Ukrainian medical stomatological academy

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PERIODONTAL DISEASES IN CHILDREN

TEXTBOOK

Poltava – 2020

УДК 616.314.17–002–053.4/.7(07)

Рекомендовано вченою радою Української медичної томатологічної академії як навчальний посібник для іноземних студентів здобувачів вищої освіти ступеня магістра, які навчаються за спеціальністю 221 «Стоматологія» у закладах Вищої освіти МОЗ України (протокол №8 від 11.03.2020 р.)

Kaskova L.F., Amosova L.I., Yanko N.V., Kulai O.O. Periodontal diseases in children: textbook. – Poltava, 2020. – 135 p.

Навчальний посібник складено у відповідності до діючої типової навчальної програми для студентів стоматологічного факультету. У посібнику розглядаються питання, що висвітлюють анатомо-фізіологічну будову тканин пародонта в різні вікові періоди. Викладено основи етіології та патогенезу найбільш поширених хвороб пародонта у дітей. Детально розглянуто аспекти клінічних проявів, сучасних методів діагностики, патогенетичної та етіотропної профілактики хвороб пародонту в дитячому віці. Подано схеми лікування хвороб пародонту в дитячого населення.

Розраховано на англомовних студентів та викладачів стоматологічних факультетів медичних вузів.

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CONTENT

Preface

Periodontal diseases in children is a detailed guide to periodontal diagnostics and treatment that takes students from assessment of periodontal patients to using of different contemporary methods of their treatment.

This book is written keeping in mind the special needs of the students such as the references, suggested readings and related landmark studies that would be helpful for undergraduates. The content is drawn up as per the syllabus of the higher education standard of Ukraine and educational program of Ukrainian medical stomatological academy in dentistry.

The textbook is organized into seven chapters; and each chapter is further subdivided into several subchapters. With the matter subdivided into smaller chapters, students will find easier to achieve their learning goals. Starting with the basics in chapter one, the text flows gradually from anatomy and physiology of periodontium to etiology, pathology, diagnosis and treatment with the inclusion of the recent advances in the field of periodontology. The effort has been made to learn the subject in a simpler and easier way by the use of tables, roenthenograms. This textbook covers relations between periodontal diseases in children and other diseases.

Hoping that the content will be enough to stimulate insight and new trains of thoughts into the subject of Periodontics in children which will be immensely educative and helpful for the students and for the faculty.

Chapter 1
ANATOMY AND PHYSIOLOGY OF PERIODONTAL TISSUES

1.1. Short development of periodontium

The *periodontium* is defined as tissues supporting and fixing the tooth, comprises root cementum, periodontal ligament, bone lining of the tooth socket (alveolar bone) and part of the gingiva facing the tooth (dentogingival junction).

Periodontium tissues start development in the antenatal period. The alveolar ridges of infants are represented by the gum pads, which are formed by thickening of the gingival mucosa. Gum pad is a duplicate mucous membrane with a large number of elastic fibers, which increases slightly and forms a ridge that provide the function of suction. In this period alveolar processes of the upper and lower jaws place on a level of the palate and the bottom of the oral cavity. The development of periodontal tissues continues with teeth eruption (M.F. Danilevsky, G.M. Vishnyak, A.M. Politun, 1981). Periodontium and alveolar processes are formed simultaneously with the root development. Their morphological formation finishes 6 months - 1 year after complete root formation.

1.2. Structure and age-related changes of gums in children

Gums or gingivae is a part of the oral mucosa that covers the alveolar processes of the jaws and surrounds the cervical portion of the teeth.

Macroscopic anatomy of the gingiva

Free gingiva (marginal gingiva): unattached portion of the gingiva that surrounds teeth in the region of the cementoenamel junction.

Gingival sulcus: V-shaped, shallow space between the free gingiva and the tooth surface. The depth is 1-3 mm for a clinically normal gingival sulcus and is measured

using a periodontal probe around the tooth. The base of the sulcus is formed by junctional epithelium.

Attached gingiva: Gingiva that is tightly connected to the cementum on the cervical third of the root and to the periosteum (connective tissue cover) of the alveolar bone. It lies between the free gingiva and the alveolar mucosa.

Interdental gingiva: the portion of the gingiva that fills the area between two adjacent teeth apical to the contact area.

1. *Papilla/Papillae:* the interdental (or interproximal) gingiva consists of two interdental papilla (one facial, one lingual)
2. *Col area: v*alley-like depression in the interdental gingiva. It lies directly apical to the contact area. The col is not present if the adjacent teeth are not in contact or if the gingiva has receded.

Mucogingival junction: the clinically visible boundary where the pink attached gingiva meets the red, shiny alveolar mucosa.

Alveolar mucosa: Movable tissue loosely attached to the underlying bone. Nonkeratinised, thin, smooth and shiny epithelium.

The layers comprising the oral epithelium are the stratum basale, stratum spinosum, stratum granulosum, and the stratum corneum. The stratum spinosum is also termed the spinous or prickle cell layer and is named for the appearance of prominent peripheral cytoplasmic processes which resemble spines at the light microscopic level. The cytoplasm is rich in tonofilaments which terminate in the attachment plaques of desmosomes which attach to adjacent cells. The granular cell layer (stratum granulosum) is characterized by the presence of keratohyalin granules which are believed to play a role in the process of keratinization. The stratum corneum is a keratinized cell layer characterized by flattened, pyknotic cells composed of tightlypacked tonofilaments. Organelles are rare and keratinization may be associated with cell lysis. As cells proceed from the basal to the keratinized layer, they become progressively flatter, organelles diminish, tonofilaments dramatically increase, and the nuclear-cytoplasmic ratio decreases with eventual loss of the nucleus. There is a shift from aerobic to anaerobic metabolism which is accompanied

by an increase of glycogen in the upper layers. Keratohyalin granules appear in the granular cell layer, and the number of desmosomes increase while the size of individual desmosomes decreases. The col is a valley-like depression of the interdental gingiva which connects facial and lingual papillae and conforms to the shape of the interproximal contact area. The connective tissue (CT) of the gingiva consists of cells, fibers, and ground substance (proteoglycans and glycoproteins). A brief review of these will be presented. Cells constitute about 5% of the CT and include fibroblasts (65%), mast cells, PMNs, macrophages, lymphocytes, and plasma cells.

In primary dentition the gingival epithelium is thin, slightly differentiated, with a slight deepening of the epithelial papillae, without phenomena of incision, and non-keratinized. Also basal membrane is thin, slightly differentiated (H.M. Mergembayeva, 1972). Zappler (1948) and Ruben et al. (1971) studied the periodontal structure in children and observed that the deciduous dentition presents more vascularized connective tissue and less organized collagen fibers. In children under 3 years of age, the oral mucosa (including the gum) has a lot of glycogen. Number of cells increases, fibers become tough with periodontium development.

At 2,5 – 3 years (the period of primary dentition aging) glycogen in the gums is not detected in contrast to other parts of the mucous membrane (lips, cheeks, soft palate, tongue). The appearance of glycogen in the gums of children elder than three years indicates the inflammation. During inflammation glycogen builds up in the cells of the prickly layer of the gums due to the impaired keratinization of the epithelium.

The gingival connective tissue in children from one to three years old has low differentiation and fewer cellular elements; collagen fibers are lost, not oriented enough, elastic fibers are absent. After age of 4 years the number of cells increases, the fibrous structures of the connective tissue gradually tighten. There are cellular elements – fibroblasts, fibrocytes, and lymphocytes in gingivae of children, but histiocytes and plasmocytes are absent.

Change dentition is characterized by next changes: with age, the thickness of

the keratin layer increases. Parts of the connective tissue protrude into the epithelium forming the papillae of the connective tissue. Basal membrane becomes tight, its permability decreses (V.E. Sklyar, 1969). Collagen slowly get mature (C. Pearse, 1962). Sanché et al. (2011) performed a descriptive analysis of infiltrating inflammatory cells in the junctional epithelium and showed that 70% of the children's samples, as well as 60% of the adult samples, did not have these types of cells.

Gums in permanent teeth period in children have mature differentiate structure and two precise functions: to isolate periodontium from environment and to provide tooth fixation. Mucous membrane of alveolar bone (gums) is keratinized, belonges to the masticatory type. Epithelium is thin and non-keratinized in the area of gingival sulcus, mesial and distal teeth surfaces (Kohl, Zander, 1961). The non-keratinized areas are sensitive to different influnces.

Gingiva presents a textured surface similar to an orange peel and is referred to as being stippled (attached gingiva). Stippling is best viewed by drying the gingiva. It is absent in infancy, first appears in children at about 5 years of age, increases until adulthood and frequently begins to disappear in old age.

Gingival sulcus (crevice) plays a crucial role in sealing off the oral environment from the periodontal tissues. Distance from the gingival margin and bottom of the gingival sulcus is the depth of gingival sulcus. The depth of gingival sulcus in children varies from 0,25 to 1,50 mm. This depth reaches 2,0 ± 0,2 mm in primary and permanent teeth (Atanasov, Jemileva, 1980).

Gingival crevicular *fluid (GCF)* or *sulcular fluid* is a serumlike fluid that seeps from the connective tissue through the epithelial lining of the sulcus or pocket. GCF flow rate reflect changes in permeability of the tissue due to inflammation. It is part of the local defense mechanism and is able to transport many substances, including endotoxins, enzymes, antibodies, and certain systemically administered drugs. It is possible use as a diagnostic aid for periodontal disease activity because the composition changes in the presence of inflammation (Gupta, 2013; Lange, Schreder, 1974; Frank, Cimasoni, 1972; Kaslick et al., 1973).

Numerous mechanisms serve to maintain clinically healthy conditions in the gingiva. The protective processes in the gingival sulcus prevent damage to gingival epithelial layers. The regular shedding of epithelial cells and the positive flow of gingival crevicular fluid may remove unattached and epithelially attached plaques, as well as toxic products of bacterial metabolism and host response.

Cells of the junctional epithelium continually dissolve and reestablish their hemidesmosomal attachments leading to the migration of defence cells and constituents of the complement system into the sulcus from adjacent venules of the dentogingival plexus. The leukocytes and the end products of the complement cascade cause osmotic lysis of the targeted microbes, leading to the release of cytotoxic products for epithelial cells. The migrated monocytes remove these metabolites from the sulcus, so a small number of microbes can be eliminated without causing damage to the epithelium. The IgG and IgA of the sulcular fluid may play a role in the opsonisation and removal of toxic bacterial products.

The gingival epithelium serves as a physicochemical barrier with its tight intercellular connection, fast regeneration and salivary cover. The gingival epithelium is also strongly associated with host defence processes of the underlying connective tissue and the adaptive defence system.

1.3. Age-related features of periodontal ligament in children

The periodontal ligament is the connective tissue that surrounds the root and connects it with the bone. It is continuous with the connective tissue of the gingiva and communicates with the marrow spaces through vascular channels in the bone.

T.F. Vinogradova (1968) claimed 7 base periods in periodontal development in children.

Primary teeth:

1) development till eruption: when the crown formation is complete, extending down to the cemento-enamel junction, beginning of root formation is seen in the form of a spicule. Next, the walls of the pulp chamber form straight lines. The root length is less than the crown height. In molars the formation of the radicular

bifurcation is seen like a calcified point or a semilunar shape. Next, the walls of the pulp chamber form an isosceles triangle. The apex ends in a funnel shape. At the commencement of periodontal ligament formation, the ligament space consists of unorganized connective tissue with short fiber bundles extending into it from the bone and cemental surfaces. Next, ligament mesenchymal cells begin to secrete collagen, which assembles as collagen bundles extending from the bone and cementum surfaces to establish continuity across the ligament space and thereby secure an attachment of the tooth to bone.

2) teeth eruption: by the time of first occlusal contact of the tooth with its antagonist, the principal fibers around the coronal third of the root, the horizontal fibers are almost completely developed. The oblique fibers in the middle third of the root are still being formed. As eruption continues, and definite occlusion is established, there is a progressive apical maturation of oblique fiber bundles.

3) growth and development of the periodontium in primary teeth.

Periodontal fibers of complete part of root pass into growth zone, where have close contact with root pulp. After complete development of root and apex the structure periodontal differentiation lasts for 1 year (N.M.Chuprynina, 1970). Periodontium in complete primary tooth is characterized by soft connective tissue, a huge number of cells and capillares. Its structure and periodontal ligament space aren't stabile.

Change bite

4) root resorption in primary teeth. Cementum and compact plate of alveolae are destoyed, periodontium transform into granulations;

5) intramaxillar development of permanent teeth;

6) eruption of permanent teeth.

Permanent bite:

7) growth and development of the periodontium in permanent teeth.

There were 5 stages of root and periodontium development after eruption characterized (M.F.Danilevskyi, G.M.Vyshnyak, A.M.Politun, 1981).

1. Stage of root length growth and marginal periodontium development (fig. 1).
2. Stage of immature root apex and marginal periodontium development (fig. 2).

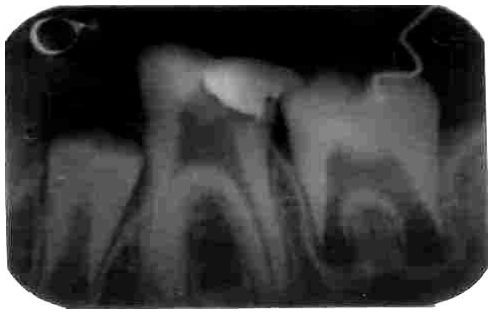

Fig. 1. Intraoral X-ray of 8 years old child. Roots of the 46 are complete in 2/3 length

Fig. 2. Intraoral X-ray of 9 years old child. Roots of the 46 are complete. Apeces are developing.

3. Stage of unclosed root apex (fig. 3).

4. Stage of closed root apex and immature apical periodontium. The apical end of the root is completely closed and the periodontal membrane has a uniform width around the tooth apex.

5. Stage of mature apical periodontium.

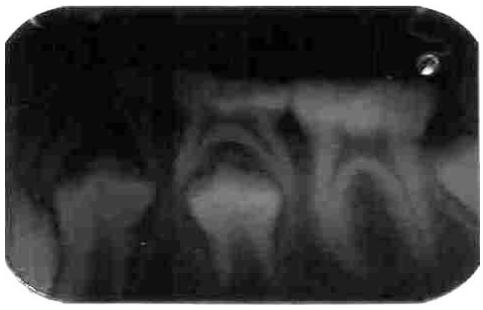

Fig. 3. Intraoral X-ray of 10 years old child. Roots of the 46 are complete. The walls of the root canal are parallel and the apical end is still partially open.

Periodontal ligament space on X-ray in complete child's tooth is wider than in adults (0,25 – 0,35 mm), mostly about tooth cervix and apex.

Principal fibres of the periodontal ligament: they are classified into several

groups on the basis of their anatomical location. Oblique fibres are the largest group among the periodontal ligaments; they extend from the cementum in a coronal direction obliquely to the bone. They bear the brunt of vertical masticatory forces and transform them into tension on the alveolar bone. Apical fibers open a sheaf. A row of fibrous bunches goes around a tooth and connect different fibers into total bunch named circular tooth ligament. It covers tooth neck as a ring, is a bottom of physiological pocket, fix teeth and prevent microflora appearance in periodontium. Crossing of periodontal fibers prevent tooth movement around its axis.

Collagen periodontal fibers in children are less mineralizated and tough, have a small number in one area in comparison to adults.

1.4. Age-related features of cementum in children

Cementum is calcified mesenchymal tissue that forms the outer covering of the anatomic root. There are four main types of root cementum: acellular, afibrillar cementum (coronal cementum), acellular, extrinsic fiber cementum (cervical two-thirds of the root), cellular, intrinsic fiber cementum (lacunas of the root at sites of cementum repair), cellular, mixed fiber cementum (apical third of the root and in furcations).

During the *primary dentition*, the cellular cement is underdeveloped and is found in the area of the root apeces in milk teeth. By the time of root resorption, the layer of cellular cement gradually increases (V.G. Vasilyev, 1973). Cement of primary teeth has a lower density compared to permanent teeth due to the fact that it contains more organic matter and has a lower degree of mineralization (P. Baer, S. Benjamin, 1974).

During the *change dentition,* the number of cementocytes increases, which is associated with root resorption.

During the *permanent dentition*, most (2/3) of the complete roots are covered with acellular cement, while the apical third of the roots – with cellular one. Thickness of cell cement slowly increases with age.

Breakthrough cement fibers connect with the periodontal fibers and penetrate

the alveolar process, providing the tooth fixation.

1.5. Structure of alveolar process in children

The *alveolar process* is defined as that part of the maxilla and the mandible that forms and supports the sockets of the teeth. It forms with the development and the eruption of teeth, and conversely, it gradually diminishes in height after the loss of teeth. The alveolar bone can be divided on the alveolar bone proper and the supporting alveolar bone.

The alveolar bone proper consists of lamellated and bundle bone. It surrounds the root of the tooth and gives attachment to principal fibers of the periodontal ligament. The lamellar bone contains osteons each of which has a blood vessel in a haversian canal. Blood vessel is surrounded by concentric lamellae to form osteon. Bundle bone is that bone in which the principal fibers of the periodontal ligament are anchored. The bundles of the principal fibers continue into the bone as Sharpey's fibers. The supporting alveolar bone consists of two parts: cortical plate and spongy bone.

At the age of 6-7 months, when the first tooth appears, the alveolar process begins to grow in height. Its active development continues with the primary bite formation: the alveolar process becomes higher, the interalveolar and interroot septae are formed, the walls of the dental alveoli thicken. The alveolar bone is formed by ossification of the protein matrix with the participation of mineral salts of calcium, phosphorus, trace elements. The alveolar process in children has a lower degree of mineralization than in adults. In children and adolescents, the spongy substance is filled with a red bone marrow with active hematopoietic function. The yellow bone marrow appears at an older age. Feature of the alveolar process in children is the presence of wide canals of osteons, thin and delicate, rarely located bone septae. The porous alveolar process contains the follicles of primary and permanent teeth.

The alveolar process is covered with periosteum, which consists of three layers in children: the adventitious, fibro-elastic and osteoblastic. The bone grows in opposition, mainly due to ossification. In adults, the osteoblastic layer changes to

fibrous, which is cell-poor and therefore has no osteogenic properties.

X-ray picture of the alveolar process in children and adolescents has certain features regarding the height of the interveal septa, the shape of their tops, the structure of spongy and compact substances (N.M. Chuprynina, 1964; I.O. Novyk, G.M.Vysgnyak, R.I. Smolyanova, 1957; N.A. Rabuchina, 1976). After complete formation of primary occlusion, the tops of the interalveolar septae are located just below the level of the enamel-cement junction. The top of the septum of the lower medial incisors is often forked. The permanent division of the septum top is observed in the upper medial incisors. In the area of the lateral front teeth, the shape of the interalveolar septa up to 4 years of age is more often rounded, then it changes to a spat. The tops of the interveal septae between the posterior teeth have a flattened trapezoidal shape. Bone pattern is a large-mottled, undifferentiated. The periodontal ligament spaces of all teeth in children and adolescents are almost twice as wide as in adults, and the cortical plates are wider but less intense (Fig.4).

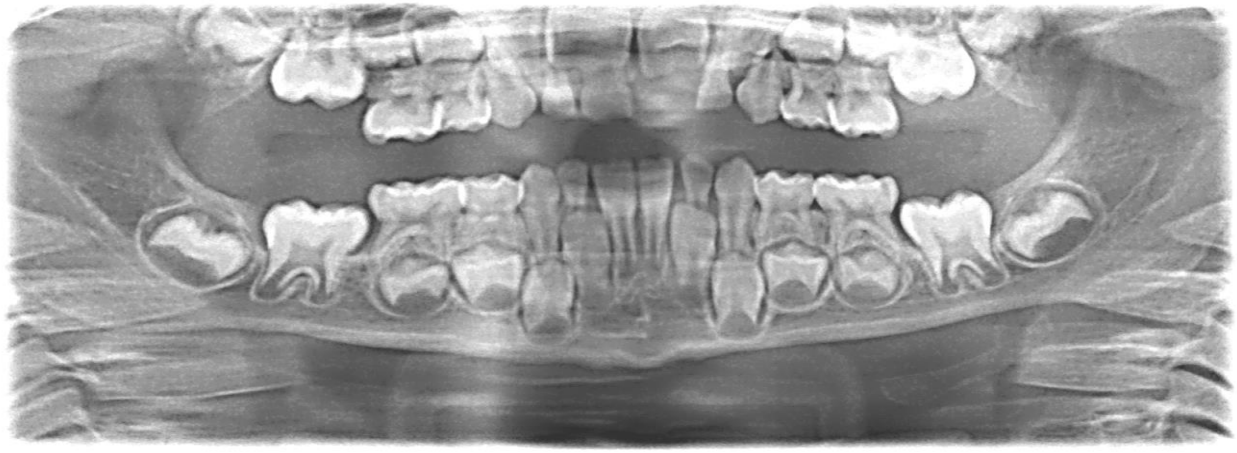

Fig. 4. Panoramic X-ray at 5 years of age

During resorption of the primary teeth roots, the interalveolar septae lose their mineral base and dissolve completely.

During the period of *mixed bite*, at the same time as the resorption of the primary teeth roots, the walls of the dental alveoli, the interalveolar and inter-root septae are also resorbed. During the eruption of permanent teeth, new dental alveoli are formed.

The formation of the interveolar and inter-root septae begins from their tops and gradually proceeds towards the jaw body. The creation of dental alveoli finishes with the complete root and periodontium formation (I.O. Novik, G.M. Vishnyak, R.I. Smolyanova, 1957; N.M. Chuprinina, 1969).

During the *eruption of permanent teeth,* the alveolar margin undergoes significant changes. Initially, the tips of the interveal septae are immature, have cut contours towards the erupting tooth, and are located above the enamel-cement junction. The periodontal ligament space near the neck of such tooth is widened (Fig. 5).

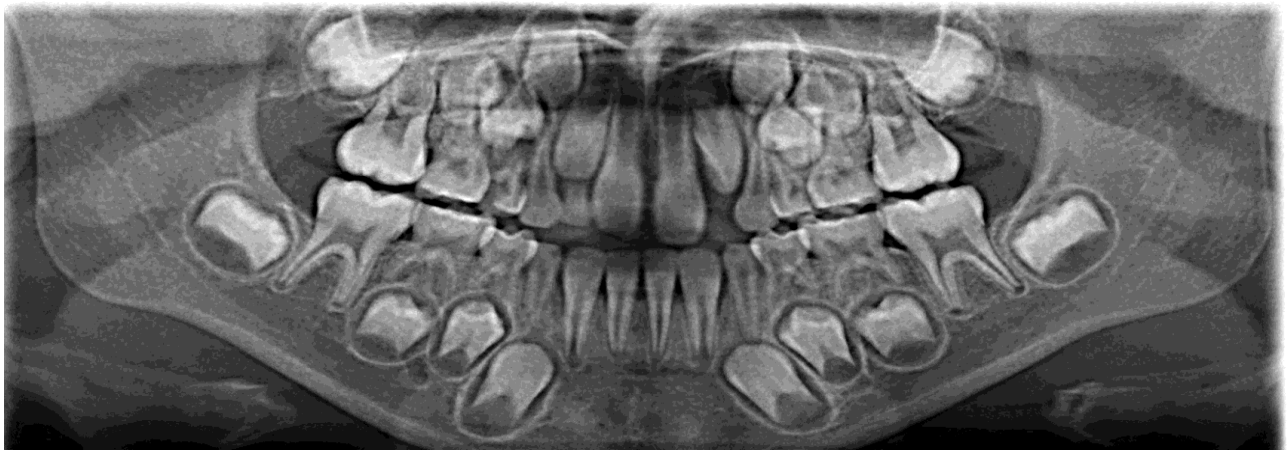

Fig. 5. Panoramic X-ray at 7 years of age (mix dentition)

With the end of the teething, the tops of the interveal septae acquire their outline and have a certain relation to the enamel-cement junction. In the area of the front teeth at the age of 7 - 8 years, the tops of the interalveolar septae are located in the area of the enamel-cement junction , at 9 - 12 years - close to it, and at 13 - 15 years – slight ly below it. Interalveolar septae in the area of chewing teeth are located more often at the level of enamel-cement junction. In the area of the lower frontal teeth, the tips of the interveal septae are more often cone-shaped, and occasionally dome-shaped. Sometimes a semi-lunar septa is found between the lower central incisors. The interalveolar septae located between the premolars and the molars resemble a truncated cone or trapezoid with a rounded or flattened tip (Figs. 6, 7, 8).

Regardless of the shape of the crest, the interdental septae are flanked by a

continuous compact lamina, which is an important indicator of their normal state.

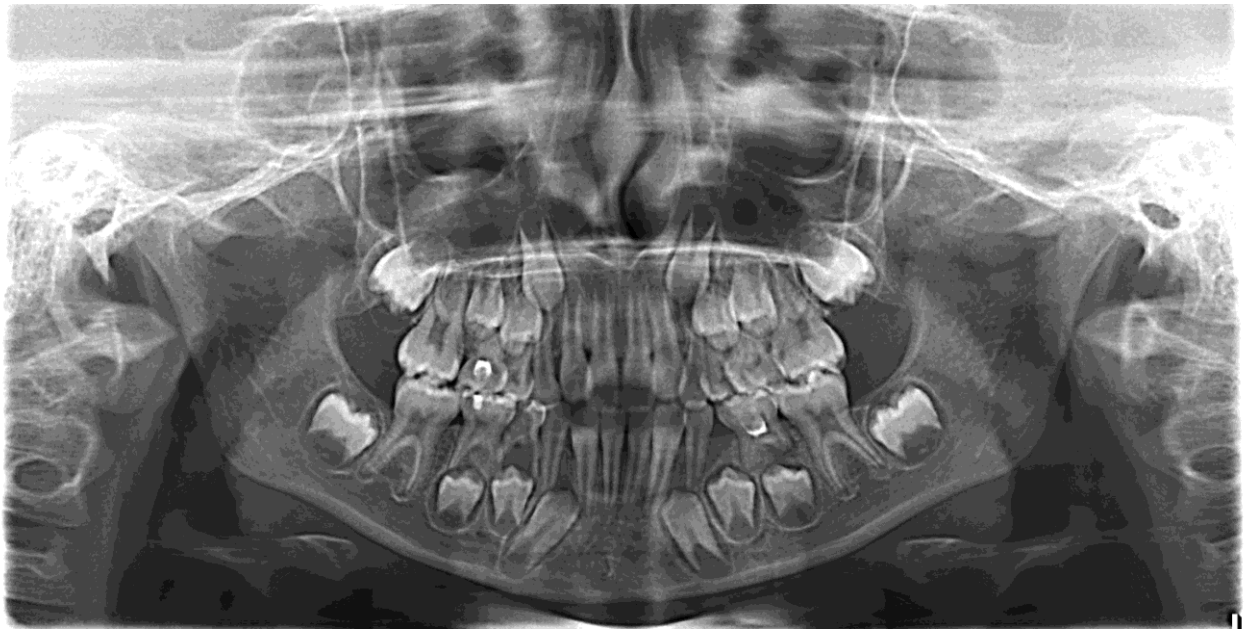

Fig. 6. Panoramic X-ray picture at 8 years of age (mix dentition)

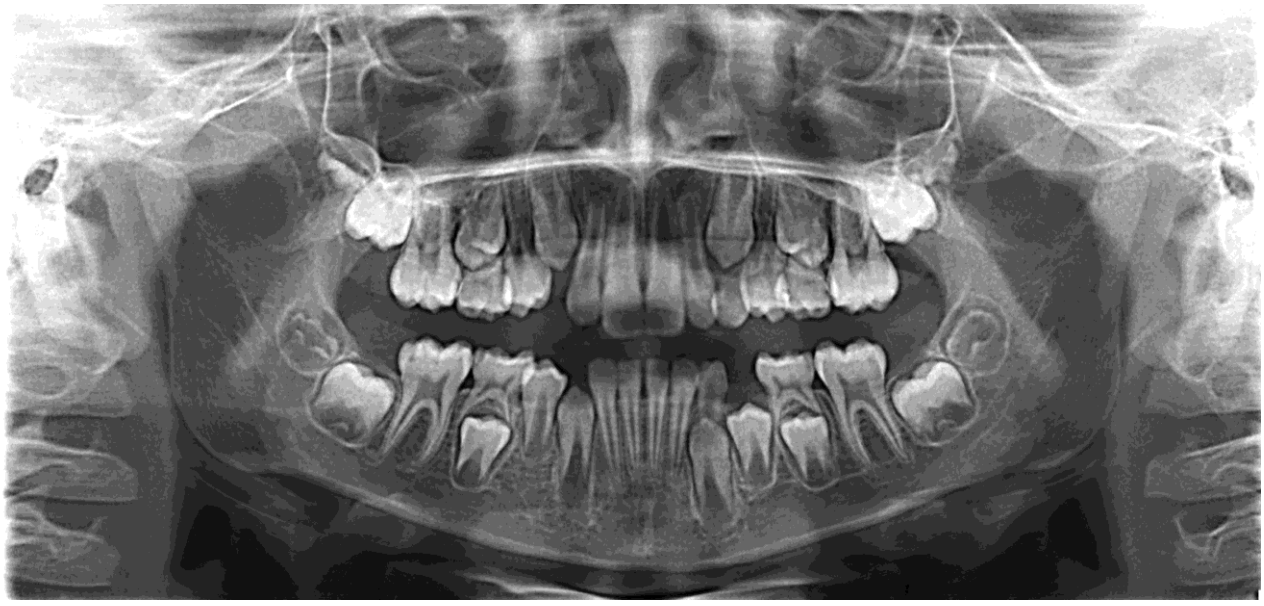

Fig. 7. Panoramic X-ray picture at 9 years of age (mix dentition)

In the period of permanent occlusion, the formed bone tissue has a pronounced spongy substance and solid plates. In children and adolescents, the spongy substance has a different trabeculae. On the tips of the septae, the trabeculae are smaller, in other areas - coarse. In the mandible anterior trabeculae are thicker, coarser and fewer than that in the maxilla with larger marrow spaces and are oriented more horizontally.

Posteriorly, the periradicular trabeculae and marrow spaces are larger than that in the anterior region.

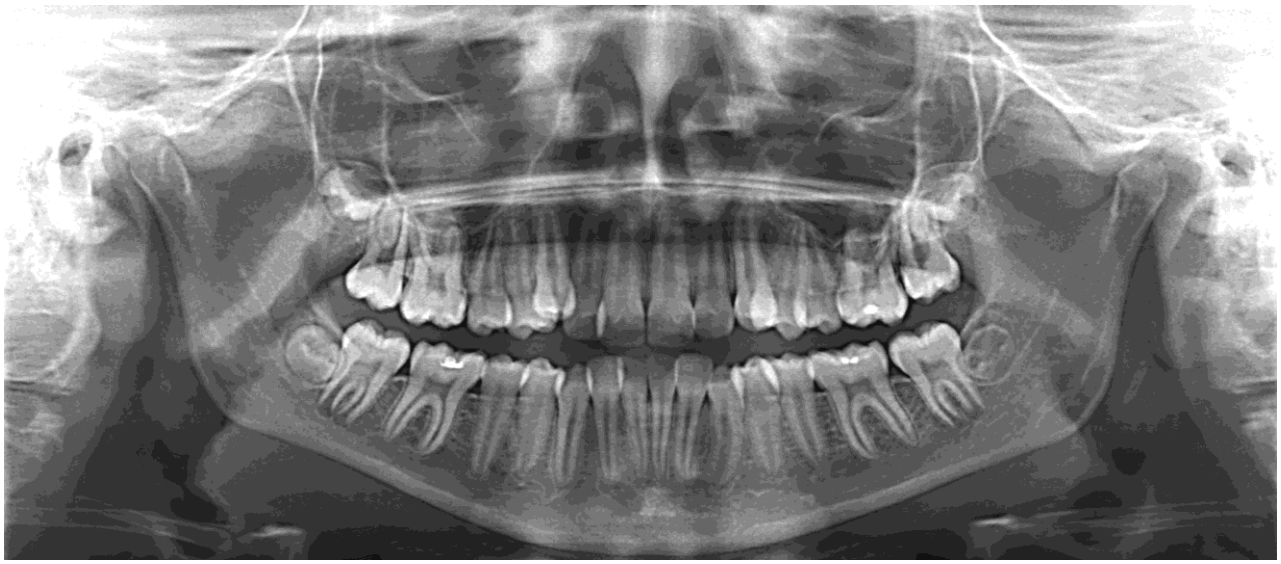

Fig. 8. Panoramic X-ray picture at 13 years of age (permanent dentition)

Cancellous bone trabeculae that form a lattice like network of intercommunicating spaces filled with bone marrow (fig. 9).

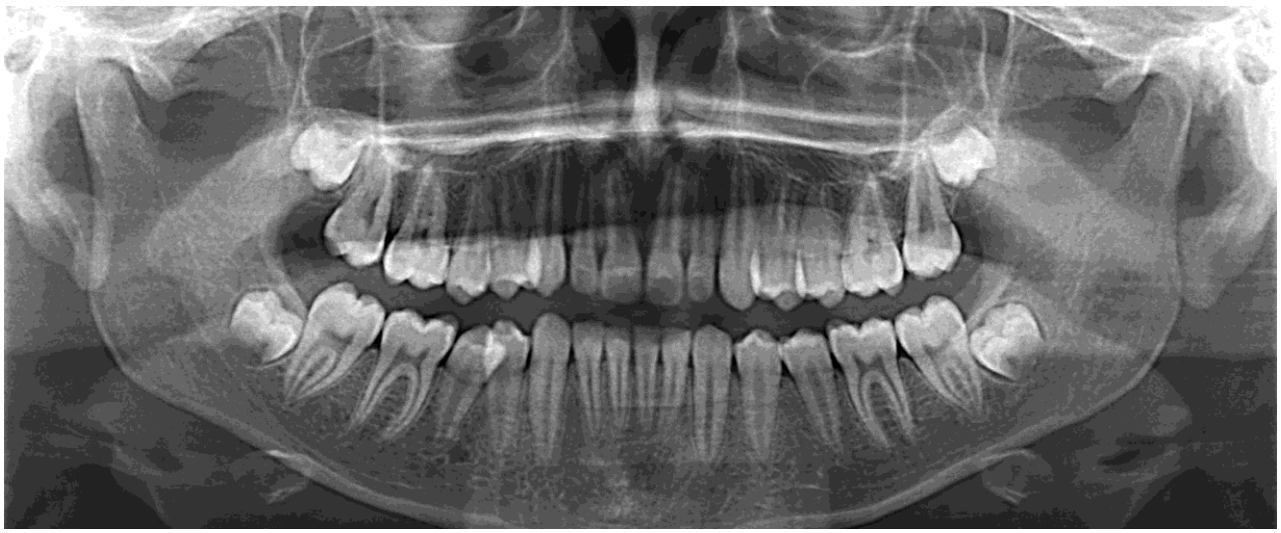

Fig. 9. Panoramic X-ray picture at 14 years of age (permanent dentition)

1.6. Blood supply of periodontal tissues

Blood supply of periodontal tissues is provided through external carotid artery. The teeth, periodontium, gums, alveolar process of the jaws are supplied by the blood

through the maxillary artery, which gives next branches: the lower alveolar artery, the posterior upper alveolar and the inferior alveolar, from which the anterior upper alveolar arteries and dental branches depart.

In contrast to adults, children and adolescents, due to structural and functional restructurisation of the periodontal tissues, exhibit instability and dynamics of age-related indeces of the blood circulation.

The degree of vascularization of some periodontal areas varies increasing from incisors to molars. The marginal part of the periodontium (gingival papillae, marginal periodontium, tips of the interveolar septae) and the apical area of teeth have intensive blood supply, the middle part of teeth - moderate supply, the medial and distal sides of the teeth have a strong blood supply (Born, 1966).

Periodontal tissues have all the structural units of the microcirculatory network: arterioles, precapillaries, capillaries, postcapillaries, venules (V.M. Uvarov, 1934; I.O. Novik, 1964, etc.). Periodontal tissues are characterized by the presence of a large number of collaterals at the expense of vascular anastomoses of the pulp micro-vessels, periodontium, bone, and gums. One of the mechanisms of pressure regulation during chewing is associated with the presence of these anastomoses. The gingival blood vessels are located almost below the basal membrane in children. With age, after the final formation of the permanent occlusion, the basement membrane is separated by a relatively thick layer of collagen fibers.

Blood circulation in periodontal tissues is regulated by neuro- and myogenic mechanisms (A.A. Prokhonchukov, N.K. Loginova, N.A. Zhizhina, 1980).

1.7. Lymph supply of periodontal tissues

Periodontal tissues have many lymphatic vessels. Lacunar extensions of the lymphatic vessels depart from the plexus in the form of glomeruli which are located under the plexus of the capillaries.

The lymphatic vessels of the gums form two plexuses: superficial and deep. The superficial plexus consists of thin vessels that connect to the deep plexus and form a mesh in the gums that surrounds the blood vessels.

Lymphatic vessels of periodontal tissues form a lymphatic network that contains lymphatic vessels of the pulp, alveolar process and gums. The lymph through the drainage vessels of the pulp and periodontium passes into the alveolar process and the jaw and flows into the regional lymph nodes.

1.8. Periodontal innervation

Periodontal innervation is provided by branches of trigeminal nerve: maxillar and mandibular.

In the thickness of the alveolar bone, the course of the nerve fibers corresponds to the vascular network.

In periodontal tissues there is a dense network of nerve fibers and a large number of receptors. The dense innervation network intergrates the tissues: nerve fibers from the alveolar bone appear into tooth pulp, cement, periodontium, and in the area of the alveolar crest they pass from the periodontium to the gingival margin and gingival papillae (MF Danilevsky, GM Vishnyak, A. M. Politun, 1981).

Chapter 2
ETIOLOGY AND PATHOGENESIS OF PERIODONTAL DISEASES

Complicating and risk factors for disease development may be etiologic, predisposing, or contributing. They are delineated as follows:

~ *Etiologic factor:* the actual cause of a disease or condition.

~ *Predisposing factor:* renders a person susceptible to a disease or condition.

~ *Contributing factor:* lends assistance to, supplements, or adds to a condition or disease.

~ *Risk factor. increases* the probability that disease will occur.

~ Etiologic, predisposing, and contributing factors may be local or systemic. defined as follows:

• *Local factor:* a factor in the immediate environment of the oral cavity or specifically in the environment of the teeth and periodontium.

• *Systemic* factor: a factor that results from or is influenced by a general physical or mental disease or condition.

2.1. Local etiologic factors for periodontal diseases development

1. Dental debris, plaque, calculus.
2. Reduced saliva production.
3. Traumatic factors: carious lesions, improper dental procedures.
4. Abnormal habits: unilateral mastication, abnormal biting habits, clenching and bruxism, mouth breathing, and abnormal sucking habit.
5. Abnormalities of structure and attachment of soft tissues in oral cavity (shallow vestible, expressed of mucous membrane bands, highly attached overdeveloped frenula of lip or tongue).
6. Factors of occlusal function.

1) Over function: a) Excessive stress on teeth; b) Insufficient periodontal

support; c) Powerful masticatory musculature;

2) Under function: a) Premature wear; b) Non-occlusion; c) Indolent mastication.

Dental biofilm consists of a complex mixture of microorganisms in microcolonies. The microbial density is very high, and it increases as biofilm ages and matures. The potential for the development of gingivitis increases with more microorganisms especially as the numbers of pathogenic outumber non·pathogenic microorganisms. The pellicle, biofilm architecture, and resulting environment promote anaerobic gramnegative bacterial growth activity.

In healthy gums biofilm consisits of *Str. mitior, Str. sanguis*, *A. naeslundii, A. viscosus, Corinebacteriae, Neisseriae, Veillonell*a. Biofilm near the gingival margin thickens as a more mature form develops.

Microorganisms in the form of biofilm are the primary etiologic agents of periodontal disease/infection:

- the type of organisms shift to gramnegative anaerobic species including Porphromonas *gingivalis* and *Tannerella forsthia* (previously known as Bac*teroides forsthus)* with the initiation of periodontal disease;
- Aggregatibacter Actinomycetemcomitans (Previously *Actinobacillus* Actinomycetemcomitans) has been associated with aggressive forms of periodontal disease;
- motile rods and spirochetes are prominent in diseased pockets versus healthy subgingival sulci.

Some microorganisms also play role in pathogenesis of periodontal disease/infection: *C.rectus, E.nodatum, F. nucleatum, P. intermedia/nigrescens, P.micros, S.intermedium T.denticola* (N.O.Savychuk, 2008).

Bacteria release of proteolytic enzymes and harmful metabolic products which damage the tissues (connective tissue fibres, fibrin, fibronectin), the components of the immune system (immunoglobulins, complement system), and structural proteins of soft tissues. Endotoxins of Gram-negative bacteria disturb blood clotting, damages the bone and complement system. Bacterial toxins can force cells to produce

cytokines directly. However, their endotoxins, and cell wall antigens (gingipain, fimbrillin, or stress proteins) can initiate pronounced immune reactions.

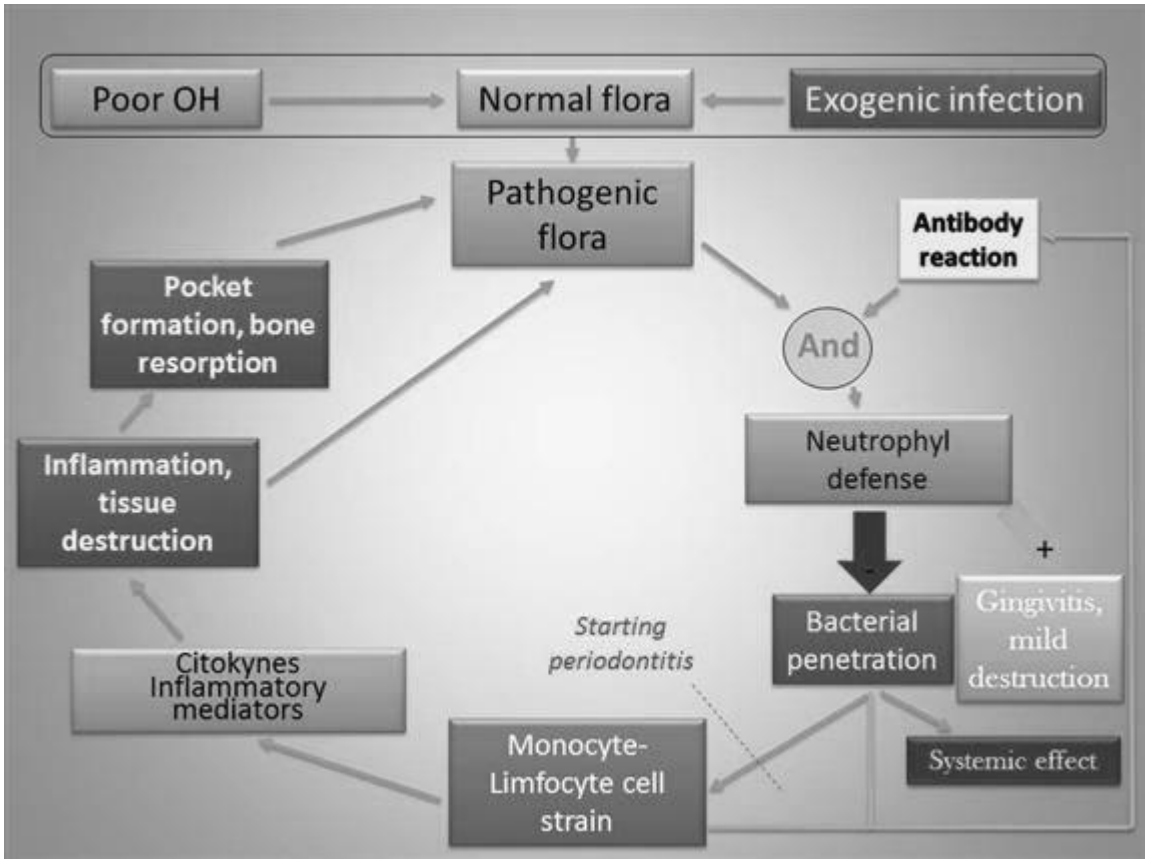

Fig. 10. Pathogenesis of periodontitis (modified from Offenbacher 1996)

Proteolytic enzymes released by PMN cells cause tissue destruction. This is followed by epithelial in-growth and granulation tissue forming, which is rich in plasma cells at the destructed area. Plasma cells continuously release inflammatory cytokines, which leads to further destruction. Enzyme release of PMN cells can be inhibited by alpha-2-macroglobuline and alpha-1-antitripsine, but a bacterial antigen of Porphyromonas gingivalis called gingipain can even destroy enzyme inhibitor proteins.

Cytokines are small molecule weight glycoproteins. Their role is information flow among cells, immune response, cell differentiation, and regulation of growth. A mediator released by a specific cell can affect neighbouring cells (paracrine) or even itself (autocrine). Cytokines can be either proinflammatory, anabolic cytokines or growth factors. The same cell is capable to produce both, depending on the triggering effect. Specific receptors are responsible for their effect.

Inflammatory cytokines express adhesion molecules, which help PMN cells migrate towards the inflammation, and fixate macrophages and lymphocytes in the connective tissue matrix. Their role is to initiate the immune response. They also play a major role in connective tissue and bone catabolism. Bone resorption is activated directly and indirectly: PGE2 production, macrophage transformation into osteoclasts, stimulating collagenase MMP enzyme production. They also inhibit new bone formation. Further cellular effects are the stimulation of T-cell and macrophage response, increasing the number of circulating PMN cells, promoting their chemotaxis, and stimulating MMP production.

Anabolic (anti-inflammatory) cytokines basically unite innate and adaptive immune subsystems by modulating T-helper cells. They reduce macrophage activity, and their production of cytokines. Anabolic cytokines also block phagocytosis and intracellular destructive processes, and antigen presentation. Through T-helper cells, they can inhibit inflammatory cytokine production, cytotoxic T-cells, and chemotactic function of the rest of T-helper cells. However, they promote activated B-cell growth and differentiation.

Growth factors play a role in the healing processes, attract differentiating cells (fibroblasts, mesenchymal cells, osteoprogenitor cells), promote differentiation, increase collagen, glucose-amino-glycane and other growth factors production and secretion. If these reparative procedures overweigh destructive procedures, regeneration (tissues as original) or reparation (tissues replacing original) takes place.

Products of the arachidonic acid cascade are a result of insults damaging the membrane, phospholipids of the membrane are transformed into arachidonic acid by phospholiase-A2 enzyme. This metabolises further into leukotrienes (lipoxigenase), prostaglandines and tromboxanes (cyclooxigenase/COX). Enzyme COX-1, is responsible for protective measures (protection of mucosa, blood clotting). The other COX enzyme (COX-2) is responsible for pain sensation by releasing prostaglandins, and for fever, by affecting the heat control centre of the hypothalamus.

Prostaglandins induce oedema by vasodilatation, potentiate the effects of cytokines, stimulate MMP production, cause bone resorption in a direct and indirect

way, and reduce the production of antibodies. Patientsh a hyper-reactive monocyte genotype are prone to extensive prostaglandin release, even under low bacterial insult.

As biofilm matures and thickens, more gram-negative anaerobic organisms appear, which are protected by the biofilm architecture and environment. Such viruses as Herpes simplex virus, Epchtain-Barr virus can closely aligned with other periodontopathogenic bacteria (N.O.Savychuk, 2008).

Dental calculus isn't met in young patients, but food debris as unstructured, loosely attached nonminerallzed particulate matter can be observed about tooth neck ar unsufficient hygiene.

Stages in the formation of dental deposites correspond periodontal changes (S.B.Ulytovsky, 1999).

1 stage – dental biofilm; healthy periodontium;

2 stage – soft plaque; periodontal status depends on the amount and the localization of plaque: either it has no effect on the periodontal tissues, or gingivitis can be revealed;

3 stage – hard dental plaque which provokes periodontal changes from inflammation, atrophy to sclerosis with distruction (resorption of interdental and interroot septae);

4 stage – soft plaque with a small amount of supragingival calculus, without destruction of dentogingival sulcus. Ginigivitis is characterized by gingival inflammation, hyperemia and bleeding of the 1st degree (minor destruction of apeces of interdental septae);

5 stage – a huge amount of supragingival calculus without the dentogingival sulcus destruction. Ginigivitis is characterized by expressed gingival inflammation, hyperemia and cyanosis. Gum bleeding of the 1-2^{d} degree without destruction of the dentogingival sulcus. Progressive resorption of apeces of interdental septae;

6 stage – a huge amount of supragingival calculus with the dentogingival sulcus destruction. Clinical manifestations are same with previous stage. In addition, periodontal pockets are revealed;

7 stage – initial stage of subgingival calculus formation.

8 stage – intermediate stage of subgingival calculus formation.

Periodontal pockets are found (4 – 7 mm), resorption of interdental septae. Bome pocket can be found. Dental calculus covers a half of the root length;

9 stage – stage of significant subgingival calculus formation.

Periodontal pockets are found more 7 mm, resorption of interdental septae. Bome pocket can be found. Dental calculus covers more than a half of the root.

The increase in the amount of dental plaque results in gingivitis. Gingivitis without any treatment will be followed by periodontal disease. In most of the cases the regular and efficient dental plaque removal prevents or heals the gingivitis.

In childhood the inflammatory processes of periodontal tissues occur as a result of chronic trauma. The causes of trauma are sharp edges of the carious cavities, improper fillings, crowns and orthodontical appliances.

All adaptive and pathological changes in the periodontium caused by overload of the masticatory function are called occlusal trauma. Unfavourable forces can appear after the adjustment of occlusion, early tooth contact and parafunction. Early contact can be the result of tooth loss, periodontitis, changes in dentition (elongation, tooth malposition or mobility) or poorly designed restorations. In case of a primary occlusal trauma, the tooth is periodontally healthy, but is subject to increased loading forces. In secondary occlusal trauma, the force might even be normal, but affected teeth are periodontally compromised.

Malpositioned teeth, craniofacial and dentofacial malformations are also provoke periodontal pathology due to impaired periodontal function, overloading of some areas and functional underloading of others ones.

The development of malocclusion pathology is often caused by bad habits, pathology of the nasopharynx (A.I.Betelman et al., 1978). These factors exxagerate the deleterious effect of malocclusion on the periodontium; the dryness of the mucous membrane (F.A. Lazutka, 1960) lead to a decrease in local immunity in periodontal tissues.

Such factors of occlusal underfunction as premature wear, non-occlusion, and

indolent mastication facilitate plaque accumulation and periodontal diseases development. This is due to a change in the physicochemical composition of the food, which has become less coarse, thermally and mechanically processed. In the last 100 years, the consumption of refined sugars that contribute to plaque formation has increased almost 25 times.

Insufficient occlusal function reduces the resistance of periodontal tissues to external stimuli, promotes the intensive formation of plaque, which leads to the development of gingivitis. dystrophic disorders of the periodontal collagen fibers and underlying bone atrophy development (Glikman, 1967).

Other factors for periodontal diseases development are shallow vestibule, expressed of mucous membrane bands, highly attached overdeveloped frenula of lip or tongue.

The vestibule of oral cavity is shallow if the distance from the marginal ginigiva to the horizontal level of the transitional fold does not exceed 5 mm, the normal - 5 - 10 mm and deep - more than 10 mm. The frenula should be gentle, flexible, movable the lips and tongue movement. Frenula can also hamper adequate oral hygiene, and its spreading into the papilla may cause a pulling effect. Pathological influence of an abnormal frenulae of the lip or shallow vestibule on the gums is detected by horizontal movement of the lip or cheeks in the area of the upper or lower jaws. Massive, dense frenulae, their high attachment, a shallow vestibule oris are the conditions for chronic functional trauma of periodontal tissues. Pronounced frenule can prohibit proper oral hygiene, highly attached frenule can damage the marginal gingiva.

The edentulous ridge suffers constant involution following teeth loss. If the tooth was affected by periodontal disease, cyst, surgical trauma or developmental disorder, its loss leads to various tissue deficiencies.

2.2. Systemic etiologic factors for periodontal diseases development

1. Faulty nutrition

2. Debilitating disease
3. Blood dyscrasias
4. Endocrine dysfunctions
5. Radiation
6. Psychogenic factors
7. Iatrogenic factors

The progression of periodontal disease is influenced by variety of factors like microorganisms, host response, systemic background, and genetic makeup of the host. Periodontitis is accompanied by endicryne dysfunctions (E.V.Borovsky et al., 1973; G.V.Vyshnyak, 1974). Hyperglycemia induces decreased chemotaxis, phagocytosis, and intracellular bacterial activity in diabetics. Defects in neutrophil function have been considered as a potential cause of bacterial infection in diabetic individuals. Ray and Orban (1950) observed that the basic structural changes in the diabetic periodontium are the degeneration of tissues and the presence of calcified bodies in and around small blood vessels of the gingiva.

The progression of periodontal disease in a hypothyroid state is more related to the negative effect of hypothyroidism on the immune system, leading to a less proficient immunogenic response to the infection.

The data Mascarenhas et al. (2003) strongly indicate that with the influence of sex hormones, children in puberty experience an exaggerated gingival inflammatory response to plaque. The impaired evolution and function of the oral epithelium due to hormone dysfunction plays a role in the pathogenesis of gingivitis. During puberty adolescents may have juvenile periodontitis and juvenile osteoporosis. Changes in the periodontal tissues are often found in girls with menstrual dysfunction due to insufficient estrogen production and bone marrow formation disorders.

In childhood, the relationship between the modeling and mineralization of the alveolar bone with the function of the hypothalamic-pituitary system is particularly pronounced. Disorders of the functional state of the system "hypothalamus - pituitary gland - glands" lead to deterioration of the mineralization into alveolar bone (N.O. Savychuk, 2008).

Many authors claimed that gingivitis mostly accompanied with diseases of digestion system. Pathological changes in periodontal tissues have been found in children with chronic gastritis, liver and gall bladder diseases, with bowel lesions (Kaskova L.F. et al., 2001; G.V. Banchenko, 1979). Helicobacter pylori can persist in periodontal pockets maintaining underlying diseases, e.g. duodenal abscess. Periodonto-pathogenic bacteria may be the aetiological factor of autoimmune inflammatory intestinal diseases (P. gingivalis).

The chronic obstructive pulmonary diseases are risk factors for periodontal pathology. Plaque accumulation is clearly an essential initial etiological factor in periodontitis, although the exact mechanisms for the relationship between oral hygiene hypotheses suggest that bacteria in oral cavity may be aspirated along with respiratory pathogens and affect adhesion of the later organisms to the respiratory epithelium, which subsequently cause lung disease (Parashar S. et al., 2018). Otherwise, periodontal disease may alter environmental conditions to permit mucosal colonization and infection by respiratory pathogens (Vadiraj et al., 2013).

Deficiency of periodontal tissues can lead to deficiency in the body of a number of vitamins: ascorbic acid, thiamine, retinol, tocopherol. Ascorbic acid deficiency disrupts collagen synthesis and metabolism of mucopolysaccharides, inhibits bone formation, reduces periodontal tissue resistance to infection. Severe ascorbic acid deficiency may be an important but not a trigger factor in the development of periodontal disease. Retinol and tocopherol deficiency affect the process of gum epithelization and thus cause inflammation. It is proved: the higher the degree of inflammation in the gums, the lower the retinol content in the serum.

The basis of the neurogenic theory of the development of periodontitis is the trophic disorders of the central and peripheral parts of the nervous system. This is confirmed by the high prevalence and severe extent of periodontal lesions in children with oligophrenia, Down disease, and other organic diseases of the nervous system. There is a close pathogenetic link between disorders of the autonomic nervous system and the development of inflammatory diseases of periodontal tissues. During adolescence, the phenomena of sharp increase of sympathetic influences are observed

against the background of formation of vagal regulation, accompanied by inflammatory and destructive phenomena in periodontal tissues. Such changes in the functional state of the organism lead to the development of local tissue dystrophy (T.N. Modyna, E.V. Mamaeva, 2006).

Current approaches to the etiology and pathogenesis of periodontal tissue diseases include the importance of such risk factors as cancer, AIDS, herpes infections, autoimmune diseases (Crohn's disease, rheumatoid arthritis), tuberculosis, syphilis, and others. They also point to age (increased likelihood with age), gender (increased likelihood in women), racial, national and other (unsatisfactory social conditions, dental anomalies, oral breathing) risk factors.

Thus, the relationship between the formation and progression of chronic somatic diseases and periodontal tissue diseases as multifactorial is realized by pathogenetically significant changes in metabolism and hemodynamics, immunological, neuroregulatory and dysbiotic disorders (N.O. Savichuk, 2008).

Chapter 3
PERIODONTAL EXAMINATION OF CHILDREN

3.1. Clinical methods of periodontal examination and revealing risk factors

Age-related changes in children, anatomic-histological and physiological features of the dento-maxillar apparatus in children and adolescents determine the peculiarity of the methodical methods of their examination in the periods of primary and permanent teeth.

Visual examination is performed to examine the condition of periodontal tissues and local etiologic factors of periodontal diseases. The examination starts from the vestibule depth measuring. Using a graduated probe, the distance from the the marginal gingiva to the horizontal level of the transitional fold is determined in millimeters. The depth up to 5 mm are considered shallow, up to 10 mm as medium, and more than 10 mm - deep.

Lip frenulum is a tiny fold consisting mucosal membranes, fibrous tissues, and muscle fibres that attach the inner lips or cheek to the alveolaris process, gingiva, and periosteum. Superior labialis frenulum is the residual embryological structure that connects upper labial tubercles to the palatine papilla and shape of triangle. Normal frenulums are attached apically on the free margin gingiva and ends on the mucogingival junction. Abnormal frenulum attachment can be detected visually by pulling the upper lip to observe the movement of papilla edge or by performing blanch test, in which upper lips are lifted and held until the area becomes ischaemic and turns pale. Attachment of labialis frenulum might induce pathologic issues and create such complications as gingivitis, gingiva recession and teeth diastema. High superior labialis frenulum attachment may create an upward pull of the healthy gingiva and prevent dental cleansing, and thus making it prone for plaque accumulation and gingivitis, and this will develop into sulcus and pocket, and eventually advanced periodontal tissues will develop. In addition, this will also lead

to local gingival recess, extreme separation or gaps in central incisive teeth and will affect patient's psychological condition. Abnormal frenulum attachment can also disturb hinder teeth movement in orthodontic care to manage central diastema and relapse after orthodontic management; furthermore, it will influence tissue healing after periodontal care.

Attachment and shape of tongue (long, narrow, short, wide) are examined. The attachment of the tongue frenulae should normally be approximately 1 cm posterior to the tongue's tip. The frenum's attachment to the inferior alveolar ridge should be proximal to or into the genioglossus muscle on the floor of the mouth. Ankyloglossia (more commonly called "tongue-tie") is a congenital anomaly characterized by an abnormally short lingual frenum, which may restrict tongue tip mobility.

During the dental examination analyse:

- the condition of the teeth (carious teeth, fillings) with the probing of their five surfaces. If necessary, conduct percussion. The tapping of a healthy tooth is painless and accompanied by a loud clear sound. Changes in the periodontium, bone resorption, change in the volume and tone of the sound, make it subdued;

- position of each tooth in the dental arch with anomalies recording (crowding, rotation around the axis, position out of the arch, etc.);

- the form of dental arches, diastema and trematae.

The hygienic condition of the oral cavity is examined using hygienic indices. The calculus is diagnosed by probing.

Gingival examination

Health gingiva in primary dentition is pink, thick, rounded, less fibrous than adult gingiva; not astighdy adapted to the teeth, may or may not have stippling. Width of the attached gingiva in children is typically between 4 and 5 mm. The healthy gingiva can be red in the case of inflammations, or pale in the case of anemia. Epithelial desquamation - the scaling of the superficial epithelium - can also be present, with patches of exposed connective tissue that bleed easily (a typical sign of desquamative gingivitis, pemphigus). The bleeding gingiva is one of the most common problems in patients with acute or chronic inflammations of the gingiva;

severe bleeding can occur at gingival inflammation associated with serious general diseases. Pain is another serious symptom associated with acute inflammations (ulcerative gingivitis). In the case of chronic inflammations, pain is usually induced (during teeth cleaning, eating, etc.). The condition of periodontal pockets must be examined thoroughly in order to distinguish true and false pockets (gingival hyperplasia may hide serious general diseases such as hemoblastosis or tumours).

In children with a healthy gingival and periodontal status the gingival margin is several millimeters coronal to the cemento-enamel junction (CEJ). The gingival sulcus may be 0.5-3 mm deep on a fully erupted tooth. In teenagers with a healthy periodontium the alveolar crest is situated between 0.4-1.9 mm apical to the CEJ (Hausmann et al., 1991).

Schiller-Pisarev iodine test is used for examination of gum inflammation. A solution which includes 1.0 iodine; 2.0 potassium iodide, and 40.0 of distilled water is applicated on gums. The intensity of coloration means the degree of gum inflammation. The straw-yellow color of the gums is considered as negative, the light brown as low positive, and the dark brown as positive.

The Schiller-Pisarev test can be given quantitatively (in points), estimating the painting of the papillae as 2 points, the marginal ginigiva - 4 points, the alveolar gums - 8 points. The total score obtained is divided by the number of teeth in the area of which the study was performed (usually six teeth). Iodine number is calculated by the formula (Savrakov's number):

$$IN = \frac{\text{Sum of marks}}{\text{Numbe of teeth}}.$$

Estimation of iodine number:

mild inflammation – till 2,3 scores;

moderate inflammation – 2,67 – 5,0 scores;

severe inflammation – 5,33 – 8,0 scores.

Gum bleeding according T.F.Vinogradova has three degrees:

I degree – rarely bleeding while teeth brushing;

II degree –bleeding while teeth brushing;

III degree – bleeding while hard fod consuming.

Examination of dento—gingival pockets

Gingival pocket or pseudopocket is formed by gingival enlargement without apical migration of the junctional epithelium. The margin of the gingiva has moved toward the incisal or occlusal without the deeper periodontal structures becoming involved. The tooth wall of the pocket is enamel. During eruption, the base of the sulcus is at various levels along the enamel. The base of the sulcus of a fully erupted tooth is near the cementoenamel junction. All gingival pockets are suprabony, that is, the base of the pocket is coronal to the crest of the alveolar bone. Sometimes it is difficult to determined initial signs of dentoepithelial attachment, so roentgenography is indicated. Periodontal pockets appear with dento-gingival attachment destruction.

3.2. Index examination of periodontium

Prevalence and intensity of periodontal diseases in population are examined in 15 years old adolescents (Tab. 3.2.1, 3.2.2).

Tab. 3.2.1

Prevalence of periodontal diseases in 15 years old adolescents

Prevalence	*Gum bleeding*	*Calculus*
low	0 – 50%	0 – 20%
middle	51 – 80%	21 – 50%
high	81 – 100%	51 – 100%

Tab. 3.2.2

Intensity of periodontal diseases in 15 years old adolescents

Intensity	*Gum bleeding*	*Calculus*
low	0,0 – 0,5 секстанта	0,0 – 1,5 секстанта
middle	0,6 – 1,5 секстанта	1,6 – 2,5 секстанта
high	≥ 1,6 секстанта	≥ 2,6 секстанта

3.2.1. Gingival indices

If the gingiva is inflamed or the sulcular epithelium is atrophic or ulcerated, periodontal probing induces bleeding in the sulcus. Probing healthy gingival tissues will not provoke bleeding. Bleeding together with colour changes is an early sign of inflammation.

Shiller's – Pisarev's test

Shiller's – Pisarev's test is conducted for chronic gingival inflammation revealing. Gums in the area of lower frontal teeth are painted with iodine solution. An amount of glycogen increases into gums in a case of hidden inflammation and could be rebealed by iodine. The light–yellow coloring evidences about negative test, the dark brown - positive.

PMA index (papillar – marginal – alveolar)

The PMA index is offered by Masser and modified by Parma in 1960. It is used for the estimation of inflammation of gums.

Scale of index estimation:

1 mark is inflammation of gingival papilla (P);

2 marks is inflammation of edge of gums (M);

3 marks is inflammation of alveolar gums (A).

PMA index is calculated in percents after a formula:

PMA= (SUM OF MARKS / (3 x NUMBER OF TEETH) x 100%.

The sum of marks concernes by addition of all greatest indexes of every tooth. Number of teeth in age to 6 years—20;

6-11 of years—24;

12-14 years — 28;

15 years and older — 30.

Criteria of estimation:

to 20% is mild degree of gingivitis;

25-50% is moderate degree of gingivitis;

higher 51% is severe degree of gingivitis.

Gingival Index (GI)

The Gingival Index (Loe and Silness, 1963) was created for the assessment of the gingival inflamation. The gingival index may be scored for all surfaces of all or 16, 11, 24, 31, 36, 44 teeth or for selected areas of all or selected teeth by probing. Code criteria:

0 – no inflammation;

1 – mild inflammation – slight change in color and slight edema;

2 – moderate inflammation (redness, edema and glazing, bleeding on probing);

3 – severe inflammation (marked redness and edema, ulceration with tendency to spontaneous bleeding).

The GI can be calculated by adding the values of each tooth and dividing by the number of teeth examined.

0.1-1.0 - mild inflammation;

1.1-2.0 - moderate inflammation;

2.1-3.0 - severe inflammation.

Sulcus Bleeding Index (SBI)

A. Purpose - to locate areas of gingival sulcus bleeding and color changes in order to recognize and record the presence of early (initial) inflammatory gingival disease.

B. Areas Examined - four gingival units are scored systematically for each tooth: the labial and lingual marginal gingiva (M units), and the mesial and distal papillary gingiva (P units).

C. Procedure:

~ Use standardized lighting while probing each of the four areas.

~ Walk the probe to the base of the sulcus, holding it parallel with the long axis of the tooth for M units, and directed toward the col area for P units.

~ Wait 30 seconds after probing before scoring apparently healthy gingival units.

~ Dry the gingiva gendy if necessary to observe color changes clearly.

SULCULAR BLEEDING INDEX (CODE CRITERIA)

0 score - healthy appearance of P and M, no bleeding on sulcus probing. Apparently healthy P and M showing no change in color and no swelling, but bleeding from suklls on probing.

1 score - bleeding on probing and change of color caused by inflammation. No swelling or edema.

2 score - bleeding on probing and change in color and sfight edematous swelling.

3 score - bleeding on probing and change in color and obvious swell-ingor.

4 score - bleeding on probing and obvious swelling.

5 score – bleeding on probing and spontaneous bleeding and change in color, marked swelling with or without ulceration.

D. Scoring

SBI for area. Score each of the four gingival units (M and P) from

SBI for tooth. Total scores for the four units and divide by 4.

SBI for individual. Total the scores for individual teeth and divide by the number of teeth. SBI scores range from 0 to 5.

3.2.2. Periodontal indeces

Periodontal index (PI)

The periodontal index was developed by Russel (1956) to establish the relative prevalence of the periodontal disease. They are based upon the signs of periodontitis and the sequence in which they usually appear— inflammation, pocket formation, and loss of function. Each tooth is scored; the scores are totalled; and this total is divided by the number of teeth present.

Score criteria:

0 *Negative.* There is neither overt inflammation in the investing tissues nor loss of function due to destruction of supporting tissue.

1 *Mild gingivitis.* There is an overt area of inflammation in the free gingivae which does not circumscribe the tooth.

2 *Gingivitis.* Inflammation completely circumscribes the tooth, but there is no apparent break in the epithelial attachment.

6 *Gingivitis with pocket formation.* The epithelial attachment has been broken and there is a pocket (not merely a deepened gingival crevice due to swelling in the free gingivae). There is no interference with normal masticatory function, the tooth is firm in its socket, and has not drifted.

8 *Advanced destruction with loss of masticatory function.*

The tooth may be loose; may have drifted; may sound dull on percussion with a metallic instrument.

Estimation periodontal scores

Clinically normal supportive tissues - zero to 0.2

Simple gingivitis - 0.3 to 0.9

Innitial destructive periodontal disease - 0.7 to 1.9

Moderate destructive periodontal disease - 1.6 to 5.0

Severe disease - 3.8 to 8.0

Ramfiord periodontal index

A method of assessing the periodontal status, first defined by Ramfjord in 1959, and used to establish the need for treatment and to evaluate the results following treatment. Only 16, 21, 24, 36, 41, 44 teeth are scored for assessment of the periodontal status of the oral cavity. Changes in color, consistency, contour; evidence of ulceration of gingiva is evaluated by a periodontal probe. The next step is recording of the crevice depth related to a cementoenamel junction. For this purpose a University of Michigan 0 probe is used. Distance from free gingival margin to the bottom of the gingival crevice or pocket on the buccal and mesial aspect of the each tooth.

Scoring Criteria

0 — Absence of inflammation

1 — Mild to moderate inflammatory gingival changes not extending all around the tooth

2 — Mild to moderately severe gingivitis extending all around the tooth

3 — Severe gingivitis, characterized by marked redness, tendency to bleed and ulceration

4 — Gingival crevice in any of the four measured areas (mesial, distal, buccal, lingual), extending apically to the CEJ, but not more than 3 mm

5 — Gingival crevice in any of the four measured areas extending apically, 3-6 mm from the CEJ

6 — Gingival crevice in any of the four measured areas extending apically more than 6 mm from the CEJ.

Ramfiord periodontal index is the total of the scores for each tooth divided by the number of teeth examined. It is used in children and adolescents after complete teeth eruption.

Community Periodontal Index (CPI)

A. Purpose

To screen and monitor the periodontal status of populations. Originally developed as the CPITN index that included a code to indicate an individual and group-summary recording of treatment needs. However, because of changes in management of periodontal disease, the treatment needs portion of the index has been eliminated. One component of a complete oral health survey designed by the WHO.

B. Selection of Teeth

The dentition is divided into sextants for recording on the assessment form. Posterior sextants begin distal to canines. 17, 16, 11, 26, 27, 37, 36, 31 ,46, 47 teeth are examined.

Adults (20 years and older)

A sextant is examined only if there are two or more teeth present that are not indicated for extraction. Ten index teeth are examined. The first and second molars in each posterior sextant. If one is missing, no replacement is selected and the score for the remaining molar is recorded. The maxillary right central incisor and mandibular left central incisor. If no index teeth or tooth is present in the sextant, then all remaining teeth in the sextant are examined and the highest score is recorded.

Children and Adolescents (7 -19 years of age)

Six index teeth are examined; the first molar in each posterior quadrant and the maxillary right and the mandibular left incisors. For children under the age of 15,

pocket depth is not recorded to avoid the deepened sulci associated with erupting teeth. Only bleeding and calculus are considered.

C. Procedure

A specially designed probe is used to record the CPI.

CODE CRITERIA

0 = Healthy periodontal tissues.

1= Bleeding after gentle probing; entire colored band of probe is visible.

2=Supragingival or subgingival calculus present entire colored band of probe is visible.

3 = 4- to 5-mm pocket colored band of probe is partially obscured.

4 = 6 mm or deeper; colored band on the probe rs not visible.

Treatment needs: TN 0: in case of gingival health (Code 0), TN 1: need for improved oral hygiene (Code 1), TN 2: need for scaling, removal of overhangs and improved oral hygiene (Codes 2+3), TN 3: complex periodontal treatment (Code 4).

Basic Periodontal Examination (BPE) index

A simplified Basic Periodontal Examination should be carried out on the following six index teeth: UR6, UR1, UL6, LL6, LL1 and LR6.

BPE codes

0 Healthy (no bleeding on probing, calculus or pocketing ≥ 3.5mm detected)

1 Bleeding on probing (no calculus or pocketing ≥ 3.5mm detected)

2 Calculus or plaque retention factor (no pocketing ≥ 3.5mm detected)

3 Shallow pocket (4 mm or 5 mm)

4 Deep pocket (≥ 6 mm)

* Furcation

The BPE is performed using the WHO 621 probe with a light probing force of 20-25 g. This has a 0.5mm spherical ball on the tip and a black band at 3.5-5.5mm to delineate healthy sulcus depth (<3.5mm) and periodontal pockets (≥ 3.5mm).

Management of index teeth according to simplified BPE Code

Code 0: No treatment required.

If BPE = 0, screen again at routine recall visit or within 1 year, whichever the sooner

Code 1: Oral hygiene instruction and prophylaxis

Code 2: Supra and subgingival scaling at selected sites in addition to oral hygiene instruction and prophylaxis. Remove plaque retention factors.

If BPE = 1 or 2, treat and screen again at routine recall or after 6 months, whichever the sooner

Code 3, 4, * Following full periodontal assessment, supplementary radiographs may be required to assist diagnosis, although the existence of false pocketing in the case of erupting teeth in the mixed and early permanent dentition must be considered as the gingival margin may be situated coronal to the cemento-enamel junction by a number of millimeters in young individuals. Other clinical signs of pathology e.g. bleeding, suppuration, tooth mobility will be pertinent to an accurate diagnosis.

After false pocketing is accounted for, young patients scoring Codes 3 should be treated as for code 2 except that more intensive treatment (including root surface debridement) may be indicated followed by a review after 3 months. Codes 4 and * are unusual in young patients and full periodontal assessment with a referral to a Specialist Periodontologist or Paediatric Dentist should be considered.

Assessment of periodontal treatment needs should be started at 7 years of age as it is rare to experience problems below this age and the index teeth are often still unerupted. Identification of periodontal disease in the primary dentition is unusual and young children with unexplained premature exfoliation or gross mobility of primary teeth or red, oedematous gingivae and /or suppuration for which no other dental cause can be seen should be referred for specialist advice.

At 7-11 years of age, in the mixed dentition phase, the index teeth should only be examined for bleeding of the gingiva, calculus and/or overhangs of fillings ie BPE codes 1 and 2 only, to avoid the problem of false pockets. In this age group both the erupting first permanent molar and later, the exfoliating second primary molar could give the appearance of periodontal pocketing.

a. *Comment*: it would be uncommon to have any true periodontal pocket at this age. If a true pocket is present, referral is recommended.

b. *Comment*: bleeding on probing even from a false pocket is indicative of the need for oral hygiene instruction.

At 12-17 years of age, the full range of BPE codes can be used on the six index teeth.

a. *Comment*: it would be uncommon to find periodontal breakdown at other teeth without the index teeth being affected.

b. *Comment*: whenever periodontal pockets are recorded i.e. BPE code 3 or 4, the alveolar bone level should be checked. Bitewing radiographs are suitable for posterior teeth. Selected periapicals are indicated for the anterior teeth.

Tooth mobility

Periodontal tissues are built up in a way to allow some oro-vestibular movement of the teeth. The extent under physiological conditions is 0.1 mm, but it can increase to about 0.5 mm during pregnancy. Physiological movement has two components: spatial organisation of rigid periodontal ligaments and mechanical flexibility of supporting bony housing. Pathological mobility is the result of quantitative and qualitative changes in the supporting tissues. Supracrestal fibres of the gingiva are destroyed by inflammatory processes. Composition changes also take place within the fibres. As a result of the spreading infection, periodontal ligaments and supporting bone gets damaged, compromising tooth anchorage. Occlusal overload can also result in tooth mobility because periodontal tissues try to adapt to the change in loading forces and neutralise the load. Widening of the periodontal space and increased mobility are typical in this condition.

0 level of mobility – physiologic movement M=0-0.2 mm,

I level of mobility – horizontal, bucco-oral movement

Physiologic<M<1 mm,

II level of mobility – horizontal, bucco-oral movement, 1 mm<M

III level of mobility – horizontal, bucco-oral movement

1 mm<M Also axial

Chapter 4
CLASSIFICATIONS OF PERIODONTAL DISEASES

4.1. Classification of XVI Meeting of Soviet Union dental scientific society (1983)

This classification divedes periodontal diseases on five groups:

1. Gingivitis- inflammation of gingival mucosa without affection of the the dentogingival junction.

Forms: catarrhal, hypertrophic, and ulcerative.

Course: acute, chronic, aggravated, remission.

Prevalence: localized, generalized.

Degree of severity: mild, moderate, severe.

2. Periodontitis- the inflammatory destructive process in periodontal tissues with disturbed integrity of the dentogingival junction.

Course: chronic, aggravated, abscessed, remission.

Prevalence: localized, generalized.

Degree of severity: mild, moderate, severe.

3. Periodontosis: - adystrophic lesion of periodontal tissues.

Course: chronic, remission.

Prevalence: generalized.

Degree of severity: mild, moderate, severe.

4. Idiopatic diseases with progressive lysis of periodontal tissues (Papillon-Lefevre syndrome, histiocytosis, hereditary neutropenia, decompensated diabetes, etc).

5. Tumors and tumor-like diseases: epulis, gingival fibromatosis, etc.

This classification has differentiated approach to the different periodontal diseases according signs of inflammation, dystrophya and tumors. The approach takes into consideration the ethiological factors that facilitates a choice of adequate treatment and prevention methods. Also standardization of base symptoms of each

disease: form, severity, clinical course, and prevalence, makes diagnostic easy.

But this classification has lacks. Localized periodontitis is considered as inflammation about 1-2 teeth after trauma. Mild, moderate and severe degree of generalized periodontitis don't show the reversible changes with their treatment.

4.2. R. S. Page and H. E. Schroeder (1983) classification

R. S. Page and H. E. Schroeder (1983) offered periodontal classification which depends on age.

I. Pre-pubertal periodontitis (till 12 years):

– localized

– generalized

II. Juvenile periodontitis (from 13 to 17 years).

III. Rapidly progressive periodontitis (from 17 to 35 years).

IV. Adult periodontitis (from 35 years).

V. Acute necrotizing ulcerative gingivo-periodontitis

4.3. The Periodontal Disease Classification System of the American Academy of Periodontology — An Update (1999)

Abbreviated version of the 1999 classification of periodontal diseases and conditions.

I. Gingival Diseases

A. Dental plaque-induced gingival diseases

B. Non-plaque-induced gingival lesions

II. Chronic Periodontitis (slight: 1-2 mm clinical attachment level (CAL); moderate: 3-4 mm CAL; severe: > 5 mm CAL)

A. Localized

B. Generalized (> 30% of sites are involved)

III. Aggressive Periodontitis (slight: 1-2 mm CAL; moderate: 3-4 mm CAL; severe: > 5 mm CAL)

A. Localized

B. Generalized (> 30% of sites are involved)

IV. Periodontitis as a Manifestation of Systemic Diseases

A. Associated with hematological disorders

B. Associated with genetic disorders

C. Not otherwise specified

V. Necrotizing Periodontal Diseases

A. Necrotizing ulcerative gingivitis

B. Necrotizing ulcerative periodontitis

VI. Abscesses of the Periodontium

A. Gingival abscess

B. Periodontal abscess

C. Pericoronal abscess

VII. Periodontitis Associated With Endodontic Lesions

A. Combined periodontic-endodontic lesions

VIII. Developmental or Acquired Deformities and Conditions

A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis

B. Mucogingival deformities and conditions around teeth

C. Mucogingival deformities and conditions on edentulous ridges

D. Occlusal trauma

4.4. The classification of periodontal and peri-implant diseases and conditions (2017)

The new classification is the outcome of a joint workshop held by the European Federation of Periodontology and the American Academy of Periodontology in Chicago in November 2017.

I. Gingival health/gingival diseases and conditions.

1. Periodontal health and gingival health.

Periodontal health can exist on an intact or a reduced periodontium. An intact periodontium is one without clinical attachment loss or bone loss, whereas a reduced

periodontium may arise either in patients with some forms of gingival recession or following crown-lengthening surgery or in a patient with a history of periodontitis. A case of clinical gingival health was established on an intact and a reduced periodontium in a non-periodontitis patient as <10% sites of bleeding on probing (BoP) and probing depths of <3 mm.

2. Gingivitis: dental biofilm induced.

- associated with dental biofilm alone;
- mediated be systemic and local risk factors;
- grug-influenced gingival enlargement.

3. Gingival diseases: non-dental biofilm induced.

a) genetic/ developmental; b) specific infections; c) inflammatory/ immune; d) reactive; e) neoplasms; f) endocrine, nutritional and metabolic, g) traumatic; h) pigmentation.

II. Periodontitis

It was concluded that there was a lack of evidence for the distinction between "chronic" and "aggressive" periodontitis. The new classification is based on four stages and three grades. Stages are assessed in terms of extent and distribution as: localised, generalised, and molar-incisor distribution.

1. **Stage 1 – Initial periodontitis** – a very incipient periodontitis with clinical attachment loss and bone loss limited to the most coronal portion of the root. In this stage, patients do not present periodontal pockets and they have not suffered tooth loss caused by periodontitis.
2. **Stage 2 – Moderate periodontitis** – periodontal destruction affects the coronal third of the root and is characterised by the presence of moderate periodontal pockets (<5mm) and the patient has not yet lost teeth because of periodontitis.
3. **Stage 3 – Severe periodontitis with potential for additional tooth loss** – A limited amount of tooth loss has usually already occurred and the presence of furcation and intrabony lesions is common. All these aspects make the treatment of this stage complex and surgical interventions are usually needed.

4. **Stage 4 – Severe periodontitis with potential for loss of the dentition** – increases the severity and complexity of the previous stage by an increased tooth loss (>5 teeth) and the presence of masticatory dysfunction, which usually requires a complex multidisciplinary treatment beyond the periodontal therapy.

5. **Grade A –** The rate of progression is low and the patient does not have risk factors.

6. **Grade B –** The expected progression.

7. **Grade C –** The patient has evident risk factors and there is a high risk of periodontal progression.

III. Other conditions affecting the periodontium.

1. Systemic diseases or conditions affecting the periodontal supporting tissues.
2. Periodontal abscesses and Endodontic-periodontal lesions.
3. Mucogingival deformities and conditions.
4. Traumatic occlusal forces.
5. Prosthesis and tooth-related factors.

4.5. International classification of diseases-11 - Mortality and Morbidity Statistics (2019)

13 Diseases of the digestive system

DA Diseases or disorders of orofacial complex

DA0B Gingival diseases

DA0C Periodontal disease

DA0C.0 Acute periodontitis

DA0C.1 Aggressive periodontitis

DA0C.2 Periodontosis

DA0C.3 Necrotising periodontal diseases

DA0C.30 Necrotising ulcerative periodontitis

1C1H Necrotising ulcerative gingivitis

DA0C.3Y Other specified necrotising periodontal diseases

DA0C.3Z Necrotising periodontal diseases, unspecified

Chapter 5
CLINICAL MANIFESTATIONS OF PERIODONTAL DISEASES

The results of epidemiological studies conducted in different countries indicate a high prevalence of periodontal diseases and their early onset. According to WHO report, chronic gingivitis in Europe is found in almost 80% of children aged 10-12 years and in almost 100% of children aged 14-15 years.

Mostly children are diagnosed with chronic catarrhal gingivitis (10 - 15%). Then its prevalence increases and almost reaches 20% in the 17-18 years olds. Periodontitis is found in 3-5% of cases, mainly in adolescents. Idiopathic periodontal diseases, as symptomatic, are more commonly manifested at an early age. Periodontosis as a dystrophical periodontal disease in children is uncommon.

According to the results of studies conducted by the staff of the Department of Pediatric Dentistry and Dental Disease Prevention of the Bogomolet's National Medical University, the prevalence of gingivitis in 15-year-old adolescents in different regions of Ukraine ranges from 51 to 100%. The prevalence of localized and generalized periodontitis in Ukraine in the same age group varies from 5.2 to 17.8% in different regions.

Therefore, a pedodontist should know the etiological factors, conditions of appearence, mechanisms of development and clinical manifestations of periodontal diseases in order to carry out their effective prevention, early diagnostics and comprehensive treatment.

5.1. Papillary gingivitis (Papillitis)

Papillary gingivitis involves the interdental papilla but not the rest of the free gingiva around a tooth.

There are three kinds of papillary gingivitis: catarrhal, ulcerative, hypertrophic and atrophic. Its clinical course can be acute or chronic.

Catarrhal papillitis

Acute catarrhal papillitis develops due to acute trauma by a hard stuff, bristles of tooth brush, and dental instruments. Cause of chronic catarrhal papillitis is local factors (improper contact of proximal filling, sharp edge of proximal caries, crown, clamp etc.).

Patients with acute catarrhal papillitis have complaints of a pain and bleeding gums when brushing teeth and eating. Pain is permanent or intermettent. Papilla is reddish and swollen. If the inflammation persists, papilla can get rounded, fibrotic; pseudopockets can develop.

Patients with chronic catarrhal papillitis have complaints of a gum burning. Papilla is cyanotic, swollen, its relief is flattened. Papilla is sensitive and bleed on palpation. Redness, cyanosis and moderate swelling of the gums are revealed.

Ulcerative papillitis

Causes of ulcerative papillitis are long-term mechanical irratants or chemical burn.

Patients with acute ulcerative papillitis have complaints of a strong pain in area of the papilla, which sometimes irradiates, and malodor. Papilla is reddish, swollen, and necrotic. Necrotic tissues are easly removed, opening bleeding surface. Regianal lymphatic nodes are swollen and painfull on palpation. General state hasn't changed.

Patients with chronic ulcerative papillitis have complaints of a unsignificant pain in area of the papilla, malodor. Papilla is congestive and covered by a grey plaque, a top of the papilla looks like cut. Necrotic tissues are difficult removed, opening bleeding surface. Probing of the gingival sulcus is painfull.

Hypertrophic papillitis

Hypertrophic papillitis has chronic course with exacerbations.

Causes of hypertrophic papillitis are such local irritant factors as improper contact of proximal filling, sharp edge of proximal caries, crown, clamp. Juvenile papillitis may develop in pubertal period.

Patients with chronic hypertrophic papillitis have complaints of dyscomfort from chemical and mechanical irritants, improper esthetics in frontal area. Gingival papilla is congestive, swollen, fill a proximal caries cavity or interdental space. Gums

are tough, a top of papilla has scalloped outline. Probing and palpation of the gingival papilla provokes insignificant bleeding.

There are 3 grades of hypertrophic gingivitis. Grade 1: Minimal enlargement of papilla covering the cervical third or less of anatomic crown. Grade 2: Moderate enlargement of gingiva extends into the half of height of anatomic crown. Grade 3: Severe enlargement of papilla than 2/3rd of the crown.

Overgrowth gingiva is traumatized by a hard food, opposite teeth causing exacerbation of chronic inflammation. This inflammation reveals as significant pain, swelling, bleeding, and brightly expressed hyperemia, exudation on palpation. Process has fast manifestations. Symptoms can fade, and papillitis transfers into chronic form.

Atrophic papillitis

Atrophic papillitis is caused by ischemia of the gingival papilla, improper exchange and blood circulation, and develops due to constant pressure of an improper filling on the gingival papilla in a case of the wrong contact point or food in a case of the contact point absence. This papillitis has chronic course.

Patients with chronic atrophic papillitis have complaints of an unpleasant feeling or significant paroximal pain due to food between teeth. Papilla is hyperemic, has a small size, a top of the papilla looks flattened. Probing of the gingival sulcus is painfull, provokes bleeding.

There are three stages of gingival papilla atrophy. At the first stage, the size of the papilla is reduced by one-third, its anatomical shape is preserved. At the second stage, the papilla atrophies to the middle third and more, the interdental space isn’t filled. At the third stage, the gingival papilla is completely absent, the adjacent gingiva is funnel-shaped.

Differential diagnosis of catarrhal papillitis is difficult, as it should be distinguished only from catarrhal gingivitis, in which the inflammation affects the gums in the area of several teeth.

In a case of ulcerative papillitis pain attacks simulate acute pulpitis. In contrast to pulpitis, rapid and timely removal of the stimulus eliminates a painful attack. If the

papilla is filled a carious cavity its probing is usually painless (in a case of pulpitis provokes a sharp pain).

Hypertrophic papillitis should be differentiated from epulid and hypertrophic pulpitis. The epulidus is denser, its surface is uneven, grainy, with areas of brown pigmentation. The epulidus is usually located on a thin leg, the bleeding is less pronounced; also radiographic changes of bone are found. The main in the differential diagnosis between hypertrophic pulpitis and gingival papillitis is a determination the source of tissue overgrowth. In a case of pulpitis the pulp grows from the tooth cavity. In case of papillitis, the gingiva grows outside the tooth. Therefore, in a case of hypertrophic pulpitis the probe can be carefully and freely held between the wall of the carious cavity and the gingival overgrowth, and in a case of papillitis the probe only reaches the point where the papilla grows into the carious cavity. Thus, in a case of hypertrophic pulpitis, the probe closes the full circle, and in a case of hypertrophic papillitis this circle in the area of the papilla overgrowth is found unclosed.

5.2. Gingivitis

Gingivitis is gingival inflammation without destruction of dental-gingival attachment. Plaque induced gingival diseases can be further divided, based on plaque being the only reason of diseases, or other factors (systemic diseases, medications, malnutrition) contributing to that. Symptoms of gingivitis are swollen gingival margins, reddish/purplish discolouration, bleeding (in the beginning to mechanical irritation, in severe cases spontaneously). Changes start at the papilla and proceed to the gingival margin. The orange skin texture of the attached gingiva can be still present. If the inflammation persists, papillae become rounded, the gingival margin and papillae can get fibrotic, pseudopockets can develop. Local factors stimulate plaque formation, or inhibit adequate oral hygiene; these factors for gingivitis can be natural or artificial. Artificial factors are restorations with improper marginal closure, micro-mechanical elements of removable partial dentures, or orthodontic appliances. Natural factors are tooth with malformations (enamel pearl, prolonged fissures) or

malpositioned, crowded teeth. A special condition is found in mouth breathers, which restricts to surfaces not covered by the lips while breathing, like incisor buccal surfaces.

Gingivitis is divided on localized and generalized. Localized process involves ginigiva in an area of few teeth, generalized inflammation spreads to few or all groups of teeth.

There are three degrees of disease. Mild form affects only gingival papillae. Moderate form affects gingival papillae and margin. Severe gingivitis is characterized by whole gingiva inflammation, including its alveolar part.

Catarrhal gingivitis

Catarrhal gingivitis in children often has chronic course. Acute catarrhal gingivitis, as a rule, occurs on background of acute infectious diseases (measles, scarlet fever, herpes, diphtheria etc.). Catarrhal gingivitis may be a symptom of these diseases and its early detection is important.

Anamnesis of catarrhal gingivitis is complaints of pain and bleeding gums when brushing teeth and eating. Clinical course has a bright redness and swelling of the mucous membrane of the gums. Index assessment of periodontal tissues: - PMA (papillary-marginally-alveolar index) index to 20% - mild severity of gingivitis from 25 to 50% - the average severity of gingivitis above 51% - severe gingivitis. Mild gingivitis has such signs as hyperemia and swelling of the gingival papillae. Moderate gingivitis has such signs as expressive hyperemia and swelling of the gingival papillae and the gingival margin, pain on palpation of these parts of gingiva. Severe degree of disease has signs of expressive hyperemia, swelling, pain, and bleeding on palpation of all parts of gingiva. Roenthenological change in alveolar bone are absent.

Medical history of chronic catarrhal gingivitis includes complaints of recurrent gum bleeding during teeth brushing. Redness, cyanosis and moderate swelling of the mucous membrane of the gums are revealed. X-ray: unclear contour of the cortical plate of the alveoli, osteoporosis of the spongy substance on the tops of the interdental septum. Index assessment of periodontal tissues can be done with PMA

and CPI. Positive Shiller-Pysarev test is an indicator of glycogen deposit in gingiva, that is a sign of chronic inflammation.

Exacerbation of chronic catarrhal gingivitis can be revealed in children with somatic diseases. In this case gums become painfull, swollen, hyperemic and bled on touch. Fatigue and subfebrile body temperature can be revealed.

Unclear complete cortical plate, osteoporosis of sponge substance on tips of interdental septae can be found on roenthgenogram. These changes in bone are recurrent and may be healed after a sucsessfull antinflammation gingival treatment.

Differential diagnostic of chronic catarrhal gingivitis is conducted with generalized periodontitis. The main signs of generalized periodontitis are such roenthgeneological changes as initial resorption amd osteoporosis of tips of the interdental septae, and widening of periodontal ligament space in the cervical part.

Long-term course of chronic catarrhal gingivitis without treatment lead to hypertrophic, ulcerative-necrotic gingivitis and generalized periodontitis development due a weak local immune state.

Desquamative gingivitis is a kind of catarrhal gingivitis which comes up in girls in the prepubertal and the pubertal periods.

Diseases together called desquamative gingivitis are characterised by bullous formation, erosions, atrophical spots, which in severe cases, peel off the underlying connective tissue, leaving a greyish-bluish unepithelialised area behind. They create major subjective complaints, especially in connection with irritative food and drink consumption (spicy foods, carbonated drinks, acidic foods, etc). This is the oral manifestation of Pemphigus vulgaris, Pemphigoid, Lichen planus, Erythema multiforme, Lupus erythematosus.

There are three forms of desquamative gingivitis: erthytematous, erosive and bullous.

Patients with erthytematous form of desquamative gingivitis complain on itching, significant pain and gingival bleeding during brushing. Clinical examination reveal that the alveolar gums are brightly hyperaemic, swollen, painful, and look denude.

Patients with erosive form of desquamative gingivitis present with a moderate pain and a bleeding during tooth brushing. Erythema, oedema and erosions are found on the attached gingiva. Gentle friction applied to the involved gingiva cause formation of blood-filled bullae, and the covering epithelium is easily removed with forceps.

Patients with bullous form of desquamative gingivitis complain on presence of the bullae on gums and the painfull erosions, difficult talking and eating. Bullous form of desquamative gingivitis is a precursor of pemphigus vulgaris and is rarely seen in children.

Hypertrophic gingivitis

Hypertrophic gingivitis – gingival inflammation related to oedematous swelling, or hyperplasia caused by overproduction of connective tissue fibres and matrix, together with thickening of the epithelium.

Gingival overgrowth

1. infection related
2. medication related (Ca-antagonist, hydantoine, Cyclosporine-A, Erythromycin, Bleomycin)
3. modified by systemic conditions (pregnancy, puberty, Vitamin-C malnutrition plasma cell infiltration, non-specific pyogenic granuloma)
4. provoked by systemic conditions (leukaemia, granulomatosus diseases, sarcoidosis)
5. provoked by tumours (malignant, benign)
6. hereditary fibromatosis gingivae
7. idiopathic

Gingival hyperplasia in frontal area of jaws often occurs due such local traumatic factors as teeth crowding, abnormal attachment of soft tissues in vestibule.

Hypertrophic ginigivitis in children has mostly chronic manifestation.

There are 3 grades of gingival enlargement. Grade 1: Minimal enlargement that is less than 1 mm increase in size gingival covering the cervical third or less of anatomic crown. Grade 2: Moderate enlargement that is 2-4 mm increase in size or

gingiva extends into the half of height of anatomic crown. Grade 3: Severe enlargement that is nodular growth and increase in size more than 2/3rd of the crown.

There are two forms of hypertrophical gingivitis: fibrous and granulating. Last one is often diagnosed in adolescents.

Children with granulating form of hypertrophical ginigivitis complain about the gingival growth, itching, moderate pain and bleeding during tooth brushing and consuming a hard food, and halitosis. Gingivae in the labial aspect are swollen, cyanotic. Gingival papillae are swollen, have wrong shape, bleed on probing. Relief of the marginal gingiva had a thickened spindle shape.

Gingival overgrowth often impairs nutrition and access to oral hygiene, resulting in an increases susceptibility to oral infection, caries and periodontal disease.

The gingival enlargement associated with anticolvusant drugs starts as a painless, bead like enlargement of the facial and lingual gingival margins. As the condition progress, the marginal and papillary enlargement unites, they may develop into a massive tissue fold covering a considerable portion of the crowns and they may interface with occlusions.

Relapse of granulating form of hypertrophical ginigivitis is characterized by strengthening of gingival hyperemia, swelling and bleeding.

Children with fibrous form of hypertrophical gingivitis don’t have complains in a case of mild form, or complain on unusual shape of enlarged gums. Gums overlap teeth crowns, and the teeth are submerged deep into the gingiva. Gums had normal colour and firm consistency without bleeding on probing. The biopsy showed dense bundles of collagen fibres with numerous fibroblasts varying from plump to spindle shape.

In case of long developed hypertrophic gingivitis osteoporosis of the spongy substance on the tips of interalveolar septae without resorption of cortical lamina are revealed during the radiography.

Differntial diagnostic of hypertrophic gingivitis is conducted with chronic catarrhal ginigivitis, gingival fibromatosis and leukemic infiltration. Fibromatosis is

characterized by slowly progressive, non hemorrhagic enlargement of the maxillary and mandibular gingiva which overlap teeth crowns to whole height. There is hypertrophy of free and attached gingiva in the labial and the lingual aspect. Gums have pale pink color, tough, without bleeding on probing. Leukemic infiltration is characterized by moderate inflammation hypertrophy of gums in the oral aspect.

Acute necrotising ulcerative gingivitis (ANUG)

Disposing factors are categorised as local and systemic ones. Systemic factors are the sympato-mimetic effect of stress, seasonality: the disease had higher occurrence in autumn and winter due to nutritional factors. It is most common among young adults with poor general health (e.g. HIV), or on the ground of malnutrition. Local predisposing factors are poor oral hygiene and the presence of plaque retentive factors. The plaque is dominated by Fusobacterium species.

Necrotising ulcerative gingivitis has acute manifestations in children. There are three degrees of this disease: mild, moderate and severe.

Children with acute necrotising ulcerative gingivitis complain on sugnificant pain and gum bleeding that strengthens during eating, bad breath. The disease associated with fever, malaise and loss of appetite, headache, insomnia, dyspepsia. The disease has 4 periods: prodromal, first clinical manifestations, significant clinical manifestations, and regress.

The prodromal period takes 1-2 days. Children with mild degree don’t have any manifestations, with severe degree complain on dry mouth, itching, and gingival pain.

Period of first clinical manifestations is characterized by catarrhal inflammation: swollen, hyperemic gums that bleed on touching. Hyperemia and swelling involves all parts of gums. Palpation of gums is significantly painfull, eating becomes difficult. Other symptoms are subfebrile temperature up to 38°C, fatigue, loss of appetite, headache, and insomnia. This stage lasts 2-4 days, in case of severe degree – 1 – 1,5 days.

In period of siginificant clinical manifestations the catarrhal inflammation disappeares and necrotic gingivae are prevalent. Duration of this period (from 5 up to

15 days) depends on disease degree and medical care. This period starts as localized gingivitis in area of 1-3 teeth (light degree). A greyish plaque is found on swollen, hyperemic gums. Painfull ulcerations with blood drops are appeared after plaque removal («blood dew» symptom). Ulcerations are typically present on the labial side. It comes with severe pain, necrosis of the papillae: blunt papillae besides the incisors and wisdom teeth, which are covered with a greyish pseudo-membrane («cut papillae» symptom). Somatic symptoms are same with period of first clinical manifestations. Bad breath is also a typical characteristic of the disease. Bleeding starts on the mechanical irritation, in later stages even spontaneously.

Due to ulcerations children keep their mouth open. There is a lot of dental plaque and calculus, tongue is covered by a plaque; salivation is found. The swollen soft painfull regional lymph nodes are also revealed.

Moderate degree of disease is characterized by generelized gingivitis, high temperature (from 37,5 up to 39^{o}C), and intoxication. A child can have loss of appetite, headache, insomnia, and dyspepsia. Gingival papillae can be necrotized completely.

Weak children have severe degree of ulcerative gingivitis. In adverse cases, contact ulcerations may be present on the pharyngeal mucosa, tongue, lips (Vincent angina), and in the most severe cases, on the buccal tissues, which can lead to perforations (cancrum oris or noma). In severe cases, systemic symptoms can develop. The disease may involve the attached gingiva and the alveolar bone (acute necrotizing ulcerative periodontitis). General state od such children is worsened due to the body temperature more than 39^{o}C and intoxication.

Roenthenologic changes in periodontal tissues are absent at acute ulcerative gingivitis.

Chronic ulcerative gingivitis develops as a result of acute process and characterized by a latent course of disease. Patients complain of permanent bleeding, painfull gingivae and malodor. Gums are hyperemic, swollen, their marginal part is ulcerative, thick and are covered with a grey plaque. Interdental spaces are open. After plaque and necrotic tissues removal the bleeding ulcerations are revealed. The

swollen soft painfull regional lymph nodes are palpated. The disease usually has a mild course, without significant complains and somatic state disorders. Roenthenologic changes in periodontal tissues are osteoporosis of the interdental septae and widening of the periodontal ligament space.

Cytologic examination of ulcerations reveals Fusobacteria, Spirochetae, Cocci, large amount of neutrophils, alone lymphocytes, plasm cells and histeocytes.

Leukocytosis and increased erythrocyte sedimentation rate are found. If ulcerative-necrotic changes in oral cavity were found the blood test is obligatory.

Differential diagnostic of acute ulcerative gingivitis is conducted with ulcerations associated with idiopathic progressive periodontal diseases and blood diseases. Diagnostic signs of ulcerations associated with blood diseases are mild inflammation of the surrounding tissues, latent course, asthenic, anaemic and hemorrhagic syndromes, and blood formula changes.

Athrophic gingivitis

Athrophic gingivitis is a dystrophic disease with gingival recession in area of one or few teeth. Its development is caused by local and general factors (insufficient blood supply of periodontal tissues, neurohumoral and other disorders). Causes of athrophic gingivitis in children are: abnormalities of structure and attachment of soft tissues in oral cavity, malpositioned teeth, and occlusal overload. Atrophic gingivitis can occur after ulcerative gingivitis.

Clinical course of athrophic gingivitis is asymptomatic, however, children may complain on iching, hypersensitivity to chemical and thermal irritants due to exposed dental cervices. There are two types of the athrophic gingivitis: localized, which name is V-shape, and diffuse.

In some case, a localized gingival recession is present only on the labial surface of the low central incisors without changes on ther lingual surface due to a short frenulum of the lower lip; there is no hyperaemia and bleeding on probing. In other cases, a habit to scratch gingiva with fingernail or improper tooth brushing provokes atrophic gingivitis. There is a loss of papilla height on the mesial aspect of the tooth and moderate gingival overgrowth on the distal aspect. The gingiva is oedematous,

hyperaemic and ulcerated. Exposed root up to 1/2 length is revealed, but dental-gingival attachment isn’t destroyed.

Diffuse atrophic gingivitis occurs rarely and presents as a gingival resession in the area of few or all teeth. Gingival mucosa is thick, swollen, its color isn't changed, pain and gum bleeding are absent. Dental cervices are gradually exposed, but teeth mobility is absent. If gums have lost relief interdental spaces are open.

Atrophic gums aren’t recovered with age, disease is developing and may finish with teeth mobility.

5.3. Periodontitis

Periodontitis is an inflammatory-destructive process that affects all periodontal tissues: mucosa of gums, periodontium, alveolar bone, and cementum of root.

The base of morphogenesis of the disease are exudative-alternative inflammation with pronounced and increasing phenomena of destruction of the alveolar bone, cementum and periodontium.

The clinical course of periodontitis can be acute, chronic, exacerbated (including with the formation of single or multiple abscesses) and remission (short-time stabilization).

Acute periodontitis is rare and usually localized. It develops, as a rule, under the influence of infections, with orthodontic interventions that cause impaired integrity of the dentition, and also due to mechanical irritation of the tissues with artificial crowns.

Chronic periodontitis is the most common in clinical practice. It takes a long time. The clinical features, the depth of the pathological process and the nature of the course depend on the general condition of the organism and its reactivity.

By reducing the reactivity of the organism, comorbidities and attachment of secondary infection, the inflammatory-dystrophic process in the periodontal tissues is exacerbated.

Under the influence of preventive and curative measures due to the increase of the body's defenses, remission or stabilization is observed.

Depending on the severity of clinical manifestations distinguish mild, moderate and severe periodontitis.

Periodontal pockets with a depth of up to 3.5 mm is a sign of mild periodontitis. Sometimes the integrity of the dental epithelial attachment is partially compromised and due to the inflammation and swelling of the gums a shallow periodontal pocket (1 - 2 mm) is difficult to find. In this case formalin test can be used: the appearance of acute pain indicates focal destruction of the attachment epithelium. The teeth are stable, but the grade I teeth mobility can be observed. The radiograph shows: widening of the periodontal ligament space around the tooth neck, the absence of a compact plate on the tips of the interdental septae and their resorption up to 1/3 of the height.

In a case of moderate periodontitis, the depth of periodontal pockets increases up to 4 - 5 mm. The grade I-II teeth mobility is observed which leads to the development of traumatic occlusion. The radiograph shows: widening of the periodontal ligament space, destruction of the cortical plate, reduction of the height of the interdental alveolar septae up to 1/2 of the height with signs of osteoporosis.

In a case of severe periodontitis, the depth of periodontal pockets increases up to 6 mm and beyond. The grade II-III teeth mobility is observed which leads to the traumatic occlusion. In addition to these changes, the radiograph shows an increase in the resorption of the interdental alveolar septae over 1/2 of their height.

The severity of periodontal tissues damage depends on the degree of their formation and morphological maturity, as well as on the functional (chewing) load. Formation of dentoalveolar anomalies, disturbance of the structure and the attachment of soft tissues create stable conditions for the development of pathological changes in the periodontium due to the permanent overload of its area. It is also facilitated by bad habits, impaired chewing, swallowing and breathing. Factors that exacerbate the severity of periodontitis include the lack of oral hygiene care. The progression of the pathological process in periodontal tissues is observed with a decrease in the immunological reactivity at somatic diseases.

Periodontitis is divided on localized and generelized. Localized periodontitis

affects at least 2 teeth: incisors and first molars. Generelized periodontitis affects at least 3 teeth other than incisors and first molars.

Localized periodontitis

It occurs on the background of disproportion in jaw growth and immature periodontal tissues under the local traumatic factors:

– improper fillings (no contact point);

– abnormalities of structure and attachment of the oral soft tissues (shallow vestibule oris, short frenulae of lips and tongue);

– malocclusion and malpositiomed teeth (teeth crowding, tortoanomaly, vestibular or oral tooth position).

Localized periodontitis is characterized by the focal inflammation (frontal or posterior) signs - swelling, hyperemia and gum bleeding. Destruction of dental-gingival junction and periodontal pockets are found with the disease development.

There are three degree of localized periodontitis: mild, moderate and severe; clinical course can be acute and chronic. Children mostly have mild degree of the chronic localized periodontitis.

Children with acute localized periodontitis complain of pain in a separate area of the gums which exacerbates during teeth brushing and eating. The pain is accompanied by bleeding, burning, gum swelling, and a bad breath.

There is a bright hyperemia and pronounced edema of the affected area of the gums, soft dental plaque, mild bleeding and pain on touch. Also periodontal pockets of different depth are revealed (depending on periodontitis severity). Serous or purulent exudate is released on palpation. The grade I-II teeth mobility is found at moderate or severe periodontitis. Schiller-Pisarev's test is negative.

Foci of diffuse osteoporosis of the spongy bone of the interdental septa in a limited area of the alveolar bone are found on radiographs, there are no signs of bone sclerosis.

Cildren with chronic localized periodontitis complain of unpleasant feeling in a particular area of the gums, itching, and periodic bleeding during brushing teeth and eating hard food. They also notice a bad breath.

There is congestive hyperemia and slight edema of the affected area of the gums, supra- and subgingival dental deposites, periodontal pockets of different depth (depending on the disease severity). Palpation of the affected area is accompanied by a mild pain and a slight discharge of serous exudate. The mobility is found with moderate or severe periodontitis. Schiller-Pisarev's test is positive.

Widening of the periodontal ligament space, destruction of the cortical plate, reduction of the height of the interdental alveolar septae and slight osteoporosis of the spongy bone of the interdental septae in a limited area of the alveolar bone are found on radiographs.

Also proliferative, and sometimes ulcerative necrotic changes can be observed. Gngival recession is revealed at long-term localized periodontitis. Further progression of the inflammatory-destructive process leads to deepening of periodontal pockets and increase of the pathological mobility of teeth.

Cytological examination of the content of periodontal pockets reveals a variety of microflora: cocci, spindly sticks, protozoa (trichomonads), spirochetes and fungi Candida.

Pathomorphological changes are determined in all periodontal tissues. In the mucous membrane of the gums there are signs of nonspecific chronic inflammation: dystrophic changes of the epithelium, diffuse lymphoid-plasmocytic infiltration of the connective tissue base of the gums with the phenomena of its surface disorganization not only in the gingival sulcus, but also in the deep areas; also epithelium growth along a root and resorption of the interdental septae under the influence of osteoclasts and macrophages.

Differential diagnostic of localized periodontitis is conducted with different forms of gingivitis that never has the alveolar bone destruction on a roenthgenogram.

Generelized periodontitis

Generelized ginigivitis occurs after chronic gingivitis, accompanied with somatic diseases and low immunity. Comparing to aggressive periodontitis, which is considered to be fast progressing, chronic periodontitis is characterized by a low to moderate rate of progression that may include episodes of rapid destruction. It is

rarely diagnosed (3 – 5% from periodontal diseases in children), mostly in adolescents.

Symptoms of generalized periodontitis:

– symptomatic gingivitis;

– periodontal pockets;

– progressive alveolar bone resorption with teeth mobility and traumatic occlusion.

There are three degree of periodontitis: mild, moderate, and severe. Clinical manifestation has exacerbation and remission stage. Children mostly have mild degree of chronic generalized periodontitis.

Children with chronic generalized periodontitis complain on malodor, gingival itching and bleeding while teeth brushing and consuming hard food. Some patients don’t have complains.

Clinical examination reveals reddened, swollen gums that bleed easily upon light touch. There is a huge amount of supragingival and subgingival dental deposites, and periodontal pockets of different depth. The gingivae are tender to palpation which provokes serous excudation from pockets. Teeth mobility is a symptom of moderate and severe periodontitis. Shiller-Pysarev test is positive.

Roentgenologic symptoms of chronic generalized periodontitis are: wide periodontal ligament space, compact plate of alveolar bone destruction, decrease in height and insignificant osteoporosis of the interdental alveolar septae affects at least 3 teeth other than incisors and first molars.

Children with relapse of chronic generalized periodontitis complain on gingival pain, malodor, gingival itching and bleeding while teeth brushing and consuming hard food. Gums are brightly hyperemic, significantly swollen, and painfull, bleed on touching; dental deposites, abscesses are also found. Periodontal pockets of different depth with granulations are revealed. The gingivae are tender to palpation which provokes serous-purulent excudation from pockets. Teeth mobility is found in patients with moderate and severe periodontitis.

Roentgenologic symptoms of relapse of chronic generalized periodontitis are:

focal diffuse osteoporosis of the interdental alveolar septae affects a significant part of the alveolar bone without sclerosis signs. Signs of resorption activation: unclear and uneven bone contours in an area of the interdental alveolar septae and teeth roots, bone pockets.

Complain in patients are absent in remission period. Disease history has periodontitis treatment. Gums are sligthy pink, toughly covered teeth. The gingivae are not tender to palpation which doesn't provoke excudation from pockets. Periodontal pockets after plastic surgery and dental deposites are absent. Exposed roots may be found. Teeth don't have mobility and fix by splinting.

Roentgenologic symptoms of remission of chronic generalized periodontitis are: no osteoporosis bone is tough, with osteosclerosis, without resorption.

In addition to the catarrhal phenomena, proliferative and ulcerative necrotic changes can be observed in children with generalized periodontitis. Further progression of the inflammatory-destructive process leads to deepening of periodontal pockets and increase of teeth mobility.

Pathomorphological changes with the development of the inflammatory-destructive process are found in all periodontal tissues. This disease is characterized by proliferation of the gingival epithelium and its germination into the periodontal pockets. The gingival basis from connective tissue and the periodontium have diffuse infiltrates with a predominance of lymphoid and plasma cells, as well as tissue basophils. The bone has a lacunar type of resorption.

Cytological examination of the periodontal pockets identifies epithelial cells with cytoplasmic vacuolation and signs of necrosis and an increase in the number of neutrophilic leukocytes and mononuclear cells. The microflora in children with generalized periodontitis is diverse. Fusobacteria, spirochetes, trichomonads are dominated, cocci and fungi are also detected at cytological examination. The microflora of the periodontal pockets varies depending on their depth and the course of the inflammatory-destructive process (chronic or exxagerated).

Reoparodontography in patients with generalized periodontitis reveals microcirculation disorders: blood stasis in capillaries, reduction of blood flow in

periodontal tissues, change in the number and shape of functioning vessels. Differential diagnostic of generelized periodontitis is conducted with different forms of gingivitis that never has the alveolar bone destruction on a roenthgenogram.

Aggressive periodontitis

Aggressive periodontitis usually develops at a young age, it is typically present in the family history and has a fast progression. Specific bacterial flora plays a major role in the destructive, fast–progressing, aggressive form of periodontitis. Host responses are generally compromised, mostly due to genetic abnormalities. Bacteria produce virulence factors and they also have special properties: leukotoxins damage PMN cells and macrophages of the immune system, the production of endotoxins stimulates the production of catabolic cytokines, bacteriocins prevent the growth of normal bacterial flora, immunosuppressive factors inhibit the production of IgG and IgM, collagenase production damages connective tissue fibres and inhibits the chemotactic migration of neutrophils preventing them from reaching the defence line.

Changes in host immune response (local, systemic) play a major role in the development of destructive diseases. A certain degree of PMN function disorder is noticeable, which prevents them from migrating and performing antibacterial functions, but it only manifests in periodontal diseases, not in any other disease. A similar dysfunction is lower mixed lymphocyte response with increased B-cell response, increased pro-inflammatory cytokine production of PMN-s and macrophages. While systemic antibody response is pronounced in the localised form, antibody levels in the blood are relatively low in generalised aggressive periodontitis.

Prepubertate periodontitis

Prepubertal periodontitis is a rare form of periodontitis which occurs in young children during or after the eruption of primary teeth. All primary teeth are affected, but the permanent dentition may or may not be affected. Functional defects of the peripheral blood neutrophils and monocytes are seen. The disease occurs in generalized and localized form, which differ in their features and progression.

In case of generalized form severe acute inflammation of the gingiva is present. There is a rapid destruction of the gingiva and alveolar bone. Skin, upper respiratory

tract infections and otitis media are frequently seen. The generalized form is usually not amenable to treatment by antibiotics.

Localized form affects few teeth. Destruction of the gingiva and alveolar bone is not as rapid as in the generalized form. Usually there is no history of frequent infections. The disease is amenable to the treatment by root-curettage and antibiotic therapy.

Juvenile periodontitis

Juvenile periodontitis is an uncommon condition characterized by severe loss of attachment and destruction of alveolar bone around permanent teeth in otherwise healthy adolescent beginning at the onset of puberty. Recent evidence suggests that Actinobacillus actinomycetemcomitans, a gram negative facultative anaerobic rod, plays a dominant role in the disease process (Nonnenmacher C.et al., 2017). Juvenile periodontitis occurs in generalized and localized form.

Localized aggressive periodontitis

Recently titled in as desmodontosis and periodontosis localized aggressive periodontitis is a kind of juvenile periodontitis. Primary symptoms are rapid loss of connective tissue and alveolar bone. Patients are considered systemically healthy. In turn the secondary symptoms are: the amount of bacterial plaque seems inconsistent with the progression of the disease, presence of large amounts of microorganisms such as *Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis* in the subgingival biofilm, impaired phagocytosis, probably the occurrence of macrophages in a state of excessive activity, showing an increased secretion of certain inflammatory mediators such as prostaglandin-2 (PGE2) and interleukin-1 beta (IL-1β), the progression of attachment and bone loss may be spontaneously self-arresting. To identify localized aggressive periodontitis, additional criteria should be adopted which are as follows: onset occurs in puberty, very high levels of antibodies against pathogenic bacteria in periodontal tissues, loss of epithelial-connective tissue attachment in the interdental spaces including the first molars and/or central incisors, amount of bacterial plaque is inconsistent with the progression of the disease.

5.4. Idiopathic diseases with progressive lysis of periodontal tissues

Idiopathic diseases with progressive lysis of periodontal tissues are associated with endicrine diseases, immunologic and heredity changes, which lead to the teeth loss despite of the treatment. These changes are named as periodontal syndrome. Genetic disorders and acquired immunodeficiency disease have a major impact on loss of periodontal tissues by influencing periodontal inflammation. Diabetus mellitus influences the pathogenesis of periodontal diseases. X histiocytosis and neoplasms can result in loss of periodontal tissues independently of periodontitis.

Indeed, often times it was and is the dentist who first suspects a severe systemic disease and guides a patient to a specialist.

Idiopathic diseases with progressive lysis of periodontal tissues:

1. Periodontal syndromes associated with blood diseases: leukemia, neutropenia, and agranulocytosis.

2. X histiocytosis: eosinophilic granuloma, Hand-Schüller-Christian disease, Letterer-Siwe disease.

3. Periodontal syndromes associated with exchange disorders: Niemann-Pick disease, Gaucher disease, Papillon-Lefevre syndrome, and uncontrolled diabetes mellitus (I type).

4. Periodontal syndromes associated with congenital diseases: Down disease, acatalasia.

Idiopathic diseases with progressive lysis of periodontal tissues have following features:

- disease development in early child age, after primary teeth eruption;
- severe gingival inflammation, acute gingival lesions, early-onset and rapidly progressive alveolar bone loss;
- loss of dento-gingival attachment and periodontal pockets appearance;
- bone lysis of alveolar bone with teeth mobility and early loss of the primary and permanent teeth;
- foci not only in alveolar bone, but in other bones and hemopoiesis organs

(spleen, lymphatic nodes etc.).

Periodontal syndrome associated with blood diseases

These blood diseases are: leukemia, neutropenia, and agranulocytosis.

Leukaemia

Acute myeloblastic leukaemia

Leukaemia is actually the appearance of young, immature white blood cell formations showing signs of atypical signs. Currently, this condition is considered to be an oncogenic bone marrow disease. The disease itself can be divided into acute and chronic forms according to differentiations between groups of leukaemias arising from the pluripotential marrow stem cells.

The aetiology is unknown, however, there is a number of factors held responsible for causing this disease (genetic causes, exposure to radiation, chemical substances, pharmaceutical products, etc.). Apart from pathologic leukocytosis, anaemia and thrombocytopenia may also accompany this disease. Oral symptoms are more common in the acute form, but may occur in chronic cases as well.

Extensive, profound ulcers develop in the oral cavity and commissure of the lips of leukaemic patients due to fusospirochetal infections, candidiasis or other infectious causes. The most common oral symptom is gingival bleeding, however, purpuras, petechiae and ecchymoses due to thrombocytopenia may develop in other mucosal regions as well. Due to the risk of haemorrhagic diathesis, no tooth extractions or any other types of oral surgeries are recommended.

Oral symptoms may be the primary signs of this disease, thus, dentists should be quite aware of this condition. Gingival and mucosal ulcers are profound; the resorption of periodontium may cause the teeth to become loose. Gingival hyperplasia may accompany oral symptoms.

Acute leucosis has such stages: initial, full development, chronic stable, accelerated, terminal.

Initial period (1 – 3 months) is characterized in children by next signs: fatigue, poor apetite, low resistance to virus and child's infections, accerelation of chronic infection foci (chronic apical periodontitis, tonsillitis et al.).

Disruption of normal hematopoiesis causes anemia, thrombocytopenia and neutropenia, leading to signs/symptoms such as pallor, fatigue, petechiae, bruising, bleeding, and fever. Extramedullary infiltration of leukemic cells can cause lymphadenopathy, hepatomegaly and splenomegaly, which are usually asymptomatic. Bone pain due to periosteal involvement is present in about 25% of children with leukemia.

One of the early signs of the disease is gum bleeding. Moreover, the degree of manifestation of hemorrhagic syndrome may be different. In some patients, bleeding occurs from mechanical stimuli, in others ones - spontaneously. Also, a tooth pain may be appear in the early stages of the disease. This pain is explained by the specific metaplasia of the pulp and periodontium cells.

The mucosa of the lips and cheeks is pale, slightly swollen. Gums are pale, plump, swollen, easily peeled from the teeth. Ulcerative gingivitis has fast development. Necrosis and ulceration of the oral mucosa initially appears on gums and diffusely extend deeply into other parts of the mucosa without any pain.

Ulcerations in the oral cavity in leukemia patients develop without reactive inflammation against a background of a pale mucosa. Secondary infection provokes pain, followed difficulty swallowing and malodor. Traumatic lesions of the mucosa are accompanied by the deep erosions and ulcers.

Hemorrhagia and ulcerations usually develop against a background of gingival hyperplasia. Hypertrophic gingivitis represents by an excessive tissue growth that more expressed from oral surface. Gingival hyperplasia is usually combined with hypertrophy of the lymphoid tonsils: pharyngeal, lingual, pharyngial. The lymph nodes are enlarged slightly, they are soft and painless on palpation.

Remission occurs under the influence of cytostatic therapy. Complete and long remission (absence of manifestations of the leukemia for 6 months or more) is possible after the treatment of lymphoblastic leukemia.

Acute lymphoblastic leukaemia

Acute lymphoid leukaemia is particularly more common in childhood (80%) than myeloid or monocytic leukaemia, however, the lymphoid type is the least

common to cause any oral symptoms. The prognosis is better than it is for myeloid leukaemias (60% of these cases result in full recovery).

Chronic myeloid leukaemia

After the manifestation of the clinical symptoms, the disease will prospectively progress for another 3–6 years. The disease usually starts with a latent period.

Symptoms of chronic myeloid leukaemia are: general symptoms causing complaints usually include stabbing or painful swelling of the spleen; however, hepatomegaly may also occur. Headaches, fatigue and occasional high temperature may develop. Patients usually seek medical care due to gingival bleeding. Lymph node swelling is quite uncommon in myeloid leukaemia, while it is common in the lymphoid type of the disease. Oral symptoms are rarely primary, and these manifestations are not pathognomic, however, gingival hyperplasia and haemorrhage may persist and even profound, persistent ulcers or the loosening of the teeth may occur. Parotid swelling is more common than it is in patients suffering from acute leukaemia. Concomitant herpes zoster, pemphigus and candidiasis may also occur. The prognosis of chronic lymphoid leukaemia is better than that of the myeloid types of the disease.

Chronic lymphocytic leukaemia

This is the type of leukaemia, which shows the slowest rate of progression (with an overall survival of 6–10 years). A characteristic sign of chronic lymphocytic leukaemia is lymph node swelling at an early stage of the disease. Generalised lymphadenopathy is commonly accompanied by splenomegaly. This disease rarely occurs in children. Apart from these symptoms, other, non-specific dermal symptoms may occur as well, such as papulous eczema and the formation of vesicles and bullae. Skin infections, advanced periodontitis, ulcers and bleeding may occur within the oral cavity.

Neutropenia

Neutropenia (or granulocytopenia) is defined as a significant reduction in the absolute neutrophil count of circulating neutrophils in the blood. Decreased bone marrow production, increased destruction by immune mechanisms and increased

clearance by the reticuloendothelial system are among the causes of neutropenia. A few congenital syndromes, such as Fanconi, Rothmund-Thomson and Shwachman-Diamond syndromes, are accompanied by primary neutropenia. Secondary neutropenia caused by infections, drugs, malignancy, or hypersplenism.

Patients with permanent neutropenia experience frequent episodes of fever, pneumonitis, skin infections, and perianal and liver abscesses, usually beginning in the early infancy.

Recurrent, painful oral ulceration and prominent generalized periodontal destruction are common oral features of the neutrophil disorders, and they may even be the initial symptoms of the disease. The ulcers may affect any part of the oral mucosa including tongue and palate. Eruption of primary teeth is accompanied by ulcerative-necrotic gingivitis, which, transforms into generalized periodontitis at 2 years' age. Primary teeth loose due to the pathological teeth mobility. Permanent teeth eruption is accompanied by hypertrophic gingivitis. Next relapse with periodontitis progression occurs after permanent teeth eruption. Children loose the all permanent teeth to the age of 12 – 14 years.

Cyclic (periodic) neutropenia is a rare disease characterized by cyclical depression of the peripheral blood polymorphonuclear leukocyte (PMNL) count at 21-day intervals. Although cyclic neutropenia usually manifests initially in infancy or childhood, it may be a familial tendency. Neutropenia is called "severe" when neutrophils are below 0.5×10^9/L and "chronic" if the condition lasts more than three months, either intermittent or permanent. When there is no underlying disease to which the neutropenia can be attributed, chronic neutropenia is designated as chronic benign neutropenia.

Cyclic changes in blood and bone marrow are named as neutropenic episodes. These episodes take 4 – 5 days and repeat with individual time interval (usually 22 – 28 days). Neutropenic episodes accompanied by deterioration in their overall health, high temperature and intoxication development.

General and oral manifestations of cyclic neutropenia are same with permanent neutropenia. In addition, the submandibular and the cervical lymphatic nodes are

enlarged.

Agranulocytosis

In this condition granular white blood cells disappear from circulation. Mielotoxic type of agranulocytosis develops due to prohibition of granulocytes by ionosation radiation and certain drugs like sulphonamides, phenylbutazone. In blood not only granulocyte count falls, also count of thrombocytes, reticulocytes and lymphocytes drops. Anti-immune type of agranulocytosis develops due to antibodies against granulocytes production. Such medicines as analgesics, diuretics, phenothiazines and anticoagulants may play role gaptens. Moreover, heredity, tuberculosis, malaria, lupus erythematosus, rheumatoid arthritis and typhoid may also induce agranulocytosis.

Person will develop fever, rigor and extreme weakness. Skin becomes pale, jaundice may develop. Urinary tract infection may develop, rectal ulcerations are common. Severe leukopenia develops and neutrophil count comes down to 0-2%.

Throat has a grey or a white plaque; there are ulcerations on tonsils and buccal mucosa. Ulcers may start bleeding. Halitosis and excessive salivation is noted. Opportunistic fungal infections may develop. Agranulocytic angina develops showing necrotizing ulceration over gingiva, soft palate and cheeks. Streptococcal sore throat also produces exudative membrane and becomes extremely painful. Pharynx may show edema and reddening.

Significant dystrophic-inflammatory changes develop in all periodontal tissues, teeth become mobile subsequently fall out.

The main differences in periodontal lesions accompained with agranulocytosis are absence of pus (in spite of the presence of deep periodontal pockets) and predominance of necrotic changes without visible local inflammatory response.

Histiocytosis X

Histiocytosis is a group of diseases, which have unknown etiology, accompanied metabolic disorders and proliferation of Langerhans' cells (immune surveillance cells) in skin, mucosa, bone marrow, and lymph nodes.

Clinical presentation characterizes a broad spectrum, typically divided into three subsets, as follows: unifocal or multifocal chronic disease of bone (eosinophilic granuloma), widely disseminated chronic disease of bone and soft tissue (Hand-Schüller-Christian disease), acute, disseminated disease with bone marrow involvement (Letterer-Siwe disease).

Histological structure of X histiocytosis ia represented by infiltrate of mononuclear cells, often with clefted nuclei. Often accompanied by a variety of other cell types, including eosinophils, lymphocytes, giant and plasma cells.

Letterer-Siwe disease

Letterer-Siwe disease represents the acute, often fulminating, form of the histiocytosis. The disease occurs in infants, usually before the age of three years. Cutaneous involvement is common and frequently the first lesions are manifested in the skin. The liver, spleen, lymph nodes, lungs and thymus are additional sites of involvement and of these, the liver appears to be the most common site affected. Oral lesions usually consist of ulceration of the oral mucosa, and diffuse destruction of the bony mandible and maxilla with loosening and premature loss of related teeth.

It manifests with the characteristic triad of exophthalmos, osteolytic lesions of the cranium and diabetes insipidus. Other manifestations, petechiae, purpura, ulcerations, lesions mimicking seborrheic dermatitis, pulmonary dysfunction, tachypnea, dyspnea and cyanosis, may appear. This clinical form may imitate cystic lesions, leukemia, lymphoma, metastasis, meningioma, and congenital processes such as encephalocele.

Alveolar bone lesions form the basis for all the associated periodontal involvement in these patients. As new osteolytic areas develop, accompanying gingival ulceration and inflammation are observed, such that all the quadrants of the oral cavity are affected to a greater or lesser degree, even though the process began initially in only one quadrant. As a consequence of the alveolar bone loss, these patients manifest gingival inflammation, ulceration, destruction of the keratinized gingiva, gingival recession, periodontal pockets and bleeding of the oral soft tissues, associated with pain and even swelling. As a result of this loss of bone support the

teeth begin to progressively move giving rise to the characteristic ‘floating teeth’, completely surrounded by a radiolucent defect accompanied by dental displacement, odontalgia and on occasions cervical adenopathies. This excessive mobility gives rise to the inevitable premature loss of these teeth.

The different types of lesions produced by LCH in the maxilla and mandible are described according to their radiographic characteristics (Madrigal-Martínez-Pereda et al., 2009):

- Solitary intra-bony lesions: localized outside the alveolar process, these are the most frequent in the initial phases.

- Multiple alveolar lesions: normally present with well- defined though not corticalized margins.

- ‘Scooped-out’ alveolar lesions: formed by bone destruction beginning below the alveolar crest, either at furcal level or at half the tooth root height and normally a part of the coronal portion of the bone crest remains intact on the mesial and/or distal margin of the damaged bone. This form of intra-bony destruction is not seen in periodontal disease, and may therefore be useful in a differential diagnosis.

- Alveolar lesions with bone sclerosis: common in inflammatory lesions of the jaws, the fact that sclerosis appears in alveolar lesions in LCH may be explained by the communication of these with the oral cavity with added infection. Thus, intra-bony lesions do not present sclerosis as they do not communicate with the oral cavity.
- Alveolar lesions with bone neoformation: formation of new bone in lesions classified as intra-bony is observed in a high number of cases. This is a relevant characteristic when differentiating LCH lesions from those of periodontal disease.

Mucosal lesions are ulcerated, ovoid or round lesions, with erythematous, inflamed borders, painful on palpation. They are localized principally in the buccal mucosa and at the back of the vestibule. They are associated with cutaneous lesions such as the typical eczematoid rash, that may be confused with a sebaceous dermatitis. Occasionally subcutaneous nodules present, therefore the initial evaluation of the patient should also include a meticulous skin examination. Some unusual cases of oral soft tissue lesions in the absence of bone lesions have been

described. The mucosal lesions are usually accompanied by enlargement of the lymph nodes which also reflects the degree of histiocytic infiltration.

When examined with the electron microscope, the cellular composition of the granuloma appears to be heterogenous. The predominant cells are histiocytes with many eosinophilic cells present, a few neutrophils with occasional basophilic metamyelocytes and true macrophages interspersed amongst them

Blood test shows anemia, eosinofilia, trombocytosis and increase of erthytrocyte sedimentation rate.

Diagnosis is based on clinical dates, roethgenelogic examination of flat bones, and biopsy from lesions. Letterer-Siwe disease has difference from generalized periodontitis that occurs in early childhood and affects skeleton bones and jaws.

Hand-Schüller-Christian disease

Chronic dissemenoid form of histiocytosis, disorder of the reticuloendothelial system.

Histologically, Hand-Schüller-Christian disease manifests four stages during its progression:

• A proliferative histiocytic phase, with collections of eosinophilic leukocytes scattered throughout the sheets of histiocytes

• A vascular granulomatous phase, with persistence of histiocytes and eosinophils; sometimes with aggregation of lipid-laden (cholesterol) macrophages

• A diffuse xanthomatous phase, with an abundance of ‘foam cells’

• A fibrous or healing phase.

The pathogenesis of Hand-Schüller-Christian disease is unknown. It may be caused due to hypersensitive reaction to unknown antigen with stimulation of histiocytes-macrophage system. Deficiency of suppressor lymphocytes, altered immunoglobulin’s autoantibodies and structural changes to thymus in all the advanced forms have been found in these patients.

Hand-Schüller-Christian disease usually affects children between 1 and 15 years old with a peak incidence between 5 and 10 years of age.

The disease has a slow course, with relapse, and three periods of its development: initial, significant changes and remission (for 3 – 4 years).

The first signs of disease are: fatigue, loss of appetite and body weight, insomnia, which are consider as usual children infectious diseases. Later papillae and spots appear on the skin of hear part of head. One third of patients have mucocutaneous lesions, most frequently infiltrated nodules and ulcerated plaques, especially in the mouth, axillae, and anogenital region. Other cutaneous manifestations include extensive coalescing, scaling, or crusted papules.

Early oral signs and symptoms, which can guide us to the diagnosis, include premature dental avulsions. The marginal gingivae can present with severe recession or hyperplasic gingival enlargement at the gingival margin. Oral manifestations include sore mouth, halitosis, gingivitis, unpleasant taste, loose teeth, and failure of extracted tooth sockets to heal. Loss of supporting bone mimics advanced periodontal disease.

Patients with Hand-Schüller-Christian syndrome (which occurs in 25% of patients with multifocal LCH) often present with recurrent episodes of otitis media and mastoiditis or with polyuria and polydipsia. The disease includes diabetes insipidus, exophthalmos, and bony defects, particularly of the cranium. Lesions may affect a variety of systems, including liver (20%), spleen (30%), and lymph nodes (50%). Systemic involvement of this disease also includes hepatosplenomegaly, and gastrointestinal tract, renal and pulmonary involvement. Central nervous system involvement includes convulsions, increased intracranial pressure, focal neurological deficits, mental retardation, hearing disturbance, and tremors. Osteolytic lesions of the long bones can lead to spontaneous fractures.

The radiographic appearance depends on the state of the lesions, varying from a unique central well-delineated lesion to a multilocullar lesion, sometimes with little sclerotic reaction due periosteal compromise. If the lesion perforates one cortical plate, it will extend to the adjacent soft tissues and will appear as a tumoral mass. The occurrence of pathological fractures of the mandible is reported with large lesions.

Recommended baseline diagnostic evaluations for disease include complete blood count with differential, reticulocyte count, erythrocyte sedimentation rate, direct and indirect Coombs test, and immunoglobulin levels. In case of anemia, leukopenia, or thrombocytopenia, a bone marrow aspirate is indicated.

The presence of periodontal syndrome and multiple osseous lesions, associated diabetes insipidus, desquamative cutaneous lesions, deficits in the cellular immune responses and the histopathology all pointed to a definitive diagnosis of Hand-Schuller-Christian syndrome.

Eosinophilic granuloma

Eosinophilic granuloma is the most benign disorders of the triad commonly known as histocytosis X. In 90% of the reported cases it appears in children under the age of ten.

The lesion may occur in the jaw and overlying soft tissues of the mouth although the skull and mandible are common regions of involvement, the femur, ribs, humerus, and other bones may also be affected.

Eosinophilic granuloma be considered a separate and distinct entity with either a unifocal or a multifocal presentation. Unifocal type is the most common and most benign lesion, more common in males and usually involves long or flat bones. The patients chief complaints are frequently bone pain, swelling and occasionally the lesions can cause pathologic fractures. The most common oral findings are soreness, swelling, ulceration, necrosis of the gingival tissues, and destruction of alveolar bone with teeth mobility and teeth loss.

There are three forms of eosinophilic granuloma in oral cavity: focal, diffuse, and generalized.

Focal lesion may be symptomless and discovered only as an incidental radiological finding. The lesion may occur in the mandible and overlying soft tissues of the oral cavity, so that the differential diagnosis between eosinophilic granuloma and other oral diseases (particularly periodontal disease) becomes imperative. The lesions are destructive and are well demarcated and roughly round or oval in outline. A large round swelling over the mandible is tender, firm, and attached to the

underlying structures. Oral examination revealed ulceroproliferative growth and multiple loose teeth around the gingivae. Microscopic examination reveals ulceration of the mucosa accompanied by extensive aggregates of histiocytes showing a reniform nucleus, nuclear grooves and eosinophilic cytoplasm. CT scan reveals a lytic lesion extending into the alveolar part of the mandible with intraoral soft tissue extension.

Diffuse lesion develops in the alveolar bone, expending the body and the ramus of mandible. Clinical course of the diffuse lesion has initial period amd period of significant changes.

In the initial period, patients complain of itching and pain in the intact teeth, redness, bleeding and ulceration of the gums. There are signs of localized lesion: symptomatic gingivitis (often ulcerative-necrotic), periodontal pockets, root exposing and teeth mobility. The X-ray exhibits bone lesions of the interdental septae.

Period of significant changes is characterized by an increase in clinical manifestations of periodontitis, which leads to loss of teeth, after which painful holes do not heal for a long time. On X-ray this period is characterized by two different pictures: bounded foci of bone destruction that have irregular shape with fissured fuzzy borders and localize in different parts of the alveolar process and the jaw body or diffuse lesions of the jaw in the form of several foci of bone destruction, merging with each other.

Generalized form of the disease is characterized by not only lesions of jaws but also other bones of the skeleton. In the oral cavity, the classic signs of generalized lesion are: symptomatic ulcerative-necrotic gingivitis, periodontal pockets with granulations and no pus from them, and pathological tooth mobility.

On X-ray this period is characterized by diffuse lesion of the jaw: few foci of bone destruction, which merging with each other, or bounded foci of bone destruction that have irregular shape with fissured fuzzy borders and localized in different parts of the alveolar process and the jaw body.

In infants, eosinophilic granuloma manifests as areas of a gray-green necrosis of the gums. After removal of the necrotized tissues, a painful erosion or ulcerative

surface is exposed which bleeds and doesn’t heal for a long time. The premature eruption of primary teeth happens later and they loose due to the deep pathological teeth mobility. With the progression of the disease, young children may have "sequestration" of the germs of permanent teeth.

Histologically, eosinofilic granuloma presents as areas of histiocytic proliferation with focal collection of eosinophils. Loose connective tissue stroma showed numerous suspended neutrophils and small mature lymphocytes.

Blood examination may reveal elevation of leukocytes and erythrocyte sedimentation rate.

Most arise in childhood; eosinophilic granuloma often arises in adolescents and adults. Jaw lesions noted in up to 20% of cases with tenderness, loose teeth (focal to segmental), gingival inflammation, and friability. Radiographic findings: bone lesions often punched out, sharply circumscribed, “floating teeth” appearance with alveolar bone involvement, skeletal survey should be performed to rule out multiple bone involvement. Eosinophilic granuloma does not lead to malignant transformation. If it expands elsewhere but bones then is called Hand-Schuller-Christian disease. The prognosis depends on the age of diagnosis and the number of foci.

Periodontal syndrome associated with exchange disorders

Periodontal syndromes associated with exchange disorders are: Niemann-Pick disease, Gaucher disease, Papillon-Lefevre syndrome, and uncontrolled diabetes mellitus (I type).

Niemann-Pick disease

Niemann-Pick disease is a sphingolipidosis, an inherited disorder of metabolism, caused by deficient sphingomyelinase activity, resulting in accumulation of sphingomyelin (ceramide phosphorylcholine) in reticuloendothelial cells.

Niemann-Pick disease inheritance is autosomal recessive and 2 types, A and B, exist. Type C Niemann-Pick disease is an unrelated enzymatic defect involving abnormal cholesterol storage. Type A is characterized by hepatosplenomegaly, failure to thrive, and rapidly progressive neurodegeneration. Death occurs by age 2

or 3 yr. Type B is more variable clinically than type A. Hepatosplenomegaly and lymphadenopathy may occur. Pancytopenia is common. Most patients with type B have little or no neurologic involvement and survive into adulthood.

Also base symptoms of Niemann-Pick disease: failure to gain weight, jundice, generelized lymphadenitis, disoders of CNS, and severe periodontal syndrome. Blood test shows pancytopenia and coagulopathy. Bones have osteoporosis foci.

Diagnosis of Niemann-Pick disease can be confirmed by DNA analysis and/or sphingomyelinase assay on WBCs and can be made prenatally by using amniocentesis or chorionic villus sampling.

Chitotriosidase is a surrogate marker for diagnosis of Gaucher and Niemann-Pick disease and is significantly increased in these disorders. In case the enzyme levels are inconclusive, the further mutation analysis can give a confirmatory diagnosis. Other biomarker as plasma lysosphingomyelin which can of potential use in diagnosis of Niemann-Pick type B and also type C.

Prognosis is unfavorable, children usually don’t live more than 5 years.

Gaucher disease

An autosomal recessive disorder resulting from pathogenic mutations in the gene that encodes glucocerebrosidase enzyme, resulting in an accumulation of glucocerebrosides in the phagocytes and central nervous system. The glycosylceramide is converted into glycosylsphingosine, with an increase in its serum levels.

There are three types of the Gaucher disease (GD): I, II, and III.

GD type I may present symptomatically at any age or sometimes remain undetected throughout life. At worst, GD is associated with massive hepatosplenomegaly, anemia, thrombocytopenia, and hemostatic defects. Bone manifestations, including pain, osteonecrosis, mineral loss, pathological fractures, and joint deformities, cause disability and impair quality of life.

Type II - acute neuronopathic Gaucher disease, manifests early at infancy and is characterized by intense CNS involvement with life expectancy less than two years. Type III - chronic neuronopathic Gaucher disease, is characterized by less

severe CNS involvement, splanchnic involvement and life expectancy that reaches adulthood.

The most prevalent finding was gross widening of marrow spaces; frank radiolucencies, endosteal scalloping, cortical thinning, root resorption, and inferior displacement of the mandibular canal or effacement of its cortices were also seen. Delayed eruption of permanent teeth is examined. Significant osteopenia, loss of the trabecular architecture of the bone, along with pseudocysts or honeycomb osseous abnormalities were observed in all cases.

Thrombocytopenia or pancytopenia are found in the blood test. Gaucher cells are detected in all tissues (bone marrow, spleen) of the affected individuals. Cytoplasm of theses cells includes hemosiderin and glucocerebrosid. Prognosis is favorable.

Papillon-Lefèvre syndrome

Papillon-Lefèvre syndrome is a rare dermatosis which etiology is still obscure; however, microbiologic, immunologic, and genetic factors are linked to its development. It is characterized by a hyperkeratosis of soles of feet and palms of the hands and severe, aggressive, and prepubertal periodontitis, leading to premature loss in both deciduous and permanent dentitions.

After the normally shaped deciduous teeth erupt, a severe inflammation of the gingiva appears, with periodontitis and pocket formation leading to a complete loss of teeth by the age of 4 to 5 years. The permanent teeth appear at normal time and are as a rule lost in the same way. The periodontal breakdown is usually a consequence of an inflammatory destruction (periodontitis) while a poor oral hygiene and the subsequent accumulation of dental bacterial plaque are contributing factors. Periodontal breakdown in a child or adolescent can be slowned down by maintaining good oral hygiene.

Cutaneous symptoms are characterized by palmar and plantar hyperkeratosis, which is normally accompanied by a moderate hyperhidrosis. Hyperkeratosis often extends to dorsal aspects of the hands and feet (transgression) as well as to the region of the Achilles tendon. Additionally, sharply delineated psoriasis-like plaques may be

present on elbows, knees, dorsal aspects of the metacarpophalangeal joints, but also elsewhere. The red colored plaques are covered by a hyperkeratotic stratum corneum or parakeratotic scales. The hair and nails are usually normal, but sparse hair was noted.

X-rays of the maxilla and the mandible show severe atrophy of the alveolar bone. The main reason is the early loss of teeth, which prevents normal jaw development. Further reason for atrophy are inflammation that incites osteolytic processes in the bone and the use of dentures.

Laboratory tests show decreased neutrophil phagocytosis. Impaired reactivity to T- and B- cell mitogens with only minimal changes in monocyte function might account for prominent gingival and cutaneous infections.

Microbiological findings show a close association between Actinobacillus actinomycetemcomitans and the periodontal disease associated with the syndrome. A actinomycetemcomitans by itself is not sufficient for the expression of periodontal disease.

Periodontal syndrome assosoated with uncontrolled diabetes mellitus (I type)

Oral sundroms and conditions associated with uncontrolled diabetes mellitus: gingivitis, periodontitis, caries, tooth loss, oral candidiasis, oral mucosal lesions such as traumatic ulcers and irritation, fibroma, impaired wound healing, xerostomia, salivary gland hypofunction, sialosis, burning mouth sensations, impairment of taste. Consensus report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions claimed that diabetes-associated periodontitis should not be regarded as a distinct diagnosis, but diabetes should be recognized as an important modifying factor and included in a clinical diagnosis of periodontitis as a descriptor.

Children and adolescents with poorly controlled type 1 diabetes appear to have more advanced gingivitis as compared to non-diabetics, despite comparable plaque indices and level of oral hygiene. The extent of gingival bleeding is most prominent in patients with newly diagnosed type 1 diabetes, and improvement of the metabolic control by intensified insulin treatment reduces the extent of gingival bleeding.

Furthermore, some studies have found that children and adolescents with type 1 diabetes have significantly deeper periodontal pockets and more extensive attachment loss than age-matched controls.

Periodontal syndromes associated with congenital diseases

Down disease and acatalasia are the most often congenital diseases acatalasia associated with periodontal syndrome.

Down syndrome

Down syndrome (DS) is associated with a range of congenital malformations, altered patterns of growth, variable intellectual disability as well as increased risk of certain chronic diseases. Down syndrome is associated with an extra chromosome of the twenty first group, for a total of 47 chromosomes.

Facial characteristics of Down syndrome are: midface hypoplasia, reduced nasal protrusion, ear malformations, epicanthal folds with slanting almond-shaped eyes (narrow palpebral tissue slanting toward the midline). The majority of persons with Down syndrome exhibit lack of supraorbital ridges and hypotelerism, nasal septum or nasal conchae deviations.

The prevalence of macroglossia, fissured tongue and protruding tongue due to the relative forward position of the mandible and open mouth is a common finding. It is possible to see alveolar bone loss in persons with Down syndrome at 6-16 years old. Additionally, the periodontal destruction occurs at a more rapid rate. These patients have a high incidence of necrotizing ulcerative gingivitis (NUG). The clinical picture of NUG in persons with Down syndrome differs from the usual symptoms in that fetid breath and exquisite pain are rarely reported. Oral conditions associated with an increase in NUG in this population include crowded dentition, traumatic occlusion, peg shaped anterior teeth, lack of root resorption in primary teeth, and incidence of high frenum attachment. Periodontal disease is one such disease which presents early and severely for those with DS. Advances in healthcare means that tooth loss from chronic periodontal disease, exacerbated by the further deterioration in self-care seen in the likely incidence of the onset of dementia.

Acatalasia

Takahara (1952) proposed that the patients with progressive gangrene of maxilla had the erythrocytes with lacked catalase activity, and this disease (acatalasia) was inherited as an autosomal recessive trait. The lesions appeared as soon as deciduous and permanent teeth erupted. Where teeth are absent, the alveolar mucosa was normal; alter extractions, wound sockets healed normally.

Patients complain of loose teeth, absence of several deciduous and permanent teeth due to extractions, and severe halitosis. Examination reveals necrotic ulcerations of the interdental papillae, and tooth mobility. Deep periodontal pockets are noted around teeth. Marked alveolar bone destruction is seen on dental radiographs in areas where gingival lesions are present.

Removal of dental plaque limited the progression of the gingival lesions, suggest that gingival damage results from a lack of catalase activity in gingiva which permits proliferation of bacteria in dental plaque and gingival inflammation.

Prognosis is favorable because periodontal therapy achieves a good status.

5.5. Periodontal neoplasmae

Benign periodontal neoplasmae: fibroma, ginigival fibromatosis, lipoma, epulis.

Fibroma

Fibroma is a reacting hyperplasia of falcon's tissue rather than a neoplasm. It also known as "irritation fibroma", "traumatic fibroma" or "fibrous no duel". It growths slowly. It appear as a well circumscribed smooth surfaced nodule about 1-2 cm in diameter. It may be firm and nodular and might be soft in nature. Variations: giant cell fibroma and peripheral ossifying fibroma.

Giant cell fibroma develops from fibrous connective tissue, mandibular or maxillary gingiva is mostly affected. This lesion is pedunculated. It has papillary or warty surface. Some may be painless, smooth nodular growth, histologically represents by multiple multinucleated giant cells.

If fibroma is clutched between teeth it may bleed. Symmetrical fibromae are placed symmetrically – on the vestibular and oral surfaces of maxilla or mandible. These fibromae are tight, elongated, have slow growth and cause difficulties during eating.

Gingival fibromatosis

It is more common in adolescents, mainly in girls, in puberty or young children with Down disease. Occasionally fibromatosis occurs during drug treatment.

Familial fibromatosis has hereditary basis—autosomal dominant. Patients often have combination of epilepsy + mental deficiency +angiofibroma.

Fibromatosis may present as focal, diffuse and total growth of gingivae. The diffuse form of the disease affects ginigiva of whole alveolar process. Total fibromatosis is characterized by the complete destruction of the alveolar processes and their replacement by fibrous enlargment that facilitates deformation in the lower part of the face and difficulty in the lips closure.

Fibromatosis characterized by slowly progressive, non hemorrhagic enlargement of the maxillary and mandibular gingiva or may be localized to either jaw. Enlargement may be bilateral. Although the enlargement is not always symmetrical, the name was given as focal gingival fibromatosis. Gingiva is grossly enlarged and pink in color, firm and lathery in consistency with a minutely rebelled surface. Secondary inflammatory changes may be present. Teeth may be delayed in emerging and speech may be Impaired. Mastication may become painful, if enlargement extends to the occlusal surface.

There are three degree of fibromatosis: in the I degree gums are covered up to 1/3 tooth crown, in the II degree gums - up to 2/3 tooth crown, in the III degree – whole crown.

Gum hypertrophy is viewed on the the vestibular and lingual (palatal) surfaces. The growth of the gingival papillae on the oral surface is more significant, papillae are round and form a hill relief surface with folds. On the lingual side, hyperthropy doesn’t change the relief of the gums. Fibromatous growth has a lobular, papillomatous appearance or a continuous growth of pale pink or pink gums. On

palpation, the growth is dense, painless, there is no bleeding on palpation.

Ginigival fibromatosis in primary bite period causes malpositions of permanent teeth. Prognosis of disease is favorable, but relapses are possible.

Lipoma

It is a tumor of adipose tissue composed of mature fat cells. These are painless lesions with cystic feeling, yellowish in color with smooth surface and without surrounding capsule. It is quite rare, usually at a young age. It is characterized by slow growth. The gums usually have a soft lipoma, fixed, tightly soldered to the underlying tissues.

Epulis

The term "epulis" is applied to any lump arising from the gingiva. Epulides are localized gingival swellings and rarely true neoplasms, metastatic carcinomas or other tumours. The fibrous epulis resembles a fibroepithelial polyp, but also usually has an inflammatory component. Epulis fissuratum may be induced by trauma from a denture flange

There three forms of epulis: angiomatosus, fibrous, and giant-cell. Angiomatous epulid has mild consistency, reddish or bluish has a smooth (sometimes lobular) surface, easily bleeds on touch. A pale pink fibrous epulid has a tough consistency, and a smooth surface.

Initially the alveolar bone is not changed. As the tumor grows into the periodontium and bone, foci of destruction in the alveolar process appear, especially in the area of the interveolar septae.

Fibromatous epulid is represented by a mature fibrous connective tissue with separate areas of bone. Ulcerative fibrous epulid with granulation growth is named granulomatous.

The angiomatous epulid is characterized by a significant growth of the blood vessels with cavities resembling the cavernous angioma and the areas of calcification.

The giant cell epulid is a connective tissue neoplasma with a large amount of giant cells that without atypical growth. The giant cell epulid has accumulations of hemosiderin (the result of hemorrhage) and some areas of ossification.

Chapter 6
TREATMENT OF PERIODONTAL DISEASES

Therapy of periodontal diseases in children should be comprehensive and strictly individualized, taking into account the etiology, pathogenesis, clinical course and pathomorphological changes in periodontal tissues and is conducted under the control of clinical, paraclinical and laboratory methods of examination. In general, it should be, first of all, etiotropic, then pathogenetic and, finally, symptomatic.

The main objectives of therapeutic measures are:

- removal of the most probable pathological factors;
- removal of local irritants;
- elimination of inflammation;
- dystrophy arrest;
- restoration of impaired periodontal tissue function;
- stimulation of the regeneration;
- preservation of the dentition as a single functional dynamic system.

Periodontal treatment consists of local and general activities.

If the child has somatic or chronic systemic diseases, the general treatment plan should be agreed with a pediatrician or specialist.

As a rule, the dentist provides comprehensive local treatment, which includes:

- elimination of unfavorable factors (removal of dental plaque, sanitation of the oral cavity);
- antiseptic and anti-inflammatory therapy;
- physiotherapy;
- surgical methods of treatment aimed at the correction of anomalies of the oral cavity;
- orthodontic methods of treatment aimed at eliminating function disorders, regulating jaw growth, eliminating bad habits;
- hygienic dental education with control;

- recommendations for the use of the best hygiene products.

Also nutrition is important, especially the proper ratio of proteins, fats, carbohydrates, minerals and vitamins in food.

6.1. Papillitis

Papillitis treatment depends on etiology, pathogenesis, character and sevetity of disease and same with gingivitis treatment.

6.2. Gingivitis

Catarrhal gingivitis

Treatment of catarrhal gingivitis is revealing and followed removal etiologic and pathogenetic factors.

Chomenko L.O. et al. offered such scheme of catarrhal gingivitis in children:

1. Professional oral hygiene.

2. Antibacterial therapy (antiseptic medicines, nytroimidasole medicines).

3. Anti-inflammation therapy (medicines that have plant origin, non-steroid anti-inflammatory drugs, inhibitors of proteolytic enzymes).

4. Physiotherapy (using different physical factors with treatment purpose).

5. Hygienic education, hygiene control and recommendations regarding oral hygiene products.

6. Removal local traumatic factors (caries treatment, periodontal plastic surgery and orthodontic treatment).

7. Maintenance therapy every 3-6 months (in a case of persistent catarrhal generalized gingivitis).

8. Specialized counsiling and treatment in pediatrician and other specialists (in a case of persistent catarrhal generalized gingivitis).

Professional hygiene in patients with chronical catarrhal gingivitis is conducted every 6 months.

Professional hygiene includes:

– professional teeth cleaning (depends on a child`s age and kind of dental deposites);

– hygienic education;

– choice and prescription oral hygiene products.

Professional removal of soft plaque in patients with catarrhal gingivitis includes the following procedures:

1. oral hygiene motivation, instruction
2. antiseptic rinsing
3. if it is required local anaesthesia is conducted
4. professional scaling
5. finish polishing of surfaces
6. fluoridation of surfaces

10% lidocaine spray or gel «Camistad» can be used for the local anaesthesia. Rinsing with chlorine-hexidine-digluconate (CHX) 0.2% solution (including the throat) or plant antiseptic is recommended before the mechanical treatment.

Scaling of calculus in patients with catarrhal gingivitis includes the following procedures:

1. oral hygiene motivation, instruction
2. antiseptic rinsing
3. if it is required local anaesthesia is conducted
4. supra-gingival scaling
5. finish polishing of surfaces
6. fluoridation of surfaces
7. correction of anatomic deficiencies

Manual instruments for removing bacterial deposits

Manual instruments are built up of three parts: handle, neck and blade. These instruments can be classified and distinguished from each other according to blade characteristics and its relation to the terminal neck. Manual instruments for plaque and calculus removal are divided into two main groups: scalers and curettes.

Nowadays the most commonly used scalers are sickle scalers and hoe scalers. Files and chisels are not used anymore, and they are not even produced by manufacturers. Sickle scalers can be distinguished from curettes on the basis of cross-

section (triangle) and ending (tip). Similarly, to universal curettes, the facial surface and the terminal neck close a 90º angle and they have two working edges.

Sickle scalers are used supra-gingivally for scaling and for removing soft tissue remnants during gingivectomy. Their sharp, narrow blade, especially that of the mini version, can be inserted in the narrowest interdental spaces. Hoe is mainly used for subgingival scaling. Sickle and hoe scalers are activated with a pulling movement.

Curettes are divided into universal and Gracey types. Their cross-section is a half-elliptical semicircle and they end in a rounded tip. Manufacturers keep developing their products, producing different blade sizes: normal, micro and macro blades are available for different indications. The neck of the instrument is either rigid or flexible. Different blade types of Gracey 14 curette: normal, atraumatic, mini profile, nano profile (Deppeler).

Several types of universal curettes exist: some consist of one single instrument, while others combine a set of instruments. They all have two working edges and end in a rounded tip. Their cross section is a half-elliptical semicircle, the terminal neck and the facial surface close a 90° angle, and the blade runs straight from the facial aspect. Instrument sets are based on tooth types (Columbia set), or jaw and tooth types (Langer set).

Universal curettes are capable of removing large deposits, but they also remove more tooth substance than Gracey curettes. They can be used for supra- and sub-gingival instrumentation and for removing granulation tissue and debriding root surface during surgery.

Gracey curettes are also called surface-specific instruments because certain instruments work well on one tooth type only or on the tooth surface. Due to blade angulation, it removes less tooth structure and leaves a smooth surface behind. This is why they are also called finishing curettes. The original set consisted of 7 instruments, to which 2 additional instruments have been added, to provide better access to the mesial and distal surfaces of second and third molars. The first 5 instruments are angled two dimensionally at the neck, the other 4 instruments are angled three dimensionally.

They only have one working edge, which is faced down. The facial surface and the neck are angled at 70°. The blade is curved and the edge is convex with a rounded tip. The Gracey minimum set contains 4 instruments (Gracey 5-6, 7-8, 11-12, 13-14). This is sufficient to treat all tooth surfaces.

Gracey instruments are indicated for root instrumentation, removing smaller deposits of calculus and root planing in both conservative and surgical therapy.

Scalers and curettes need sharpening prior to every use. This can be done manually with Arkansas stone or with the help of a machine. The point of sharpening is to remove an even layer from one of the surfaces of the working edge. To perform correct sharpening, one must know the angles of the instruments well to be able to reproduce it. Curvature of the blades must also be recognized and followed when sharpening, especially with Gracey instruments. Sharpening must always be performed after disinfecting, which is followed again by disinfecting and sterilisation.

CORONAL POLISHING OF THE TEETH

Tooth polishing is the smoothing of all exposed tooth surfaces with a rubber cup, a brush, or by an air polisher driven by a slow-speed hand piece or water unit. Traditionally, it follows scaling and root planning.

The primary objective of polishing is to remove extrinsic stain and supragingival plaque. The rationale for this procedure includes improving the appearance of dentition, demonstration of standard of oral cleanliness for the patient to attain on a daily basis, and motivating the patient to improve plaque control, as well as the belief that the outcome of high-quality periodontal service should be a plaque free mouth.

Rubber cups and polishing brushes are used in the hand-piece (slow speed, without water spray). The use of the brush should be confined to the crown to avoid injury to the gingiva and cementum. An ideal prophylactic paste should combine good cleaning ability with simultaneous polishing (morphological smoothing of dentin and enamel surfaces). In addition, the agent should cause minimal abrasion and surface roughness of dental hard tissues.

The abrasives are of various particle sizes and are categorised as fine (1–45 μm), medium (74–105 μm) or coarse-grade (74–177 μm) prophylaxis pastes. They are normally colour-coded for convenience. These abrasives can increase the roughness of the dental hard tissues as well as the restoration surfaces but are necessary for effective plaque and stain removal. Some pastes also contain sodium fluoride or stannous fluoride as a desensitising agent.

Tooth polishing strips are used for polishing the proximal surfaces of teeth that are inaccessible to other polishing instruments.

- thin flexible backing with aluminium oxide coating
- centre-gapped strip with two abrasive grades per strip which allows interproximal access
- different types (different colours mark different size of granules).

Air polishing devices

An automated polishing method, sometimes called air-powered polishing or jet polishing has been available for the past three decades. This method uses finely powdered sodium bicarbonate as an abrasive, which is delivered under pressure through a narrow nozzle of a specialised handpiece surrounded by a mist of warm water. The resulting aerosol is propelled against the tooth surface to remove plaque and extrinsic stain by mechanical abrasion. The advantage is a remarkably thorough process of deposit removal accomplished in a short time, although the long-term effect of it on oral health status has not been researched yet. The abrasive is quite fine and despite the pressure of application, minimal loss of enamel is resulted when used according to manufacturer directions. However, significant loss of root structure may occur even with careful use. Air-powder polishing removes less tooth structure than manual instruments.

The disadvantages of air-powder polishing include the creation of a significant aerosol, epithelial abrasion when applied too close to the gingival margin, a salty taste, and a mild stinging sensation in other areas of the mouth caused by the deflected spray.

Air-powder polishing is contraindicated for patients with respiratory diseases such as chronic obstructive pulmonary disease, asthma and emphysema, for patients who wear contact lenses or are on a low sodium diet, on composite restorations, around the margin of cast restorations, and on demineralised enamel.

Using the proper technique with this polishing method minimises the side effects and prevents patient injury. The handpiece should be held with a modified pen grasp but no fulcrum is needed because no pressure is required during a stroke. The tip is positioned 4 to 5 mm away from the tooth surface and is kept in constant motion using circular, brush-like strokes directed at the middle third of the anatomical crown. Avoid long, continuous use, and allow the patient to rinse the mouth at appropriate times. The spray is directed at an 80-degree angle to facial and lingual surfaces of the posterior teeth and at a 60-degree angle for the anterior teeth. A 90 degree angle is used only on occlusal surfaces. Rub vaseline onto the patient's lips. Use cotton rolls for soft tissue retraction, use suction tube/ exhaustor. Use the powder spray only indirectly in the area of composite fillings, root cement demineralised enamel. After the treatment, inform the patient to avoid smoking and consuming tooth colouring foods in the first 2–3 hours.

Final stage of professional hygiene is fluorine treatment: varnish applications in the clinic and fluorine containing toothpastes, mineral water, and salt as well as home treatment.

Antiseptic medicines are often used for mouthwashes and rinses of gingival pockets. Medicines that have plant origin (Rotocan, Salvin, Novoimanin, Romazulan, Chlorphyllyptum, calendula and eucallyptus tincturae) are utilized in periodontology, but substances with chlorine as chlohexidine, metronidazole, triclosan are the most effective.

For antibacterial therapy of chronic cattharal gingivitis a combination of chlorhexidine and metronidazole (gel «Metrogyl Denta») is used. It is recommended as application on gingivae or rinces of gingival pockets 2 times a day for 20–30 min.

Medicines, which have plant origin, are used for mouthrinces: folia salvii sheets, flores chamomillae, herba hyperici, cortex quercus et al. If plant medicines

aren’t effective non-steroid anti-inflammatory drugs (NSAIDs) are recommended as applications (1% mephenaminate paste, 1% sodium diclophenac gel). Prepared ex tempore solution of arificial lysozyme is used for rinses. Such inhybitors of proteolytic ferments as contrical and trasylol are utilized for treatment of moderate and severe degree of chronic generelized gingivitis.

Physiotherapy (treatment with use of different physical factors) in practice of periodontologist isn`t conducted in the presence of general and personal contraindications.

Physiotherapy for chronic catarrhal gingivitis includes carbon dioxide hydrotherapy (10-15 visits for 10 minutes daily or in a day). Iontoophoresis of 1% galascorbin solution or 5% ascorbic acid solution with 1% nicotinic acid solution (10 sessions daily or on a day) are conducted. The duration of electrophoresis depends on the individual electric current resistance.

The presence of roentgenological changes in the long-term course of chronic catarrhal gingivitis is an indication for electrophoresis of 10% calcium gluconate solution or 2.5% calcium glycerophosphate solution (5 - 6 visits) and 1% sodium fluoride solution (4 - 5 visits).

Caries treatment, change of improper restorations, plastic surgery of a high attachment of the lip frenula and orthodontic treatment is conducted after minimization of the gingival inflammation.

Maintenance therapy in children with catarrhal gingivitis consists of regular check-ups each 3-6 months for the disease prevention, and oral hygiene control. A main element of the maintenance therapy is professional oral hygiene.

Hypertrophic gingivitis

Treatment of hypertrophic gingivitis depends on the etiological factors, the pathogenesis of the disease, its clinical form and course, as well as the degree of hyperplasia of the gingiva.

In a case of diffuse hypertrophic gingivitis, a child should be examined and consulted by an appropriate specialist: the pediatric endocrinologist - if the cause is a hormonal disbalance in the prepubertal or pubertal period and the psychoneurologist

– in a cause of use of antiseizure medicines.

Chomenko L.O. et al. offered such scheme of granulating form of hypertrophic gingivitis in children:

1. Professional oral hygiene.

2. Antibacterial therapy (antiseptic medicines, nytroimidasole medicines).

3. Anti-inflammation therapy (medicines that have plant origin, non-steroid anti-inflammatory drugs).

4. Physiotherapy (using different physical factors with treatment purpose).

5. Hygienic education, hygiene control and recommendations regarding oral hygiene products.

6. Removal local traumatic factors (caries treatment, periodontal plastic surgery and orthodontic treatment).

7. Maintenance therapy every 3-6 months (in a case of persistent catarrhal generalized ginigivitis).

8. Specialized counsiling and treatment in pediatrician and other specialists (gynecologist, neuropathologist, endocrinologist et al.).

The firt stage – professional oral hygiene. Prophy brushes aren't used to prevent trauma of hypertrophic gingiva.

For antibacterial therapy of granulating form of hypertrophic gingivitis chlohexidine, metronidazole, their combination (gel «Metrogyl Denta»), and medicines that have plant origin (Salvin, Novoimanin, Romazulan, Chlorphyllyptum, calendula and eucallyptus tincturae are utilized.

Non-steroid anti-inflammatory drugs (NSAIDs) are recommended for inflammation monimization in patients with hypertrophic gingivitis. These drugs are used locally (1% mephenaminate paste, 1% sodium diclophenac gel). Medicines that have plant origin can be used for mouthrinces: folia salvii sheets, flores chamomillae, herba hyperici, cortex quercus et al.

1% iontophoresis with glascorbine, rinces with decoctions of folia urtica, achillea millefolium can be used in a case of siginificant gingival bleeding.

After inflammation minimization next kind of the treatment can be used –

sclerosis therapy.

Products that have plant origin are used for the grade I-II gingivitis treatment (Maraslavin, Befunginum as instillation in false pockets, Chelidonium as oral rinse). Iontophoresis with 10% Calciui chloridum (8 – 10 visits) is also effective.

Iontophoresis with lydase or ronidase (15-20 visits in a day) is recommended for the grade I-II hypertrophic ginigivitis. In a case of the grade III hypertrophic gingivitis or ineffective medicamentous treatment such surgical methods as cryodestruction, diatermocoagulation, gingivectomy are utilized.

Vacuum massage (6-10 sessions a day), hydrotherapy with carbon dioxide (10-15 sessions for 10 minutes daily or in a day) can be used as physiotherapeutical treatment. After elimination of the inflammation, darsonvalisation by the spark method (15 - 20 sessions) is conducted. To improve metabolism in periodontal tissues heparin ointment or electrophoresis (ultra-phonophoresis) of heparin solution (10 - 12 sessions) is also prescribed.

Removal of traumatic factors is conducted in a case of minimization of the gingival inflammation. Maintenance therapy in children consists of regular check-ups each 3-6 months for the prevention disease, and oral hygiene control. A main element of the maintenance therapy is professional oral hygiene.

Chomenko L.O. et al. offered such scheme of fibrous form of hypertrophic gingivitis in children:

1. Professional oral hygiene.

2. Antibacterial therapy (antiseptic medicines, nytroimidasole medicines).

3. Physiotherapy (using different physical factors with treatment purpose).

4. Hygienic education, hygiene control and recommendations regarding oral hygiene products.

5. Removal local traumatic factors (caries treatment, periodontal plastic surgery and orthodontic treatment).

6. Orthodontic treatment if it is nessesary.

Anti-inflammation therapy is not indicated for fibrous form of of hypertrophic gingivitis in children.

Ulcerative (ulcerative) gingivitis

Treatment of ulcerative-necrotic gingivitis depends on the features of the disease pathogenesis, the severity and nature of its clinical course, as well as the child's age.

Children with mild and moderate degree of ulcerative gingivitis are recommended to consume an energy-rich soft food easily with digestible proteins and vitamins (porridge, fish, poultry meat), as well as numerous vitamins and enveloping drinks (acidic, fruit drinks).

Also polivitamins with ascorbinic acid, vitamins A, P, E and group B could be prescribed. If body temperature is more than 38–38,5°C non-steroid anti-inflammatory medicines are recommended.

Metronidazole is recommended for patients with moderate ulcerative gingivitis depending on child`s age. Duration of therapy depends on clinical symptoms and cytologic results in dynamics.

Severe degree of disease is treated with a pediatrician into hospital. Hospital therapy includes desintixocation with salt and plasma exchange solutions (Natrii Chloridum 0.9%, Rheopolyglukin etc.). Metronidazole, broad-spectrum antibiotics are prescribed intravenously or orally.

Local treatment includes elimination of conditions for the fuzospirillar infection due to removal of the necrotic tissues and the dental plaque, and metronidazole, chlorhexidine ointment or solution which affect the anaerobic microflora.

Chomenko L.O. et al. offered such scheme of fibrous form of ulcerative gingivitis in children:

1. Anesthesia.

2. Necrotic tissues removal (ferments).

3. Antibacterial therapy (antiseptics, nytroimidasole medicines).

4. Anti-inflammation therapy (medicines that have plant origin, non-steroid anti-inflammatory drugs).

5. Tissues regeneration stimulation (keratoplastics).

6. Hygienic education, hygiene control and recommendations regarding oral hygiene products.

7. Removal local traumatic factors (caries treatment, periodontal plastic surgery and orthodontic treatment).

First stage of the treatment includes applications of 10% lidocaine spray or gel «Camistad» as local anaesthesia.

Applications of proteolytic enzymes (trypsin, chymotrypsin, chymopsin, terilitin), which have necrolytic and mucolytic action, are used for the removal of the necrotic tissues. It is advisable to use complex medicines containing proteolytic enzymes and antibiotics (Lingesin ointment) for the large lesions.

After application of enzyme medicines, mechanical removal of necrotized tissues by a cotton swab moistened with the antiseptic solution. Irrigation and aerosol inhalations are also recommended for the passive abruption of the necrotized tissue. Chlorine-containing (chlorhexidine, hexitidine) and nirofuran derivatives (furagin) are most commonly used.

Acute symptoms are really painful, therefore the reestablishment of adequate oral hygiene can only be managed carefully, step by step. The first session should be limited to careful supra-gingival debridement, supplemented with 3% hydrogen-peroxide irrigation, which helps remove necrotic tissues. As hydrogen-peroxide releases oxygen during its decomposition, it is effective in eliminating dominant Fusobacterium species. Since individual oral hygiene is difficult to perform when in pain, hydrogen-peroxide or CHX mouthwashes are recommended for home use. The medication prescribed should be Metronidazol (3x250 mg for 7 days). The next session should be scheduled in two days to start actual cause-related therapy. After this, the patient is checked on day 7 and every week thereafter, until oral hygiene is re-established. If the alveolar bone is involved, surgical correction might be necessary.

Medicines of the group nitroimidazole are prescribed as applications on the affected areas of the gingiva or instillations in the gingival pockets. In case of severe course of ulcerative-necrotic gingivitis, it is recommended to use broad-spectrum

antibiotics (lincomycin, tetracycline, etc.).

Non-steroidal anti-inflammatory medicines use for inflammation minimization: sodium mefenaminate (0.1 - 0.2% aqueous solution), diclofenac-sodium (1% gel or jelly "Dicloran", 1% emulsifier "Voltaren"), piroxicam (1% cream, 0.05% gel), as well as herbal remedies: sage leaves, chamomile flowers, calendula, St. John's wort, nettle leaves, etc.

The use of keratoplastic agents to accelerate tissue regeneration and stimulate epithelialization is advisable only after cleaning ulcerative surfaces from necrotic masses and fibrin deposits, as well as after eliminating the inflammatory response of the surrounding tissues. The most commonly used keratoplastics are oil solutions of vitamins A and E, the multivitamin complex "Aevit", rosehip oil, carotolin, liniment "Aloe", Kalanchoe juice, jelly "Solcoseryl", etc.

The elimination of local contributing factors is done only after the processes of tissue epithelialization are completed.

6.3. Periodontitis

Localized periodontitis

See scheme of periodontal treatment plan.

0 - Preliminary phase (systemic pre-tratment, emergence treatment)

Phase – Initial (non-surgical) therapy

Re-evaluation

Corrective phase (surgery, restorations)

Phase - maintenance therapy

Scheme of periodontal treatment plan

"0" preliminary phase

Some precautions are required related to the management of systemic diseases (antibiotic prophylaxis, prevention of bleeding, adjustment or change in medications if necessary) beside the treatment of symptoms of acute pain.

Initial therapy

The goal of the therapy is to eliminate aetiological factors by means of non-surgical methods: removing dentogingival plaque, correction of plaque retentive factors, improper fillings. The aim of this phase is to motivate patients, and to teach them individual forms of oral hygiene. Periodontal health is also influenced by endodontic diseases; therefore, endodontic treatment should be performed at least partially in this phase. It involves chemo-mechanical treatment of the canals, and at least a provisional root canal filling to stop the infection. To achieve a better healing outcome, mobile teeth may be splinted for the duration of the treatment.

Re-evaluation

Re-evaluation is performed by measuring periodontal parameters 6–8 weeks after treatment. This timeframe is necessary to evaluate regenerative potential of the tissues, motivation of the patient, and efficiency of non-surgical treatment. If the inflammation could not be reversed, and it is not possible to provide individual and professional oral hygiene, corrective phase is the next step. Patient motivation and cooperation is a prerequisite for this phase. If the recorded parameters are not acceptable, the hygienic phase may be repeated to increase patient motivation. If it is still not successful, palliative treatment is to be performed with the appropriate restorative therapy.

The corrective phase

The most important part of the corrective phase is periodontal surgery. During periodontal surgery, aetiological factors remaining form non-surgical therapy can be eliminated to provide the circumstances for reparation or regeneration by establishing ideal conditions for individual and professional oral hygiene. If regenerative treatment is performed, some of the damaged tissues go through complete healing, and thus improving tooth prognosis. Orthodontic treatment can be a part of the

correction phase. Its goal is not only to establish ideal contact between the teeth and to correct defects compromising individual oral hygiene, but it may also influence neighbouring bone volumes, and this way correcting some defects. With orthodontic extrusion, a less traumatic crown lengthening, with an extraction, vertical bone gain can be achieved for optimal implant placement. Plastic surgery of shallow vestibule or incorrect attachment of lip frenulae are conducted in the corrective phase for children.

Final step of the corrective phase is restorations. Restorative treatment consists of procedures (direct or indirect restorations, prosthetic treatment, insertion of dental implant, etc.) which reconstruct the dental arch according to the requirements, such as loading, aesthetics, phonetics and functions.

Maintenance therapy

The described procedures provide a stable dental condition with which teeth can be preserved in their place for a long time. To maintain healthy conditions and prevent progression, regular check-ups are needed to be done, and treatment performed if necessary. The frequency of check-ups varies depending on the patient's motivation, nature of the disease, and presence of aetiological factors. Besides regular professional oral hygiene treatment, re-motivation, and re-instruction, some corrective treatments may also have to be performed again, or in case of recognising development of a new disease.

Professional oral hygiene treatment in patients with periodontitis includes the following procedures:

1. oral hygiene motivation, instruction
2. supra-gingival scaling
3. sub-gingival instrumentation
4. subgingival scaling
5. root planing
6. subgingival (closed) curette
7. elimination of plaque retentive factors
8. correction of overcontoured fillings

9. correction of overcontoured crowns
10. provisional treatment of carious lesions
11. provisional endodontic treatment
12. correction of anatomic deficiencies
13. closure of open contact points

Mechanical removal of dental deposits

Scaling means the elimination of dental plaque and its mineralised form (calculus). It can be done manually or with machined instruments as well. Usually highly effective ultrasonic devices are used first: the cooling spray helps to wash off removed deposits and blood, this way keeping the working area clean and visible. Tips of ultrasonic and sonic scalers have all or lateral active sides (Fig.11). For larger supra-gingival calculus removal sickle shaped scalers or universal curettes are recommended. For the instrumentation of sub-gingival areas, Vector or Gracey curettes are favoured.

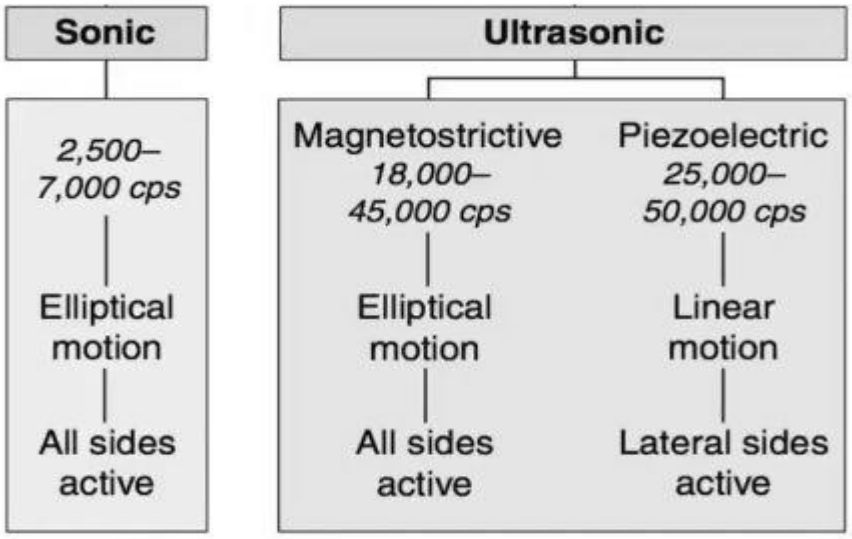

Fig.11. Kinds of machined scalers

Professional oral hygiene procedure

10% lidocaine spray or gel «Camistad» can be used for the local anaesthesia.

- rinsing with chlorine-hexidine-digluconate (CHX) 0.2% solution (including the throat) before mechanical treatment

Removal of sub-gingival deposits and root planing can be done quadrant-wise, or as „full mouth disinfection" (Marc Quirynen et al, 1995). This means mechanical instrumentation in the entire mouth within 24 hours, combined with the use of antiseptics against persisting bacteria in the „ecological gaps". The literature describes several interventions under the same definition, some with solely mechanical therapy and some combined with antiseptics (full-mouth disinfection).

Full mouth disinfection

Non-surgical mechanical plaque and calculus removal cannot eliminate the entire biofilm and it has no effect on bacteria in the „ecological gaps". Bacteria persisting in surface irregularities, cementum lacunae, fissures along the CEJ, on the epithelium and papillae of the tongue predispose to reinfection. Non-instrumented pockets are also possible sources of infection.

The concept of „full mouth disinfection" is based on the concept above, combining observations and theories of several authors:

- scaling and root planing in 2 sessions, within 24 hours
- rinsing with chlorine-hexidine-digluconate (CHX) 0.2% solution (including the throat) before mechanical treatment
- rinsing pockets with 1% CHX gel after instrumentation
- scrubbing the tongue for 1 minute with 1% CHX gel
- oral rinsing at home, twice a day for half a minute with 0.2% CHX solution for 2 months to prevent reinfection.

According to data in the literature, patients with poor oral hygiene and advanced periodontal infection benefit from the procedure. It significantly reduces the number of microorganisms in the periodontium and in the whole mouth, reduces the chance of reinfection and the risk of additional surfaces getting infected.

Root planing: Its goal is to prepare denuded, hyper-mineralised, intoxicated cementum for reparation and regeneration. Only the superficial layer of the porous cementum gets infected, therefore extensive removal of the cementum is unacceptable. Excessive instrumentation of the cementum can result in cervical sensitivity, possibly even pulp necrosis.

Root instrumentation can be carried out with machined instruments (ultrasonic or sonic device, Vector), but the final step must always be the checking of surface irregularity. Since there is a lack of visual control, palpation with a hand-instrument (Gracey curettes) is always recommended.

Removal of plaque retentive sites and polishing of surfaces

It is well documented in literature that a significantly greater amount of bone resorption can be found around overdimensioned, deep marginal closure restorations than around the contra-lateral unrestored tooth.

During the initial therapy, precise marginal closure provisionals need to be prepared, since the level of marginal gingiva will change after the treatment. In some instances, the removal of the restoration is not an option. The correction of restoration margins with a rotary instrument can be a solution, but this is mostly associated with aesthetic compromises. In case the preparation of the crown was convex (the equator of the tooth is wider than the cervical portion), marginal correction is not applicable, the restoration has to be removed. If the fillings are undercontoured or there is a gap at the margin, the restoration has to be changed.

The most common rotary instruments are polishing devices, brushes, rubber cups used with slow motion contra-angle hand pieces, perpendicular to the tooth surface. Special prophylactic hand pieces are also available, in which screwed devices can be inserted. Polishing is done with polishing pastes. Abrasive granules polish the surface, while a carrier substance is used to prevent overheating of the tooth. Granule size determines the field of application: (170-250 micron) removal of discolouration, surface irregularities, (120 micron) removal of plaque and smaller particles, (40 micron) final polishing of composite surfaces.

Air-abrasive systems can be used both for calculus removing and surface polishing. Some of them are attached to the turbine hose, some are separate devices, sometimes combined with built-in ultrasonic scalers. Aluminium-oxide, baking soda, glycine base, hydroxycarbonate-apatite, sodium-phosphosilicate or calcium-carbonate powder (27-50 micron) does the cleaning. They can be applied supra-gingivally but some new instruments can be used sub-gingivally as well (Air-FlowPerio).

Abrasive material needs to be sprayed at a 55º (palatal surface of incisors) - 80º angle. Proper suction is important to prevent the inhalation of the powder and any damage to soft tissues.

The final stage of professional hygiene includes fluoridation of surfaces using fluorine-based varnishes to prevent teeth hypersensitivity.

Pharmacotherapy

In moderate and mild chronic periodontitis (1-2 medium depth pockets/quadrant, 4-5 pockets/mouth), local antibiotics can be effective. For local use, tetracycline substances are the most common (ointment), but Metronidazol based local antibiotics (gel «Metrogil denta») are also available. CHX based local medications are also useful («Angileks», «Sebidin»). Long-time released medications are also utilized (25% metronidazole gel «Elyzol», films «Diplen denta» with metronidazole or chlorhexidine. The main requirements are continuous release of the active compound (7-14 days) and total resorption of the carrier.

Natural antibacterial substances (Novoimanin, Sangviritrin) can be used as the mouthrinces and the applications in children with peiodontitis.

Non-steroid anti-inflammatory drugs *(*NSAIDs) aren't recommended for general use in patients with periodontitis because of their side-effects. However, in post-operative care specific PGE2 inhibitor vinegar-acid substitutes (Indometacin, Diclofanac) play an important role, together with Propion-derivatives (Naproxen, Ibuprofen, Flurbiprophen). These drugs can be utilized locally (1% mephenaminate paste, 1% sodium diclophenac gel).

Tetracycline group was used successfully against the localized form of aggressive periodontitis to inhibit the reproduction of Aggregatibacter actinomycetemcomitans (50-100 mg/day), due to its high excretion concentration in the sulcular fluid. It has been observed that even in a low dose (40 mg/day), it is still effective in blocking collagenase enzymes.

The use of physiotherapeutical treatment promotes the activation of metabolism in the periodontal tissues and the normalization of their trophism. Carbon dioxide hydrotherapy includes 10-15 sessions for 10 minutes daily or in a day. Apply

iontophoresis of 1% galascorbin solution or 5% ascorbic acid with 1% nicotinic acid solution (10 sessions daily or in a day). The duration of the iontophoresis session depends on the individual sensitivity to current.

With progressive bone resorption of the alveolar process, iontophoresis with 10% calcium gluconate or 2.5% calcium glycerophosphate (5 - 6 sessions) and 1% sodium fluoride (4-5 sessions), is effective.

Maintenance therapy in children with localized periodontitis consists of regular check-ups each 3-6 months for prevention disease, and oral hygiene control. A main element of the maintenance therapy is professional oral hygiene.

Generalized periodontitis

The treatment plan for generalized periodontitis depends on the presence of a concomitant somatic disease in the child, its nosological form and the clinical course, and is made after careful examination of the child together with a pediatrician, endocrinologist or other specialist.

Professional oral hygiene treatment scheme is same with localized periodontitis and is dealt with plaque removing and scaling.

After local anaesthesia supragingival scaling and plaque elimination can be done in one session, but sub-gingival instrumentation needs more sessions to be effective. One efficient system for appointment planning is by quadrants or sextants with local anesthesia for more advanced disease at 1-week intervals to permit patient learning and progressive healing. With less severe periodontitis and a compliant patient, two quadrants on the same side (maxillary and mandibular arches) may be completed at an appointment. Systemic antibiotic treatment cannot replace mechanical plaque control; it is only additional treatment. The key to antibiotic treatment is to prevent selection and reproduction of resistant strains. The criteria of antibiotic application must be observed. Antibiotics may only be used to supplement oral hygiene procedures after the diagnosis of an active, progressing periodontal infection, possibly after identifying periodonto-pathogenic strains. Considering the characteristics of typical mixed infections, combined therapy is applied in most cases to achieve a synergistic effect of the medications: Metronidazol+Amoxicillin, or in

case of Penicillin allergy, Metronidazol may be combined with Ciprofloxacinnal or Cefuromexatil. For monodrug therapy, the first choice is Metronidazol, if no Aggregatibacter actinomycetemcomitans strains can be isolated, then Clyndamycin, for non-specific infection, Doxycyclin.

If a significant amount of fungi Candida was found in periodontal pockets topically 1% cream «Clotrimazole», gel with miconazole, 1% cream «Micogal» (omoconazole) can be used.

Antibiotics immobilized on biopolymer media are used for topical application at generalized periodontitis: thread with tetracycline impregnation (25%), doxycycline gel (8.5%), films "Diplen denta L" and "Diplen denta K", etc. These agents are able to maintain a high concentration of the antibiotic in periodontal pockets over a long period of time without significantly increasing its level in the blood.

To normalize the microflora of the oral cavity and other biotopes of the body in complex therapy of patients with generalized periodontitis probiotics can be prescribed, which include probiotic microorganisms and a nutrient medium for their growth. Today in dental practice, Lactobacterin, Bifidumbacterin, Acylact, Symbiter are widely used.

Such NSAD as sodium mefenaminate (1% paste), diclofenac-sodium (1% gel or jelly "Dicloran", 1% emulsifier "Voltaren"), piroxicam (1% cream), as well as plant remedies: sage leaves, chamomile flowers, marigolds, St. John's wort are chosen. Galascorbin (1% aqueous solution), decoctions of nettle leaves, yarrow herbs are used in a case of severe hemorrhage.

If a purulent exudate is present in periodontal pockets, such proteolytic enzyme inhibitors as Contrical, Trasylol are topically used.

For the restoration of impaired metabolism in patients with generalized periodontitis, oral administration of calcium, fluorine, as well as complex agents containing macro-, trace elements and vitamins are recommended.

An important part in complex treatment of generalized periodontitis have physiotherapeutical methods which activate metabolism in the periodontal tissues and

normalize their trophic. Carbon dioxide hydrotherapy (10 - 15 sessions for 10 minutes daily or in a day) can be recommended. Electrophoresis with 1% galascorbin solution or 5% ascorbic acid with 1% nicotinic acid solution (10 sessions daily or in a day) is recommended to decrease gum bleeding. The duration of the electrophoresis session depends on the individual current sensitivity. Electrophoresis with 10% calcium gluconate or 2.5% calcium glycerophosphate (5-6 sessions) and 1% sodium fluoride (4-5 sessions) are recommended in a case of the alveolar bone resorption. A vacuum massage is recommended for symptomatic hypertrophic gingivitis. Atrophic ginigivitis is an indication for the vibration therapy. Finally, they recommend the use of D'Arsonval current (application technique), diathermy, paraffin, ozokerite applications.

Photodynamic therapy

The goal of photodynamic therapy is to eliminate bacteria from limited access areas, where mechanical therapy would be insufficient or not even possible. The principle is that a tissue friendly material is exposed to light, as a result it releases nascent oxygen, which can cause substantial chemical damage to microorganisms.

As a light source both visible light and laser light sources are used. Devices used in the dental practice are special instruments or diode lasers, either in the visible or in the laser light spectrum. Photoinitiators can be pigments (e.g.: metilene-blue, toluidine-blue), chlorine substances, porphyrin, xanten, monoterpen, depending on the light source.

To begin the treatment, a photoinitiator fluid (gel) needs to be injected in the pocket through a cannula, which has to be lit with a light transmitting fibre inserted in the pocket. Side-effects can be thermal damage or cytotoxic effect of the photoinitiator, but to our best knowledge, temporary discoloration of the gums is the only problem expected.

Photodynamic therapy does not replace mechanical therapy, but can be an efficient supplement. In vitro studies showed substantial antibacterial effect, but in vivo experiments are not sufficient enough to draw positive conclusions clinically: an adequate number of clinical studies and meta-analysis still needs to be conducted.

Use of lasers in non-surgical therapy

Lasers are special light sources, which use stimulated emission to produce a light beam. Laser beam is coherent (in time and space), almost parallel, mono-coloured light, which can achieve high density of energy, even at a long distance. Laser beam is an electro-magnetic wave, which consists of a one wavelength component. Laser light is amplified by stimulated emission, this way more photons are produced in the laser beam than absorbed. Applying the energy level needed for stimulated emission on the laser is called pumping. Different energy types are suitable for pumping: light energy (flashing light, another laser), electric energy (gas discharge) or chemical energy (chemical reaction). Classification of lasers, regarding laser agent is showed in Tab.6.3.1:

Tab.6.3.1

Properties of lasers used in dentistry

	Laser type	Wave length, nm	Indication (periodontology)
Diode lasers	Indium-gallium-arsenide-phosphorus	655	Photodynamic therapy
	Gallium-arsenide	685	Photodynamic therapy
	Gallium-alumonium-arsenide	810	Soft tissues vapourization, coagulation, plaque removal
	Indium-gallium-arsenide	980	Soft tissues vapourization, coagulation, plaque removal
Gaz laser	CO_2	10600	Soft tissues vapourization, deepithelization
Solis state laser	Neodymium-doped Yttrium, Aluminum, Gamet	2780	Soft currete, cutting
	Erbium-doped Yttrium, Aluminum, Gamet	2940	Calculus, plaque removal

Lasers work in a continuous or pulsating manner. Conduction of the laser light generally goes through glass fibre. In case of the highly available diode laser, the working end must be activated by a coloured material (occlusion paper, cork) when cutting or vaporising, but decontamination of tooth or implant surfaces can be performed without activation. CO2 lasers and certain Er:YAG lasers conduct light through fixed supports. The summarised properties of dental lasers can be seen in the figure below.

Effects of lasers on the biofilm have been studied both in vitro and in vivo. Obvious advantages of Er:YAG lasers have been discovered. This type is even capable of removing solid, mineralised substances. Disadvantages are its size and retail price. The effects of small-sized diode lasers on the biofilm are questionable. Their disadvantage is that working at an 810-nm wavelength, they produce much heat. An interesting field of application of these lasers is experimentally discovered biostimulation effect: it stimulates the proliferation of ligamental and gingival fibroblasts.

HEALING AFTER NON-SURGICAL TREATMENT

After non-surgical treatment, in optimal conditions, the periodontal pocket is filled with a blood clot. On the soft tissue side a 0.2 mm wide necrotic zone develops, where leukocytes accumulate. Their task is to clean the wound, protect it against bacterial forces and stimulate healing process. Healing starts within 8-24 hours after intervention: secretion of inflammatory mediators, beginning of cell proliferation, transformation of the blood clot. Epithelisation starts from the marginal gingiva, under the protection of the leukocyte infiltrated necrotic zone, covering the healthy portion of gingival connective tissue. At day 7, most of the connective tissue is covered by new epithelium and a connection is built between the epithelium and the root surface. Considering that complete removal of the biofilm is technically impossible and bacteria will persist in surface irregularities and dentine tubules to a certain extent, a constant inflammatory reaction is present. If there is a persistent large supply of bacteria (e.g.: failing restorations), the healing process cannot proceed

and pocket depth will remain the same. The rest of the process includes maturation of the tissues.

Clinical attachment level will be disposed 1-2 mm coronally from the baseline situation, while pocket probing depth can decrease by 2-4 mm on average, owing to the long junctional epithelium and regression of the inflammatory swelling. Marginal gingiva will shift 1-2 mm apically causing recession. Connection between the gingiva and the root surface is not regenerated attachment (new attachment) but long junctional epithelium formed by epithelial cells of the gingival margin.

Maintenance therapy in children with generalized periodontitis consists of regular check-ups each 3-6 months for prevention of the disease, and oral hygiene control. Roenthgenologic examination is conducted 2 times a year, after 1 time a 3 year if clinical activity isn't present. Microbiological examination of the periodontal pockets is indicated for ineffective conventional treatment. A main element of maintenance therapy is professional oral hygiene.

6.4. Idiopathic diseases with progressive lysis of periodontal tissues

In general, dental treatment consisted of scaling, root planning and extraction under corticosteroid and antibiotic cover. Early communication with patients, parents and guardians regarding the limitations of dental care in preventing tooth loss and other dental sequelae including prosthetic limitations is important. The responsibility for meticulous oral hygiene and the support and development of healthy dental behaviors by the parents and caregivers should be emphasized. Some patients with Down sundrome may be able to effectively brush their own teeth, but most will not have the hand coordination and/or ability to follow the complex sequence of events required for effective plaque removal. Watching the patient or caregiver brush the patient's teeth may help determine any additional guidance needed. Demonstration of oral hygiene techniques may be beneficial. With high caregiver turnover, some patients may not receive consistent home care and may need continued re-introductions to home care at subsequent visits. Flossing aids may prove beneficial.

Early dietary counseling can also be of benefit in preventing the nutritional problems commonly encountered in this population.

Tab. 6.4.1.

Periodontal diseases associated with genetic disorders

Disorder	**Etiology**	**Clinical oral signs/symptoms**	**Therapy**
Familial and cyclic neutropenia	ELANE mutation, cyclical decrease in the number of circulating neutrophils	Oral ulcers Gingival inflammation Severe periodontitis	Mechanical debridement monthly and during neutropenic episodes chlorhexidine rinsing
Down syndrome	Trisomy chromosome 21, reduced chemotaxis and impaired phagocytosis	Gingivitis Necrotizing ulcerative gingivitis Severe periodontitis Tooth mobility	Preventive treatment and periodontal therapy
Leukocyte adhesion deficiency syndrome	Defects in adhesion receptors of the white blood cells and impaired phagocytosis	*Type 1:* Severe gingival inflammation Rapidly progressive periodontitis *Type 2:* Chronic severe periodontitis	Periodontal treatment with or without antibiotics, often followed by extraction of primary or permanent teeth
Papillon-Lefèvre syndrome	Mutation of gene encoding for cathepsin-c,	Aggressive periodontitis Premature loss of	Conventional periodontal treatment with

	impaired neutrophil function	teeth	antibiotics according to the dentition involved and extraction of primary teeth 6 months prior to eruption of permanent teeth if indicated
Chédiak-Higashi syndrome	Mutation in LYST gene	Oral ulcerations Severe gingivitis Early-onset periodontitis	Challenging periodontal therapy
Histiocytosis syndromes	Abnormal proliferation of bone marrow-derived histiocytes	Periodontitis Alveolar bone loss replaced by soft tissue Oral ulceration Premature loss of teeth	Conventional periodontal therapy and surgical therapy in mandibular lesions in some cases
Severe congenital neutropenia	ELANE and HAX1 mutations	Gingival inflammation Increased probing depth Severe alveolar bone loss in both dentitions	Scaling and root planing, use of antimicrobial agents
Hypophosphatasia	Mutation in tissue nonspecific alkaline phosphatase	Absence of root cementum Premature exfoliation of	Possible extraction of primary teeth and more conservative

	activity	deciduous teeth	treatment on permanent teeth

6.5. Periodontal neoplasmae

Surgical periodontal treatment - gingivectomia, diathermocoagulation or cryodestruction. The prognosis is not always favorable, relapses are possible. The issue of teeth preservation is solved on the basis of clinical and radiological data.

Tab.6.5.1

Non-Plaque-Induced Gingival Conditions & Lesions in Young Patients

Aetiology	Specific cause	Condition/lesion	GDP/Refer
Infective lesions	Viral	Herpangina	GDP – r
		Hand Foot & Mouth	GDP – r
		Herpes Simplex I (primary)	GDP – r
		Herpes Simplex I (secondary)	GDP – r
		Molluscum Contageosum	Refer
	Fungal	Candidosis	GDP – r
		Linear gingival erythema (candidosis)	Refer
	Deep mycoses	Aspergillosis	Refer
		Blastomycosis	Refer
		Coccidiomycosis	Refer
		Cryptococcosis	Refer
		Histoplasmosis	Refer
		Geotricosis	Refer
Genetic conditions	Fibromatosis Anatomic variations	Hereditary Gingival Fibromatosis	GDP – r GDP

		Delayed Gingival Retreat	
		Coeliac Disease	Refer
Systemic diseases that manifest within the gingivae	Haematological disease		
	Benign conditions	Agranulocytosis	Refer
		Cyclical Neutropenia	GDP - r
		Familial Benign Neutropenia	GDP - r
	Malignant conditions	Myelodysplastic Syndromes	Refer
		Myeloid leukaemia	Refer
		B-cell Lymphoma	Refer
		Hodgkins Lymphoma	Refer
	Granulomatous inflammations	Crohn's Disease	Refer
		Sarcoidosis	Refer
		Melkersson-Rosenthal syndrome	Refer
		Wegener's Granulomatosis	Refer
		T.B.	Refer
		Disseminated Pyogenic Granulomata	Refer
	Immunological conditions	Hypersensiyivity reactions	GDP - r
		Lichen Planus	Refer
		C1-esterase Inhibitor Deficiency/ Dysfunction (angioedema)	Refer

Trauma	Thermal	Burns	GDP
	Chemical Physical	Ulceration	GDP
		Gingivitis artefacta	Refer
Drug-induced	Immune complex reactions		
		Erythema multiforme	Refer
		Lichenoid drug Reactions	GDP – r
	Cytotoxic drugs	Methotrexate	Refer
		Doxycycline	Refer
		Hydroxychloroquine	GDP
	Pigmenting drugs	Doxycycline	GDP
		Oral Contraceptive	GDP
		Antimalarials	GDP
	Anti-retroviral drugs	Anti-HIV Drugs (VII nerve neuropathy)	Refer

GDP = manage in practice; Refer - to refer a patient to a physician, GDP – r = manage in practice but refer if concerned or complications arise.

From ILC Chapple Table 6.1 in Clerehugh V, Tugnait A, Chapple ILC. Periodontal management of children, adolescents and young adults. Quintessence Publishing Co. Ltd., London, 2004.

Chapter 7
PREVENTION OF PERIODONTAL DISEASES

7.1. Base stages of periodontal diseases prevention

Prevention of periodontal diseases is a part of prevention in dentistry. Prevention is devided on three categories: primary, secondary and tertiary (WHO, 1978).

Primary prevention is a set of social, medical, hygienic and educational measures targeted on the prevention of periodontal diseases by removal of causes of their origin, and improvement of immune system resistance in general. Its main goal is to protect the level of population's health quality by means of all possible measures, techniques and methods.

Secondary prevention is targeted on early diagnostics of periodontal diseases, removal of their first symptoms, prevention of recurrences, progresses of pathological processes or complications of diseases. Secondary prevention is a part of a rehabilitation program. The main goals and methods of the first and second prevention may look the same, but there are certain differences in criteria of their efficiency estimation.

Tertiary prevention is a set of measures targeted on prevention of periodontal diseases prior to occurrence of their severe forms (or stages), as well as prevention of their exacerbation, and decrease of temporary disability risks.

7.2. Methods of periodontal diseases prevention

Methods of periodontal diseases prevention could be devided on endogenous and exogenous, medical and non-medical.

Endogenous non-medical periodontal diseases prevention includes balanced nutrition as a sourse of good metabolism. It is essential to pay special attention on qualitive and quantative correction of protein content in a diet because proteins

stimulate calcium adsorbtion due to the formation of its soluble substances with aminoacids. Protein deficiency during the child's development "programs" the defective bone structure. Intake of a sufficient amount of minerals and vitamins, especially vitamins E and P in the composition of vegetable oils, which are natural antioxidants and play an important role in prevention of the periodontal diseases, is important for a child.

The basis of *endogenous medical prevention of periodontal diseases* is the use of fluorine medicines, which strengthen the structure of calcium-organic complexes in the hard tissues of the teeth and bone, normalize phosphorus-calcium metabolism. Interest in the use of fluorine substances for prevention of the periodontal diseases arise due to the data on their efficacy in the treatment of systemic osteoporosis (Belashov, 1982).

To prevent the development of hypocalcaemia, isolated use of fluorine substances should be avoided, while fluorine and calcium medicines should be administered at the same time following various ways of their introduction. Fluoride medicines should be administered to children in areas with a low fluoride content in drinking water according to fluoride dose for the prevention of dental caries.

Endogenous medical prevention of periodontal diseases is also performed in cases of malnutrition, especially deficiency of ascorbic, nicotinic acids, vitamins B in winter and spring, or in the presence a case of somatic disease (together with a pediatrician).

Exogenous non-medical prevention of periodontal diseases provides oral hygiene, intensive chewing of hard food at the end of the meal, limited consumption of carbohydrates, slow drinking of milk, tea.

Exogenous medical periodontal disease prevention is a topical application of fluorides and professional hygiene, which includes dental examination, removal of soft dental deposits, tartar, polishing teeth and filling.

7.3. Measures of periodontal diseases prevention

Periodontal diseases prevention measures can be collective and individual. Collective prevention includes general and special measures.

General measures:

- following a healthy lifestyle;
- toughening up;
- sport activity;
- rational balanced nutrition;
- prevention and treatment of infectious and somatic diseases.

Special measures:

- hygienic education of the population;
- therapeutic and preventive measures for the purpose of rehabilitation of the oral cavity.

Forms of collective prevention include:

- active sanitary and educational work in organized children's groups;
- active sanitary and educational work through the press, radio and television.

Measures of individual prevention of the periodontal diseases are age-related.

In the prenatal period, the formation of functionally complete periodontal tissues is facilitated by the health care of the pregnant woman and her nutrition. The main forms of preventive work at this stage are active sanitary and educational work in women's consultations.

In infancy, measures to ensure the correct function of suction are important for the prevention of periodontal disease. This promotes a proper growth and development of the jaws, the formation of complete periodontal tissues. The duration of baby feeding should be 20 - 30 minutes. If the duration of feeding is reduced and the infant does not have time to satisfy the sucking reflex, a child has favorable conditions to develop bad habits of sucking the tongue, fingers, etc. In this case, after feeding, it is necessary to give the pacifier, which can play the role of an individual product for the prevention bad habits in young children (T.F. Vinogradova, 1988).

From the age of 6 months, when the baby begins to feed, it is important to remove the food from a spoon with lips. This is one of the conditions for moving the mandible forward.

During early childhood, the main preventative measures to prevent the periodontal disease are aimed at the formation of full-fledged chewing function. As the teeth erupt (from 6 to 9 months), rough food (apple, carrots, dried fruits) should be introduced into the baby's diet.

During the formation of primary bite impaired function of swallowing, breathing, speech lead to the development of anomalies and damage of the periodontal tissues.

The presence of improper attachment of the lip and tongue frenulae leads to improper growth of the jaws, formation of a shallow vestibule of the oral cavity, development of malocclusion, which lead to the early emergence of the periodontal diseases. Therefore, it is important to make early diagnosis and treat such anomalies.

It is necessary to monitor the condition of the primary teeth at this age and to examine defects that provides normal development and maturation of the periodontal tissues.

After teething (2-2.5 years), the child should be taught oral hygiene skills (mouthwash, teeth cleaning). In the preschoolchildren (3-6 years), in addition to the mentioned factors, the prevention and treatment of malocclusion is the main task in the prevention of periodontal diseases.

It is also important to increase the activity of the organs and tissues of the oral cavity by expanding diversity and increasing the number of natural stimuli. The intensity of chewing is increased by introducing into the child's diet raw vegetables (carrots, radish, cucumbers, cabbage, green onions). Parents need to teach children the habit to finish eating with fruit, which improves the self-cleaning of the oral cavity.

During the period of mixed bite, the main direction of preventive work is the elimination of harmful factors that disrupt the growth and formation of dental rows. Such factors are bad habits, a shallow vestibule oris, improper attachment of the lip

and tongue frenulae, impaired breathing, swallowing, and speech. It is possible to appoint myotherapy and therapeutic gymnastics for the purpose of regulation of formation of jaws and dental arches.

Miogymnastic exercises:

1. In a case of disposture and for health breath training – morning gymnastic complex.

2. Normalization of swallowing:

а) The patient is instructed to practice correct swallowing pattern by placing the tip of tongue on the palate, close teeth, close lips and swallow with tongue in that position.;

б) same exercise with a sipe of water;

в) tutting;

г) yawning;

д) throat rinsing.

Exercises for orbicularis oris and circumoral muscles:

a) Upper lip should be stretched over the lower lip in an attempt to touch the chin;

b) Pump hot salt water back and forth behind the lips 4 to 5 times and spit it out and repeat it. Use half a glass of water at a time. This exercise has salubrious effect on hypertonicity;

c). The above exercise can be done using breath, instead of water. Force air behind the lips as forcibly as you can, hold it for a moment and then release it;

d) Button tug of war: Two 1½ inch flat buttons are taken, thick thread is passed between them. One button is held by the patient and the other by another person. The button should be held in position by the lip pressure. Gently, the pressure can be increased. The patient should not tilt or do abrupt movements, as it can hurt them.

d) exercises with appliences:

- Shonkher`s vestibular plate;
- Dass` activator;
- Friel`s disc;

- Wind toy.

3. Exercises that move mandible forward:

- Slowly move mandible forward till incisal inclination;
- Same exercise moving head on the right, left.

4. Exercises that raise mandible:

- close lips, clench teeth, increase pressure on teeth by contraction of chewing muscles in central occlusion;
- same exercise with stick, eraser.

The purpose of muscle exercises is to provide a normal structure and function in the orofacial musculature, as they are important elements of aiding growth and development of the periodontal tissues. Treatment of the dentofacial anomalies and the malooclusion is performed by special devices- orthodontic appliances.

It is known that a high percentage of periodontal inflammation in children is caused by a poor oral hygiene, especially during the mixed bite. Therefore, it is very important for prevention of the periodontal diseases to teach children oral care using the interdental hygiene means.

In order to prevent periodontal inflammation, it is important to treat teeth at time and completely.

It is important to perform gum massage and mouth irrigation at home. Finger massage, or gum massage, plays an important role in the complex of therapeutic and preventive measures. Right massage increases the gingiva resistance to harmful irritants, promotes the tightening of the oral mucosa, improving its metabolism. In addition, it is easy to perform self-massage, and this can be done independently by the patient.

Self-massage technique. Using the forefinger and thumb, grasp the external (labial, buccal) and palatine (lingual) surfaces of the alveolar process of the jaw. Usually, the massage starts from the center of the jaws, gradually moving the fingers on their lateral surfaces. The movements of the fingers start from the base of the alveolar process to the teeth (on the upper jaw from top to bottom, on the lower jaw - from bottom to top). Massage movements of the fingers should not cause pain, and

the force of movement of the fingers is regulated on the basis of this perception. The massage movements of the fingers should be circular. Massage is performed sequentially on each jaw.

Gum massage should be performed regularly, at least once a day, preferably before sleep, after brushing your teeth and rinsing your mouth. After the massage, the mouth should be rinsed again with warm water. The duration of gum massage should not exceed 7 - 8 minutes.

Before the finger massage, dental deposites should to be removed .

During the period of permanent bite, preventive measures to prevent the periodontal diseases should be aimed at:

1) elimination of pathological situation in the oral cavity:

- since the most significant and controlled indicator of the periodontal disease in children is an excessive bacterial contamination of the oral cavity (plaque, pockets). The oral hygiene, which includes local medical interventions using antibacterial medicines, oral hygiene education, monitoring their compliance and ongoing patient motivation, is the most effective. Individual hygiene measures should be aimed at the rational care of the oral cavity with the help of special therapeutic and prophylactic toothpastes and tooth elixirs;

- elimination of permanent trauma (replacement of low-quality fillings and improper crowns, dentures);

2) training of the vessels of the maxillofacial area with the help of special gymnastic complexes for mimic and chewing muscles, finger massage of the gums;

3) therapeutic measures in the oral cavity that do not cause additional irritation and do not disturb microbial symbiosis and physiological environment;

4) rational nutrition.

7.4. Factors supporting periodontal health in children

The factors that support periodontal health in children are considered (N.A. Savichuk, 2008):

1) physiological development and age correspondence of structure and functions of the periodontal tissues:

- age-related development of the periodontal morphological structure;
- maturity of the vessel structures and the histogematic barrier;
- maturity and functional activity of the system of colonization resistance - local (system of local immunity and microecology of periodontium) and general (system of general immunity and microecology of different loci of the organism);
- the maturity and adequacy of the immune-endocrine regulation;

2) adequate functional load of the periodontal tissues;

3) sufficient self-cleaning of the teeth and the interdental spaces;

4) balanced nutrition which provides the needs of the body in nutrients, micro- and macroelements;

5) effective and regular oral hygiene;

6) physiological formation of endocrine functions in puberty;

7) favorable environmental and social living conditions;

8) absence of chronic traumatic factors;

9) absence of chronic diseases of organs and systems;

10) absence of bad habits;

11) harmonious psychological and physical development.

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