intra-arch splinting of teeth during parafunction

2. In patients who have a normal periodontium and demonstrate heavy occlusal wear of parafunctional origin, or who have a history of fracturing restorations or teeth, an occlusal/night guard may be prescribed as an ablative shield. Force control may not be as much of an issue in these patients as tooth protection, particularly in cases where teeth have normal amounts of periodontal support. This appliance allows the patient to continue engaging in parafunctional activity without additional occlusal wear. Instead of irreversible tooth wear as a consequence of parafunction, the appliance is ablated.

3. In patients who have mild MPD and where muscle deprogramming is required before occlusal adjustment, an anterior bite plane may be constructed covering the lingual surfaces of the six maxillary anterior teeth. A Hawley labial wire is commonly included in the appliance to stabilize tooth position. A lingual platform establishes occlusal contact with the six mandibular anterior teeth, increases the occluding vertical dimension, and "discludes" all posterior teeth. The net effect of this appliance is that noxious tooth contacts are prevented while the patient wears the appliance. The expected outcome should be the reduction of myospasm in the muscles of mastication (Figures 74 and 75).

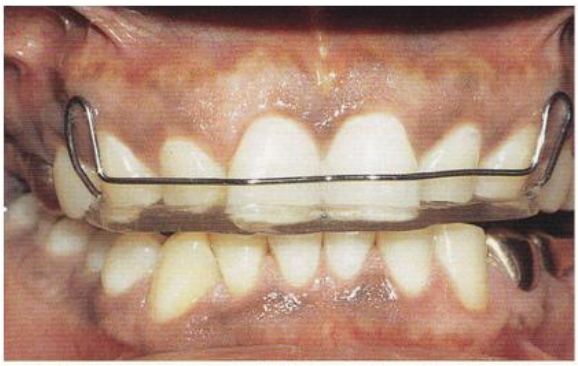


Figure 74. A clinical photograph of an occlusal appliance with a Hawley wire. this appliance is best suited for short periods of muscle deprogramming.

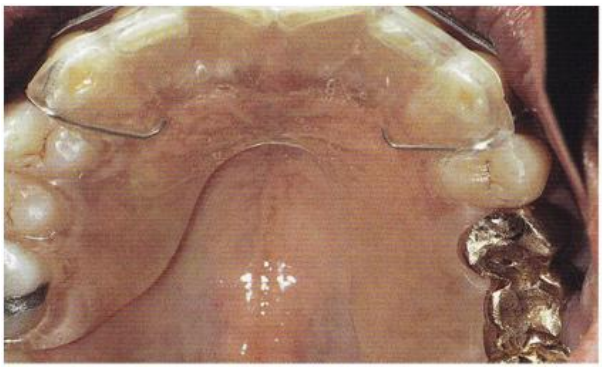


Figure 75. The anterior bite plane makes contact with the mandibular anterior teeth only. Posterior teeth not contained in the appliance are subject to supraeruption.

Because anterior bite plane appliances are reasonably simple to fabricate and usually require less chair time to adjust than the full occlusal appliances, it is tempting to use these appliances for long term force control. One consequence of this approach may be the extrusion of un-opposed teeth and unexpected additional occlusal problems. It is recommended that anterior bite-plane appliances be used only for short-term muscle deprogramming.

Force control has been a tradition in the treatment of periodontitis. Initially, it was believed that forces acting on teeth produced gingival recession and attachment loss and that periodontal therapy was incomplete unless these forces were eliminated or at least controlled. Today, the paradigm is that occlusal trauma and marginal periodontal diseases are probably distinct clinical entities. Occlusal trauma is treated with force control and periodontitis is treated by infection control.

The sequence of treatment for each is critical. Effective treatment of marginal periodontal inflammation may decrease some of the signs and symptoms of occlusal trauma. In addition, reactive repositioning (closure of open contacts) may take place after inflammatory disease control. There is also evidence that untreated inflammatory disease may impact negatively on the repair of occlusal trauma. In some cases, occlusal therapy may not be necessary after inflammation control.

Question 8. Splints in periodontal therapy

1. Definition

Dental splinting: it is defined as the joining of two or more teeth into a rigid unit by means of fixed or removable restorations/devices. Splint by definition is an appliance used for immobilization of injured or diseased parts.

A periodontal splint: it is an appliance used for maintaining or stabilizing mobile teeth in their functional position. The main objective of splinting is to promote healing and to increase the patients comfort and function.

There are two schools of thought regarding the use of splinting.

Harmful Aspects

- It creates an environment for plaque accumulation.

- Since functional movement of the tooth within the socket is not possible it may lead to ankylosis.

Beneficial Aspects

- Since they are splinted to the neighboring healthy teeth, mobility during mastication is prevented.
- Non-mobile teeth heal faster than mobile teeth.

2. Objectives of splinting

1. Provides rest.

2. For redirection of forces - the forces of occlusion are redirected in a more axial direction over all the teeth included in the splint.

3. For redistribution of forces - redistribution ensures that forces do not exceed the adaptive capacity of the periodontium.

4. To preserve arch integrity - splinting restores proximal contacts, reducing food impaction and consequent breakdown of the periodontium.

5. Restoration of functional stability - restores a functional occlusion, stabilizes mobile abutment teeth and increases masticatory efficiency.

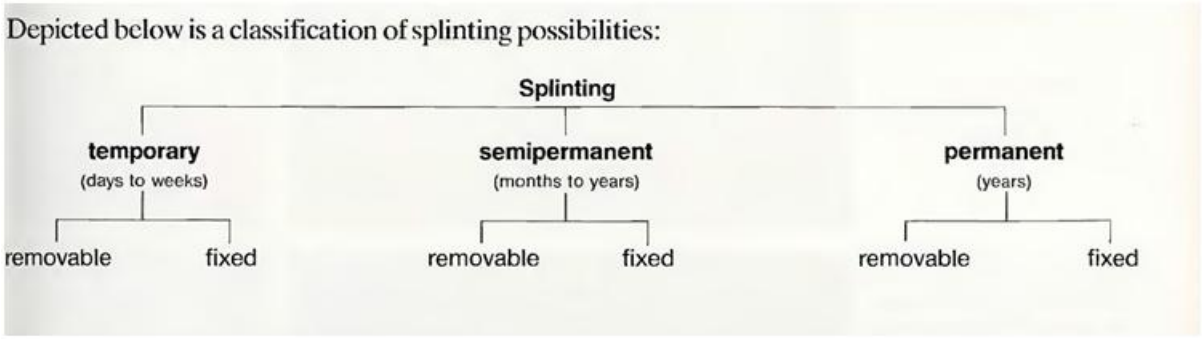
6. Psychologic well-being - gives the patient freedom from mobile teeth there by giving him a sense of wellbeing.

7. To stabilize mobile teeth during surgical, especially regenerative therapy.

8. To prevent the eruption of teeth without an antagonist.

3. Classification of splints

According to the Period of Stabilization



According to the Type of Material

1. Wirewound
2. Plastic (acrylic)
3. Composite. At present, modern armatures used for splinting teeth, according to their chemical composition, are divided into two large groups:
   a) materials based on inorganic matrix - ceramics and fiberglass (Glass Span, Splint-it, USA, Fiber Splint, Switzerland);
   b) materials based on organic matrix - polyethylene ("Ribbond", "Connect", Kerr, USA).

4. Principles of splinting

1. Inclusion of sufficient number of healthy teeth: It is suggested that the healthy teeth included in the splint should have double the root surface area of the mobile teeth to be splinted. Since the posterior teeth are multi-rooted the number of healthy teeth to be included in the splint in the posterior segment will be less as compared to the anterior.

2. Splint around the arch: Muscles of the lips, cheek and tongue exert some forces on the teeth. Based on the direction of such forces applied on the teeth, the dental arch can be divided into two posterior sextants and an anterior sextant. In the posterior sextant the tongue pushes the teeth buccally and the muscles of the cheek counter act it by pushing them lingually. When the splint is confined to any one sextant, the splinted teeth tend to tilt lingually or outwards depending on the muscular forces. Such a collapse of the splinted sextant can be prevented by including few teeth from the adjacent sextant. This is termed splinting around the arch.

3. Coronoplasty may be performed to relieve traumatic occlusion.

4. The splint should be fabricated in such a way as to *facilitate proper plaque control.*

5. Splint should be aesthetically-acceptable and should not interfere with occlusion.

5. Indications and contraindications of splinting

Indications

1. It stabilizes moderate to advanced tooth mobility that can not be reduced by other means and which has not responded to occlusal adjustment and periodontal therapy.
2. When it interferes with normal masticatory function.
3. Facilitates scaling and surgical procedures.
4. Stabilizes teeth after orthodontic movement.
5. Stabilizes teeth after acute dental trauma, e.g. subluxation, avulsion, etc.
6. In order to prevent tipping and drifting of teeth.
7. Prevent extrusion of unopposed teeth.

Contraindications

1. Moderate to severe tooth mobility in the presence of periodontal inflammation and/ primary occlusal trauma.
2. Insufficient number of firm/sufficiently firm teeth to stabilize mobile teeth.
3. Prior occlusal adjustment has not been done on teeth with occlusal trauma or occlusal interference.
4. Patient not maintaining oral hygiene.

Advantages

1. May establish final stability and comfort for patient with occlusal trauma.
2. Helpful to decrease mobility and accelerate healing following acute trauma to the teeth.
3. Allows remodelling of alveolar bone and periodontal ligament for orthodontically, splinted teeth.
4. Helpful in decreasing mobility thereby favoring regenerative therapy.
5. Distributes occlusal forces over a wider area.

Disadvantages

1. All the splints hamper patient's self care. Accumulation of plaque at the splinted margins can lead to further periodontal breakdown in a patient with already compromised periodontal support.
2. Number of studies have shown that splinting does not actually reduce tooth mobility (once the splint is removed).
3. The splint being rigid acts as a lever with uneven distribution of forces, even if one tooth of the splint is in traumatic occlusion, it can injure the periodontium of all the teeth within the splint.
4. Development of caries is an unavoidable risk. Thus, it obviates the need of excellent oral hygiene in the patient.

In conclusion splinting decreases mobility there by improving the health of the tooth. It is important to note that if used incorrectly or not managed properly it may fail to achieve the desired results.

Test control

1. Causes of development of primary traumatic occlusion:

a) increased bite on seals, inserts, single crowns or bridges;
b) incorrect choice of the number of supporting teeth in bridges;
c) the non-rational arrangement of clasps, especially in clasp prostheses;
d) forced orthodontic treatment;
e) loss of many teeth;
f) all of above.

2. Pathological mechanisms in the development of secondary traumatic occlusion are:

a) resorption of bone tissue of the alveolar process;
b) destruction of the periodontal ligament of the tooth;
c) increased extra-alveolar part of the teeth;
d) pathological mobility and movement of teeth;

e) loss of interdental contact and unity of dentition;
f) all of the above.

3. On X-rays, combined traumatic occlusion reveals:

a) resorption of bone tissue of the alveolar process only in the area of congested teeth;
b) resorption of the bone tissue of the alveolar process is revealed throughout the dentition;
c) uneven expansion of the periodontal gap;
d) foci of rarefaction of bone tissue in the peri-vertebral region of congested teeth;
e) excessive deposition of cement in the apical third of the root;
f) general discontinuity of the cortical plate;
g) presence of denticles in the cavity of the tooth;
h) all of the above.

4. Supractress is:

a) undesirable occlusal contact, preventing the teeth from closing in the central occlusion, sliding in it from other positions of the lower jaw;
b) undesirable occlusal contact, preventing the teeth from closing in the central occlusion;
c) undesirable occlusal contact, preventing the teeth from closing in all kinds of occlusion;
d) premature

5. Types of supracontacts are:

a) centric;
b) on the work side;
c) on the balancing side;
d) in the anterior occlusion;
e) all of the above.

6. The objectives of selective teeth polishing:

a) elimination of the traumatic situation in periodontium by distributing the functional load to as many teeth as possible;
b) distribution of load along the axis of teeth;
c) removal of the pathological activity of the masticatory muscles;
d) elimination of balancing and hyperbalancing supracontacts;
e) creation of stable stable central occlusion;
f) all of the above.

7. At the first visit during the grinding of teeth according to Jenkelson, early occlusal contacts are revealed:

a) I class in distal occlusion;
b) II class in the central occlusion;
c) III class in distal occlusion;
d) I class in the central occlusion;
e) III class in the central occlusion.

8. What type of occlusal trauma is characterized by a strict localization of pathological changes in the area of one or more teeth?

a) primary;
b) secondary;
c) combined;
d) all of the above.

9. What type of occlusal injury is characterized by periodontal damage most often in the area of all teeth?

a) primary;
b) secondary;
c) combined;
d) all of the above.

10. What type of occlusal injury is characterized by a delay in the erosion of the teeth?

a) primary;
b) secondary;
c) combined;
d) all of the above.

LESSON 8. PERIODONTOLOGY AND ORTHODONTICS. PERIODONTITIS IN ADULTS WITH DENTOALVELAR DEFORMITIES: DIAGNOSES, CLINIC MANIFESTATIONS, PECULIARITIES IN PLANNING OF COMPLEX TREATMENT.

The questions to be studied for the learning of the topic:

1. The influence of dentofacial anomalies and deformations on the occurrence of periodontal disease.
2. Features of clinic and diagnostics of complex periodontitis in adults with dentofacial deformities.
3. The principles of orthodontic interventions in patients with diseases of the periodontium.
4. The features of the plan of complex treatment.

Question 1. The influence of dentofacial anomalies and deformations on the occurrence of periodontal disease

The periodontium is a particular system, the main biological function of which is the absorption of mechanical energy that occurs during chewing, the uniform distribution on the bone tissue of the alveoli. In response to functional load, which is the dominant forcing factor in the periodontium occur reactive power. An indispensable condition for normal periodont is the equality of active and reactive forces.

Improve chewing pressure, on the one hand, in terms of training can lead to functional adaptation of the periodontium, but on the other hand, when reducing an individual's stamina can lead to pathological changes. All types of loading and micro-movements that counteract the collagen fibers. Occurs proprioceptive regulation and transformation of the masticatory pressure, perceived in a biologically compensated load. The fibers of the periodontal ligament involved in the work in a certain sequence, depending on the magnitude and direction of the force, the original position of the tooth. The most evenly this is done by action on the teeth "axial forces", i.e. the vertical load. Vertical loading is more physiological than the horizontal at which a different pressure zone in the periodontium. In addition, the value is the location of load application to the crown of the tooth. In case of unfavorable ratio of clinical crowns and intra-alveolar part of the tooth, if the place of application of force is away from the pivot point of the tooth, it is displaced as a result action. The load is increased in the presence of defects of dentition, which often leads to the repositioning of the remaining teeth, and deformities of dentition. Thus, dentoalveolar anomalies and deformations contribute to the incorrect distribution of load on the periodontium and way lead to overloading of the latter, i.e. presence of primary traumatic occlusion. The tension in the periodontium increases almost 20 times during the load of the teeth in the protrusion. (G.P. Sosnin).

Should distinguish between the anomaly and deformation. Under the anomaly understand developmental disorders of form and function of the dentition. Deformation - change in shape and function of the dentition, due to acquired pathological processes. Anomalies of the dentition in turn divided into congenital and acquired. Dentofacial anomalies occur in 50% of children and 30% of adolescents and adults. The anomalies of the teeth-jaw system is not resolved in childhood, of course, retained in adults. The clinical picture of malocclusion in adults is more difficult than in children because the symptoms of the disease join the loss of teeth, deformation of dentition and jaws, the functional overload of parodontium disorders and occlusion. These changes lead to traumatic occlusion, which in turn causes the complex development of chronic periodontitis.

Question 2. Features of clinic and diagnostics of complex periodontitis in adults with dentofacial deformities.

The complex chronic periodontitis in adults with dentofacial deformities characterized by complaints of mobility and displacement of teeth, bleeding and sore gums, bad breath, difficulty chewing of food. Also characterized by the presence of complaints from the temporomandibular joint (crunching, clicking, pain, etc.).

An objective examination of patients with complicated chronic periodontitis defined pronounced chronic inflammation of the gums. Often, on examination, there are abundant over-and subgingival dental plaque. The color of the gums, usually bright red with a bluish tint (congestion hyperemia), the consistency of the gum loose, pasty, shape and contour of the gingival papillae modified, the surface smooth and shiny. Often there is a change in the position of the gingiva relative to the enamel-cement border. In addition, characterized by the presence of bleeding. Symptom of bleeding gums is evaluated with a light (with a force of 0.25 N) probing the gingival groove, for 30 seconds after probing.

In complex periodontitis, developed on the background of the anomalies of the jaws and dentition, secondary deformation occur more often and are much heavier.

The key clinical parameters of complex chronic periodontitis in adults with dental and facial deformities are:

- ✓ the presence of chronic inflammatory process in periodontal tissues;
- ✓ significant destruction of bone tissue (more than 1/3 length of the root);
- ✓ the presence of deep periodontal pockets (5 mm or more);
- ✓ traumatic occlusion;
- ✓ displacement of teeth and deformation of the dentition;
- ✓ abnormal mobility of the teeth.

The examination of the adult patient has a number of features, due to the fact that an anomaly of dentition is combined with partial adentia.

The examination of the patient begins with a study of the appearance, however, all adult patients need to examine the temporomandibular joints. During the inspection of the person of an adult, measure the height of the lower third of the face, profile of face, angle of the mandible, studying the movement of the lower jaw when opening and close your mouth.

The study of the oral cavity begins with inspection of the mucosa. Adult patients meet acute and chronic diseases of the mucous membranes, which can serve as a contraindication to the use of intraoral appliances. Then examine the ratio of dentition. It should be remembered that improper interdigitation can be the consequence not only of dentoalveolar anomalies and deformations of the dentition in their destruction. Examine the alveolar apophysis, size of apical base, the height of the sky, studying the mucous membrane, etc. To determine the exact shape of the anomaly, the differential diagnosis and study of pathogenesis requires the use of special methods of research of dentofacial anomalies, diagnostic study models, x-rays of the teeth and temporomandibular joints, x-ray analysis of the facial skeleton, the analysis of soft tissue facial profile, electromyographic study of masticatory muscles, etc.

Question 3. The principles of orthodontic interventions in patients which have diseases of the periodont

The indications to this kind of treatment of great importance is the ability of the patient to maintain oral hygiene at an appropriate level. It is also necessary to take into account the fact that the adult patient can be bridges and if they are sintered, the problems of fixing orthodontic braces does not occur. If the prosthesis metal, the position and the inclination of unfavorable, then they are better off. During orthodontic treatment for adults should also be aware of potential issues with the temporomandibular joint, as in children and dysfunction occurs incomparably less.

The simplest method of treatment dentoalveolar anomalies in adults is alignment occlusal surface dentition by filing of teeth hard tissues and further prosthetics of their different orthopedic constructions.

Many authors point to the need for complex treatment of dentoalveolar anomalies and deformations. Some researchers recommend to include in the complex treatment of dentoalveolar anomalies formed permanent dentition physiotherapy. In particular, according to the author, the use of magnetophoresis Trilon "B" reduces the duration of active orthodontic treatment 2.1 times and avoids the surgical preparation of the alveolar process. The appointment the period of retention of magnetic therapy or magnetophoresis calcium lactate reduces tooth mobility of 1.6 times, and shortens the course of treatment 1.2 times.

The surgical treatment of dentofacial anomalies is not neglected. The essence of surgical treatment is resection of the excess bone, plastic surgery

of the frenulum, osteotomy, decortication, compactosteotomy with subsequent orthodontic treatment. More broadly, apparently, should be applied surgery - removal of teeth to form a correct dentition as a radical intervention in case of discrepancy between the size of jaws, the number and size of teeth.

Question 4. The features of the plan of complex treatment.

After the diagnosis to plan a comprehensive treatment determine the prognosis of diseases of the periodontium, with the inclusion of planning orthodontic measures.

Orthodontic measures in patients with diseases of periodontium are carried out in three directions:

1. The reallocation of space in the dentition in case of wrong position of teeth.
2. Correction of the vertical length of the alveolar ridge.
3. The increase in the thickness of the alveolar ridge.

The reallocation of space in the dentition in case of wrong position of teeth is performed orthodontic will moving teeth to their optimal position in the dental arch.

The correction of the vertical length of the alveolar ridge is carried out by vertical traction (extrusion) the tooth, which gives the opportunity to aesthetically improve the alveolar ridge to the orthopedic treatment, as well as conditions for implantation in the region "hopeless pulling teeth". Orthodontic traction teeth to eliminate intraosseous defects or increase of the crown of individual teeth.

The teeth with root fracture save by orthodontic traction of the periodontal. In these teeth hold fibrotomiya e i.e. excision of circular collagen fibers in the region of the crown part of the tooth.

The increase in the thickness of the alveolar ridge is carried out by orthodontic re-placement of teeth in the horizontal direction. Horizontal movement is indicated for teeth with intraosseous defects. The movement of teeth is performed to build bone in the defect area. Vertical (which restores) and resorption of intraosseous defects reduce or eliminate adequate orthodontic tooth movement.

After an effective anti-inflammatory therapy indicate the teeth with uncertain prognosis or "bad". During the period of orthodontic treatment of "hopeless" teeth can be saved to enhance the support (anchorage).

Orthodontic treatment is carried out only after the formation of persistent skill to care for the oral cavity, and also after the elimination of inflammation in periodontal tissues. In the process of orthodontic treatment to control the level of hygiene of the oral cavity every 3 months and if necessary carry out a professional dental cleaning, together with other curative and preventive measures.

Each control periodontal examination record data and assess the state of periodontal tissue, including the degree and prevalence of inflammation, depth of the tooth-gingival pockets, the sensitivity of the periodontium, bleeding gums, tooth mobility, intensity and prevalence of receding gums, the bone level introalveolar partitions, circulation, electroodontometry teeth.

After the removal of the orthodontic apparatus recommend a special care of the oral cavity, to prevent rotation of the usual excessive force when brushing. This gives you the opportunity to delay the rapid progression of recession gums. For patients with diseases of the periodontium choose the orthodontic appliances that meets not only aesthetic characteristics but also indifferent to the tissues of the periodontium. In this regard, preference is given to sapphire (ceramic) braces in the anterior area; in other areas metal braces are used.

Test control

1. Absolute contraindications to orthodontic treatment in adults are:

a) decompensated diseases of the cardiovascular system;
b) bone system diseases that reduce bone repair;
c) endocrine system diseases;
d) blood diseases;
e) all of the above.

2. Relative contraindications to orthodontic treatment in adults are:

a) poor oral hygiene;
b) precancerous and malignant diseases of the oral cavity;
c) the presence of metal implants of other organs;
d) diseases of the temporomandibular joint;
e) bruxism.
f) all of the above

3. Indications for orthodontic treatment of periodontal diseases in adults are:

a) Popov-Godon phenomenon;
b) tremas and diastema due to tooth displacement;
c) deep bite;
d) deep bite, progenia complicated by secondary deformation of the dentition.
e) all of the above

4. For the effective treatment of dentition anomalies in adults need to know:

a) what structural changes in the dentition are necessary to eliminate;
b) what morphological restructuring of the dentition is possible in adults.

c) all of the above

5. The meaning of surgical treatment before orthodontic treatment is

a) resection of excess bone.
b) plastic bridle.
c) decortication.
d) compact osteotomy.
e) all of the above

6. The key clinical parameters of chronic complex periodontitis in adults with dentoalveolar deformities are:

a) the presence of a chronic inflammatory process in periodontal tissues;
b) significant destruction of bone tissue (more than 1/3 of the root length);
c) the presence of deep periodontal pockets (5 mm or more);
d) traumatic occlusion;
e) tooth displacement and deformation of the dentition;
f) pathological tooth mobility.
g) all of the above

7. Deformation is

a) disturbances in the development of the form and function of the dentition
b) the presence of a chronic inflammatory process in periodontal tissues
c) a change in the form and function of the dentofacial system due to acquired pathological processes.

8. Orthodontic measures in the complex treatment of periodontal diseases can be included at the age of:

a) up to 35 years old;
b) after 18 years;
c) at any age;
d) from 18 to 25 years old.

9. The purpose of orthodontic treatment in patients with periodontal diseases is:

a) the achievement of full fissure-tubercular contacts between antagonistic teeth, symmetry and the absence of gaps between the teeth;
b) improvement of aesthetic indicators;
c) redistribution of chewing load;
d) improved posture.

10. The main method of treatment of dentoalveolar anomalies in adults is:

a) orthodontic method;
b) surgical method;
c) orthopedic method;
d) orthopedic-surgical method.

LESSON 9. FEATURES IN PLANNING THE COMPLEX TREATMENT OF PERIODONTAL DISEASE IN ADULTS USING ORTHODONTIC, IMPLANT AND ORTHOPEDIC MEASURES. AESTHETIC PERIODONTOLOGY

The questions to be studied for the learning of the topic:

1. The primary goals of treatment for periodontitis
2. Initial therapy
3. Corrective phase
4. Orthodontics
5. Function - occlusal therapy
6. Reconstruction – Prosthodontics

Question 1. The primary goals of treatment for periodontitis

The primary goals of treatment for periodontitis are:

1. Elimination of gingival inflammation
2. Elimination of symptoms of disease activity (exudate, pus, hemorrhage) in periodontal pockets
3. Cessation of bone loss and attachment loss
4. Elimination or reduction of periodontal pockets that represent potential sites of re-infection
5. Prevention of recurrence of inflammation
6. Achieving new attachment to the root surface by means of regeneration of periodontal tissues
7. Improvement of gingival contour
8. Creation of optimal, functional occlusal relationships
9. Stabilization of mobile teeth.

Towards the achievement of these treatment goals there are many clinically proven and scientifically tested concepts of therapy. All such concepts have in common the attempt to remove the supra- and subgingival microorganisms (plaque control) that elicit disease, as well as the creation of a clean, smooth, endotoxin-free and bioacceptable root surface. These are the elements of initial therapy.

Beyond these similarities of orientation, the various treatment concepts and treatment goals may differ according to the form and severity of disease. A variety of standard surgical procedures may be indicated. The most important aspect of surgical intervention is that it provides the opportunity for the dentist to treat the root surface with direct vision, as well as to correct morphological irregularities of gingival and periodontal structures. This is called the corrective phase of therapy.

Functional therapeutic measures such as occlusal adjustment by selective grinding or orthodontics, stabilization or splinting are only undertaken if some functional disturbance (e.g., occlusal trauma) is

accelerating the progress of periodontitis and if increasing tooth mobility is noted.

Phase's periodontal therapy:

1. The initial (cause-related) phase
2. The corrective phase
3. The recovery (reconstructive) phase
4. The supportive phase

Question 2. Initial therapy

Every patient must traverse the initial therapy phase. For gingivitis and for periodontitis, initial therapy is often the only treatment necessary. The aim is to control plaque. Periodontal disease is an infection due to the presence of plaque biofilm, therefore, disruption of the plaque biofilm and control of plaque is the key to success. More complex treatments will always fail in the absence of effective plaque control.

The dentist must accomplish the following:

1. Informing the patient about his periodontal disease and its treatment (case presentation), as well as motivating him concerning disease control
2. Instruction in oral hygiene, including use of the toothbrush and other aids for interdental hygiene
3. Prescription of medicaments in special cases only, e.g., topical chlorhexidine rinses or systemic administration of tetracycline or metranidazole
4. Supragingival calculus removal (gross scaling and tine debridement)
5. Elimination of iatrogenic irritants, polishing old restorations, creation of conditions that will foster interdental hygiene (access)
6. Elimination of natural plaque-retentive areas such as grooves, depressions or irregularities on crown and root surfaces, furcations, and crowding of teeth
7. Subgingival plaque and calculus removal, root planing, and soft tissue curettage
8. Functional treatment (bite guard, selective occlusal grinding)
9. Arch stabilization via splinting of excessively mobile teeth.

After removal of supragingival deposits and iatrogenic irritants, treatment of the periodontal pocket may begin.

Scaling: the process by which plaque and calculus - are removed from both supra- and subgingival tooth surfaces (fig. 76).

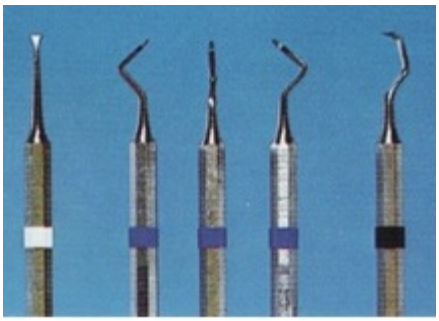


Figure 76. Scalers

Root planing: the process by which residual embedded calculus and portions of the cementum are removed from the roots to produce a clean, hard, smooth surface that is free of endotoxin (fig. 77).

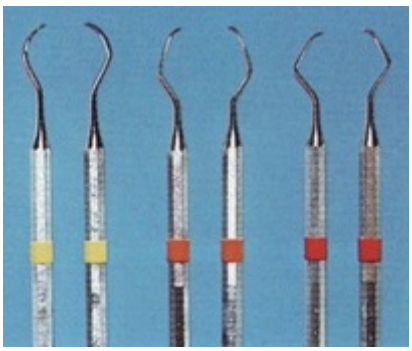


Figure 77. Curettes (yellow – Universal; orange – Anterior; red – Posterior)

Gingival curettage (soft tissue curettage, closed curettage): the process by which the pocket epithelium and infiltrated subepithelial connectives are removed without reflecting flaps, i.e., without direct vision of the surfaces being treated. Gingival curettage is classified as a surgical procedure.

Open curettage: the process by which thorough scaling, root planing, and removal of diseased pocket epithelium and connective tissue are accomplished with direct vision, after an inverse bevel incision and conservative reflection of marginal gingiva

Closed curettage without subsequent surgical intervention is indicated in cases of mild to moderate periodontitis with pockets of 6 mm or less. During a re-evaluation of the case several weeks or months later, the dentist can decide whether local surgical intervention maybe required. In severe, advanced periodontitis, curettage in the form of subgingival debridement may be considered as pretreatment for the subsequent surgery, as wound healing is enhanced.

Question 3. Corrective phase

The corrective phase is designed principally to restore function and, where relevant, aesthetics. It includes:

- further nonsurgical therapy
- periodontal access surgery
- regenerative surgery,

- mucogingival surgery
- resective surgery, e.g. Gingivectomy
- treatment of furcation lesions

The aims of this phase are:

1. To eliminate pathological periodontal pockets, or to create a tight epithelial attachment where the pocket once existed.
2. To arrest loss of, and in some cases improve, the alveolar bone support.
3. To create an oral environment which is relatively simple for the patient to keep plaque-free.

Applications of periodontal surgery

- Provide access for root surface instrumentation. Direct vision of the root surface is possible. This is particularly helpful with furcation defects.
- Result in a site which is accessible for cleaning.
- Correction of gingival overgrowth by gingivectomy.
- Create new periodontal attachment in the case of regenerative procedures.
- Improved aesthetics and function following gingival recession by root coverage techniques.

Contraindications to periodontal surgery:

- Poor plaque control.
- Systemic disease, e.g. uncontrolled diabetes, severe cardiovascular problems, bleeding disorders. Liaise with GMP and specialist.
- As smoking outcome of Rx, some periodontologists have limited Rx in those continuing to smoke. While periodontal surgery hardly has the public impact of cardiac surgery, the ethical problem is the same.
- Teeth with poor long-term prognosis.

Types of Periodontal surgery

1. The modified Widman flap
2. The apically repositioned flap
3. Osseous surgery:
    - Osteoplasty is conservative recontouring of the bone margin (i.e. non-supporting bone).
    - Ostectomy is excision of bone aimed at eliminating infra-alveolarpocketing,
4. Gingivectomy
5. Guided-tissue regeneration
6. Mucogingival surgery

Periodontal surgery may be categorized as:

- ✓ Gingivcctomy/gingivoplasty (GV/GP)
- ✓ Flap procedures with conservative flap reflection (Modified Widman flaps/Ramfjord technique)
- ✓ Flap procedure with flaps reflected completely, permitting various possibilities for flap repositioning
- ✓ Combinations of methods and special operations

✓ Mucogingival surgery.

The indication for gingivectomy/gingivoplasty is limited. GV/GP is used in cases of pronounced gingival enlargement/overgrowth, shallow suprabony pockets, localized "minor operations," and in combination with flap surgery. The GV/GP is contraindicated for treatment of infrabony pockets and osseous thickening, and when attached gingiva is narrow or absent.

The modified Widman procedure (Ramfjord technique, partially reflected flap) is the most universally applicable periodontal surgical modality. It is particularly indicated for treatment of mild to moderate periodontitis.

The fully reflected mucoperiosteal flap (with or without vertical incisions) is indicated in severe periodontitis with irregular bone loss within an arch segment and for advanced furcation involvement. In such cases, osteoplasty may also be performed, as well as intraosseous implants.

Special periodontal surgery includes wedge excisions, implants of various types into bony pockets, treatment of isolated furcation involvement, and extraction of hopeless teeth with simultaneous revision of periodontal supporting tissues of the adjacent teeth. These procedures are usually performed in conjunction with flap operations.

Mucogingival surgery is indicated only in cases exhibiting advanced gingival recession where attached gingiva is lacking or where frenum attachments radiate into the marginal gingiva.

Periodontal surgery - regenerative techniques

Guided-tissue regeneration (GTR). The recognition that epithelium migrated along the root surface before any other cell type, after periodontal surgery, and created the long junctional epithelium which prevented new attachment, created the possibility that prevention of migration of epithelium would allow new connective tissue attachment

Guided-tissue regeneration. Bone grafts may be used in combination with a barrier membrane. Grafts from inorganic bovine bone matrix (Bio-oss®) are in widespread use. Synthetic bone substitutes are also used (e.g.PerioGlas®).

Guided-tissue regeneration. GTR is essentially interposing a barrier to epithelial migration prior to completion of surgical or non-surgical therapy. Non-resorbable membranes Resorbable membranes (e.g. Bio-gide®,Vicryl®) Bio-absorbable flowable polymer (Atrisorb FreeFlow) GTR can be used alone or with bone grafts or enamel matrix derivatives

Enamel matrix derivatives (EMD). Emdogain is a product containing porcine EMD proteins in a propylene glycol alginate gel. Enamel matrix proteins (e.g. amelogenin) are found in Hertwig’s sheath and induce root formation in the developing tooth. Locally applied enamel matrix proteins

may help form acellular cementum, the key tissue in the development of a functional periodontium.

Indications. GTR is useful for treatment of 2- or 3-walled intrabony defects, furcation defects, recession defects. But work best in cases with 3 walled defects or grade II furcations. Case selection is important in success of regeneration techniques. They limited use in generation of new bone for implant placement. Good oral hygiene is essential. Smoking has an adverse effect on outcomes. Careful and scrupulous surgical technique requires.

Mucogingival surgery. Mucogingival surgery includes those techniques aimed at the correction of local gingival defects

Indications. Where change in the morphology of the gingival margin would improve plaque control, e.g. presence of high fraenal attachments or deep areas of recession. Areas where recession creates root sensitivity or aesthetic problems. A very thin layer of attached gingiva overlying a tooth, which is to be moved orthodontically: the evidence for this is somewhat anecdotal.

Grafting is subdivided into:

Free grafts which are completely removed from their donor area. Free gingival grafts, commonly of palatal mucosa and connective tissue are taken and grafted to donor sites prepared by incising between attached and alveolar mucosa. A template may be used to harvest the correct amount of tissue. Mucoperiosteum is exposed at the recipient site and the harvested tissue is sutured carefully over this. A *subepithelial connective tissue graft* from the palate gives a better aesthetic result. At the recipient site it is covered by a coronally advanced flap. Pedicle grafts are not separated from their blood supply. Commonly used pedicle grafts are the laterally repositioned flap, coronally repositioned flap, and the double papilla flap. These techniques may be of some value in very narrow areas of isolated gingival recession. Technically, of course, these are flaps not grafts. Can be used in conjunction with EMD.

Question 4. Orthodontics

A casual relationship between dental malocclusion and periodontitis is difficult to document. However, dental crowding may make plaque removal difficult, and this favors the progression of inflammatory disease in the periodontal supporting tissues. One must differentiate between malocclusion that has existed since complete eruption of the permanent dentition and tooth drifting that occurs over a period of time, perhaps as a consequence of periodontitis. The latter may be due, at least in part, to the periodontal disease process itself, and includes tipping, rotation, extrusion and especially protrusion of the maxillary anterior teeth. Realignment of teeth that have drifted in a dentition manifesting periodontitis can usually be accomplished by means of simple orthodontic movement.

The factors involved in the development of dental malocclusion following loss of periodontal support are not always obvious. Possible causes include oral parafunctions such as tongue thrust or lip biting, occupation-related peculiarities such as holding nails or pins between the teeth, and tipping of adjacent teeth into an extraction site resulting in occlusal imbalances. It has also been speculated that the granulation tissue present in deep periodontal pockets may exert a force that causes drifting, or that the intact fiber structure on the side of a tooth opposite a deep pocket may exert a pull that results in tipping or drifting of a tooth.

Orthodontic treatment of a pcriodontally compromised dentition should never be started until the initial therapy phase is completed and the infection has been brought under control. The rationale for various treatment options should be considered: Is the proposed tooth movement for functional or esthetic reasons? Could the patient's problem be solved by other means, for example by reconlouring teeth (odontoplasty) to correct functional, morphologic or esthetic problems? Is a prosthetic solution feasible? If the final decision is to go ahead with orthodontic treatment, an array of possible methods including simple wire ligatures, removable plates and fixed appliances is available. The choice will depend in large measure on the diagnosis, the goals of therapy and the difficulty of the tooth movement desired. Another factor in selection of treatment modality is the concerns of the patient, especially the adult patient, who is often not prepared or willing to submit to longterm orthodontic therapy with fixed appliances. Thus, compromises often have to be made. In a dentition that has been ravaged by periodontal disease, orthodontic treatment represents a particular trauma for the remaining supporting structures. It has been amply demonstrated that orthodontic treatment results in temporarily increased tooth mobility, which is greatly accelerated by occlusal trauma. For this reason, if the destruction in the periodontium is severe, major orthodontic treatment may be contraindicated.

Question 5. Function - occlusal therapy

The significance of occlusal trauma in the initiation of periodontitis was a source of controversy for decades. Today, we know from experimental studies that abnormal occlusal forces can elicit neither gingivitis nor periodontitis. However, the progression of an already present (active) periodontitis may be accelerated

Definition: occlusal trauma is defined as "a microscopic alteration of the structures adjoining the periodontal ligament that manifests clinically as a (reversible) elevation of tooth. The histologic changes include circulatory disturbances, edema and hyalinization of periodontal ligament fibers, mild inflammatory infiltrate, and nuclear pyknosis of osteoblasts, cementoblasts and. The periodontal ligament space adapts by becoming wider, and assumes

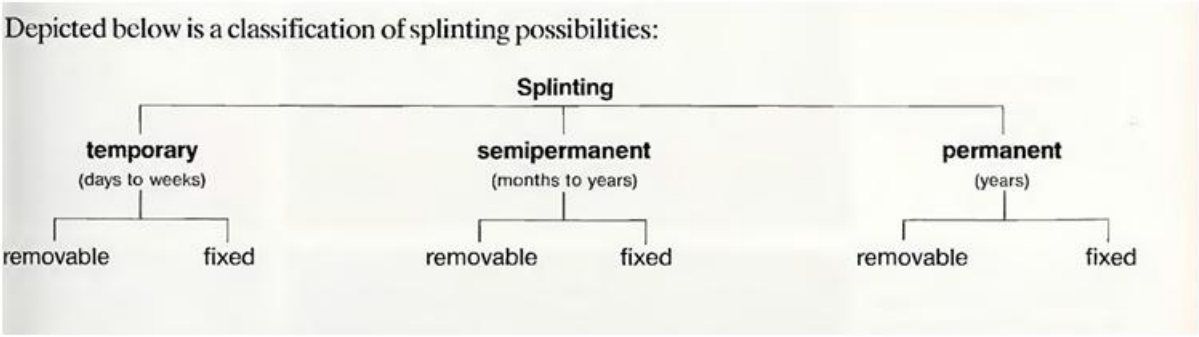
an hour-glass shape. There are no histologic changes in the supracrestal (gingival) collagen fibers nor in the junctional epithelium. The histologic alterations in the periodontium are completely reversible if the cause of the trauma is eliminated. Tooth mobility also returns to normal when the etiology is removed.

Adaptation to unphysiologic loading: If occlusal trauma persists over a long period of time, the tissues of the periodontium may adapt to the insult, even without any treatment. The periodontal ligament space remains widened, but its histological structure is normal in appearance. Tooth mobility remains elevated, but does not increase.

Progressive tooth mobility from unphysiologic loading: If abnormal occlusal forces are heavy and persistent, tooth mobility may continue to increase. Therapeutic elimination of the cause of the trauma may be possible by selective occlusal grinding, inserting a bile guard, splinting of teeth (stabilization), or reconstruction (e.g.,bridgework).

A. SPLINTING – STABILIZATION

Indications for the various types of splinting Temporary or semipermanent splinting is indicated for severely mobile teeth before or during periodontal therapy. Such splinting can reduce treatment trauma. Semipermanent or permanent splinting may be used to stabilize highly mobile teeth that impair the patient's chewing. Orthodontic retention may also be viewed as a type of semipermanent/permanent splinting. Permanent splinting is employed during complex oral rehabilitation where abutments are highly mobile or where a few abutments must support the entire prosthesis, particularly when such abutment teeth have minimal periodontal support, but have been successfully treated periodontally. If such teeth are not splinted, the danger of progressively increasing tooth mobility exists



Temporary Splinting. The simple wire ligature may serve as a fixed splint for a few days to several weeks. Wire ligatures are seldom used today, primarily because of the esthetic considerations. A more commonly used fixed temporary splint is the acid-etch composite resin splint without tooth preparation. Such a splint can be applied quickly and easily with the rubber dam in place; however, it is a temporary measure because adhesion of the

resin to tooth structure is not very strong without the additional mechanical retention provided by a cavity preparation grooves etc. Fracture of the splint is com-i mon if more than 3-4 teeth are included in a single splinted unit. A removable temporary splint may be fabricated of clear acrylic pulled under vacuum over a study model. Such splints are often indicated for temporary stabilization of individual teeth for short periods of time. This type of splint was formerly used as a "bite guard" in the treatment of oral parafunctions, but with very little success.

Semipermanent Splinting - Anterior Area. The most commonly used fixed semipermanent splint in the anterior area is the acid-etch composite resin splint applied after tooth preparation. It may serve for several months or even years. Often it is possible to remove old anterior restorations and utilize the cavity preparations in the splint. The technique of application is identical to placement of a composite resin restoration using the acid-etch pretreatment. In the mandibular anterior area, the intracoronal resin splint incorporating polyester fibers has proved useful. Light-polymerized resin is generally used for this type of splint because of its long working time. Removable semipermanent splints may be fabricated as cast chrome-cobalt alloy frameworks incorporating finger clasps for retention. This type of splint generally is indicated only for wear at night, as a retention appliance after orthodontic treatment or after surgical procedures.

Semipermanent Splinting - Posterior Area. The most common indication for semipermanent splinting in the posterior areas is for interim stabilization of highly mobile teeth subjected to heavy occlusal forces. Such splinting may be necessary before, during or after periodontal therapy, when the long-term prognosis for the mobile teeth has not yet been established. This sort of "handicraft" temporary splint is inexpensive and easy to fabricate. A popular method involves use of composite resin reinforced with stiff steel wire. Since composite resin is highly susceptible to abrasion, the dentist may elect to incorporate amalgam or ceramic centric "stops" in the occlusal surface of the splint

B. OCCLUSAL SELECTIVE GRINDING

Occlusion can be adjusted by minor reshaping of the biting surfaces of the teeth so that they receive less force. This procedure is occlusal selective grinding.

Goals of occlusal selective grinding:

- elimination of occlusal trauma
- creation of symmetrical left/right chewing function
- prevention or elimination of parafunctions
- adjusment of occlusion after orthodontic treatment
- adjusment of occlusion before prosthetic replacement

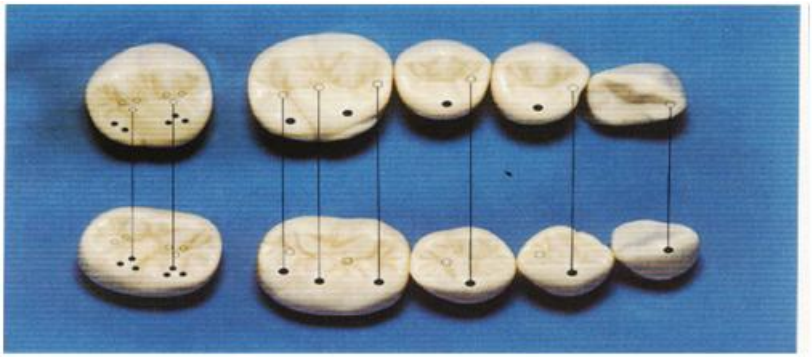


Figure 78. Contact of antagonists in intercuspation

Premature contacts in centric relation arc detected using the "tapping test". If the test reveals that at least three pairs of opposing teeth on each side of the arch make contact at the same instant, and if an interference-free slide in centric of no more than 1mm occurs, no corrective occlusal adjustment is indicated. If only one or two pairs of teeth makes contact initially, and/or if the subsequent slide to centric is greater than 1 mm or has a lateral component, selective grinding is indicated. An experienced dentist can perform selective grinding immediately. Only in difficult cases or with complex, expansive rehabilitation is it necessary to first mount the case in an articulator to study the precise interarch relationship. The goal of selective grinding is the creation of freedom in centric, i.e., free horizontal guidance of the cusps between CR and CO.

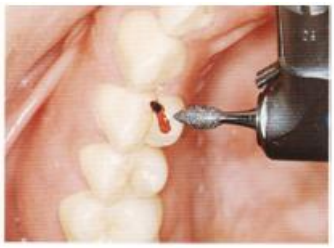


Figure 79. Premature contact

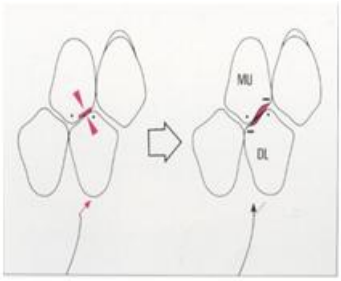


Figure 80. Premature contact on cuspal inclines

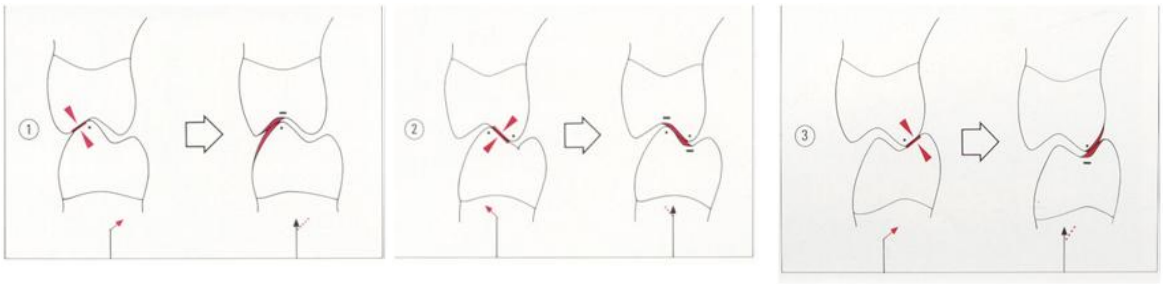


Figure 81. Buccal-lingual inclines

Question 6. Reconstruction – prosthodontics

The splinting that is effected by fixed bridgework does not, in and of itself, have any therapeutic advantage. For this reason, splinting per se is not

included in a prosthetic treatment plan unless there are specific reasons to do so, such as increased tooth mobility that disturbs the patient, or mobility that exhibits increasing severity due to occlusal trauma that is difficult to eliminate.

The insertion of a prosthetic replacement must not be associated with trauma to the gingiva or periodontium. Most important in this regard is that the bridge or denture 1) not promote plaque retention and 2) permit the patient to practice optimum oral hygiene. Consideration must be given to the characteristics of different types of prostheses (temporary and definitive):

1. Fixed bridgework Crown margins supragingival; crown contour; interdental and sub-pontic areas accessible for hygiene; functional, atraumatic occlusion.
2. Removable prostheses using telescope crowns or precision attachments Telescope crown margins supragingival; attachments accessible for hygiene; occlusion functional but atraumatic; major and minor connectors should not create plaque-retentive nitches.
3. Cast framework partial dentures Clasps (hold abutments bodily; occlusal rests; no continuous splinting); interdental areas accessible for hygiene; major and minor connectors should not create plaque-retentive areas; functional but atraumatic occlusion.

   Any fixed or removable temporary or definitive replacement must be planned and executed with regard for its possible effects on periodontal supporting structures. This is particularly important in the periodontitis patient, who often wears some type of temporary appliance throughout the entire treatment and healing phases of therapy.

Test control

1. Phases of periodontal therapy:

a) the initial
b) restorative
c) the final
d) corrective

2. Scaling is:

a) stabilization of mobile teeth
b) the process by which supra- and subgingival deposits are removed
c) gum massage
d) removal of granulation tissue
e) treatment of the root surface

3. Root planning is:

a) treatment of the root surface
b) removal of granulation tissue

c) splinting of mobile teeth
d) a variety of mukogingivalnoy operation

4. Methods of periodontal surgery are:
a) V-shaped excision of tissues
b) scaling
c) root planing
d) operation by widman
e) regenerative methods

5. Types of materials for bone replacement are:
a) autogenous bone tissue
b) xenogenous bone tissue
c) allograft
d) alloplastic material
e) all of the above

6. The purpose of setting the scientific and technological revolution is:
a) mechanical obstruction
b) suppression of rapid ingrowth of epithelium
c) does not give the connective tissue of the gums contact with the root surface
d) gives time for the restoration of periodontal structures
e) closure of aesthetic defects on the oral mucosa

7. Types of resection methods are:
a) gingivotomy
b) gingivectomy
c) operation by widman
d) root planning
e) all of the above

8. The purpose of mukogingival operations are
a) increase the width of the attached gingiva
b) closure of recession sites
c) deepening of the mouth cavity
d) removal of bone pockets
e) all of the above

9. Types of splinting of mobile teeth are:
a) temporary
b) semi-permanent
c) all of the above

10. Results of teeth polishing are:

a) elimination of premature contacts
b) elimination of aesthetic disorders
c) creating a smooth root surface
d) elimination of deep fissures on the masticatory surface
e) all of the above

LESSON 10. PHYSIOTHERAPY METHODS FOR TREATING PERIODONTAL DISEASE: INDICATIONS AND CONTRAINDICATIONS. PECULIARITY IN CHOOSING PHYSIOTHERAPEUTIC METHODS AND MEANS, DEPENDING ON DEVELOPMENT STAGE OF INFLAMMATION IN PERIODONTAL TISSUES.

The questions to be studied for the learning of the topic:

1. Indications and contraindications to physiotherapy in periodontology.
2. Physical methods of treatment.
   2.1. Drug electrophoresis.
   2.2. Darshonvalization.
3. Laser therapy.
4. Physiotherapeutic treatment methods of gingivitis.
5. Physiotherapeutic treatment methods of periodontitis.

Question 1. Indications and contraindications to physiotherapy in periodontology

Indications. Physiotherapeutic treatment methods in dentistry are applied taking into account the individual phases of inflammation and are aimed at certain parts of the pathological process.

Alteration stage (for an analgesic effect): electrosleep, acupuncture, electroreflexotherapy, laser therapy, hydrotherapy in the form of baths

Ultra-high-frequency (UHF) treatment, ultraviolet irradiation, laser therapy and hydrotherapy are shown at the ***exudation stage*** with the aim of an analgesic effect, improve tissue trophism, increase immunobiological processes, bactericidal action, to improve the maturation of abscesses. Hydrotherapy is used in the form of rinses (herbal extract, silver ionic solution, Borjomi mineral water), irrigations, oxygen foam cocktails.

All methods of physiotherapeutic treatment are used at the ***proliferation stage***.

Table 23. Contraindications to the use of physiotherapy of periodontal diseases

Common	1. Malignant and benign neoplasms. 2. Individual intolerance to electric current. 3. Cardiovascular diseases in the decompensation stage. 4. Myocardial infarction and after it (6 months). 5. Blood diseases. 6. Pregnancy. 7. Specific diseases. 8. Idiopathic diseases with progressive lysis of periodontal tissues.
Local	1. Acute inflammatory diseases of the oral mucosa.

	2. Proliferative processes on the mucous membrane of the oral cavity (papillomatosis, circumscribed hyperkeratosis, rhomboid glossitis). 3. All forms of leukoplakia.

Question 2. Physical methods of treatment

In the treatment of generalized periodontitis are essential physiotherapy techniques and treatments, some of which we have already described in the sections covering the methods of removal of dental plaque and treatment of gingivitis. In the treatment of generalized periodontitis are essential physiotherapy techniques and treatments, some of which we have already described in the sections covering the methods of removal of dental plaque and treatment of gingivitis.

Physiotherapy treatments can provide sufficiently effective and non-invasive effects on the affected area with minimal risk of side effects. Physical therapy can reduce the load on the dentist, depriving him of the routine work that does not require high skills. Natural therapies acting on reflex, and neurohumoral mechanisms that stimulate redox processes in the periodontium, reduced metabolism, improves trophic tissues and metabolism. As a result, they improve blood and lymph circulation, inhibit the growth of granulation tissue, reduce inflammation and congestion. Physical factors increase the activity of the elements of connective tissue, phagocytic activity of leukocytes, enhancing local immunity and accelerate the regeneration process.

The effectiveness of integrated treatment of any disease (including periodontal disease) increased 10 subject to such general principles of health care using natural factors (Ulaschyk V.S, 1992).

1. the dialectical unity of theory and practice (deep knowledge of pathogenic aspects of the disease, on one hand, and the mechanisms of action of physical factors - on the other);
2) universality, which is based on a single (reflex) mechanisms of action of all therapeutic physical factors and violations of self-regulation body during illness);
3) Unity etiotrop, pathogenic and symptomatic approaches (appointment of such method would simultaneously weakene or eliminate the influence of etiological factor, and would have acted for major pathogenic links and key symptoms);
4) small doses, which can stimulate the body's own defenses to provide regulatory and toning effect on various systems and cause homeostatic effect;
5) the adequacy of the actions for which duration methodic application and the main parameters physical factor must meet the nature and severity of the pathological process phases;

6) Individualization of physical therapy (treatment of a specific disease in a given patient and appointment procedures, taking into account the patient's biorhythms);
7) dynamism treatment (taking into account the patient given time and the dynamics of the pathological process);
8) the complexity of the action (including attitudes about the system as pathological basis of disease) in pathological determinant in this system is particularly important in chronic diseases;
9) variation of parameters (changes in the course of treatment of conditions of physical therapy, diametric characteristics);
10) sequence (strict accounting of the entire spectrum of pre-treatment in patients who received physiotherapy).

A number of preventive measures for periodontal disease are important physiotherapy. They are shown for almost all forms and stages of the disease and are widely used in various stages of diagnosis, comprehensive treatment, preventive and rehabilitative for the individual steps of the process and pathogenetic links for symptomatic treatment. Some physical factors directly affect the cells and tissues. In addition, they are all rich with receptor field irritating the mucous membranes of the mouth, nose or any other part of the body, providing reflex action, a positive effect on the nervous system, its vegetative division and hemodynamics, resulting in periodontal improvement of blood and lymph circulation, trophic and metabolism, abnormal granulation oppressed growth, decrease inflammation and congestion, increased activity of the elements of connective tissue, phagocytic activity of leukocytes and elements of the reticulo-endothelial system, accelerating the regeneration, and others.

Types of physiotherapy procedures:

- ✓ galvanization and electrophoresis with aloe extract, ascorbic acid, heparin, nicotinic acid, etc.;
- ✓ diadynamic currents;
- ✓ amplipulse;
- ✓ darsonvalization gum;
- ✓ UHF in oligothermal dose;
- ✓ GNL to the area of gums;
- ✓ massages of gums;
- ✓ hyperbaric oxygen therapy;
- ✓ local hypo-hyperthermia;
- ✓ ultrasound therapy.

2.1. Drug Electrophoresis

Valuable feature of physiotherapy is to stimulate non-specific reactivity of the tissues and the body's defenses, pathogenetic orientation of physical methods in the treatment of various diseases. Acute inflammation in

periodontal tissues in the early stages shows types of physical therapy, reducing the permeability of blood vessels, stimulating the flow of fluid from the source of ignition. For effects on humoral regulation of the pathological process in order to reduce the education of biologically active compounds, there are useful techniques contributing to the stabilization of cell membranes, thus limiting the formation of hydrolases and their transition into the fabric.

Physical therapy is performed after removal of dental plaque and relief of acute inflammation. There are some treatments such as UV, hydrotherapy, laser, aeroionotherapy etc. In some cases they can be done from the beginning of complex treatment. In the treatment of periodontal disease, often electrophoresis input of drugs in tissues by direct electric current is used.

When performing electrophoresis, active electrodes are placed upon the gingival margin through hydrophilic pad soaked with drugs. Passive electrode is fixed in the hand or forearm. Gasket passive electrode moistened with tap water or isotonic sodium chloride solution. Amperage set individually, but not more than 0.1-0.3 μA per 1 cm^2 active area of the electrode. Duration is 10-20 minutes. Number of sessions is 10-12. Assign electrophoresis in acute and those that worsened chronic catarrhal and hypertrophic gingivitis, periodontitis after elimination of active inflammation and periodontal disease.

In hypertrophic gingivitis recommended electrophoresis 10% solution of calcium chloride alternately with the anode and cathode. It provides deposit in gum tissue calcium, chlorine and then to anti-inflammatory (calcium) of which provides sustained clinical benefit. For influence the exudation processes: electrophoresis with 5% solution of ascorbic acid, 1% solution of vitamin P, trypsin, ribonuclease (1 mg/ml) solutions are introduced from the anode. Aqueous solutions of aloe extract, 1% nicotinic acid solution, viproxin, heparin solution (1:15) are recommended to be introduced from the cathode.

During periodontal disease is recommended electrophoresis 1-2-4% sodium fluoride, 2.5% solution of calcium glycerophosphate, while hyperesthesia of hard tissue of teeth is thiamine chloride solutions with novocaine. Electrophoresis of these drugs improves mineral metabolism and trophic periodontal tissues, reduces the effects of osteoporosis bone. If you enter the complex preparations consisting of oppositely charged ions, electrophoresis is carried out alternately in one day with a negative in the second or the positive pole. More pronounced therapeutic effect is noted in electrophoresis drugs in focal dosed vacuum. With this method, the depth of penetration of the drug through the oral mucosa is increased by 3-5 times.

Vacuum electrophoresis carried out using electron system (LHC) and a set of different electrodes (vacuum cell). The method allows you to enter in

the periodontal tissues of various drugs, including ions of calcium, phosphorus, fluorine and other trace elements. Sometimes appointed by electrophoresis in combination with other treatments (UHF, microwave therapy, UV, etc.). In addition, it is effective to use electrophoresis in a magnetic field, for example, magnetoelectrophoresis.

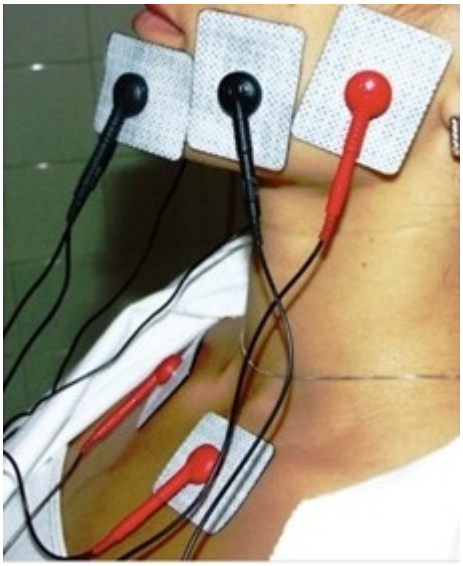


Figure 82. Patient with electrodes to promote healing and stimulate lymph drainage (left side view).

2.2. Darsonvalization

Darsonvalization is electric currents produced by the devices' «Iskra-1» and «Iskra-2». The device "Spark" is high-generator (150 kHz) AC low power (0,015-0,02 A), high voltage (to 20 kV), which gives greatly damped discharge. To set the device attached vacuum glass electrodes of different shapes.

D'Arsonval currents inhibit the sensitivity of peripheral nerve receptors, providing an analgesic effect, reduce itching in the tissues, dilate blood vessels, improve trophic tissues, relieve spasm of blood vessels, increase the migration of leukocytes, etc.

Darsonvalization is indicated for chronic gingivitis, generalized periodontitis, periodontal disease.

Treatment D'Arsonval currents spend in quiet mode and spark. In the first case imposes an electrode directly on the skin or mucous membrane, and the effect of the current on the fabric is weak. If spark stimulating effect of the current is stronger due to slippage sparks through the air gap to 0.5-1.0 cm during treatment using silent discharge electrode is slowly moved along the gingival margin, avoiding the spark discharge through dental hard tissues. The procedure lasts 20 minutes. The course of treatment consists of 10-20 sessions every day or every other day. It is recommended that the form be combined with UFO purpose. Spark diathermy coagulating action provides and promotes the formation micronecrosis. This property is used for the treatment of hypertrophic gingivitis.

Question 3. Laser therapy.

A. Laser basics. Lasers produce light energy that can be absorbed by a target tissue. The absorption process produces a thermal reaction in that tissue. Depending on the instrument's parameters and the optical properties of the tissue, the temperature will rise and various effects will occur. The results can be useful in the treatment of periodontal disease. In general, most non-sporulating bacteria, including anaerobes, are readily activated at temperatures of 50 degrees C. The inflammatory soft tissue present in periodontal disease can be removed with a temperature of 60 degrees C; moreover, hemostasis can also be achieved within the same heat parameters. Laser excisional or incisional surgery is accomplished at 100 degrees C, where vaporization of intra- and extra cellular water causes ablation, or removal of biological tissue. This is the temperature at which calculus could be removed from the root surface.

There are many different types of lasers and each produces a specific color or wavelength of light; however, the light from nearly all of the available dental lasers is invisible to the human eye. Each wavelength has a somewhat unique effect on dental structures, due to the specific absorption of that laser energy in the tissue. Some lasers are only absorbed by blood and tissue pigments, while others are only absorbed by water as well as "hard" tissue, like enamel, dentin, bone, and calculus. (11)

More specifically, the wavelengths can be categorized into three groups:

1) **Erbium lasers (Er,Cr:YSGG and Er:YAG)** can be used for calculus removal as well as for soft tissue debridement, because of the their excellent absorption in apatite crystals and water.

2) **Carbon Dioxide lasers** also easily interact with free water molecules in soft tissue as well as vaporizing the intracellular water of pathogens.

3) **Diode and Nd:YAG** wavelengths would target inflammatory tissue and pigmented pathogens.

Clearly, all of these lasers can disinfect and detoxify periodontal tissues. These lasers can successfully and safely be used on a wide range of the population such as children and pregnant women unlike some prescribed and/or sulcularly delivered medicaments. Unlike those medicaments, the patient will not experience allergic reactions, bacterial resistance, or untoward side effects when the laser is used.

There are significant differences in the depth of penetration of dental laser wavelengths: The Erbium wavelengths are absorbed on the surface of the tissue with a depth as little as 5 microns and Carbon Dioxide's radiation will travel about 0.5 millimeters; whereas the diode and Nd:YAG can penetrate up to a few millimeters. The diode and Nd:YAG lasers use a small diameter (300 micron) flexible glass fiber for laser transmission; however,

Erbium and Carbon Dioxide lasers utilize either rigid quartz or sapphire tips or metal cannulas.

B. Initial Periodontal therapy applications of various laser wavelengths

Erbium. The Erbium lasers are effective in removing calculus, as well as producing pocket depth reduction. Several references indicate safe and effective root substance removal comparable with conventional instrumentation; moreover, there were no negative thermal effects on the pulpal tissue. Furthermore, these lasers are highly bactericidal against P. gingivalis and A. Actinomycetemcomitans, as well as effective in removing lipopolysaccharides and other root surface endotoxins.

Carbon Dioxide. This wavelength is a very effective in removing soft tissue, and is used for removal of diseased and inflamed tissue, with good hemostasis and bacterial reduction. However, only one clinical study has been published to show that this laser can decrease probing depth, and thus it is not yet widely used for initial periodontal therapy. Carbon Dioxide's chief disadvantage is that the current emission mode's irradiation produces severe thermal damage and carbonization to the root surface. New modalities are under development to address this problem.

Diode and Nd:YAG. These lasers are only used for treatment of the diseased periodontal soft tissue, allowing significant bacterial reduction and removal of the inflammatory products while creating excellent hemostasis. As mentioned above, these instruments employ a flexible fiber optic delivery system that allows the clinician easy and safe access around the periodontal pocket. The wavelengths are transmitted through water and are very poorly absorbed in apatite crystals, making them an excellent choice to use in a periodontally involved sulcus that has dark inflamed tissue and pigmented bacteria. These lasers are contraindicated for calculus removal not only because of their ineffective absorption but also because of the possibility of heat build-up due to the interaction with darkly colored deposits.

C. Treatment planning and protocol. Therapy should always begin in the area with the deepest pocket depths and progress to the more shallow ones. On subsequent appointments, the clinician should plan to relase the previously treated sites with a bacterial reduction setting which reduces the bacterial load and enhances the healing process.

The first one hour period consists of the following steps:

- ✓ Anesthesia as needed (topical or injected)
- ✓ Calculus removal employing an Erbium laser and/or an ultrasonic scaler with antimicrobial irrigant
- ✓ Detailed hand instrumentation to finish the hard deposit elimination
- ✓ Laser bacterial reduction of soft tissue

- ✓ Laser coagulation of the treatment sites
- ✓ Ultrasonic antimicrobial irrigation
- ✓ Post-op instruction/home care instruction

For patients requiring multiple visits, the laser is used with a bacterial reduction setting on previously treated sites with the fiber calibrated to account for healing that has takenplace since the previous appointment.

D. Soft tissue laser treatment. Calculus removal is very straightforward whether a laser, ultrasonic, or a hand scaler is used. Following scaling, the author recommends a diode or Nd:YAG laser be used for the soft tissue phase of treatment, and those wavelengths' fibers must be calibrated prior to performing bacterial reduction. After tooth debridement, each pocket is probed to recheck the architecture and reconfirm the depth. The probe is then placed next to the laser fiber assembly and the fiber length is adjusted in length (figure 83).

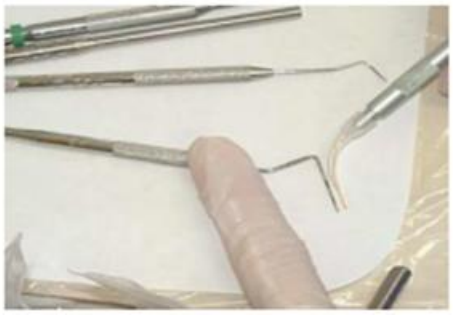
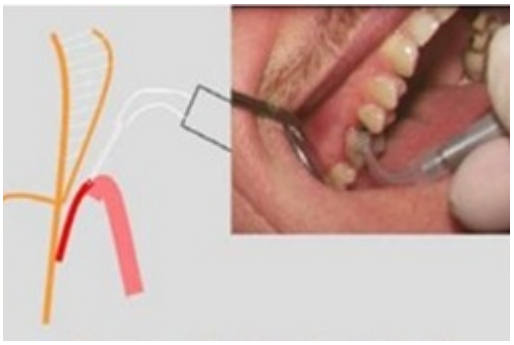


Figure 83. Calibration of laser fiber 1mm less than pocket depth

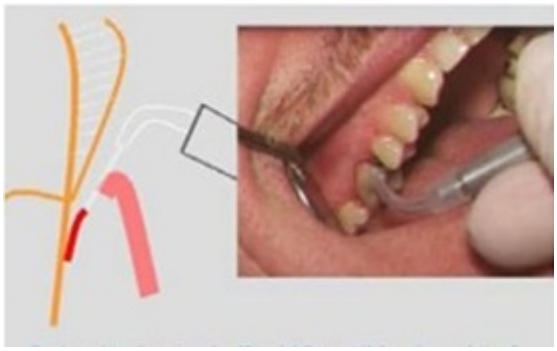
The calibration depth is 1 mm shorter than the pocket. This measurement is important because the laser energy will penetrate through the tissue and the adjustment will minimize any interaction with the epithelial attachment. At subsequent therapy appointments the fiber calibration is 2 mm less than the initial pocket depth to take into account the healing that takes place from the apical end of the pocket after initial therapy.

E. Laser Fiber Placement and Therapy. The laser fiber's design allows energy emission at the tip, thus making the fiber "end cutting," and the clinician must maintain contact with the inflamed epithelial lining of the pocket. The fiber is placed on the tissue at the top of the sulcus which directs the laser energy away from the tooth structure. (figures 84, 85)



Start at the of the sulcus and aim the fiber at the diseased tissue, not toward the tooth structure

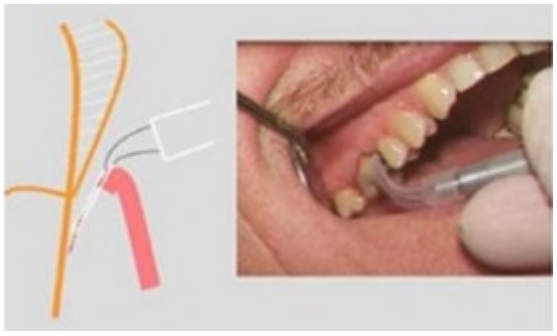
Figure 84. The laser is placed at the top



Begin to lase, keeping the fiber fairly parallel to the surface of the epithelium and adjacent inflammatory connective tissue

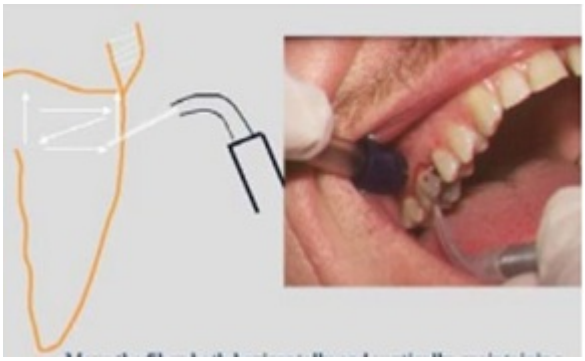
Figure 85. The laser is activated and of the sulcus in contact with the soft tissue the fiber is oriented parallel to the long axis of the tooth.

The fiber is moved both horizontally and vertically, and contact is maintained with the soft tissue down to the calibrated depth (figures 86, 87).



Continue lasing until the calibrated depth is reached

Figure 86. The laser is moved horizontally reached



Move the fiber both horizontally and vertically, maintaining contact with the soft tissue at all times. Inspect the fiber frequently and wipe off accumulated tissue

Figure 87. The calibration depth is vertically along the soft tissue.

The distal end of the fiber must be inspected frequently, any accumulating tissue and debris must be wiped off to avoid the laser's energy from heating up the darkly colored material (figure 88). Bacterial reduction is finished when signs of fresh bleeding occur (figure 89). The doctor would then proceed with the adjacent tooth, continuing the above steps.

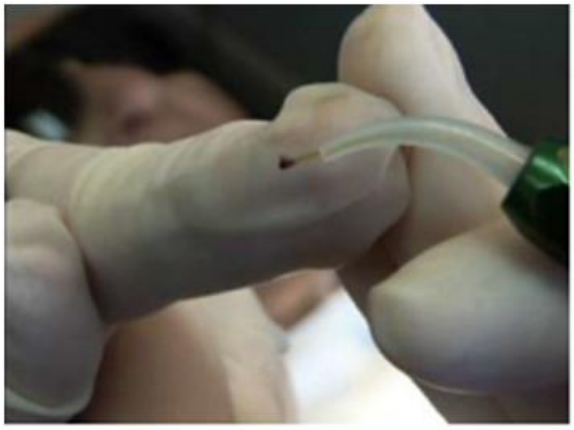
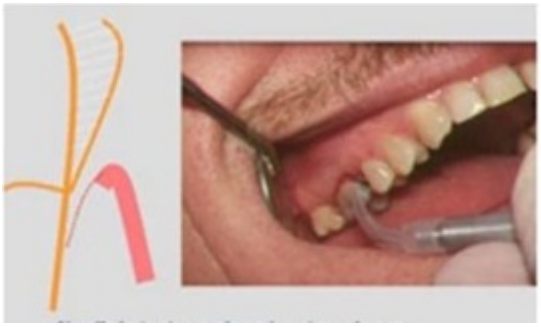


Figure 88. Acumulated debris



Usually lasing is complete when signs of a new wound site (fresh bleeding) have appeared

Figure 89. Bacterial reduction is wiped off completed when fresh bleeding occurs

When this first laser step; has been completed on all teeth scheduled to be treated on that appointment, the laser energy is then changed to a coagulation setting and the fiber is held in contact with the tissue with the same motion from the top of the sulcus to the bottom until the bleeding stops. Laser therapy is now completed.

D. Post Operative Instructions. It is important that the patient perform adequate oral hygiene following laser treatment. In addition to soft brushing and careful flossing, additional aides might be suggested. The lased site is not particularly fragile, but spicy or grainy foods that would irritate healing tissue should be avoided. Generally mild analgesics should easily manage any discomfort. Rinsing and irrigation can begin immediately, but light swishing is recommended for the first few days.

Question 4. Physiotherapeutic treatment methods of gingivitis.

Table 24. Types and characteristics of physiotherapeutic methods used for the treatment of gingivitis

Method	***How to use***
Acute gingivitis	
Hydrotherapy	8-12 procedures (furatsillin, decoctions, tinctures of medicinal herbs are added to the water for the procedure. Irrigation with water saturated with carbon dioxide is used).
Short-wave ultraviolet (SWU)	5-6 procedures
Hydroionotherapy	inhalation with anesthetics and anti-inflammatory drugs
Finger and vibration	after acute processes

massage	
Laser therapy	the parameters which have anti-inflammatory effects - the power density is 100-200 mW / cm2; exposure is 2-4 minutes on irradiation zone.
Simple marginal gingivitis	
Hydrotherapy	irrigation is carried out with water saturated with carbon dioxide, oxygen. Irrigation can be carried out with mineral, sulfide, sea, rhodon waters in a sanatorium (10-15 procedures). Oral baths with a 0.06% chlorhexidine solution are prescribed for 3 minutes in the morning and evening after teeth brushing for the period of training in oral hygiene (7-10 days).
Short-wave ultraviolet (SWU)	10-15 procedures every other day in combination with hydrotherapy
Electrophoresis	15-20 procedures. After the elimination of active inflammation. 10% calcium chloride solution, 5% ascorbic acid solution, 1% vitamin P solution, 5% solution in the presence of bleeding and exudation are used for electrophoresis.
Darsonvalization	10-20 procedures daily or every other day (used to relieve venous stasis and edema in combination with UV radiation, as an anaesthetic and antipruritic).
Diadynamophoresis	4-5 procedures daily or every other day (the duration of the session during treatment is increased from 5 to 15 minutes).
Finger massage	once a day at bedtime after teeth brushing for 5-7 minutes
Vacuum Therapy	4-6 hematomas are done in different areas of the gums during each session. The next visit is prescribed after 3-4 days, when the hematomas resolve. 8-12 procedures, duration is 5-7 minutes. Used during congestion and edema.
Laser therapy	Laser radiation parameters are 1-50 mW / cm^2, exposure is from 20 s to 2 minutes per irradiation zone.
Hyperplastic gingivitis (edematous form)	
Hydrotherapy	✓ irrigation with water saturated with carbon dioxide, oxygen with the addition of an antiseptic, ✓ rinse with sodium chloride solution, chamomile decoction, furatsillin.
Electrophoresis	It is recommended to carry out with a 10% solution of calcium chloride alternately (from the anode and

	cathode) for providing an anti-inflammatory effect. Electrophoresis with a 5% solution of ascorbic acid, 1% solution of vitamin P, trypsin, ribonuclease (1 mg / ml) solutions from the anode is used to influence exudation processes. Aqueous solutions of aloe extract, 1% nicotinic acid solution, viproxin, heparin solution 1:15 is recommended to be introduced from the cathode.
SWU	8-10 procedures
Darsonvalization	10-20 procedures to eliminate the proliferative reaction of gum tissue
Phonophoresis	12 procedures, duration is 10 minutes. 15% dibunol solution in sunflower oil.
All types of massage	Applied after the elimination of inflammatory processes
Hyperplastic gingivitis (fibrous form)	
Point diathermocoagulation of the gingival papillae	The root needle is introduced into the tissue of the gingival papilla to a depth of 3-5 mm, coagulation is carried out for 2-3 s. Power is 6-7 W. 3-4 papillae coagulate at the same time. 4-5 papillae under anesthesia carry out coagulation in one visit.
Hydrotherapy	It is introduced from the cathode.
Electrophoresis	rinse with weak solutions of antiseptics (0.25% chloramine solution, 0.1% decamine solution)
Paraffin treatment	10-15 procedures. Melted paraffin is applied to the dried gums from the vestibule with a special syringe or napkins (8-10 layers of gauze for 10 minutes).
Cryodestruction	Carried out in case of 3rd degree hyperplastic gingivitis (exposure temperature is 60-140^{0}C, exposure is 35-44 s).
Ulcerative necrotic gingivitis	
Irrigation with water saturated with carbon dioxide, oxygen	antiseptics (furatsillin, ethacridine lactad, etc.) are added to remove necrotic tissues. Spray irrigation by antiseptic solutions is carried out after mechanical removal of tissue decay. Rinses with a 0.25% solution of chloramine 4-6 times a day, a 0.05% solution of chlorhexidine bigluconate.
SWU	combined with irrigation water on the same visit. 6-8 procedures.
Aerosol therapy	inhalipt spray is used (before irrigation it is necessary to rinse the mouth with warm water, remove necrotic deposits, keep the drug in the oral cavity for 5-7

	minutes)
UHF electric field	appointed in an athermic dose or microwaves in the case of a pronounced reaction from the lymph nodes. Power is 1-3 W. Duration is 5-7 minutes

Question 5. Physiotherapeutic treatment methods of periodontitis.

Table 25. Types and characteristics of physiotherapeutic methods used for the treatment of periodontitis

Method	***How to use***
Acute periodontitis	
Irrigation with solutions of medicines	mineral water is used for irrigation: sulfide, carbonic, water with a 1% solution of romazulan, with decoctions of herbs (chamomile, sage, St. John's wort); water subjected to magnetic processing.
Oral baths with antiseptics	furacilin, potassium permanganate, rivanol) are prescribed 3 times a day after meals for 5-10 minutes. The solution temperature is 34-35°C.
Short-wave ultraviolet (SWU)	10 procedures daily.
UHF electric field	athermic dose or low heat dose is prescribed for resorption of abscess formation. Power is until 30 W. Duration of one procedure is 10 minutes. 4-10 procedures.
Microwave therapy	✓ Local abscess formation: 4-8 procedures daily. Duration is 5-7 minutes. Power is 1-3 W. ✓ Faster removal of inflammation and bleeding: affect with microwaves on both cheeks on the right and left for 5 minutes (output power is 7 W). Electrophoresis of gums with vitamin B_1 for 20 minutes is carried out immediately after microwave therapy. Current density is 0,2 mA/cm^2, course is 12 procedures.
Fluctuating currents	are used in case of exacerbation of processes with purulent discharge from dental pockets; the duration of the procedures is 12-15 minutes, the course of treatment is 6-8 procedures. Fluctuorization is combined with the electric field of UHF and microwave therapy in case of severe inflammatory processes in the periodontal tissues.
Laser therapy	Severe inflammatory processes before surgery and after surgery (helium-neon laser).
Chronic periodontitis	
Ultrasound	to remove dental plaque

Hydrotherapy	irrigation with mineral waters, with medicines (1% solution of romazulan, 2% percent solution of citral, decoctions of chamomile, sage, St. John's wort). 8-10 procedures
Hydroionotherapy	aeroionization is carried out for 10 minutes at the rate of 100-150 billion light negative ions per procedure; course of treatment is 10-15 exposures daily or every other day (the procedure is prescribed for hypertension)
Darsonvalization	in case of venous stasis or edema. 10-15 procedures. Duration is 10 min for each jaw
Magnetotherapy	magnetic mouthguard is used for 20-30 days and has an anti-inflammatory effect. It is permissible to use rinsing with magnetized water with medicines
Electrophoresis	➢ bleeding gums: electrophoresis of calcium, vitamins C and P is prescribed for 15-20 procedures ➢ for anti-inflammatory action: heparin from the cathode (5 ml of heparin is dissolved in 30 ml of distilled water). 15 procedures. Duration is 20 min. ➢ electrophoresis of tannin, zinc or copper from a 3% sulfate solution of these trace elements is used in case of gum itching. ➢ trypsin solution electrophoresis for 20 min, treatment course is 20 procedures are used in case of pus from pathological gingival pockets
Electrosleep	10-20 procedures. Duration is 20-60 min. In case of a disorder of the central nervous system
Vacuum Therapy	is prescribed if there are no abscesses. 6-8 procedures
Auto massage	is prescribed after the removal of dental plaque
Periodontal atrophy	
Electrophoresis	electrophoresis of calcium (10% solution of calcium gluconate), fluorine (2% solution of sodium fluoride), phosphorus (2.5% solution of calcium glycerophosphate) is indicated in case of increased sensitivity of the teeth neck. 20 procedures daily. Duration is 20 min.
Darsonvalization	is used in the initial stage of periodontal atrophy (high-frequency discharge has a tonic effect on periodontal vessels and is an electric massage)
Automassage	is recommended in the morning after teeth brushing. The gums are massaged with the index finger, placing it on the transitional fold at the base of the interdental papilla. Movements are made to its apex down, circular. Duration is 3-5 minutes
Hydromassage	Duration is 7-10 minutes. 20 procedures. The stream of

	liquid with carbon dioxide or water (pressure is 1.5-2 atmospheres) is directed to the periodontal tissue at a distance of 20-30 cm. The procedure is performed by the patient.
Vibromassage	2 courses of vibration massage per year. Duration is until 10 minutes. The procedure is carried out using a vibrator, which moves vertically from the transition folds to the neck of the tooth.
Oxygen therapy	10-12 injections. 1 injection contains 5 ml of oxygen. Blister forms at the injection place, which resolves within 20-30 minutes. It is also possible to saturate the tissues with oxygen using cotton swabs moistened with hydrogen peroxide, potassium permanganate. It is possible to use irrigation with these solutions, their aerosol spraying (water jet or drug solution is enriched with oxygen).

Test control

1. Contraindications to the appointment of physiotherapeutic treatment are:

a) malignant neoplasms
b) diseases of the blood
c) dysfunction of the liver and kidneys
d) cardiovascular and respiratory insufficiency
e) all of the above

2. The effect of physiotherapy on the human body is:

a) anesthetic effect
b) reduction of the inflammatory process
c) improve trophism of tissues
d) strengthens the reparative processes
e) reduces the immunity

3. Types of physiotherapy procedures are:

a) electrophoresis
b) open curettage
c) gum massage
d) laser treatment
e) antibiotic therapy

4. Medicinal electrophoresis is:

a) the action of pulsed high-frequency current of small force and high voltage

b) the effect of a low-voltage direct current and drugs introduced with it into tissues
c) the effect of an alternating sinusoidal current of high frequency and high voltage
d) effect of low-energy laser radiation
e) effect of uv rays on the tissue

5. Sclerosing effect with medicinal electrophoresis is provided by:
a) 10% calcium chloride solution
b) Vitamins C, PP
c) Calcium gluconate
d) Heparin
e) Metronidazole

6. Therapeutic effect of laser therapy is
a) anti-inflammatory action
b) bactericidal and bacteriostatic action
c) decongesting action
d) anesthetic action
e) all of the above

7. Methods of laser therapy are
a) contact
b) labile
c) non-contact
d) stable
e) temporary

8. Types of therapeutic gum massage are:
a) vacuum
b) vibrating
c) hydromassage
d) automassage
e) all of the above

9. Automassage is:
a) irrigation of the oral cavity with decoctions of herbs
b) creation of a vacuum on the surface of the mucous membrane of the gum 300-400 mm hg.
c) finger self-massage
d) effects of mechanical oscillations of low frequency on the process of microcirculation
e) all of the above

10. Errors in carrying out physiotherapeutic treatment

a) violation of technique and methodology
b) underestimation of concomitant pathology
c) reduction of the inflammatory process
d) untimely appointment of physiotherapeutic treatment
e) increased immunity

Test answers

Questions									
1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
***7 semester*. Lesson 1**									
a,b,d,e	a	a	e	a	c	b	a,b,d,e,f	e	d
Lesson 2									
b,d	b	b,c, d	a, c	a,e	a,c,e	b	a,c	a,c,e	a,b
Lesson 3									
c	a	b	b	g	d	e	c	d	b
Lesson 4									
f	b	a	f	a	a,b	a	a	a	a
Lesson 5									
a,b,c, d	d	a,b	e	a,b	f	f	d	f	b
Lesson 6									
a	c	a	b	b	c	a	g	j	b
Lesson 7									
a	b	a	c	e	e	g	b	a	c
Lesson 8									
g	h	i	h	b,d,e	b,d,e	i	f	f	f
Lesson 9									
b	a,b,d	a,b,c,d	b,c	b,c	b	c,d,e	c,d	e	c
Lesson 10									
b	d	b	a	a	c	c	a	b	a
Lesson 11									
a,b	a,b	a	a	c	b	c	d	b	d
Lesson 12									
c	b	c	a	a	a	a,b,c,d	b,c	a	a,b
Lesson 13									
a	c	a	a	a	a	d	a	a	d
***8 semester*. Lesson 1**									
b,c,d	b,d	b	b	a,b	a,c	a,e	a,c,e	a,c,e	a,c
Lesson 2									
c	a	a	a	a	e	a,c,d,f	e	a,d	a,c,d
Lesson 3									
e	a	c	e	e, c	a,e,f	a	a	d	e
Lesson 4									
b	d	a	b	a	f	c	c	b	c
Lesson 5									
c	a	a	a	c	e	a,c,e	a,b	d	e
Lesson 6									
a,b	b,c,d	c	a,b	e	d	a,c	c,d	c	a,b

Lesson 7									
f	f	h	a	e	f	c	b,c	a	b,c
Lesson 8									
e	f	e	c	e	g	c	c	a	a
Lesson 9									
a,b,d	b	a	a,d,e	e	a,b,c,d	a,b	a,b,c	c	a
Lesson 10									
e	a,b,c,d	a,c,d	b	a,d	e	a,b,c,d	e	c	a,b,d

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Учебное издание

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Редактор Н.Э. Колчанова
Компьютерная верстка С.В. Суслина

Подписано в печать _____2020. Формат 64x84 $^{1}/_{16}$.
Бумага типографская № 2. Гарнитура_____. Усл. печ. л._____.
Уч.-изд.л._____. Тираж_____ экз. Заказ №_____.

Издатель и полиграфическое исполнение: УО «Витебский
государственный медицинский университет».
ЛП №02330/453 от 30.12.2013
пр. Фрунзе, 27, 210602, г. Витебск