Ryazan State Medical University
named after academician I.P. Pavlov

The Department of Therapeutic and Pediatric Dentistry

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DISEASES OF PULP AND PERIODONT

Textbook
for 4th , 5th years students of the faculty of dentistry
with English translation service

Ryazan, 2019

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This edition is designed for foreign English speaking students, it contains the information about structure of periodontium, pathomorphology, ethiology, pathogenesis, clinical manifestations, differential diagnostics, treatment and prophylaxis of periodontitis. Approved by scientific Methodical Council of RyazSMU and recommended for publishing.

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Кафедра терапевтической и детской стоматологии

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БОЛЕЗНИ ПУЛЬПЫ И ПЕРИАПИКАЛЬНЫХ ТКАНЕЙ

Учебное пособие
для студентов 4, 5 курсов
стоматологического факультета, обучающихся
с сервисом перевода на английский язык

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Данное учебное пособие разработано для иностранных студентов, обучающихся с сервисом перевода на английский язык, в нем содержится информация о строении, патоморфологии, этиологии, патогенезе, клинических проявлениях, дифференциальной диагностике, лечении и профилактике периодонтита. Утверждено учебно-методическим советом ФГБОУ ВО РязГМУ Минздрава России и рекомендовано для печати.

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CONTENTS

INTRODUCTION

Periodont is a part of parodontium complex. It consists of connective tissue which surrounds the tooth, located between alveol and tooth cementum.

Apical periodontitis is the inflammation of periradicular tissues caused by persistent microbial infection of the root canal system of the affected tooth. The infected and necrotic root canal system is a selective environment for the causative organisms, which grow mostly in sessile biofilms and also as aggregates, coaggregates, and planktonic cells suspended in the fluid phase of the canal. A biofilm is a community of microorganisms of one or more species that are embedded in an extracellular polysaccharide matrix and are attached to a solid surface against planktonic organisms, which are free-floating single microbial cells in an aqueous environment. It should be understood that sessile microorganisms that are protected in biofilms are 1000 times more resistant to antimicrobial agents than the same organisms in planktonic form.

There is a lot of interest in this problem because this disease is very common and prevalent clinically. Old methods of treatment of caries and pulpitis may lead to periodontitis genesis. It is important to remember: There are no blocked root canals! It is very important to use modern methods of diagnostics and treatment of dental diseases.

Good knowledge of teeth anatomy, and their peculiarities are the base of excellent treatment. In modern world there are many modern instruments, materials, methods of canals preparation and treatment. All these measures help dentists achieve good treatment of periodontitis.

The aim of this manual is to study modern information about pathhistology, aetiology, pathogenesis, clinical picture, diagnostics and treatment of apical periodontitis. This book is good for foreign students studying therapeutic dentistry in English language.

1. Structure and functions of periodontium

Periodontium - is a connective tissue, situated in the periodontal fissure between the root of tooth and alveolar bone.

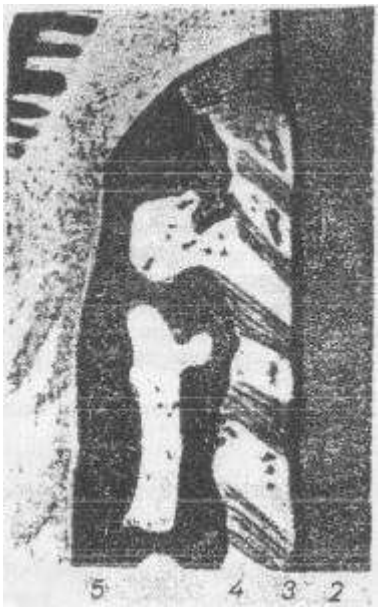


Fig. 1. Periodontium.
1- gingival;
2- tooth;
3- cementum;
4- periodontium;
5- alveola.

The formation of periodontium ends a year after the complete development of root apex. The size of the periodontal fissure is an alterable notion and it may change alongside with the tooth development, its functioning , and age as a result of a pathologic process. According to Sherbakov (1994) width of periodontal fissure is different in different parts of alveolar bone: it is 0,23 mm in the orifice of alveoli, 0,16-0,19 mm in cervical part, 0,08-0,14 mm in median part, 0,16-0,19 mm in apical part, and in the bottom of alveoli it is 0,23-0,28mm. So periodontal fissure has form of “sand glass” because of such sizes. It explains micro-movements of tooth inside alveoli. Periodontal fissure can become about 0,1 mm if antagonist-tooth is extracted because of decreased power of periodont. But if there is a big load on tooth, periodonteum becomes thicker and periodontal fissure is wide.

The periodontal connective tissue consists of three basic components: ligaments, cellular elements, basic substance with blood and lymphatic vessels and nerves.

Periodontal ligaments

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 narrow spaces through vascular channels in the bone.

Periodontal ligament contains collagen fibers that form thick oriented bunches and form several basic groups. The intervals between them are filled with thinner branched collagen bunches which form three dimentional network. Apart from collagen fibers, periodontal ligament contains a network of oxytalan fibers. There are no mature elastic fibers in human periodontal ligament, according to some scientists, this is connected with quick renovation of periodontal ligament. But this idea is doubtful, because elastic fibers are found in periodontal ligaments of many animals and it is also characterized by a high speed of renovation.

The most important elements of the periodontal ligaments are the principal fibers, which are ***collagenous***, arranged in bundles, and have a wave form when viewed in longitudinal sections. Terminal portions of the principal fibers that are inserted into cementum and bone are termed Sharpey's fibers. The principal fiber bundles consist of individual fibers that form a continuous anastomosing network between tooth and bone.

Collagen fibers consist of collagen fibrill bunches of typical structure. The peculiarity of collagen fibrills in periodontal ligament is that they have small diameter (near 55 nm., which is 2-4 times less than in tendon), and during the life it reduces from 70 to 30 nm, Collagen fibers are characterized by a slightly wave motion, thus they are capable of slight lengthening and tightening. Due to this they can provide restricted motions of a tooth.

One end of collagen fiber bunches is attached to cementum and the other one is embedded in the bone of alveolar processus. Moreover their terminal parts in both tissues are called perforating (Sharpeev) fibers. Perforating fibers of bone are usually thicker than those of cementum. Fiber ends in cementum are partially mineralized. Separate fibers are tangled into each other and can be shorter than the bunch.

According to some observations, the bunches of collagen fibers in periodontal ligament are represented by two components, one component leaves from the bone (alveolar fibers), the other one leaves from cementum (tooth fibers). The fibers of both parts are tangled with each other approximately in the middle of periodontal ligament forming intermediate plexus. There exists an idea that such structure of periodontal ligament provides optimal conditions for its reconstruction in conformity with changing state and dynamic loading.

Collagen is a protein composed of different amino acids, the most important of which are glycine, proline, hydroxylysine, and hydroxyproline. The amount of collagen in a tissue can be determined by its hydroxyproline content.

Collagen biosynthesis occurs inside the fibroblasts to form tropocollagen molecules. These aggregate into microfibrils that are packed together to form fibrils. Collagen fibrils have a transverse striation with a characteristic periodicity of 64 nm; this striation is caused by the overlapping arrangement of tropocollagen molecules. In collagen types I and III these fibrils associate to form fibers and in collagen type I the fibers associate to form bundles.

Collagen is synthesized by fibroblasts, osteoblasts, odontoblasts, and other cells. There are several types of collagen, all distinguishable by their chemical composition, distribution, function, and morphology. The principal fibers are composed mainly of collagen type I, whereas reticular fibers are collagen type III. Collagen type IV is found in the basal lamina.

The molecular configuration of collagen fibers provides a tensile strength greater than that of steel. Consequently, collagen imparts a unique combination of flexibility and strength to the tissues where it lies.

The principal fibers of the periodontal ligament are arranged in six groups: transseptal, alveolar crest, horizontal, oblique apical, and interradicular:

1. ***Transseptal Group.*** Transseptal fibers extend interproximally over the alveolar crest and are embedded in the cementum of adjacent teeth. They are a remarkably constant finding and are reconstructed even after destruction of the alveolar bone has occurred in periodontal disease.

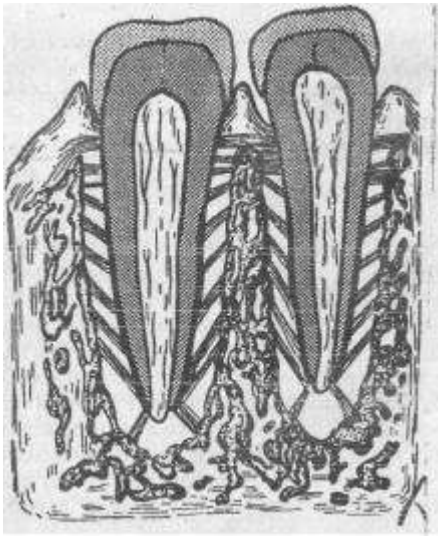
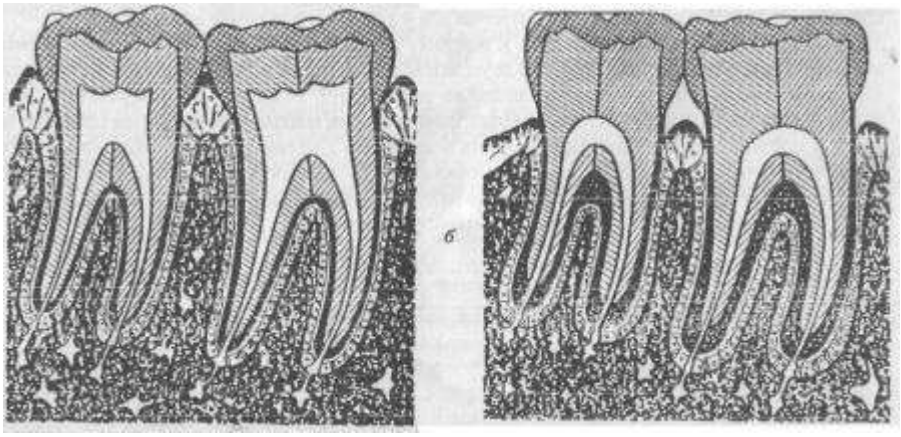


Fig. 2. Direction of collagen fibers in different regions of periodontium.

2. ***Alveolar Crest Group.*** Alveolar crest fibers extend obliquely from the cementum just beneath the junctional epithelium to the alveolar crest. They prevent the extrusion of the tooth and resist lateral tooth movements. Their incision does not significantly increase tooth mobility.
3. ***Horizontal Group.*** Horizontal fibers are situated deeper than alveolar crest fibers near the entrance to the periodontal area. They are perpendicular to the surface of the root and alveolar bone. Horizontal fibers form the so - called circulatory ligament that also includes transseptal fibers connecting neighbouring teeth and situated under the apex of alveolar processus.
4. ***Oblique Group.*** Oblique fibers, the largest group in the periodontal ligament, extend from the cementum in a coronal direction obliquely to the bone. They bear the brunt of vertical masticatory stresses and transform them into tension on the alveolar bone.
5. ***Apical Group.*** The apical fibers radiate from the cementum to the bone at the fundus of the socket. They do not occur on incompletely formed roots. These fibers prevent lateral movements of tooth.
6. ***Interradicular Fibers***. These fibers fan out from the cementum to the tooth in the furcation areas of multirooted teeth. In multi rooted teeth, they connect the root in the area of bifurcation with the inter root septum crest towards which they are directed partially horizontally and partially vertically.
7. ***Circular fibers.*** These fibers surround cervical part of tooth.

8. ***Free fibers of gum.*** They are directed from the cervical part of tooth to the gum. In periodonteum of teeth, elastic fibers can be found but in less amounts. These fibers are thin and often are situated in periodonteum of incisors and canines between cervical fibers.



A B

Fig. 3. Changes in periodontium during life.
A – structure of periodontium of young man;
B - structure of periodontium of old man: obliteration of tooth cavity, thickness of cementum, change of bone structure and vertical movement of tooth.

One peculiarity of periodonteum is the arrangement of a high amount of ***oxitalan fibers***. They are situated in cervical and apical parts of tooth. Amount of oxitalan fibers is increased because of a hugh functional load. Oxitalan fibers can be connected with blood vessels and form “oxitalan-blood structures”. These fibers are part of the receptor mechanism of periodonteum which controls vessels.
There are also argyrophilic fibers in periodonteum of teeth especially in multirooted teeth.

Oxytalan fibers form bunches with the diameter 0,5-1,0 mkm. Their length reaches several millimeters. They are parallel to the root and situated near it or in the central part of periodontal area. Having vertical direction, they form a three dimentional network around the root that penetrates the bunches of collagen fibers perpendicularly. These fibers are most numerous in the area of the neck of a tooth. They are tangled into cementum but evidently they are not fixed on the bone, Oxytaian fibers are supposed to deform reversibly,

participate in regulation of blood flow corresponding to the function of the tooth,

Other well-formed fiber bundles intersect at right angles or splay around and between regularly arranged fiber bundles. Less regularly arranged collagen fibers are found in the interstitial connective tissue between the principal fiber groups; this tissue contains the blood vessels, lymphatics, and nerves.

Cellular elements

Four types of cells have been identified in the periodontal ligament: connective tissue cells, epithelial rest cells, defense cells, and cells associated with neurovascular elements.

Connective tissue cells include fibroblasts, cementoblasts, and osteoblasts. ***Fibroblasts*** are the most common cells in the periodontal ligament and appear as ovoid or elongated cells oriented along the principal fibers and exhibiting pseudopodia like processes. These cells synthesize collagen and have also been shown to possess the capacity to phagocytose, "old" collagen fibers and degrade them by enzyme hydrolysis. Thus, collagen turnover appears to be regulated by fibroblasts. Also they form connective capsule around the influammated region.

In physiological conditions when periodontal ligament is damaged, fibroblasts constantly replace each other due to different precursor cells having perivascular position, they migrate towards bone or cementum. Some fibroblasts of periodontal ligament turn into myofibroblasts in the process of differentiation. These cells are supposed to be able to play an essential part in dental eruption. The number of fibroblasts decreases in older people.

Cementoblasts are situated on external surface of root and produce secondary cellular cement. These cells are of various (more often cubic or branched) form. At the period of active cementum formation they form uninterupted layer producing cementoid or precementum (uncalcified organic cement matrix) that is subjected to mineralization

Osteoblasts produce bone tissue of alveoli. When they are active, they can form an uninterrupted layer and produce osteoid realizing its migration.

Osteoclasts and odontoclasts are big multinuclear cells of hematogenic origin with well developed lysosomic apparatus. They are situated in gaps on the surface of the bone and root of a tooth, thus destroying solid tissues. Odontoclasts are often called cementoclasts, but the first term is more preferable, because these cells participate in both cementum and dentin destruction.

The presence of osteoclasts and odontoclasts indicates resorption of tissues. And unlike osteoclasts, odontoclasts are not constant cellular element of periodontal ligament and cementum. They appear only during the resorption of roots in temporary teeth, under the influence of excessive forces upon the root during orthodontic replacement of a tooth, and at some pathalogic states connected with resorption processes in root tissues.

Labrocites (granular cells) are situated between blood vessels. There is a high amount of big granules inside labrocites. These granules produce biologically active substances like heparin and histamine. These substances regulate penetration of basic substance of periodonteum and carry out protective function.

Plasmatic cells are situated in cervical region of teeth. Often they can be found in marginal part of periodonteum. Their amount is increased if there is a chronic inflammatory process. They have oval or round form. These cells can produce antibodies. Granular cells are formed from plasmatic cells.

Macrophages (histiocytes) have round or irregular form. They can be free or fixed. They form collagen fibers in normal, but if there is pathological process they remove microorganisms and other harmful products. Macrophages, fat cells and leukocytes (mainly eosinophilic granulocytes, lymphocytes and monocytes) are contained mainly in interstitial connective tissue of periodontal ligament. They provide development and process protecting reactions. Their contents increase immediately during different inflammatory processes.

The epithelial rests of Malassez form a mesh-work in the periodontal ligament and appear as either isolated clusters of cells or interlacing strands, depending on the plane in which the microscopic section is cut. Epithelial Remenants of Melassez are formed in the period of root formation after decay of Hertwing's epithelial root sheath and epithelium of tooth plasty.

Epithelial cells originally form dense network surrounding root at the distance of 30-40 mkm., which gradually reduces. Epithelial tissues of recently erupted teeth are perforated cell layers that subsequently look like a network of epithelial bands. In grown up people this net finally decomposes and forms isolated epithelial islands (Remenants of Melassez), first they are numerous in the apical part of the root and in older age they are more numerous in the neck (where they can contact with epithelium of fixation). Epithelium of these residues is the most numerous in the second decade of human life, then their quantity reduces; the process of decay of epithelial bands network is very active in the period that lasts from 10 to 30 years.

There exists an opinion that epithelial cells of the residues interact with fibroblasts and they are able to produce some biologically active substances, glycosaminglicanes and enzymes, they can phagocytose and digest collagen fibrills. When epithelial Remenants of Melassez expand, they can be sources of cysts and malignant tumors development. During chronic inflammation in periodontal ligament surrounding tooth apex, epithelium expantion in the contents of cellular infiltrates (periapical granulomes), is seen in 90% of cases.

Little differentiated cells of mesenchimal origin are situated near small blood vessels. They are sources of cell's renovation in periodontal ligament. However its still unclear what is the common precursor for fibroblasts, osteoblasts, cementoblasts or if each line of cells has its own precursor. New formation and differentiation of cells in periodontal ligament are balanced by their migration from the ligament & their death.

The periodontal ligament may also contain calcified masses called ***cementicles,*** which are adherent to or detached from the root surfaces. Cementicles may develop from calcified epithelial rests; around cementum or alveolar bone traumatically displaced into the periodontal ligament; from calcified Sharpey's fibers; and from calcified, thrombosed vessels within the periodontal ligament.

Ground Substance

The periodontal ligament also contains a large proportion of ground substance filling the spaces between fibers and

cells. It consists of two main components: glycosaminoglycans such as hyaluronic acid and proteoglycans, and glycoproteins such as fibronectin and laminin. It also has a high water content (70%).

There are two kinds of glycosaminoglycans: acidic glycosaminoglycans (hialuronic acid, heparin) which carry out protective and trophic function; neutral glycosaminoglycans which carry out protective function (prevent penetration of microorganisms and toxins inside perodonteum).

Functions

The functions of the periodonteum are physical, formative and remodeling, nutritional, and sensory.

Physical Function

The physical functions of the periodontal ligament entail (1) provision of a soft tissue "casing" to protect the vessels and nerves from injury by mechanical forces, (2) transmission of occlusal forces to the bone, (3) attachment of the teeth to the bone, (4) maintenance of the gingival tissues in their proper relationship to the teeth, and (5) resistance to the impact of occlusal forces (shock absorption).

Resistance to the Impact of Occlusal Forces (Shock Absorption). Three theories relative to the mechanism of tooth support have been considered: the tensional theory, the viscoelastic system theory,and the thixotropic theory.

The tensional theory of tooth support ascribes to the principal fibers of the periodontal ligament the major responsibility in supporting the tooth and transmitting forces to the bone. When a force is applied to the crown, the principal fibers first unfold and straighten and then transmit forces to the alveolar bone, causing an elastic deformation of the bony socket; finally, when the alveolar bone has reached its limit, the load is transmitted to the basal bone. Many investigators find this theory insufficient to explain available experimental evidence.

The viscoelastic system theory considers the displacement of the tooth to be highly controlled by fluid movements, with fibers having only a secondary role. When forces are transmitted to the tooth, the extracellular fluid passes from the periodontal ligament into the marrow spaces of bone through foraminae in the cortical layer. These perforations of the lamina dura link the periodontal ligament with the

cancellous portion of the alveolar bone and are more abundant in the cervical third than in the middle and apical thirds.

After the depletion of tissue fluids, the fiber bundles absorb the tighten. This leads to blood vessels stenosis; arterial back-pressure causes ballooning of the vessels and passage of blood ultrafiltrates into the tissues, thereby replenishing the tissue fluids.

The thixotropic theory claims that the periodontal ligament has the rheologic behavior of a thixotropic gel (i.e., the property of becoming fluid, when shaken or stirred and then becoming semisolid again). The physiologic response of the periodontal ligament may be explained by changes in the viscosity of the biologic system. According to Schroeder, the presence of organized collagen fibers makes this theory untenable.

Transmission of Occlusal Forces to the Bone. The arrangement of the principal fibers is similar to a suspension bridge or a hammock. When an axial force is applied to a tooth, there is a tendency toward displacement of the root into the alveolus. The oblique fibers alter their wavy, untensed pattern, assume their full length, and sustaining the major part of the axial force. When a horizontal or tipping force is applied, there are two phases of tooth movement. The first is within the confines of the periodontal ligament, and the second produces a displacement of the facial and lingual bony plates. The tooth rotates about an axis that may change as the force is increased.

The apical portion of the root moves in a direction opposite to the coronal portion. In areas of tension, the principal fiber bundles are taut rather than wavy. In areas of pressure, the fibers are compressed, the tooth is displaced and there is a corresponding distortion of bone in the direction of root movement.

In single-rooted teeth, the axis of rotation is located in the area between the apical third and the middle third of the root. The root apex and the coronal half of the clinical root have been suggested as other locations of the axis of rotation. The periodontal ligament, which is shaped like a “sand glass”, is narrowest in the region of the axis of rotation. In multirooted teeth, the axis of rotation is located in the bone between the roots. In compliance with the physiologic mesial migration of the teeth, the periodontal ligament is thinner on the mesial root surface than on the distal surface.

Formative and Remodeling Function

Cells of the periodontal ligament participate in the formation of cementum and bone, which occur in physiologic tooth movement; in the accommodation of the periodontium to occlusal forces; and in the repairment of injuries. Variations in cellular enzyme activity are correlated with the remodeling process.

Cartilage formation in the periodontal ligament, although unusual, may represent a metaplastic phenomenon during repairment of this ligament after injury or a result of some toxins such as uranium.

The periodontal ligament is constantly undergoing remodeling. Old cells and fibers are broken down and replaced by new ones, and mitotic activity can be observed in the fibroblasts and endothelial cells. Fibroblasts form collagen fibers and may also develop into osteoblasts and cementoblasts. Therefore, the rate of formation and the differentiation of fibroblasts affect the rate of formation of collagen, cementum, and bone.

Radioautographic studies with radioactive thymidine, proline, and glycine indicate a very high turnover rate of collagen in the periodontal ligament. The rate of collagen synthesis, as established in the rat molar, is twice as fast that in the gingiva and four times as fast as that in the skin. There is also a rapid turnover of sulfated glycosaminoglycans in the cells and amorphous ground substance of the periodontal ligament.

It should be noted that most of these studies have been performed in rodents, and information on primates and humans is scarce.

Nutritional and Sensory Functions

The periodontal ligament supplies nutrients to the cementum, bone, and gingiva by way of blood and provides lymphatic drainage.

The periodontal ligament is abundantly supplied with sensory nerve fibers capable of transmitting tactile, pressure, and pain sensations by the trigeminal pathways. Nerve bundles pass into the periodontal ligament from the periapical area and through channels from the alveolar bone that follow the course of the blood vessels. They divide into single myelinized fibers, which ultimately lose their myelin sheaths and end in one of four types of neural termination: free endings (which have a tree-like configuration); Ruffini-like

corpuscles(located primarily in the apical area); coiled forms (found mainly in the midroot region); and spindle-like endings (which are surrounded by a fibrous capsule and located mainly in the apex).

2. Vascularization and innervation of supporting structures

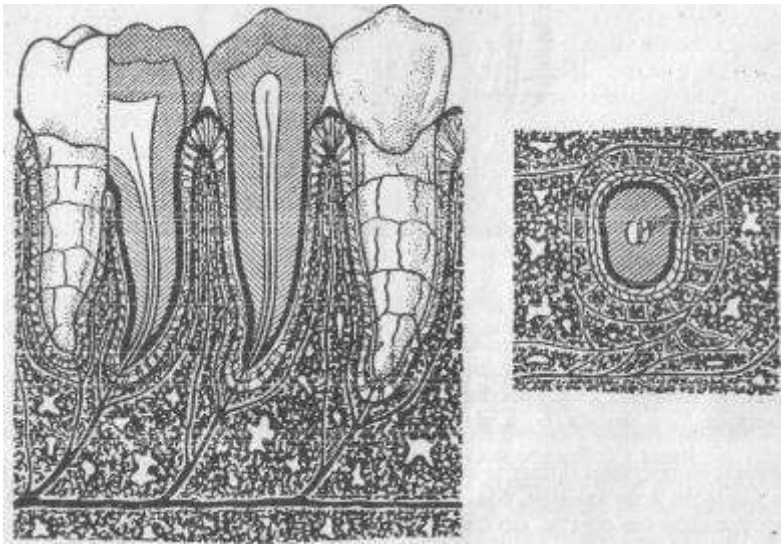
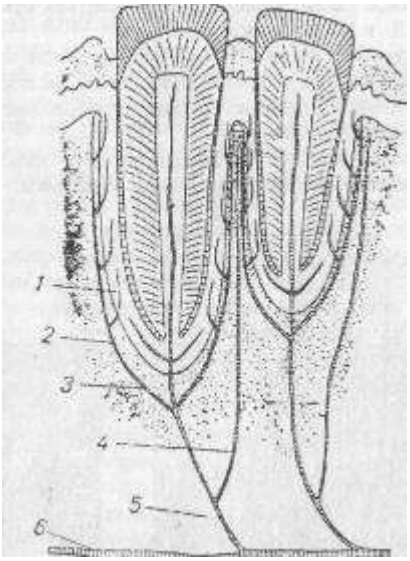


Fig. 4
Vascularization of periodonteum.

Periodontal ligament is characterized by intensive blood supply and thus high activity of renovation in cellular and non cellular components.The vascular supply to the bone enters the interdental septa via nutrient canals together with veins, nerves, and lymphatics. Dental arterioles, also branching off the alveolar arteries, send tributaries through the periodontal ligament, and some small branches enter the marrow spaces of the bone via the perforations in the cribriform plate. Small vessels emanating from the facial and lingual compact bone also enter the marrow and spongy bone.

The blood supply to the supporting structures of the tooth is derived from the inferior and superior alveolar arteries to the mandible and maxilla respectively, and reaches the periodontal ligament from three sources: apical vessels, penetrating vessels from the alveolar bone and anastomosing vessels from the gingiva. The apical vessels give off branches supplying the apical region of the periodontal ligament before entering the dental pulp. The transalveolar vessels are branches of the intraseptal vessels that perforate lamina dura and enter into the ligament. The intraseptal vessels continue on to vascularise the gingiva; these gingival vessels in turn anastomose with the periodontal ligament vessels of the cervical region.



Pic. 5 Scheme of vascularization.
1 - rami perforantes alveolaris, 2 - a. periodontalis longitudinalis, 3 - rami interalveolaris, 4 - a. interalveolaris, 5 - a. dentalis, 6 - a. alveolaris. (по И.Г. Лукомскому).

The main sources of blood supply are the upper and the lower alveolar arteries.

The major part of arterioles (with a diameter less than 100 mkm), that penetrate it from medullary areas of interdental and inter root parts of alveolar processus through bone foramens (Volkman canals), situated at different levels of alveolus. Such alveoli are more in the posterior teeth than in frontal teeth and more in lower than upper teeth.

Blood is also supplied through the branches of dental artery . These branches go from periapical part of the ligament towards the gum and the branches of supraperiostal arteries in mucous membrane cover alveolar processes.

The comunication of the vessels in periodontal ligament with pulpal vessels going through additional root foramens is very important because of the possible ways of spread of infection.

The vessels within the periodontal ligament are contained in interstitial spaces of loose connective tissue between the principal fibers and are connected in a net-like plexus that runs longitudinally and closer to the bone than to the cementum. The blood supply increases from the incisors to the molars; is it’s highest in the gingival third of single-rooted teeth, less in the apical third, and least in the middle; is equal in the apical and middle thirds of multirooted teeth; is slightly greater on the mesial and distal surfaces than on the facial and lingual; and is greater on the mesial surfaces of mandibular molars than on the distal.

Venous Drainage

The venous drainage of the periodontal ligament accompanies the arterial supply. Venules receive the blood via the abundant capillary network; there are also arteriovenous anastomoses that bypass the capillaries. These are more frequent in apical and interradicular regions, and their significance is unknown.

Lymphatic drainage

The lymphatic system of periodontal ligament is weakly developed. Thin walled lymphatic capillaries begin blindly in the friable fibrous connective tissue. They go to collective lymphatic vessels that have valves and go together with veins, some of these veins are directed towards gum, and other veins perforate.

Lymphatics, supplement the venous drainage system. Those draining the region just beneath the junctional epithelium pass into the periodontal ligament and accompany the blood vessels into the periapical region. From there they pass through the alveolar bone to the inferior dental canal in the mandible or the infraorbital canal in the maxilla and then to the submaxillary lymph nodes.

Innervation of periodonteum.

Periodontal ligament is innervated with both afferent and efferent fibers. Afferent nerves have 2 sources. Peripheric branches moving away from the dental nerve up to its entrance into apical foramen form the first source. These fibers reach gingiva. Nerve branches penetrating foramens of inter dental and inter root bone septum (Volkmann's canals) and directed towards the apex of root or that of crown form the second source of afferent fibers. The fibers of both sources are mixed forming nerve plexus of periodontal area. It includes the thick bunches of fibers that are parallel to the long axis of root and also the thin bunches. Terminal branches and separate fibers move away from thin bunches.

Nerve endings are mostly mechanical and pain receptors. They are oval incapsulated corpucles, plastic, fusiform and leaf like structures or more often thin dendrite branched free endings. The highest concentration of nerve endings is typical in the area of root apex except upper incisors. Their endings are distributed with equally high density in the apical and adjacent to the crown parts of root.

3. **Classifications of periodontitis:**

Classification by .G.Lukomsky (1955) according to pathologic physiological and morphological changes in periodontium during inflammation:

1. Acute periodontitis:
    a. acute serous
    b. acute purulent
2. Chronic periodontitis:
    a. chronic fibrous
    b.chronic granulating
    c.chronic granulematous
3. Acute chronic periodontitis
4. Radicular cyst.

Numerous terms and classifications have been used to describe apical periodontitis. Periapical lesions, apical granuloma/cysts, periapical osteitis, and periradicular lesions are frequently used synonyms. Although the term periradicular includes inflammation of the furcational and lateral locations, it does not distinguish pulp derived periodontitis from marginally spreading lesions. The limitations of the various names and the arguments for using the term apical periodontitis have been discussed elsewhere.

Because apical periodontitis is an inflammatory disease, it can be classified on the basis of symptoms, etiology, histopathology, and so on. The World Health Organization classified apical periodontitis under “diseases of periapical tissues” and subdivided it into several categories based on clinical signs.

However, this useful classification system does not take into account the structural aspects of the desceased tissues. Because the structural framework forms the basis for an understanding of the disease process, a histopathologic classification is used here. (Table 1). This system is based on four factors: (1) the distribution of inflammatory cells in the lesion; (2) whether epithelial cells are present or absent; (3) whether the lesion has been transformed into a cyst; and (4) the relationship of the cyst cavity to the root canal of the

affected tooth. Following definitions which are based on structure differ from clinical definitions.

World Health Organization Classification of Diseases of Periapical Tissues (1995)

Table 1

Code number	Category
K04.4	Acute apical periodontitis
K04.5	Chronic apical periodontitis (apical granuloma)
K04.6	Periapical abscess with sinus (sentoalveolar abscess with sinus, periodontal abscess of pulpar origin)
K04.60	Periapical abscess with sinus to maxillary antrum
K04.61	Periapical abscess with sinus to nasal cavity
K04.62	Periapical abscess with sinus to oral cavity
K04.63	Periapical abscess with sinus to skin
K04.7	Periapical abscess without sinus (dental abscess without sinus, dentoalveolar abscess without sinus, periodontal abscess of pulpar origin without sinus)
K04.8	Radicular cyst (apical periodontal cyst, periapical cyst)
K04.80	Apical and lateral cyst
K04.81	Residual cyst
K04.82	Inflammatory paradental cyst.

Acute apical periodontitis is an acute inflammation of the periodontium of endodontic origin; it is characterized by the presence of a distinct focus of neutrophils in the lesion. It is said to be primary, or initial, when the inflammation is short-lived and starts in a healthy periodontium in response to irritants. It is labeled secondary, or exacerbating, when the acute response occurs in an already existing chronic apical periodontal lesion. This latter form is also reffered to as periapical flare-up, or phoenix abscess. The lesions can be epithelialized or nonepithelialized.

Chronic apical periodontitis is a long-standing inflammation of the periodontium of endodontic origin; it is characterized by the

presence of granulematous tissue predominantly infiltrated with lymphocytes, plasma cells, and macrophages. The lesions can be epithelialized or nonepithelialized.

Periapical true cyst is an apical inflammatory cyst with a distinct pathologic cavity completely enclosed in an epithelial lining so that no communication to the root canal exists.

A periapical pocket cyst is an apical inflammatory cyst containing a saclike, epithelium-lined cavity that is open to and continuous with the root canal. **(Pathways of the pulp. 9th edition. Stephen Cohen, Kenneth M. Hargreaves. Mosby, 2006.)**

According to etiologic sign:

a. infectious
b. traumatic
c. drug-induced, toxic

4. **Aetiology and pathogenesis of perodontitis**

Aetiology of periodontitis:

1. ***Infection;***

Microorganisms can reach the dental pulp via several routes. Openings in the dental hard tissue wall caused by caries, clinical procedures, or trauma-induced fractures and cracks are the most common origins of pulpar infection. However, microbes also have been isolated from teeth with necrotic pulps and apparently intact crowns. Endodontic infections of such teeth are preceded by pulp necrosis. Some have suggested that bacteria from the gingival sulci or periodontal pockets might reach the root canals of these teeth through blood vessels of the periodontium. However, microorganisms are unlikely to survive the immunologic defenses between the marginal gingival and apical foramen.

An alternative possibility is that the teeth, although clinically appearing intact, may have microcracks in the hard tissues that allow entry of bacteria. Pulp infection also can occur through exposed dentinal tubules at the cervical root surface, as a result of gaps in the cemental coating. Some suggested that bacteria remaining in infected

dentinal tubules may be a potential reservoir for endodontic reinfection.

Other researches claim that microbial infection reaches the necrotic pulp via the general blood circulation, a process called anachoresis. However, when the bloodstream was experimentally infected, bacteria could not be recovered from the root canal system unless the root canals were overinstrumented during the period of bacteremia. Evidence that further discredits anachoresis as a potential source of necrotic pulp infection comes from a study by MÖller and others in which all experimentally devitalized pulps (n=26) in monkeys remained sterile for longer than 6 months.

Ecomorphology of endodontic flora

An infected necrotic root canal system acts as a selective habitat for the causative organisms. Morphologically, the flora consists of a mixed microbial population of cocci, rods, spirochetes, and long filamentous organisms. Numerous dividing forms of cocci, rods, and yeast cells generally can be identified by TEM; these forms are a sign of organism's vitality at the time of fixation. However, these organisms are not uniformly distributed throughout the canal. The microbes can exist as aggregates of one microbial type, coaggregates of several forms, and planktonic cells, suspended in the fluid phase of the infected necrotic root canal. However, the undisturbed intracanal flora of an infected tooth with primary apical periodontitis is mostly organized as a matrix-embedded collection of multispecies of organisms in microecosystems that may be immobilized on the dentinal wall.

In contemporary microbiological terms, this means that apical periodontitis is caused by an intraradicular accumulation of certain microorganisms that are ecologically organized into protected sessile biofilms composed of cells embedded in a hydrated exopolysaccharide complex that cannot be eradicated by host defenses.

2. ***Trauma*** (by endodontic instruments, injury, filling material, post, harmful habits, pathology of orthopedic treatment, complications after filling of crown)

Chronic traumatic periodontitis may be caused by slight but repeated trauma, by wrong applied filling , orthogenic (direct) occlusion, by regular pressure of a cigar mouth-piece or wind

instruments, by bad habits (cross-biting threads, pressing the tooth with a pencil and so on). Chronic inflammatory process forms only when vascular-nervous fascicle is injured alongside with the periodontal injury during a trauma.

3. **Medicaments** (arsenic, formaline, phenol)
4. **Allergy** (antibiotics, sulfanilamides)
5. **Several factors.**

Pathogenesis and histopathology

Infectious periodontitis develops as a result of immediate introduction of pathogenic microorganisms into periodontium from the dental canal, rarely by hematogenic and lymphogenic way, more seldom inflammation displaces to periodontium as a result of pathologic process in the alveolar appendage from adjacent pathologic foci of the teeth. At pulp's gangrene, accompanied by long - term periodontium's irritation, acute apical periodontitis is in fact an exacerbated chronic periodontitis (Weis 1965). Acute apical periodontitis lasts from 2-3 days till 2 weeks and in its course, one may single out 3 stages each requiring a different treatment.

The first stage is characterized by hyperemia (vascular impairments), by vascular dilation, edema of periapical part of periodontium. Sometimes bleedings occur. Leukocytic infiltration is scarcely expressed. Clinical pains are absent and appear only at hot food intake. The patient feels heavy sensation, tension in the tooth. The tooth seems to be longer, than others, it is painful to bite on it.

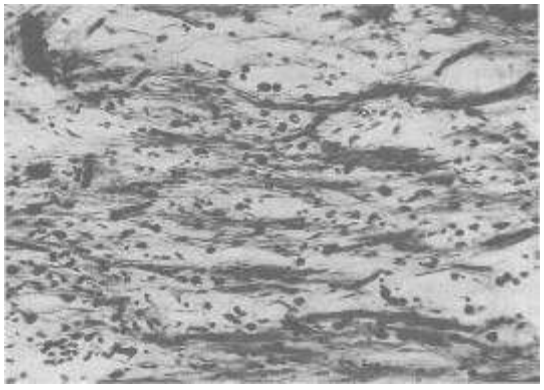


Fig.6 Leukocytic infiltration of periodontium (acute apical periodontitis, phase of exudation).

The second stage is serous inflammation. The edema of periapical part of periodontium is apparently expressed, loose connective tissue is impregnated by

serous exudates, fiber bundles (fascicles) are loosened. There are many bleeding foci, central leukocytic and hystiocytic infiltration.

The process spreads through an opening in the internal alveolar foramina, on haversian canals, reaching periosteum and gingival mucous membrane. Clinical pain is constant. It enhances while biting on the injured tooth.

The third stage is purulent. Leukocytic infiltration in medullary cavity is evidently expressed. Subsequently purulent liquefaction of tissues occurs.

Acute traumatic periodontitis may be caused by a single trauma impact on periodontium (bruise, hurt, injury). Trauma may be inflicted at pulp's extirpation by a pulp-extractor, any other instruments at root canal treatment. Traumatic periodontitis may occur at invasive treatment of the tooth, at wrong made crown.

Mechanisms of traumatic periodontitis development are various, depending on whether physical destruction of periodontal tissues took place or only their irritation.

Structures and reactions resistant to trauma are typical of periodontium. They provide compensation of disturbances caused by a trauma. That is why, isolated traumatic injury of apical periodontium is connected with the development of apical inflammation. Acute drug - induced toxic periodontitis develops at overdosage or long term action of arsenic paste, after application of formalin-containing paste on the pulp's stump, irrigation of the canals with strong antiseptic solutions , also at using toxic filling pastes. To drug-induced periodontitis we also refer periodontitis developed as an allergic reaction. It occurs when antibiotics are introduced into the root canal, also during sulfonamides and some antiseptics introduction. Drug-induced toxic periodontitis is also observed at deep carious cavities filling with quickly hardening plastics, cements without isolating liners. Changes in periapical tissues may occur at periapical tissues damage caused by drugs. Arsenic acid damages protoplasm of cells. If the time of arsenic acid is a little prolonged, acute inflammatory reaction develops in periapical tissues. If arsenous acid action lasts for several days, it entails necrosis of periapical tissues.

Strong antiseptics of low concentration cause irritation of apical periodontium, which leads to the development of acute inflammatory reaction.

The microbial and host factors outlined previously form the background for developing a cohesive view of the pathogenesis of apical periodontitis and the shifting nature of the histopathology of various classes of lesions. The structural components of the lesions depend on the balance at any time between the microbial factors and the host defenses. The morphologic description of apical periodontitis based on a zonal pattern (originally described for inflammation induced in bone) does not seem to represent the structural variation seen in most periapical lesions. In fact, great structural heterogenity is the norm for apical periodontitis, particularly among chronic lesions.

Initial apical periodontitis

Usually is caused by microorganisms or their biproducts residing in or invading the periapical tissue from the apical root canal. However, it also may be induced by accidental trauma, injury from instrumentation, or irritation from chemicals or endodontic materials; any of these mechanisms can provoke an intense, short-lived host response that may be accompanied by clinical symptoms such as pain, tooth elevation in some cases, and tenderness to pressure on the tooth. Such initial, symptomatic lesions are viewed as the acute apical periodontitis (acute periradicular periodontitis). It should be understood that, except for the outcome, no clear clinical distinction exists between aseptic and infectious inflammation.

Histopathologically, tissue changes generally are limited to the apical periodontal ligament and the neighboring spongiosa. These changes are characterized by hyperemia, vascular congestion, edema of the periodontal ligament, and extravasation of neutrophils. Neutrophils are attracted to the area by chemotaxis, induced initially by tissue injury, bacterial products, and complement factor. Because the integrity of the hard tissues (bone, cementum, and dentine) has not yet been disturbed, the periapical changes are undetectable radiographically. If some noninfectious but irritating agents have induced the inflammation, the lesion may subside and the structure of the apical periodontium will be restored by healing.

When infection occurs the neutrophils do not only attack and kill the microorganisms but also release leukotrienes and prostaglandins. There is much amount of neutrophils and macrophages transport into the area, and the macrophages activate osteoclasts. In a few days the

bone surrounding the periapex can be resorbed, and a radiolucent area may be detectable at the periapex. This initial, rapid bone resorption can be prevented by administration of indomethacin, which inhibits cyclooxygenase and thus suppresses prostaglandin synthesis. Neutrophils die in great numbers at the inflammatory site and release enzymes from their "suicide bags" causing destruction of the extracellular matrices and cells. The self-induced destruction of the tissues in the "battle zone" prevents the spread of infection to other parts of the body and also provides space for the development of reinforcements, arriving in the form of more specialized defense cells as the battle becomes a protracted war.

During later stages of acute response, macrophages begin to appear at the periapex. Activated macrophages produce a variety of mediators; of these, the proinflammatory and chemotactic cytokines are particularly important at this stage. These cytokines intensify the local vascular response, osteoclastic bone resorbtion, and effector-mediated degradation of the extracellular matrices and can put the body on general alert by endocrine action to sharply raise the output of acute-phase proteins and other serum factors by hepatocytes.

The acute response can be intensified, particularly in later stages, by the formation of antigen-antibody complexes. The acute early lesion may take any of several possible courses: spontaneous healing may occur; the infection may intensify and spread into the bone (alveolar abscess); the lesion may open to the exterior (fistula or, more appropriately, sinus tract formation); or it may become chronic.

Chronic apical periodontitis

If the irritants (e.g., bacteria or their products) are not removed, the neutrophil-dominated early lesion gradually shifts to a macrophage-, lymphocyte-, and plasma cell-rich lesion encapsulated in a collagenous connective tissue. To use a battlefield analogy, this type of asymptomatic, radiolucent lesion can be seen as a "lull" after the intense, high-casualty battle in which neutrophils died in great numbers but the invaders into the periapex were temporarily beaten and the enemy front held back in the root canal.

The macrophage-derived proinflammatory cytokines are powerful lymphocyte stimulators. Although the quantitative data on the various types of cells found in chronic periapical lesions probably are far from representation, investigations based on monoclonal

antibodies tend to suggest a predominant role for T lymphocytes and macrophages. Activated T cells produce a variety of cytokines that down-regulate the output of the proinflammatory cytokines, leading to suppression of osteoclastic activity and reduced bone resorbtion. On the other hand, the T cell-derived cytokines may concomitantly up-regulate the production of connective tissue growth factors, with stimulatory and proliferative effects on fibroblasts and the microvasculature. T-helpers population may participate in this process.

The option to down-regulate the destructive process explains the absence of retarded bone resorbtion and the rebuilding of the collagenous connective tissue during the chronic phase of the disease. Consequently, chronic lesions can remain “dormant” and symptomless for long periods without major changes in the radiographic status. However, at any time the delicate equilibrium prevailing at the periapex can be disturbed by one or more factors that may favor the microbes in the root canal. The microbes advance into the periapex, and the lesion spontaneously becomes acute with recurrence of symptoms) exacerbating apical periodontitis, phoenix abscess). As a result, microorganisms can be found extraradicularly during these acute episodes with the possibility of rapid enlargement of the radiolucent area. This characterictic radiographic feature is the result of apical bone resorbtion that occurs rapidly during the acute phases, with relative inactivity during the chronic periods. The disease therefore does not progress continuously but rather in discrete leaps after periods of “stability”.

Chronic apical periodontitis used to be referred to as dental or periapical granuloma. Histopathologically, it consists of a granulomatous tissue with infiltrate cells, fibroblasts, and a well-developed fibrous capsule. Serial sectioning shows that about 45% of all chronic periapical lesions are epithelized. When the epithelial cells begin to proliferate, they may do so in all directions at random, forming an irregular epithelial mass in which vascular and infiltrated connective tissue becomes enclosed. In some lesions the epithelium may grow into the entrance of the root canal, forming a pluglike seal at the apical foramen. The epithelial cells generate an “epithelial attachment” to the root surface or canal wall, which under TEM reveals a basal lamina and hemidesmosomal structures. In random

histologic sections, the epithelium in the lesion characteristically appears as arcades and rings. The extraepithelial tissue consists predominantly of small blood vessels, lymphocytes, plasma cells, and macrophages. The connective tissue capsule consists of dense collagenous fibers that are filmly attached to the root surface, which means the lesion may be removed in to with the extracted tooth.

Cystic apical periodontitis

Periapical or radicular cysts generally are considered a direct sequela to chronic apical periodontitis, but not every chronic lesion develops into a cyst. Although the reported incidence of cysts among apical periodontitis lesions varies from 6% to 55%, investigations based on meticulous serial sectioning and strict histopathologic criteria show that the actual incidence of the cysts may be well below 20%. As was stated previously, radicular cysts are divided into two distinct categories: those that contain cavities completely enclosed in epithelial lining and those that contain epithelial-lined cavities that are open to the root canals. The latter type, originally described as a “bay cyst”, now is called a periapical pocket cyst.

More than half of cystic lesions are apical true cysts, and the remainder are apical pocket cysts. In view of the structural difference between the two types, the pathogenic pathways leading to their formation may differ in certain respects.

Periapical true cysts

The process of true cyst formation has been discussed as occurring in three stages. During the first phase, the dominant cells “rests of Malassez” are believed to proliferate, probably under the influence of growth factors released by various cells in the lesion. During the second phase, an epithelium-lined cavity forms. Two long-standing hypothesis exist regarding the formation of the cystic cavity. The “nutritional deficiency theory” is based on the assumption that the central cells af the epithelial strands are deprived from their source of nutrition and consequently undergo necrosis and liquefactive degeneration. The accumulating products attract neutrophilic granulocytes into the necrotic area. Such microcavities containing degenerating epithelial cells, infiltrating leukocytes, and tissue exudate coalesce to form the cystic cavity lined by stratified squamous epithelium. The “abscess theory” postulates that the proliferating epithelium surrounds an abscess formed by tissue necrosis and lysis

because of the inherent nature of epithelial cells to cover exposed connective tissue surfaces.

During the third phase of true cyst formation, the cyst grows by same mechanism that has not yet been adequately clarified. Theories based on osmotic pressure have receded to the background in recent years, and research attention has shifted in favor of finding a molecular basis for the cystogenesis.

The fact that the apical pocket cyst, which has a lumen open to the necrotic root canal, can grow eliminates osmotic pressure as a potential factor in the development of radicular cysts. Although no direct evidence is yet available, the tissue dynamics and the cellular components of radicular cysts suggest possible molecular pathways for cystic expansion. The neutrophils perishing in the cystic lumen provide a continuous source of prostaglandins, which can diffuse through the porous epithelial wall into the surrounding tissues. The cell population in the extraepithelial area includes numerous T lymphocytes and macrophages, which are known to produce a battery of cytokines. The prostaglandins and the inflammatory cytokines can activate osteoclasts, culminating in bone resorption. The presence of effector molecules also has been reported in human periapical cysts.

Histopathologically, an apical true cyst has four major components: cyst cavity, epithelial cyst wall, the extraepithelial tissue, and the collagenous capsule. The cavity, completely enclosed in an epithelial lining, generally reveals necrotic tissue and sometimes cholesterol clefts and erythrocytes (the presence of the erythrocytes probably is due to hemorrhage). The thickness of the stratified squamous epithelium can vary from a few cell layers to several layers. Scanning electron microscopy of the inner surface of the cyst wall reveals flat epithelial and globular cells (i.e., the surface of the epithelium and neutrophils protruding through the intercellular spaces). The basal cell side of the epithelium is irregular, forming ridges. The tissue between the epithelial lining and the fibrous capsule usually consists of numerous blood vessels and infiltrating cells, predominantly T lymphocytes, B lymphocytes, and plasma cells. Neutrophils, which are numerous in the epithelial lining are rarely found in the extraepithelial area.

Periapical pocket cyst

Periapical pocket cysts contain an epithelium-lined pathologic cavity that is open to the root canal of the affected tooth. A periapical pocket cyst probably is initiated by the accumulation of neutrophils around the apical foramen in response to the bacteria in the apical root canal. Biologically, a pocket cyst constitutes an extension of the infected root canal space into the periapex. The microluminal space becomes enclosed in a stratified squamous epithelium which grows and forms an epithelial collar and around the root tip. The epithelial collar forms an "epithelial attachment" to the root surface, sealing off the infected root canal and the microcystic lumen from the periapical milieu and the rest of the body. Microorganisms at the apical foramen attract neutrophilic granulocytes into the microlumen by chemotaxis. However, the pouchlike lumen, biologically outside the body milieu, acts as a "death trap" for the external neutrophils. As the necrotic tissue and microbial products accumulate, the saclike lumen enlarges to accommodate the debris, forming a voluminous diverticulum of the root canal space into the periapical area. **(Pathways of the pulp. 9th edition. Stephen Cohen, Kenneth M. Hargreaves. Mosby, 2006.)**

The scheme of pathogenesis of periodontitis: microorganisms+endotoxines -----degranulation of labrocytes (histamine, heparin) +++activation of system of complement (penetration of vessels increases) ------ number of macrophages and lymphocytes increases ----- they produce lysosomal enzymes ----- activation of osteoclasts ------ destruction of periodont.

Arsenic ----- destruction of protoplasm of cells
Strong antiseptics ----- coagulation of periodont

5. Pathophysiology of periodontitis

Acute serous periodontitis (K04.4): Periodontium is thick, swollen. Inflammation hyperemia is seen under microscope. There is infiltration by leucocytes. There are histiocytes andlymphocytes in the regions of infiltration. Blood vessels are dilated.

Acute purulent periodontitis (K04.4): Periodontium is swollen. Periodontium's tissue is impregnated with exudate. There are perivascular infiltrates. After this small abscesses are formed. Purulent region appears in the center of which there is a nonstructural mass.

There are signs of destruction in bone near periodontium. Superficial layers of cementum are resorbed. Quick resorption of bone tissue causes penetration of pus under periosteum. Soft tissues are also involeved in inflammated process. Some scientists are sure that purulent periodontitis is the initial stage of osteomyelitis.

Chronic fibrous periodontitis (K04.5): periodontium is thick, there is hypercementosis of alveoli. There is little amount of cells, but much amount of fibers in the healthy part of periodontium.

Chronic granulating periodontitis (K04.5): root apex is without soft tissues, dark grey colour. At the beginning of inflammation there is much amount of leukocytes in exudates, after some time – hystiocytes. Cementum, dentin and bone are destroyed.

Chronic granulematous periodontitis (K04.5): proliferated process is regional. At the beginning of formation of granulated tissue in the region of apex there are lymphocytes. There is much amount of cells, collagen fibers are destroyed. Granulated tissue destroys bone and a cavity with bone cells is formed. Fibrous capsule is formed because of collagen fibers growth. Cells of granuloma: plasmatic cells, leukocytes, epithelial cells and fibroblasts and others.

6. Clinical manifestations and differential diagnostics

The basic symptom of acute apical periodontitis is constant localized pain. Character of pain and other signs of disease depends on type of exudate: serous or purulent. Clinical manifestations depends on phase of acute inflammation. There are two main phases.

6.1. Clinical picture of acute apical periodontitis (K04.4)

There are two phases of the acute process: intoxication and exudation. Phase of exudation is divided into serous and purulent.

Phase of intoxication.

Patient complains on constant localized pain increasing during mastication and touch of the tooth. Patient can show this tooth.

The tooth was treated before or there was carious cavity during long time (history of disease). Patient was sick or there were other reasons of decrease of immunity (history of life).

Objectively: face is symmetric, oral mucous near the affected tooth is pale pink, tooth is not changed in color. A deep carious cavity (or tooth under filling), formed by pigmented dentine, communicating

with a dental cavity. Probing and reaction to cold are painless. Percussion is weak painful. There are no changes on X-ray. EOD is 100 mA.

Phase of exudation. In this stage complains depend on character of exudate.

Serous exudates: Patient complains on spontaneous, constant, localized pain, which increases during mastication and touch of the tooth. Patient can show the affected tooth. There is "sense of growing tooth".

Objectively: Body temperature is normal. General condition is normal. Face is symmetric. Lymphatic nodes are normal or slightly increase on the side of affected tooth, painless during palpation and mobile. There is hyperemia and oedema of oral mucous near affected tooth (not always), it's palpation is painful. Symptom of vasoparesis is positive. Tooth is mobile, tooth is changed in color. If there is carious cavity it is communicated with pulp cavity. Probing of this cavity and reaction to cold are painless. Percussion is painful.

The noted symptom of vasopressor according to Lukomsky: excavation remains after pressing gingiva with the head of condenser, this excavation is surrounded with the areas of anemia which quickly gets bright red colour.

Serous phase of acute periodontites can turn into **purulent** which is continued about 20 days.

Patient complains on pulsating, irradiating constant pains, increasing at a slight touch of the tooth (so patient doesn't close mouth). General weakness, headache and raised body temperature are observed.

The tooth was treated before or there was carious cavity for long time (history of disease). Patient was sick or there were other reasons of decrease of immunity (history of life).

Objectively: Face is asymmetric. Gingiva in the area of affected tooth and neibouring teeth is edematous and hyperemic, palpation intensifies pain. Regional lymphatic nodes are enlarged and tender at palpation. The tooth is mobile and changed in color. A deep carious cavity, formed by a pigmented dense dentine, is communicated with a dental cavity widely (under filling). Probing and reaction to cold is painless. Percussion is strong painful. There is widening of periodontal space on X-ray. EOD is 100 mA. In severe cases the

general reaction is pronounced fever, intoxication and sometimes weak heart activity, leukocytosis with left shift increase in speed of erythrocyte sedimentation (ESR) can be observed.

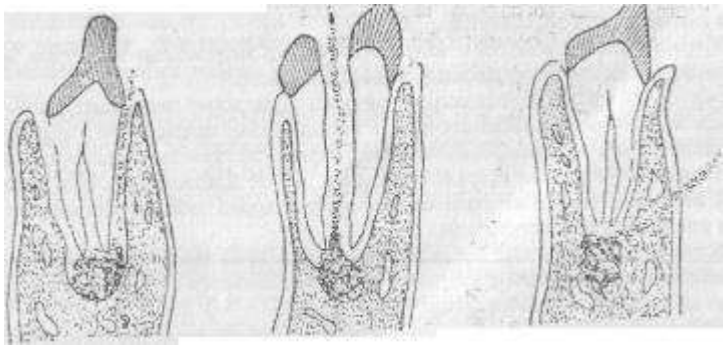


Fig. 7 Outflow of purulent exudate.
A – through periodontium;
B – through root canal;
C – through fistula.

When purulent exudate comes under periosteum pain is decreased. Patient complains on pain in jaw, general weakness. So periostitis or sometimes phlegmon or osteomyelitis is formed. The above described phenomena increase with accumulation of purulent exudate in the area of root apex. At this stage of purulent apical periodontitis a patient needs urgent help. If there is no help, exudate "searches for an exit" out of the closed periapical area. The process which is excruciating for a patient can last for several days.

This is the stage of greater tension, when all signs of disease reach the climax. As soon as purulent exudate finds way out, usually through bone under periosteum (other ways of purulent exudate drainage are seldom observed in clinical practice), both the symptoms and pain syndrome get weaker. When exudate finds way out, the patients sometimes stop complaining of toothache, body temperature reduces, sometimes normalizes. At this stage of apical periodontitis when percussion may seem painless. But palpation remains painful and it shows subperiosteal or subgingival abscess. The stage of a patient distinctly improves.

Subsequently the course of disease depends on the part of jaw or submandibular area where purulent exudate expands. Localization of subperiosteal abscess depends on anatomo-topographical peculiarities of affected area in alveolar bone and first of all it depends on the affected tooth. When central incisors and canines are affected, abscess is formed in the labial sides, and it is formed in the palatal side when upper lateral and palatal roots of molars are affected. It should be

taken into consideration that in case of acute purulent apical periodontitis, abscess of facial soft tissues and oral cavity fundus can develop. As a rule acute purulent apical periodontitis results in chronic inflammation.

Differential diagnostics of acute serous periodontitis (K04.4):

Table 2

Sign	Acute serous periodontitis	Acute purulent periodontitis	Exacerbation of chronic periodontitis	Acute diffuse pulpitis
Complaints	Constant localized pain increasing during mastication and touch of the tooth.	General weakness, headache, raised body temperature are observed.Pulsating, irradiating constant pains, enhancing at a slight touch of the tooth.	Depend on stage of acute process (serous or purulent). Pain during mastication. Sense of grow tooth.	Spontaneous, cramping pains (with long painful periods and short painless periods), night pains, long pains caused by all types of irritators, long-term pains, irradiating pain.
History of disease	After eruption of teeth	After eruption of teeth	After eruption of teeth. Tooth was treated or there were such exacerbations	After eruption of teeth. Patient has such complaints more than 2 days.
History of life	Involvement increases with a decrease of fluorine in drinking water	Involvement increases with a decrease of fluorine in drinking water	Involvement increases with a decrease of fluorine in drinking water	Involvement increases with a decrease of fluorine in drinking water

Objectively	Tooth is not changed in color. A deep carious cavity, formed by pigmented dentine, communicating with dental cavity.	Face is asymmetric. The mucous membrane is hyperemic, swollen, fluctuation is determined. Regional lymph nodes are enlarged and tender at palpation. The tooth is mobile, is changed in color. A deep carious cavity, formed by pigmented dense dentine, communicating with dental cavity widely (can be under filling).	Hyperemia and oedema of oral mucous near affected tooth. Tooth is changed in color. A deep carious cavity, formed by pigmented dentine, communicating with dental cavity. There can be filling. **Symptom of vasoparesis** is positive. Regional lymph nodes are enlarged and tender at palpation.	A deep carious cavity, formed by softened light dentine, not communicating with dental cavity.
Probing	Painless	Painless	Painless	Painful on the whole bottom.
Reaction to cold	Painless	Painless	Painless	Painful long-term.

Percussion	Painful	Strong painful	Painful	Painless
X-ray	-	-	Changes which characterize form of chronic periodontitis.	-
EOD	100 mA	100 mA	100 mA	25-30mA

Differential diagnostic of acute purulent periodontitis (K04.4):

Table 3

Sign	Acute purulent periodontitis	Acute serous periodontitis	Exacerbation of chronic periodontitis	Acute odontogenic osteomyelitis	Purulent cyst
Complaints	General weakness, headache, raised body temperature are observed. Asymmetry of face. Pulsating, irradiating constant pains, enhancing at a slight touch of the tooth.	Constant localized pain increasing during mastication and touch of the tooth.	Depend on stage of acute process (serous or purulent). Pain during mastication and percussion. Sense of grow tooth.	General weakness, headache, raised body temperature (39-40) are observed. Asymmetry of face. Pain in jaw. Pain during mastication and percussion.	General weakness, headache, raised body temperature are observed. Asymmetry of face.
History of disease	After eruption of teeth	After eruption of teeth	After eruption of teeth. Tooth was treated or there were such	After eruption of teeth.	After eruption of teeth

			exacerbations		
History of life	Involvement increases with a decrease of fluorine in drinking water	Involvement increases with a decrease of fluorine in drinking water	Involvement increases with a decrease of fluorine in drinking water	Involvement increases with a decrease of fluorine in drinking water	Involvement increases with a decrease of fluorine in drinking water
Objectively	Face is asymmetric. The mucous membrane is hyperemic, swollen, fluctuation is determined. Regional lymph nodes are enlarged and tender at palpation. The tooth is mobile, changed in color. A deep carious cavity, formed by	Tooth is changed in color. A deep carious cavity, formed by pigmented dentine, communicating with dental cavity.	Oral mucous near this tooth is hyperemic. Tooth is changed in color. A deep carious cavity, formed by pigmented dentine, communicating with dental cavity. There can be filling. Symptom of vasoparesis is positive. Regional lymph nodes are	The mucous membrane is hyperemic, swollen, fluctuation is determined (buccal and lingual sides are affected). Teeth are mobile. A deep carious cavity, formed by softened light dentine, not communicating with dental cavity. Regional lymph nodes are	The mucous membrane is hyperemied, swollen, fluctuation is determined (buccal and lingual sides are affected). Teeth are mobile. A deep carious cavity, formed by softened light dentine, not communicating with dental

	pigmented dense dentine,communicating with dental cavity widely (under filling).		enlarged and tender at palpation.	enlarged and tender at palpation.	cavity. Regional lymph nodes are enlarged and tender at palpation. There is **Vensane symptom** (loss of sensitivity of bone and oral mucous near affected tooth.
Probing	Painless	Painless	Painless	Painless	Painless
Reaction to cold	Painless	Painless	Painless	Painless.	Painless
Percussion	Strong painful	Painful	Painful	Painful (and teeth which are near affected tooth)	Painful (and teeth which are near affected tooth)
X-ray	-	-	Changes which characterize form	Regions of resorption of	Cyst with destruction of

			of chronic periodontitis.	bone	bone tissue near the cyst
Electroexcitability	100 mA	100 mA	100 mA	100 mA	100 mA

Acute periodontitis can be differentiated with periostitis and local parodontitis (marginal periodontitis). Parodontitis: pathological dento-gingival pocket, pus from pocket, gums bleeding, normal EOD, specific changes on X-ray.

6.2. Chronic apical periodontitis (K04.5)

Chronic forms of apical periodontitis are the most frequent, because chronic apical periodontitis is a kind of progress of caries. Very often development of chronic apical periodontitis is due to imperfect methods of treatment of pulpitis.

Chronic forms of apical periodontitis are characterized by poor symptoms because proliferative phenomena and weak exudation are predominant in these forms of the disease. Teeth with chronic forms of apical periodontitis are considered as foci of bacteria, more often streptococcal sensibilization of organism.

Unlike acute periodontitis, chronic apical periodontitis is characterized by phenomena of proliferation - reproduction of tissue cells (fibroblasts, histiocytes) and formation of connective tissue fibers. Exudative phenomena are also observed, but recede into background. They are more expressed during exacerbation.

Clinical picture of chronic fibrous periodontitis

There are no complaints. Patient can have sometimes complaints about carious cavity. The tooth was treated before or there was carious cavity during long time (history of disease).

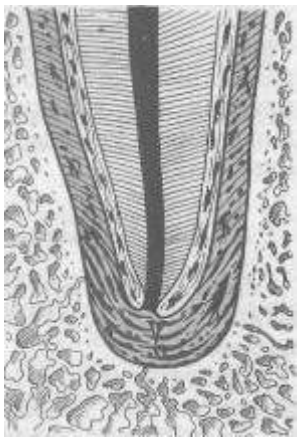


Fig. 8 Chronic fibrous periodontitis.

Objectively: Oral mucosa is pale pink. Tooth is changed in color (it can be under filling). A deep carious cavity, formed by pigmented dentine, communicating with a dental cavity. Probing and reaction to cold are painless. Percussion is painless. There is widening of periodontal space in the region of apex on X-ray. EOD is 100 mA.

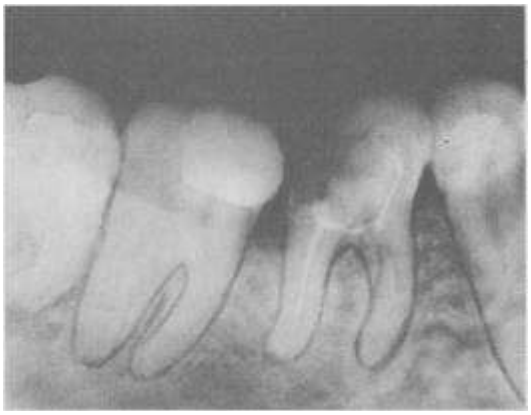


Fig. 9. Chronic fibrous periodontitis on X-ray

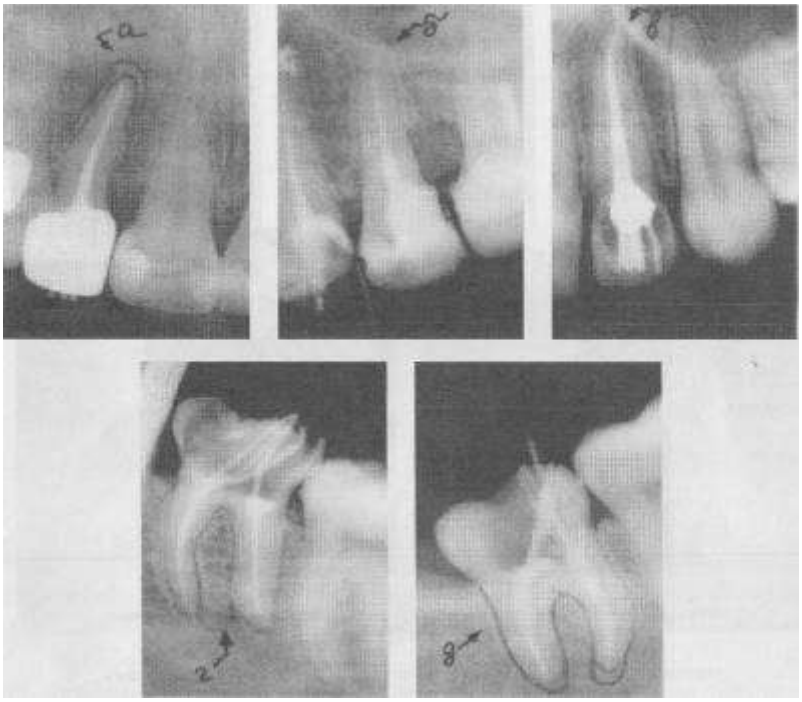


Fig.10 Chronic fibrous periodontitis.

Clinical picture of chronic granulating periodontitis

There are no complaints. Patient have sometimes complaints about carious cavity, about fistula on mucous or skin. The tooth was treated before or there was carious cavity during long time (history of disease). Patient says that he had spontaneous pain and pain from irritators before(history of disease). The patients complains of periodical pain or unpleasant feeling in the area of the affected tooth. Unpleasant feelings in the area of teeth are noted sometimes, the tooth is different from the others. Medical history shows that the tooth was

painful for a relatively long time. In the past there were attacks of pain the tooth was sensitive to temperature changes etc. Over the last period of time the tooth gets higher than the others, then the gingiva gets swollen. Some people have fistula on gingiva or on face. Very often patients indicate the previous treatment of the tooth.

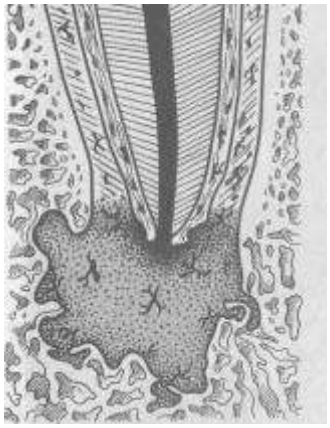


Fig.11 Chronic granulating periodontitis.

Objectively: The tooth is changed in color. A deep carious cavity, formed by pigmented dense dentine, communicating with dental cavity widely (under filling). There is fistula on oral mucosa. Probing is painless (there can be pain and bleeding in apical part of root canal because of granulating tissue); symptom of vasoparesis is positive.

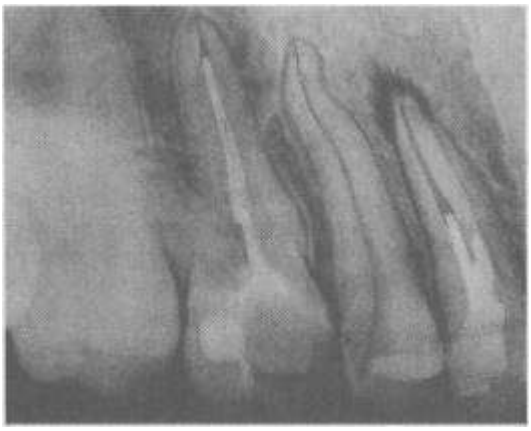


Fig.12 Chronic granulating periodontitis on X-ray

Reaction to cold is painless. Percussion is painless. There is destruction of bone like “fire”. Sometimes there can be shortness of root because of cement and dentine resorption. EOD is about 160 mA.

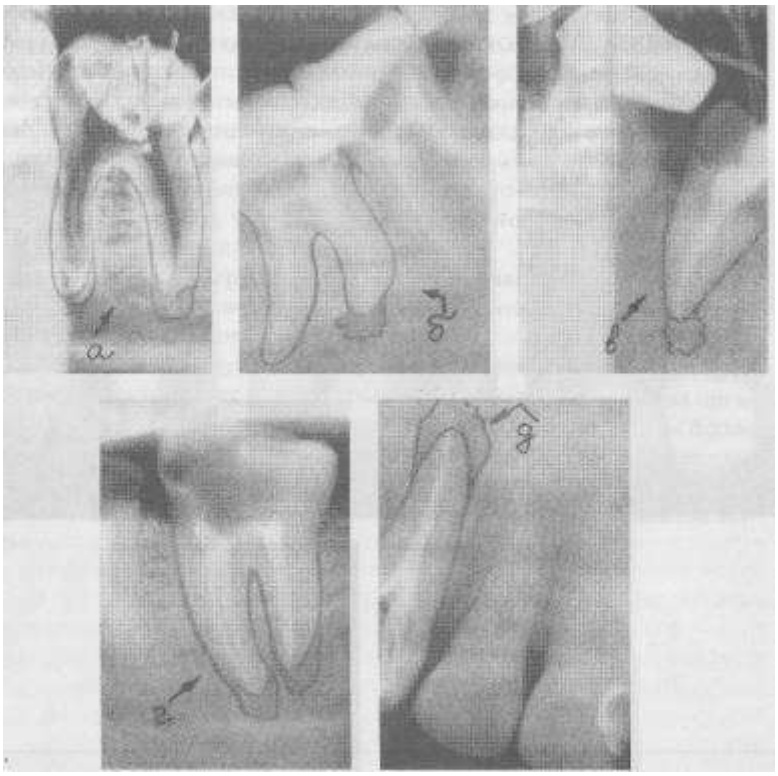


Fig.13 Chronic granulating periodontitis

The morphologic characteristic of this diseases is replacement of dense fibrous tissue by granulating tissue, where cellular elements are predominant such as fibroblasts, histiocytes and leukocytes. This is the typical way of fistular passage formation, that opens either on mucous membrane of gingiva or on skin.

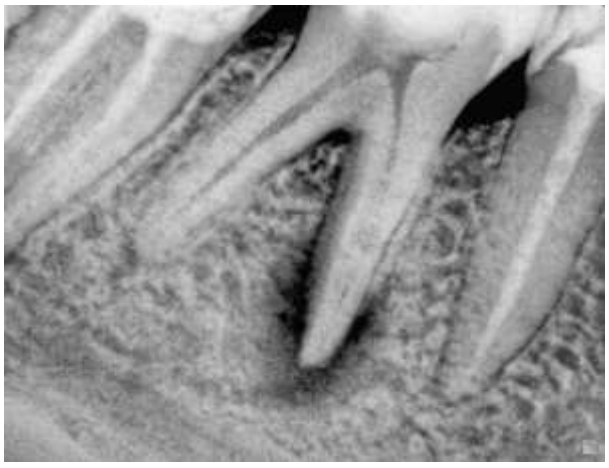


Fig.14 Chronic granulating periodontitis 3.6.

Together with alternative phenomena resulting in destruction of soft and solid tissues in periodontal ligament restorative processes take place. There is fibration of granular tissue, new formation of cementum in the root and in the bone tissue. If the process isn't acute, granular tissue is relatively rich in fibers, fibroblasts and other

connective cells. Long course of chronic granulating periodontitis results in development of granular tissue, some parts of it turn to scars, and the others are infiltrated by cellular elements.

Clinical picture of chronic granulomatous periodontitis

There are no complaints. Patient has sometimes complaints of carious cavity. Sometimes the patients complain of periodical weak pain in the area of the affected tooth and difficulties on chewing food. Sometimes patients realize swelling in the periapical area. The tooth was treated before or there was carious cavity during long time (history of disease). Medical history notes pain that stopped independently or after treatment.

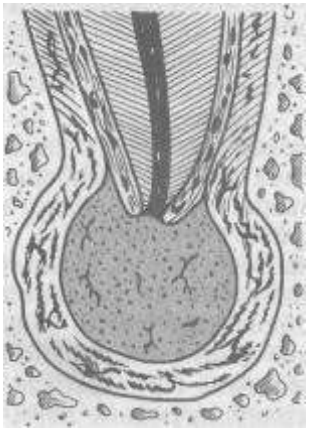


Fig.15 Chronic granulomatous periodontitis

Objectively: Tooth is changed in color. Tooth can be intact or under filling. There is deep carious cavity, formed by pigmented dentine, communicating with dental cavity. Tooth can be filled. Probing is painless; symptom of mobility of root is positive. Reaction to cold is painless. Percussion is painless. There is destruction of bone like “ball”. EOD is 100 mA.

Gingiva is usually unchanged, sometimes it is slightly edematous. Sometimes fistular passage can be found. The tooth is often covered with soft plaque or calculus. Root pulp is devitalized, fetoid purulent exudate is found. Probing of root canals is painless. Percussion is painless but it can cause unpleasant feeling. "Root shivering" is noted on percussion, it is felt with fingers at the root apex. Lymph nodes are usually not palpable. Radiograph shows defect of bone tissue that is round with well defined borders.

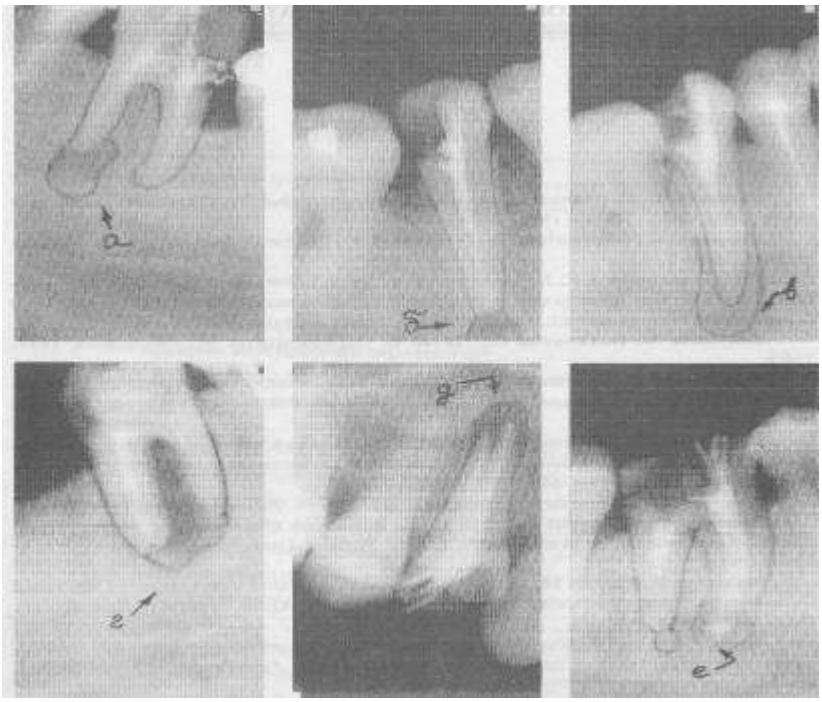


Fig.16 Chronic granulomatous periodontitis on X-ray.

Granulomatous apical periodontitis can become acute. When root canals are permeable the disease is treated endodontically. If chronic granulomatous periodontitis can't be treated, root cyst can develop.

From morphological point of view, granuloma is a focus of granular tissue of various maturity, limited with connective membrane. Granuloma is round or oval and it is closely connected with root apex.

The membrane of granuloma is a thick connective tissue, its fibers are arranged concentrically and form dense capsule. Long course of chronic granulomatous periodontitis without acute stages can result in scarring of granuloma. And frequent acute attacks of chronic granulomatous periodontitis result in autolysis and formation of fissural cavity in the centre of granuloma; these cavities have the tendency to fuse. The cavities covered with epithelium increase in size progressively. Such granulomas are called epithelial or cystogranulomas. If this process has developed for years, root cyst can form.

Chronic granulomatous periodontitis is a more stable and less active form compared with the chronic granulating periodontitis. Phenomena of proliferating character prevail here over phenomena of

inflammatory swelling and hyperemia. At granuloma a partial replacement of periodontium with granulation tissue is surrounded by fibrous capsule, whose fibers traverse immediately into tissues of perodontium. The central part of granuloma contains a large number of plasmocytes, and also hematogenic and histiogenic cells.

There are simple non-epithelial, complex epithelial granulomas and cystogranulomas.

In most cases chronic granulomatous peridontitis does not manifest itself, except during periods of exacerbation. In some cases it may give a sign of chronic granulating periodontitis (fistula, hyperemia, swelling of gingiva)

Like in case of chronic fibrous periodontitis, the diagnosis of chronic granulamotous periodontitis is formed on the basis of roentgenologic data. Chronic forms of periodontitis are differentiated: with median caries, chronic gangrenous pulpitis, chronic osteomyelitis and chronic maxillary sinusitis.

6.3 Clinical picture of exacerbation of chronic periodontitis (Periapical abscess with sinus K04.6, Periapical abscess without sinus K04.7)

Patient complains on localized constant dull pain, strong pain from touching by tongue and mastication.

Objectively: The mucous membrane is hyperemic, swollen, fluctuation is determined. Symptom of vasoparesis is positive. There is deep carious cavity communicating with tooth cavity. Probing is painless. There can be mobility of tooth of 2-3 degree. EOD is 100 mA. Sometimes there can be fistula with pus, palpation of fold is painfull.

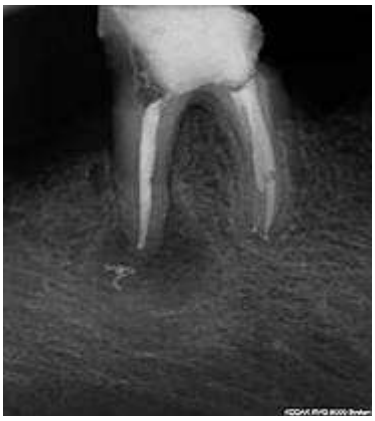
Exacerbation of chronic periodontitis can be differentiated with acute periodontitis, exacerbation of local parodontitis, trigeminal neuralgia, chronic periodontitis.

6.4. Clinical picture of radicular cyst (K04.8)

Cyst develops slowly if left without treatment for a number of years. However it develops and can expand into maxillary sinus cavity and compress mandibular nerve. Root cyst developing in the lower jaw can cause its pathologic fracture.

From the morphologic point of view root cyst is unicellular formation with fluid in it. Membrane of cyst consists of two distinctly

limited layers - epithelial, directed towards cavity lumen, and connective layer that surrounds cyst from the outside. They distinguish the third layer, which have the form of friable connective tissue between epithelial and connective layers. There exists an opinion that intermediate layer can be observed only during inflammation.



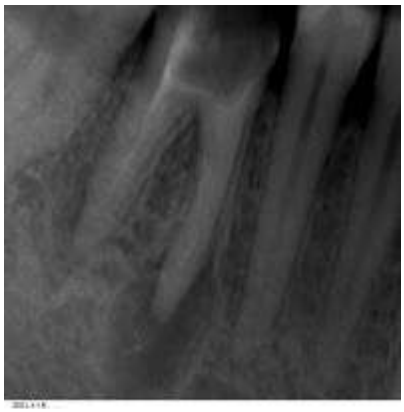


Fig. 17 Radicular cyst on X-ray.

Epithelial layer is multilayer flat epithelium that consists of 4-12 layers of cells. This epithelium usually doesn't keratinize. Epithelial membrane is to some extent infiltrated with polymorphonuclear leukocytes and lymphoid cells. Such infiltration often penetrates all layers of cystic membrane, that results in its thickening.

Connective membrane of cyst consists mostly of collagen fibers, situated concentrically. Cyst cavity is filled with transparent fluid that contains cholesterol crystals. This fluid gets turbid and purulent on suppuration. The process of suppuration is accompanied with cystic membrane and resorption of osteal bed of cyst.

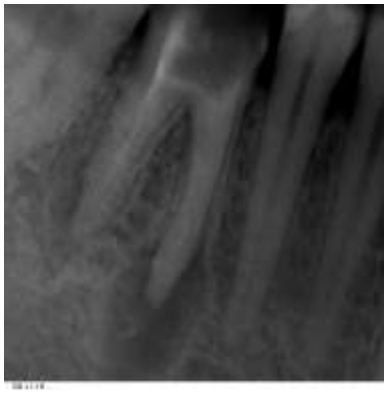


Fig.18 Radicular cyst on X-ray**.**

The patients usually have no complaints. Unpleasant feelings and difficulties on chewing are connected with the affected tooth. Only when relatively big cyst develops, a patient can complain of protruding bone in the area of muccobuccal fold, more seldom from the palatal side, the complaints can be connected with the sense of paresthesia in the half of lower lip.

Objectively: Tooth is discoloured, the crown is to some extent destroyed, root canal probing is painless, pulp decay is seen, when it is removed light yellow fluid can discharge. Percussion is painless, sometimes it causes unpieasant feelings. Odontometry confirms necrosis of pulp and can show decrease sensitivity to electric current compared to adjacent intact teeth. Palpation can reveal crunch. Roentgenogram shows round or oval sharply defined radiolucency. Roots are merged in it, and they are situated in the form of fan. Root cyst can suppurate. In this case clinical picture reminds of typical features of acute or acute attack of apical periodontitis. In such cases roentgenogram shows violation of distinct cyst's borders. If root canals are permeable and organism resistance is well, root canals can be treated endodontically. In unfavorable conditions it can be subject to surgical treatment. **Symptom of pergament crack** is positive. There is region of bone destruction 0,5 sm and more.

According to morphological structure cyst is single-chamber with liquid. The cover of cyst consists of two layers: epithelial (inside the cavity) and connective (on epithelial layer). There is third layer (soft connective tissue) between two layers. Some scientists claim that this layer exists only during inflammation. Epithelial layer is multilayer flat epithelium which consists of 4-12 layers. Connective layer consist of collagen fibers. Cavity of cyst is full of transparent liquid with crystals of cholesterol. Purulent cyst is full of pus. There is breach of cyst covering and resorbtion of bone in the region of cyst.

Differential diagnostics of chronic fibrous and granulating periodontitis (K04.5)

Table 4

Sign	Chronic fibrous perodontitis	Chronic granulating periodontitis	Chronic granulomatous periodontitis	Chronic gangrenous pulpitis
Complaints	There are no complaints. Patient can have sometimes complaints about carious cavity.	There are no complaints. Patient can have sometimes complaints about carious cavity, about fistula on mucous or skin.	There are no complaints. Patient can have sometimes complaints about carious cavity.	There are no complaints, pain can be from hot irritators.
History of disease	After eruption of teeth; tooth can be treated for pulpitis before	After eruption of teeth; there was pain in this tooth several months ago	After eruption of teeth; tooth can be treated for pulpitis before	After eruption of teeth. There was pain several days ago.
History of life	Involvement increases with a decrease of fluorine in drinking water	Involvement increases with a decrease of fluorine in drinking water	Involvement increases with a decrease of fluorine in drinking water	Involvement increases with a decrease of fluorine in drinking water
Objectively	Tooth is changed in color. A deep carious cavity, formed by	The tooth is changed in color. A deep carious cavity, formed by	Tooth is changed in color. A deep carious cavity, formed by pigmented dentine,	A deep carious cavity, formed by dense pigmented dentine,

	pigmented dentine, communicating with dental cavity.	pigmented dense dentine,communicating with dental cavity widely (under filling). Fistula on oral mucosa.	communicating with dental cavity. It can be filled.	communicating with dental cavity widely.
Probing	Painless	Painful; symptom of vasoparesis is positive.	Painless	Painless, deep probing is painful.
Reaction to cold	Painless	Painless	Painless	Painless.
Percussion	Painless	Painless	Painless; symptom of mobility of root is positive.	Painless.
X-ray	Widening of periodontal space in the region of apex	Destruction of bone like "fire"	Destruction of bone like "ball"	-
Electroexcitability	100 mA	160 mA	160 mA	60-90mA

Differential diagnostics of chronic granulomatous periodontitis (K04.5)

Table 5

Sign	Chronic granulomatous periodontitis	Chronic fibrous perodontitis	Chronic granulating periodontitis	Chronic gangrenous pulpitis	Radicular cyst
Complaints	There are no complaints. Patient can have sometimes complaints of carious cavity.	There are no complaints. Patient can have sometimes complaints of carious cavity.	There are no complaints. Patient can have sometimes complaints of carious cavity, of fistula on mucous or skin.	There are no complaints, pain can be from hot irritators.	There are no complaints. Patient can have sometimes complaints of carious cavity.
History of disease	After eruption of teeth; tooth can be treated for pulpitis before	After eruption of teeth; tooth can be treated for pulpitis before	After eruption of teeth; there was pain in this tooth several months ago	After eruption of teeth. There was pain several days ago.	After eruption of teeth; tooth can be treated for pulpitis before
History of life	Involvement increases with a decrease of	Involvement increases with a decrease of	Involvement increases with a decrease of	Involvement increases with a decrease of	Involvement increases with a

	fluorine in drinking water	fluorine in drinking water	fluorine in drinking water	fluorine in drinking water	decrease of fluorine in drinking water
Objectively	Tooth is changed in color. A deep carious cavity, formed by pigmented dentine, communicating with dental cavity. It can be filled.	Tooth is changed in color. A deep carious cavity, formed by pigmented dentine, communicating with dental cavity.	The tooth is changed in color. A deep carious cavity, formed by pigmented dense dentine,communicating with dental cavity widely (under filling). Fistula on oral mucosa.	A deep carious cavity, formed by dense pigmented dentine, communicating with dental cavity widely.	Tooth is changed in color. A deep carious cavity, formed by pigmented dentine, communicating with dental cavity. It can be filled. Cyst can be palpated on the alveolar bone, symptom of "parchment crackle".

Probing	Painless	Painless	Painfull; symptom of vasoparesis is positive.	Painless, deep probing is painfull.	Painless
Reaction to cold	Painless	Painless	Painless	Painless.	Painless
Percussion	Painless; symptom of mobility of root is positive.	Painless	Painless	Painless.	Painless.
X-ray	Destruction of bone like "ball"	Widening of periodontal space in the region of apex	Destruction of bone like "fire"	-	Destruction of bone like "ball" more then 5 mm.
Electroexcitability	160 mA	100 mA	160 mA	60-90mA	160 mA

Differential diagnostics of radicular cyst (K04.8)

Table 6

Sign	Chronic granulomatous periodontitis	Radicular cyst	Follicular cyst	Adamantinoma
Complaints	There are no complaints. Patient can	There are no complaints. Patient can have sometimes	There are no complaints.	There are no complaints.

	have sometimes complaints of carious cavity.	complaints of carious cavity.		
History of disease	After eruption of teeth; tooth can be treated for pulpitis before	After eruption of teeth; tooth can be treated for before pulpitis	-	-
History of life	Involvement increases with a decrease of fluorine in drinking water	Involvement increases with a decrease of fluorine in drinking water	-	-
Objectively	Tooth is changed in color. A deep carious cavity, formed by pigmented dentine, communicating with dental cavity. It can be filled.	Tooth is changed in color. A deep carious cavity, formed by pigmented dentine, communicating with dental cavity. It can be filled. Cyst can be palpated on the alveolar bone, symptom of "parchment crackle".	It is not connected with apex of root. It is in body of jaw. There is an intact permanent tooth inside the cyst.	It has many cellular structures. It is not connected with tooth.
Probing	Painless	Painless	-	-
Reaction to cold	Painless	Painless	-	-
Percussion	Painless; symptom of mobility of root is	Painless.	-	-

	positive.			
X-ray	Destruction of bone like "ball"	Destruction of bone like "ball" more then 5 mm.	There is a tooth in the cyst inside the bone.	There is cyst in the body of jaw.
Electroexcitability	160 mA	160 mA	-	-

7. Treatment of periodontitis

7.1. General principles of treatment of periodontitis.

There are **conservative, surgical and mixed** methods of periodontitis treatment. The choice of method depends on the clinical picture, character, time of pathologic process development, general well-being of the organism. One must take into consideration reasons for the inflammation, reliability of the crown patency of the root canals.

Treatment of periodontitis, is not limited to treatment of the tooth, which is the reason of this disease, it also includes removal of the foci of infection from organisms, prevention of organism sensitization and prevention of inflammation in maxillofacial area and diseases of internal organs.

It is necessary to stop the inflammatory process in periapical tissues when treating apical periodontitis. The inflammation should not extend.

There are ***special indications*** for conservative treatment of apical periodontitis:

- acute, chronic and exacerbation of chronic periodontitis;
- radicular cyst till 1,0-1,5 cm in size.

Absolute contraindications for conservative treatment:

- radicular cysts with the diameter more than 1,5 cm. or cyst that grows into maxillary sinus, mandibular canal etc.

- septic reaction in spite of opening of tooth chamber and cutting the mucogingival fold;

- 3rd degree of mobility of teeth.

- deep periodontal pockcts communicating with periapical region.

Relative contraindications:

- a part of a broken instrument in the canal;

- frequent exacerbations of chronic periodontitis in multirooted teeth with narrow curvatured canals;

- perforation of root or tooth;

- teeth which have been treated before but they are source of progressive disease;

- teeth filled with zinc-phosphate cement.

It should be noted that improvazation of endodontic instruments and infra- canal medicaments reduce these contradictory effects. Methods of treatment such as root amputation, hemisection, resection of root apex, sectioning of a tooth, and replantation should be considered as alternative methods which are used when endodontic treatment is impossible.

7.2. Pain relief in treatment of periodontotitis.

As the pulp is non vital, the main endodontic manipulations are painless. If pain is present, it is quite bearable. So the use of anesthetics as a rule isn't necessary.

Preparation of hard tissues is painful if there is acute attack of periodontitis, because periapical tissues are irritated.

Anesthesia also encounters some difficulties:

- decreased effects of local anesthetics in the area of inflammation.
- in cases of periostitis, injections are painful and provide dissemination of infection.
- anesthetics quickly migrate from the area of inflammation.
- local anesthesia can accompany reactive conditions connected with general conditions of a patient (strong pain, overfatigue).

Perfect technique of solid tissues preparation can increase anesthetic effect. This technique includes the following: usage of sharp burs, fixation of the tooth with fingers, slight touch with rotary bur, interupt preparation should be ruled out.

Air rotor allows to remove filling and open the cavity of the tooth and provides painless preparation.

If anesthesia is still necessary, conduction anesthesia is preferable. There is a great variety of medicines for local anesthesia nowadays, so it is possible to choose anesthetic electively. In some cases (inadequate reaction of a patient, intolerance to local anesthetics), general anesthesia or a combination of local anesthesia and sedation can be possible.

7.3 Treatment of acute apivcal periodontitis (K04.4)

During acute apical periodontitis treatment one must first of all provide the first medical aid:

- remove pain,
- give exudate drainage,
- eliminate the source of the infection and intoxication.

One must prevent spreading the process further to the deep hard and soft tissues and eliminate pathologic process. If there are general problems doctor must prescribe anti-inflammatory and desensibilising therapy.

Treatment of acute infectious periodontitis depends on phase of process: intoxication or exudation. For treatment of acute serous periodontitis it is not necessary to widen apical hole while for acute purulent periodontitis it is necessary.

Treatment of acute periodontitis is a complicated task. Medical manipulations consist of active clearing of the whole organism, in removing the focus of inflection, in prevention the organism's sensitization, in prevention of inflammatory processes development in the maxillofacial area, internal organs diseases.

A system of macrocanals, a system of microtubules, pathologically changed periapical tissues are the main objectives of treatment of chronic apical periodontitis.

Scheme of conservative treatment of acute periodontitis in stage of intoxication (serous stage):

1. Anesthesia (if it is necessary)

2. Isolation of tooth by Rubber dam

The use of the rubber dam is mandatory in root canal treatment. Developed in the 19th century by S.C. Barnum, the rubber dam has evolved from a system that was designed to isolate teeth for placement of gold foil to one of sophistication for the ultimate protection of both patient and clinician. The advantages and absolute necessity of the rubber dam must always take precedence over convenience and expediency (a rationale often cited by clinicians who condemn its use). When properly placed, the rubber dam facilitates treatment by isolating the tooth from obstacles (e.g., saliva, tongue) that can disrupt

any procedure. Proper rubber dam placement can be done quickly and will enhance the entire procedure.

Fig. 19 Optidam Intro Kit Anterior

The rubber dam is used in endodontics because it ensures the following:

1. patient is protected from aspiration or from the swallowing of instruments, tooth debris, medicaments, and irrigating solutions.

Fig.20 Rubber dam

2. clinician is protected from ligitation because of patient aspiration or swallowing of an endodontic file.

3. a surgically clean operating field is isolated from saliva, hemorrhage, and other tissue fluids. The dam reduces the risk of cross contamination of the root canal system, and it provides an excellent barrier to the potential spread of infectious agents.

4. soft tissues are retracted and protected.

5. visibility is improved. The rubber dam provides a dry field and reduces mirror fogging.

6. efficiency is increased. The rubber dam minimizes patient conversation during treatment and the need for frequent rinsing.

The dentist should be aware that in some situations, especially in teeth with artificial crowns, access into the pulp system may be difficult without first orienting root structure to the adjacent teeth and periodontal tissues. Radiographically, the coronal pulp system is often obstructed by the restoration, and as a result the dentist may misdirect the bur during access. In these cases it may be necessary to locate the canal system before placing the dam. Therefore the dentist can visualize root topography, making it easier to orient the bur toward the long axis of the roots and prevent perforations. Once the root canal system is located the rubber dam should be immediately placed.

Armamentarium: the main part of the rubber dam system is the dam itself. These auticlavable sheets of thin, flat latex come in various thickness (e.g., thin, medium, heavy, extraheavy, special heavy) and in two different sizes. For endodontic purposes, the medium thickness is probably best because it tends to tear less easily, retracts soft tissues better than the thin type, and is easier to place than the heavier types. However, a thinner gauge may be desirable to decrease tension if retainer placement is questionable or if the retainer is resting on a band. The dam is also manufactured in various colors, ranging from light yellow to blue to green to grey. The darker-colored dams may afford better visual contrast, thus reducing eye strain. However, the lighter-colored dams, because of their translucency, have the advantage of naturally illuminating the operating field and allowing easier film placement underneath the dam. Depending on individual preference and specific conditions associated with a tooth, the clinician may find it necessary to change the colour and thickness of the rubber dam used.

For patients with latex allergies, a nonlatex rubber dam is available from Coltene/Whaledent, Inc. This powder-free, synthetic dam comes in one size and in one thickness (medium gauge). It has a shelf life of 3 years but has only one third the tensile strength of a latex dam. Other companies provide nitrile rubber dams.

Another component of the rubber dam system is the rubber dam frame, which is designed to retract and stabilize the dam. Both metal

and plastic frames are available, but plastic frames are recommended for endodontic procedures. They appear radiolucent, do not mask key areas on working films, and do not have to be removed before film placement. The Young's rubber dam frame (plastic type), the Star Visi frame, and the Nygaard-Ostby (N-O) frame are examples of radiolucent frames used in endodontics. New to endodontics is a specially designed foldable plastic frame, with a hinge to facilitate film or sensor placement without disengaging the entire frame. Although metal frames can be used, their radiopacity tends to block out the radiograph. If removed, this may result in destabilization of the dam, the salivary contamination of the canal system and negating the disinfected environment that was previously attained.

Fig.21 Composition of modern rubber dam

Rubber dam clamps or retainers anchor the dam to the tooth requiring treatment or, in cases of multipleteeth isolation, to the most posterior teeth. They also aid in soft-tissue retraction. These clamps are made of stainless steel, and each consists of a bow and two jaws. Regardless of the type of jaw configuration, the prongs of the jaws should engage at least points on the tooth. This four-point clamp-to-tooth relationship stabilizes the retainer and prevents any rocking , which in itself can be injurious to both hard and soft tissues.

Clamps are available from a variety of manufacturers and are specifically designed for all classes of teeth with a variety of anatomic configurations. For most uncomplicated endodontic isolations, the

clinician's basic armamentarium should consist of winged clamps, a butterfly-type clamp for anterior teeth, a universal premolar clamp, a mandibular molar clamp, and a maxillary molar clamp. The wings, which are extensions of the jaws, not only provide additional soft-tissue retraction but also facilitate placement of the rubber dam, frame, and retainer as a single unit.

Other retainers are designed for specific clinical situations in which clamp placement may be difficult. For example, when minimal coronal tooth structure remains, a clamp with apically inclined jaws may be used to engage tooth structure at or below the level of free gingival margin. Retainers with serrated jaws, known as tiger clamps, also may increase stabilization of broken-down teeth. Another type of retainer, the Silker-Glickman clamp, should also be included in the dentist's armamentarium. Its anterior extension allows for retraction of the dam around a severely broken-down tooth, and the clamp itself is placed on a tooth proximal to the one being treated.

The remaining components of the rubber dam system include the rubber dam punch and the rubber dam forceps. The punch has a series of holes on a rotating disk from which the clinician can select according to the size of the tooth or teeth to be isolated. The forceps holds and carries the retainer during placement and removal.

Methods of rubber dam placement.

As mentioned earlier, an expedient method of dam placement is to position the bow of the clamp through the hole in the dam and place the rubber over the wings of the clamp (a winged clamp is required). The forceps stretch the clamp to maintain the position of the clamp in the dam, and the dam is attached to the plastic frame, allowing for the placement of the dam, clamp, and frame in one motion. Once the clamp is secured on the tooth, the dam is teased under the wings of the clamp with a plastic instrument.

Another method is to place the clamp, usually wingless, on the tooth and then stretch the dam over the clamped tooth. This method offers the advantage of enabling the clinician to see exactly where the jaws of the clamp engage the tooth, thus avoiding possible impingement on the gingival tissues. Gentle finger pressure on the buccal and lingual apron of the clamp before the dam is placed can be used to test how securely the clamp fits. Variations of this method include placing the clamp and dam first, followed by the frame, or

placing the rubber dam first, followed by the clamp and then the frame.

Fig.22 Rubber dam in oral cavity

A third method, the split-dam technique, may be used to isolate anterior teeth without using a rubber dam clamp. This technique is not only useful when there is insufficient crown structure, as in the case of horizontal fractures, but also it prevents the possibility of the jaws of the clamp to chip the margins of teeth restored with porcelain crowns or laminates. Studies on the effects of retainers on porcelain-fused-to-metal restorations and tooth structure itself have demonstrated that there can be significant damage to cervical porcelain, as well as to dentin and cementum, even when the clamp is properly stabilized. Thus for teeth with porcelain restorations, ligation with dental floss is recommended as an alternate method to retract the dam and tissues, so the adjacent tooth can be clamped.

In the split-dam method, two overlapping holes are punched in the dam. A cotton roll is placed under the lip in the mucobuccal fold over the tooth to be treated. The rubber dam is stretched over the tooth to be treated and over one adjacent tooth on each side. The edge of the dam is carefully teased through the contacts on the distal sides of the two adjacent teeth. Dental floss helps carry the dam down around the gingiva. The tension produced by the stretched dam, aided by the rubber dam frame, secures the dam in place. The tight fit and the cotton roll help produce a relatively dry field. If the dam has a tendency to slip, a premolar clamp may be used on a tooth distal to the three isolated teeth or even on an adjacent tooth. The clamp is placed over the rubber dam, which then acts as a cushion against the jaws of the clamp.

3. Preparation of carious cavity.

Carious cavity is opened by sterile spherical or fissure bur. Necrotomy is conducted with maximal approach to dental cavity by short interrupted movements avoiding too much pressure on the tooth. The stage is finished with removal of overhanging edges of enamel and repeated antiseptic processing of carious cavity.

4.Opening and widening of pulp cavity (if it is necessary)

Using the bath of antiseptics the roof of carious cavity is opened (if it is necessary) by a sterile spherical bur of a medium size without much pressure with interruptive movements. It's done in the area of the horn of pulp. Then it's removed along the border with carious cavity by a sterile fissure bur. This stage is finished with repeated antiseptic processing and removal of overhanging edges of dentin.

5.Widening of orifices of canals (if it is necessary):

Instruments are: "Gates gliden", "Largo", "Orifice opener" drills.

"Gates gliden" has short working surface in the form of a drop, on a long thin stem. It's used for dilatation of orifices and the initial 1/3 of a canal.

Drills are produced in 6 sizes – 1, 2, 3, etc. and are marked by rings on holders (1 – 6). "Largo" and "Orifice opener" has long working surface, which comes in a hard stem. They are used not only for dilatation of canal orifices but also for opening of a coronal part of upper 1/3 of a root canal in single-rooted teeth as well as for palatal and distal canal of multi-rooted teeth.

6. Determination of working length of a canal

Every part of endodontic treatment is controlled by a measurement of the instrument's penetration depth into the canal. This length is typically determined in millimeters. It is measured from a point on the tooth's coronal surface that is within the clinician's field of view. It varies from the complete canal length to some arbitrarily determined point near the termination of the canal space.

It's conducted with the help of X-ray, apex-locator, a table, tactile and visual methods. At determination of length of anterior group of teeth the cutting edge is used, while posterior teeth - buccal cusps are used.

Significance of working length Working length determines the extent of canal cleaning and shaping that will be accomplished. This measurement limits the penetration depth of subsequent instruments

and determines the ultimate form of the shaping process. Cleaning and shaping can have no greater precision than the working length. It is extremely important to make an accurate determination. The most clinically relevant working length landmark is the apical constricture, regardless of whether it is in dentin or cementum.

The constricture is the narrowest point of the canal, and therefore the narrowest diameter of the blood supply. Beyond the constricture, the canal widens and develops a broad vascular supply. Therefore, from a biologic perspective, the constricture is the most rational point at which to end the canal preparation, since the existence of a functional blood supply controls the inflammatory process. Intraradicular termination of the cleansing process leaves a canal content interface equal in area to the total inflammatory process (1:1). Termination beyond the constricture provides a greater area of blood supply than the irritant interface. Extraradicular termination of the working length can theoretically provide a hemisphere of vascular support to the inflammatory process. That gives a numerically superior advantage to the inflammatory process. The surrounding vital tissues have more capacity to destroy irritants and restore the area to a biologically functional state. Thus, cleaning and shaping upto the apical constricture completely eliminates pathogenic canal contents and allows the inflammatory healing mechanisms to complete.

Radiographic. The most commonly used method of determining the length of a canal is radiography. The clinician starts by placing a file to the preestimated length and then exposes a film. The location of the instrument tip is read from this film and any necessary changes in length are made. Changes greater than 0.5 mm should be verified by an additional radiograph. The exact canal preparation depth depends on the technique and philosophy of the operator. The periodontal ligament space typically is used to identify the apical termination of the canal.

This point includes the expanding portion of the canal beyond the constricture; consequently, techniques routinely make an allowance. The preparation length is shortened from the full length to the periodontal ligament space by at least 0.5 mm initially. Greater adjustments are recommended in some techniques.

Often dentists can use special formula for determination of working length according to radiographic method.

$$\frac{\text{Natural length of tooth (Tn)}}{\text{R length of tooth (Tr)}} = \frac{\text{Natural length of instrument (In)}}{\text{R length of instrument (Ir)}}$$

For example, the length of instrument (In) is 15 mm, R length of tooth (Tr) (according to X-ray) is 21 mm, the length of instrument on X-ray (Ir) is 15 mm.

$$\text{Tn (working length of tooth)} = \frac{\text{In x Tr}}{\text{Ir}} = \frac{15 \times 21}{15} = 21 \text{ mm}$$

Electronic.

Apex locators may be used to determine the canal length. The unit is connected to a file that can be inserted to the end of the canal , and a second lead is attached to the oral mucosa. The pulp is extirpated, the canal is irrigated and dried, then the attached file is inserted to the apex.

Fig.23 Apex locator

This precaution eliminates ionic conduction, which can give a premature indication that the apex has been reached. The more recent impedance models apparently are not as sensitive to ionic solutions as the older resistance-based units. The dryness is perhaps not as important in that case. Apex locators are most helpful in placing the first length determination file. At that time the working length must be estimated from a preoperative radiograph. This method requires some clinical experience to be used successfully; controlling the first file

into the canal with an apex locator reduces guesswork. Over time use of an apex locator can guide the clinician as he or she develops tactile sense. The clinician must be careful to avoid contaminating the file while connecting the electrical lead and inserting the measurement instrument. The file can be placed into the canal at some distance before the lead is attached, to reduce the chance of accidental contamination. From there the file is carefully oscillated and gently pressed toward the apex. As it approaches the foramen, the electrical resistance or impedance gradually increases and reaches the calibrated level as it contacts vital tissues. The location is verified by slow withdrawal and reinsertion.

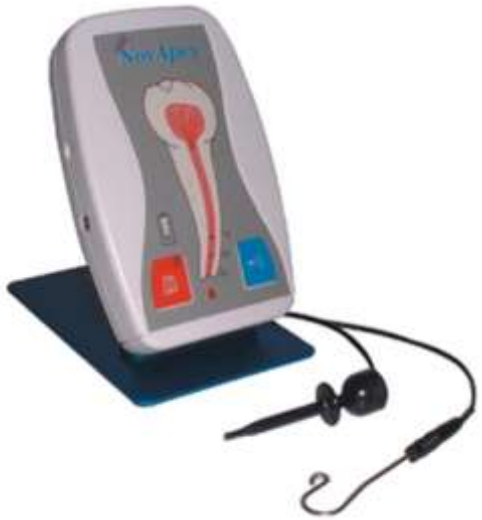


Fig.24 Apex locator “Nov Apex”, USA

The indicator is carefully observed during this action to ensure that the same apical indication is given. If the point repeats several times, it is typically a reliable measurement. Some clinicians recommend leaving the file at this indicated depth and exposing a radiograph to verify the position. Used in conjunction with a radiograph, the locator is an effective adjunct. Accidental passage through an accessory canal would indicate contact with the periodontal ligament space; however, the length would be inappropriate. A radiograph would illustrate the need for additional adjustment. Electronic apex locators are especially useful when treating teeth with calcified pulp chambers, as a minute perforation can be discerned before it is enlarged.

Tactile.

The experienced clinician develops a keen tactile sense and can gain considerable information from passing an instrument through a

canal. This ability must be developed, and for clinicians beginning a career certain information may speed the development of such ability. Following access, when interferences in the coronal third of the canal are removed, the observant clinician can detect a sudden increase in resistance as a small file approaches the apex. Careful study of the apical anatomy discloses two facts that make tactile identification possible: (1) the unresorbed canal commonly constricts just before exiting the root, and (2) it frequently changes course in the last 2 to 3 mm. Both structures apply pressure to the file. A narrowing presses more tightly against the instrument, whereas curvature deflects the instrument from a straight path. Both consume energy, and the sensitive hand can detect a sudden change in pressure required to accomplish insertion. The awareness of this change may be enhanced by the use of a file that is larger than the expected constricture. When the coronal two-thirds of the canal is constricted, clinicians cannot discern with accuracy what they feel because the file may be binding coronally rather than apically. As preparation develops space in the coronal two-thirds (i.e., radicular access), the quality of tactile information improves. At that point files bind only in the apical area, and resistance must be in that region. When only the tip of the file binds in the canal it becomes a sensitive instrument with which the experienced clinician can accurately determine passage through the foramen. At this point, it also has access to pass through apical accessory canals.

Paper point evaluation.

Once the preparation is complete, a paper point may yield more than a dry canal. After the moisture has been removed, the point may be used to detect apical moisture and/or bleeding. A bloody or moist tip suggests an overextended preparation. Further assessment of the apical preparation and working length should be made in this event. The point of wetness often gives an approximate location to the actual canal end point. A wet and/or bloody point may also indicate that the foramen has been zipped or the apex perforated during preparation. These conditions would require additional canal shaping in addition to adjustment of the working length.

Radiographs, electronic apex locators, tactile sense, and paper point evaluation used in harmony ensure that the final shaping and obturation will extend the full length of the canal.

7. Removal of putride mass

8.Instrumental and medical processing of root canals

Irrigants and other intracanal medicaments are necessary adjuncts that enhance the antimicrobial effect of mechanical cleansing and thus overall clinical efficacy. Several studies have shown that large areas of canal walls, particularly in the apical third but also in ribbon-shaped and oval canals, cannot be cleaned mechanically, meaning that microorganisms present in these untouched areas could survive. Residual bacteria and other microorganisms exist both in these hard-to-reach spaces and in dentinal tubules. Chemical disinfection is an important cornerstone of a successful outcome, because it reaches bacteria or fungi present in dentinal tubules and in the crevices, fins, and ramifications of a root canal system. In one study, investigators prepaed root canals, irrigated with saline solution, and sampled before, during and after instrumentation. They then cultivated and counted colony-forming units. These researchers found that with instrumentation alone, progressive filing reduced the number of bacteria, regardless of whetner rotary or stainless steel hand instrumentation was used. However, no technique resulted in bacteria-free canals. They found that instrumentation combined with saline irrigation mechanically removed more than 90% of bacteria in the root canal. Many authors have stressed the importance of using antimicrobial irrigants during chemomechanical preparation to ensure complete disinfection.

Fig.25 Manual instruments for root canals' preparation.

Substances that have been used to rinse and chemically clean root canals have different purposes, such as dissolution of soft and hard tissues, antimicrobial effect against bacteria or other microorganisms in the root canal, and inactivation of bacterial lipopolysaccharides. These substances also should be as nontoxic as possible to protect the periradicular tissues. Unfortunately, solutions that are toxic for bacteria cells frequently are toxic for human cells as well; therefore, care must be taken to avoid extrusion of irrigants into periapical regions.

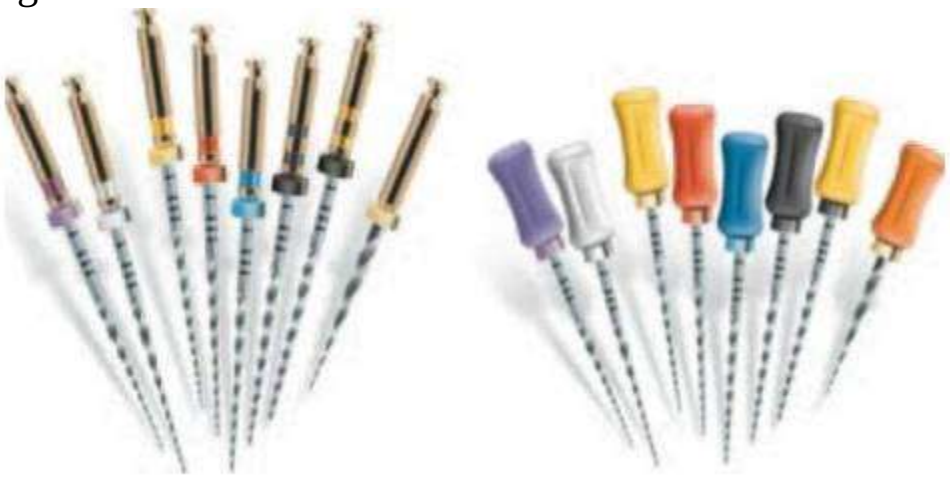


Fig.26 ProTapers for root canals' preparation

Another critical factor is the volume of irrigant. In a study evaluating the effect of different amounts of fluids, the volume of irrigant was found to affect the cleanliness of the root canal. NaOCl and EDTA administered in larger volumes produced significantly cleaner root canal surfaces than smaller volumes. The choice of an appropriate irrigating needle, therefore, is also important. Although larger gauge needles allow the irrigant to be flushed and replenished more quickly, the wider needle diameter does not allow cleaning of the apical narrower areas of the root canal system. Excess pressure or wedging of needles into canals during irrigation, with no possibility of backflow of the irrigant, should be avoided under all circumstances to prevent extrusion of the irrigant into periapical spaces. In juvenile teeth with wide apical foramine or when the apical constriction no longer exists, special care must be taken to prevent resorption or overpreparation of the root canal.

Fig.27 Instruments Mtwo for root canals' preparation

When bacterial samples test negative after treatment, the prognosis is improved. During mechanical root canal preparation, endodontic instruments are used to clean and enlarge root canal systems. Rotating instruments have an additional, advantageous "Archimedes screw' effect by which debris is transported in an apicocoronal direction. Even when simple saline was used as an irrigant, a ten fold to 1000-fold reduction of the bacterial load through mechanical instrumentation was demonstrated.

However, as noted earlier, instrumentation alone does not produce a bacteria-free root canal. In one study, dentin samples tested positive in most of the teeth after mechanical instrumentation even when bacteria had been eliminated from the root canals in some cases. Bacteria persisted in seven root canals despite mechanical cleaning and saline irrigation during five consecutive appointments. Moreover, teeth with a high number of bacteria in the initial sample remained infected despite being treated five times. In another study, teeth that caused symptoms tended to have more bacteria than teeth with no clinical symptoms.

Orstavik and Haapasalo investigated the effect of endodontic irrigants and dressings in standardized bovine dentin speciments that were infected with test bacteria. They found that bacteria were capable of colonizing the canal lumen and dentinal tubules. In the specimens used, E. fecalis rapidly infected the whole length of the tubules, whereas Escherichia coli penetrated approximately 600 ɱm. They also found that IKI appeared to be more effective at destroying bacteria than NaOCl, which was more effective than chlorhexidine.

Other investigators have explored the effects of sodium hypochlorite (with and without EDTA), chlorohexidine, and hydrogen peroxide in varying concentrations when used in sequence or in

combination as endodontic irrigants. They found that chlorohexidine and sodium hypochlorite were similarly effective in eliminating the bacteria tested. Synergistic effects were observed for some of the irrigants (e.g. , chlorohexidine and iodine potassium iodide.

Both of the preceding studies used infected dentine specimens; dentine is an important factor in disinfection because certain concentrations of calcium hydroxide solution, sodium hypochlotite, chlorohexidine, and iodine potassium iodide are inactivated or their activity is reduced by dentine powder.

The endodontic irrigation solution with the best proteolytic effect is NaOCl, even though it does not meet all the requirements of an ideal irrigant. It is readily available, inexpensive, and a widely used irrigation solution. Given sufficient time, NaOCl is a powerful solvent of necrotic pulp tissue and organic debris and has excellent antimicrobial properties. Necrotic tissue and debris are dissolved by the break-down of proteins into amino acids through free chlorine in NaOCl. Concentrations used clinically range from 0,5% to 5,25% . However, because free chlorine is the important component, the solution must be replenished frequently during preparation to compensate for lower concentrations and to constantly renew the fluid inside the root canal. This is even more important when the root canal is narrow and small, and the files must carry the NaOCl to the apical third during instrumentation. A 1% solution is effective at dissolving tissue and providing an antimicrobial effect. The use of commercial household bleach in its undiluted form (5,25%) cases substantial necrosis of wound surface areas and may result in serious clinical side effects. It is diluted in 1:1 or 1:3 ratios with water to produce a 2,5% or 1 % solution; both are suitable for clinical endodontic use.

As stated earlier, irrigation needles should never be wedged into canals during irrigation to avoid extrusion and serious damage to periapical tissues. Higher concentrations of NaOCl are more aggressive toward living tissue and can cause severe injuries when forced into the periapical area.

Such accidents can be prevented by marking the working length on the irrigation needle with a bend or a rubber stop and by passively expressing the solution from the syringe into the canal. The needle should be continuously moved slightly up and down. It should remain loose in the canal, allowing a backflow of fluid. The goal is to rinse

the suspended, concentrated dentinal filings out of the pulp chamber and root canals as new solution is brought down into the most apical areas by the endodontic instrument and the capillary effect.

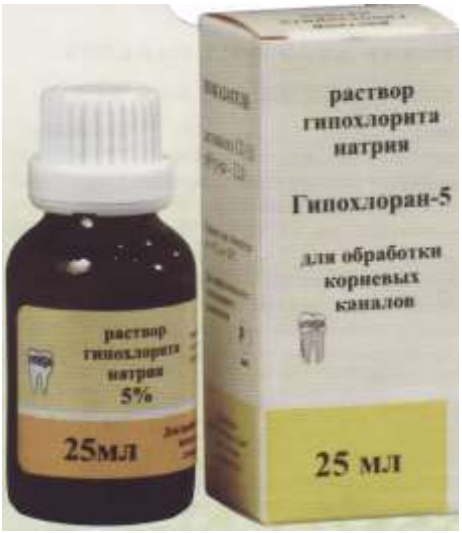


Fig.28 Sodium hypochlorite, 5%.

Patency files should be used carefully and should not be extended farther than the periodontal ligament, because they are possible sources of irrigant extrusion.

In one study, heating increased the antibacterial action of NaOCl. This can be done in several ways; for example, after the solution has been drawn into the irrigating syringe, a syringe warmer can be used (e.g., Syringe Warmer /Vista Dental Products). Heating also enhanced the antibacterial effectiveness of CHX and Ca(OH)2 solutions. A 0,5% NaOCl solution heated to 45° C dissolved putride mass as efficiently as 5,25% solution used as the poitive control. Heating to 60°C resulted in almost complete dissolution of tissue. Studies have shown that 1 minute at 47°C is the cutoff exposure at which osteoblasts can still survive; however, higher temperatures may in fact be sufficient to kill osteoblasts and other host cells. Also warming of NaOCl to 50°C or 60°C increases collagen dissolution and disinfecting potential, but it may also have severely detrimental effects on NiTi instruments, causing corrosion of the metal surface after immersion for 1 hour.

An increase in the temperature of the irrigant may be a reason to include ultrasonic devices in canal irrigation; these devices also increase the tissue-dissolving capabilities of sodium hypochlorite. In

one study the peak irrigant temperature during use of an ultrasonic device reached 45°C near the file tip but remained at 32°C on the outer root surface of teeth prepared to a size #45. Another reason for using ultrasonic devices might be enhancement of canal cleanliness. However, some authors have reported no beneficial effect with ultrasonics, neither in debriding root canal walls nor in reducing bacterial counts.

Fig.29 Sodium hypochlorite, 3%.

Smear layer management

EDTA is a decalcifying, chelating agent used as a gel or a 17% buffered solution during instrumentation of root canals. It acts as a chelator with calcium ions and removes the dentinal debris produced on the root canal walls during preparation. It thus opens dentinal tubules, allowing better penetration of disinfectants.

Whenever the wall of a root canal is instrumented, whether by hand or rotating instruments, the parts of a dentin wall touched by an instrument are covered by a surface layer called the smear layer. The smear layer, which consists of dentin shavings, cell debris, and pulp remnants, can be described as itself having two separate layers; a loose superficial deposit and an attached stratum that extends into the dentinal tubules, forming occluding plugs.

For some time clinicians and researchers paid little attention to the smear layer, partly because it was a thin superficial layer (1 to 5 ɱm) that might be present or not, depending on the type of instrument and the sharpness of its cutting blades. Also, because acids and chelating agents dissolve the smear layer, it was removed and from

routinely processed specimens. Smear layers are not seen in unprepared canal areas, which may have calcospherites, buttonlike structures that are abundant on intracanal surfaces.

Some authors have reported that an overlying smear layer delays but does not eliminate the effect of medicaments. Other contend that a smear layer may adversely affect disinfection and may also increase microleakage after canal obturation. Although organic substrate in a smear layer may serve as a nutrition source for some species of bacteria, some have suggested that, conversely, a smear layer can act as a beneficial barrier, preventing microorganisms from entering the dentinal tubules when a root canal is colonized by bacteria between appointments. The potential of intracanal disinfectants has been evaluated in vitro after removal of the smear layer with a combination of 5,25 % sodium hypochlorite and 17% EDTA. The decalcifying effect of EDTA is self-limiting; therefore the solution must be replaced at intervals. EDTA can help open very narrow root canals and can decalcify to the depth of approximately 50 ɱm. Because the smear layer consists of organic and inorganic components, alternating use of NaOCl and EDTA is most effective.

Fig.30 RC-prep (EDTA).

Liquid disinfectants were effective against E. fecalis in dentinal tubules up to depth of 400 ɱm. microbiologic analysis of split root

halves showed that early removal of the smear layer resulted in significantly higher bacterial counts. In contrast, other researchers have acknowledged that the smear layer, while acting as a barrier, might block irrigation solutions from entering the dentinal tubules. Moreover some bacteria (e.g., Bacteroides gingivalis and Treponema denticola) have the potential to dissolve smear layer proteins, thereby producing gaps, which could promote both coronal and apical microleakage and bacterial multiplication.

Some reported that the presence of the smear layer had no significant effect on apical leakage in dye penetration test. Others described an improved seal after removal of the smear layer. The latter study, which used a coronal leakage model, found a significantly decreased incidence of bacterial penetration (30% versus 70%) when the canals were irrigated with 17% EDTA and 5,25% NaOCl before obturation. In obturated root canals, a remaining smear layer led to bacterial leakage in 60% of the samples versus no leakage when the smear layer was removed. Other authors had similar results after smear layer removal with EDTA solution alone. Another investigation described many lateral canals in the apical thirds of the root canals systems cleaned with a barbed broach wrapped with MTAD-soaked cotton and showed less erosion than when EDTA was used. Other studies have found that a stronger bond was present when the smear layer was removed, and a statistically significant reduction of microleakage was measured. However, another investigation reported increased apical microleakage of the filled root canal after removal of the smear layer.

For the time being, root canals must be mechanically enlarged. Lager apical preparations enhance the efficacy of irrigation, and the additional use of ultrasonic energy during cleaning and shaping may also increase the efficacy of endodontic irrigants. Ultrasonics used passively in canals with sufficiently large apical preparation may reach and better clean any instrumented canal area. One investigation studied the debriding ability of 2,5% NaOCl in canal recesses. In 10 of 11 cases, these researchers found significantly cleaner histologic sections after ultrasonically activated irrigation. In the ultrasonically treated group, the bacteria count was reduced by 99,8%. However, hand filing alone reduced the bacteria count by 99,3%; therefore the improvement using ultrasonic therapy was limited. With ultrasonics,

root canals are debrided by shear stresses produced between the irrigant and the canal wall, with subsequent cell disruption.

Acoustic streaming of the irrigation fluid through ultrasonic treatment has been suggested as a method of improving cleanliness. However, this effect occurs mainly in the most coronal levels; the apical areas were least affected by activated irrigation. Because the amplitude of the oscillation is greatest at the instruments tip, attenuation and constraint most significantly affect the apical part where the diameter of the canal is smallest.

Some investigators used an irrigation time of 1 minute for each of EDTA and NaOCl. These authors stated that the use of ultrasonic energy for irrigant activation did not improve debridement compared with control groups. However, bacterial species show varying degrees of susceptibility of ultrasonication.

9. Control X-ray

10. Drying of a root canal.

For drying of root canals it's better to use paper points which are made of natural Japanese rice paper. They quickly absorb fluid, preserving their form. It's better to use these points in sterile packs.

11. Temporary filling by paste with anti-inflammatory and antiseptic action (f.e. powder of calcium hydroxide with 0,05% chlorohexidine, Metapaste, Metapex, Vitapex and others) or turunda with antiseptic liquid (scientists don't recommend).

12. Hermetic bandage (Septopak(Septodont)) It is necessary to remove bandage if pain will increase.

Fig. 31 Calcium-containing temporary filling material for root canals

2 VISIT

1. Removal of hermetic bandage

2. Medical processing of root canal

3. Drying of root canal

4. Filling of a root canal

The success of treatment of a canal depends on method of canal preparation and filling material.

Fig. 32 Material on the base of epoxide resins.

Gutta-percha point is used of similar size with that of the last file. It's moved along the canal walls without reaching the apex 1-2 mm. Before putting gutta-percha pins in a canal it's recommended to put them in filling material. It provides its easy sliding in the canal and good obturation in the apical area of root.

Then the point is condensed by vertical movements in the apical direction. It's used for pushing gutta-percha to the apex. Then the point is condensed to the canal walls by lateral movements (lateral condensation) with the help of "Finger spreader".

The construction of lateral condenser with cut tip is similar to that of vertical one. But it has a pointed tip, it's introduced into a canal, moved as deep as possible, and then by lateral movements gutta-percha is pressed to the canal wall. After the removal of lateral condenser there is space in its place where the next point can be placed. Gutta-percha points are introduced till full filling of a canal. The excess of points is cut by a hot instrument.

5. Control X-ray. They check fullness of obturation and dense adjacency of material to the canal walls.

6. Isolating liner.

7. Restoration of teeth after endodontic treatment.

8. Grinding and polishing.

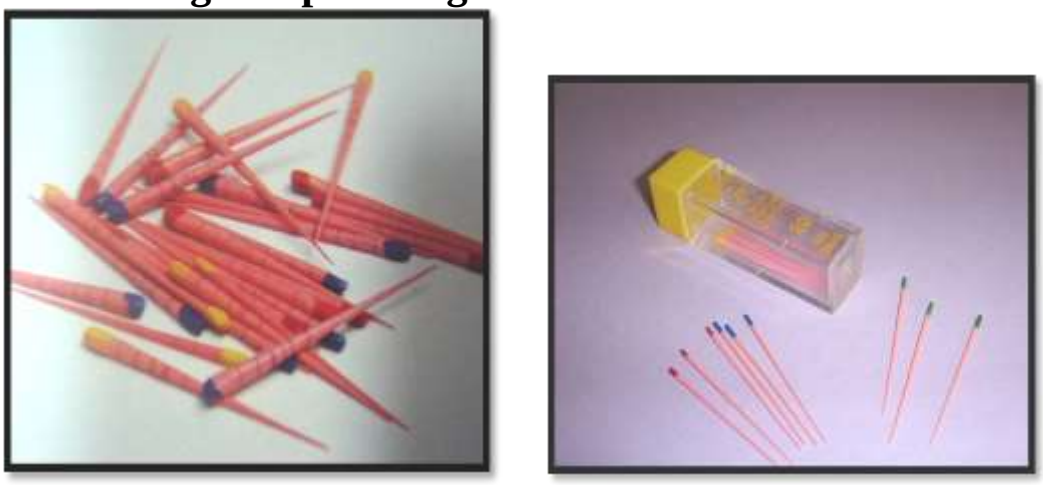


Fig. 33 Gutta-percha

Pecularity of treatment of acute medicamentous periodontitis.

If it is intoxication as a result of overdose of arsenic paste it is necessary to remove coronal and root pulp and to irrigate the canal with the solution that neutralizes or reduces the influence of this paste. Untra-canal cotton with the solution of or 1 % iodinole solution, is left in the canal for 48 hours. If irritation of periodontal ligament is due to acids and alkaline or strong antiseptics, the preparations of contradictory influence are used for neutralization. Non irritating medicines are used after cleaning and shaping of a canal, these medicines (iodinol, hydrocortisone etc.) are left in the canal under artificial dentine. Antihistamine preparations and non narcotic analgetics are preferable. During the second appointment root canals are processed and filled to the apex if these are no symptoms.

Treatment of acute purulent periodontitis

Acute purulent periodontitis treatment must be sparing. As a rule, in case of acute purulent periodontitis the main thing is to give exuadate's drainage. The drainage may be performed through the root canal, through a cut in the transitional fold, through the gingival recess (if it is present), or through the alveolar socket. The drainage through the root canal is the most favorable.

Before this procedure there are several preliminary steps.

1 VISIT:

1. **Anesthesia.**

Touching the tooth at acute periodontitis is painful, that is why different methods of pain elimination exist.

Nowadays doctors use sophisticated techniques at solid tissues preparation : sharp diamond burs, well-regulated headpiece, fixation of the injured tooth with the free hand, use of air-turbine drilling machine. If pain relief is necessary, one must prefer block anesthesia. If acute inflammation has not spread to the soft tissues - one must use infiltrational anesthesia. Sometimes narcosis is perfomed .

Nowadays doctors pay much attention to patients; premedication to remove fear, anxiety, and tension. Seduxen (diazepam) is the most effective drug of sedative premedication. It may be introduced in a 2,0 - 0.5 % solution. One may carry out oral single premedication at 5-15 mg of Seduxen uptake.

Before removing products of disintegration from the root canal, one must deactivate putrid mass by help of antiseptic solutions to prevent infected masses from getting to periodontium. Removal of the products is carried out in fractions, each time introducing the pulp-extractor deeper to the root canal under the antiseptic bath.

2. **Partial instrumental and medical processing of root canals**
3. **Opening of apical hole (if it is necessary)**

If after putrid mass evacuation from the canal exudate release is not marked, one must process the canal medicationally and open the apical fissure with a bur under antiseptic bath. Exudate release contributes to pain relief and the stop of inflammatory process. At subperiosteal abscess being present evacuation of exudates through the root canal is not sufficient. In this case one must cut the periostium and leave drainage. One must not wait till pyogenic abscessis formed, the cut must be done as soon as the swelling or hyperemia appears.

At diffusional process the drainage is done through the periodontal fissure with incision of circular ligament.

After the drainage antiseptic treatment of the gingival recess is carried out, drainage of the periodontal fissure through the alveolar socket is done by means of the tooth extraction.

The tooth is not filled to provide elimination of exudates. Patient must isolate tooth use cotton during meals and he must gargle oral cavity after meals. Horizontal cutting in the region of transitional fold is used if periodontitis is complicated by periostitis. Drainage by rubber stripes is used after it.

Treatment of acute periodontitis with hardly permeable or impermeable canals is as a rule done surgically, if exudate outflow is impossible through the canal. Muccobuccal fold is cut, the focus is drained or tooth is extracted. When inflammation is removed, there is an attempt to prepare the canals, if it is impossible, the tooth should be extracted.

Doctor must prescribe medicines for **GENERAL TREATMENT** if manifestations of intoxication increase:

- antibiotics

- Ciprolete 500 mg 3 times a day; Amoxicillin 500 mg 3 times a day – every 8 hours (first dose 1000 mg); Amoxiclav 625 mg 3 times a day.
-
- 

Fig.34 Amoxicillin, 500 mg.
-

If patient has allergy to amoxicillin he should use Clindamycin (to anaerobes), Dalacin C 300 mg, after this 150 mg 4 times a day. Aeritrimycin is not effective to anaerobes.

Doctor can see effectiveness after 24-48 hours. If penicillins are not effective we can add Metronidazol to Amoxicillin – 250mg 3 times a day or Augmentin can be used (it is less effective than Amoxicillin).

- Antihistamine medicines;

Diozolin 0,1x 2 times a day (Suprastin 1 tabletx 3 times a day, Tavegil 1 tablet 2 times a day);

- Analgetics (Ibuprofen 400 mg 3 times a day);
- Polyvitamines;
- Gargling use warm hypertonic solution of sodium hydrocarbonate 4 times a day.

Fig.35 Suprastin**.**

Fig.36 Diazolin

Fig.37 Ibuprophen

2 VISIT: after 3-5 DAYS

During the second visit the doctor's policy is determined by subjective sensations and objective patient's condition.

1. **Removal of hermetic bandage**
2. **Instrumental and medical processing of root canals**

3. **Antiseptic processing of a root canal**
4. **Drying of a root canal, probe with iodinol (it shows presence of pus inside a root canal)**
5. **Putting tampon with medicament or filling of root canal using temporary filling material (if probe with iodinol is negative). 1-2 days**
6. **Hermetic bandage**

3 VISIT (if patient has no complaints) after 3-5 DAYS

1. Removal of hermetic bandage
2. Medical processing of root canal
3. Drying of root canal and probe with iodinol
4. Filling of a root canal
5. Control X-ray.
6. Isolation liner.

They check fullness of obturation and dense adjacency of material to the canal walls.

6. Hermetic bandage.

Fig.38 Calcium-containing temporary filling material.

IV VISIT:

1. Removal of hermetic bandage.
2. Restoration of teeth after endodontic treatment.
3. Grinding and polishing

7.4. Treatment of chronic periodontitis

Successful treatment of periodontitis depends on permeability of root canals. In this case, good obturation is achieved and the source of intoxication and infection is removed. Only the root canal allows to influence inflammative process in the periapical tissue.

Scheme of treatment of chronic fibrous periodontitis (K04.5)

1. **Isolation of tooth use rubber dam.**
2. **Preparation of carious cavity** (or filling if the tooth is filled).
3. **Opening and widening of pulp cavity (if it is necessary**
4. **Widening of orifices of canals (if it is necessary)**
5. **Determination of working length of a canal**
6. **Elemination of putrid mass**
7. **Instrumental processing of root canals**
8. **Medical processing of root canal**
9. **Drying of root canal and probe with iodinol**
10. **Filling of a root canal**
11. **Control X-ray.**
12. **Isolation liner.**
13. **Permanent filling.**
14. **Grinding and polishing of the filling.**

Scheme of treatment of the destructive forms of chronic periodontitis (granulating, granulematous) (K04.5)

Both granulating and granulomatous periodontitis are characterized by destructive changes of periapical area and the products of decay (biogenic amines) are dangerous since they cause chronic intoxication and sensibilization.

Chronic periodontitis has negative influence on the organism, and it can cause or aggravate common somatic diseases, such as rheumatism, rheumatic carotids and nephritis. Thus method of treatment of periodontitis depends on general condition of organism.

In case of chronic periodontitis macro- & microcanals and pathologically changed periapical tissues are subjected to therapeutic intervention.

Traditional conservative method of treatment of chronic periodontitis consists of creating depot of biologically active substances that influence the process of reparative osteogenesis, It is not recommended to remove medical pastes behind the limits of root canal when treating chronic fibrous periodontitis.

1 VISIT

1. Isolation of tooth use rubber dam.

2. Preparation of carious cavity (or filling if the tooth is filled).

1. **Opening and widening of pulp cavity (if it is necessary**
2. **Widening of orifices of canals (if it is necessary)**
3. **Determination of working length of a canal**
4. **Elemination of putrid mass**
5. **Instrumental processing of root canals**
6. **Medical processing of root canal**
7. **Drying of root canal and probe with iodinol**
8. **Filling of a root canal with temporary filling material (often on the base of Ca-hydroxide)**
9. **Control X-ray.**
10. **Temporary filling.**

2 VISIT after 2-4 weeks

1. **Removal of temporary filling.**
2. **Instrumental and medicamentous processing of root canals.**
3. **Drying of root canals**
4. **Filling of root canals with permanent filling materials (sealer and filler)**
5. **Control X-ray**
6. **Isolation liner**
7. **Permanent filling**
8. **Grinding and polishing.**

A dentist must control the result of treatment by the help of X-ray after 6, 9, 12 months.

Pecularities of treatment of chronic periodontitis in single rooted teeth

If single rooted teeth have well permeable canals, fibrous, granulating, granulomatous periodontitis and radicular cyst (diameter less than 2 cm), root canal treatment & filling is done in one or two appointment. First, carious cavity is prepared (on lingual (palatal) surface in incisors & canines and on masticatory surface in premolars). Prepared cavity provides good access to root canal for endodontic instruments. Step - by - step removing of pulp under antiseptic in order to avoid its penetration into tissues near the both. Then root canals is cleaned with cottons saturated with solutions of enzymes and anticeptics. Infected dentine is removed. When the canal is irrigated for the second time with medicines and dried, intracanal cotton with anticeptic is left there & covered with temporary bandage. In 2-3 days the bandage is removed. If exudate remains, canal is cleaned again, if the canal is clean, it is processed with ethyl alcohol, dried & filled. If is very important to know if the canal is permeable or not. If the canal is fully permeable it can be filled. Filling material should fill the whole canal and close apical foramen very well. In most cases it is enough for the restoration of periapical tissues, destroyed as a result of pathologic process (granulating or granulomatous periodoniitis & sometimes root cysts). It proves good regenerative ability of periodontal ligament (unlike pulp). Fistural passage is absolute indication for treatment of periodontitis in single rooted teeth.

Fistula is an exit for exudate, thus root canal filling with, filling material pushed behind the apex doesn't cause acute attack of inflammation. All manipulations are done in the usual order but in one appointment. The relative indication for one - appointment - treatment of chronic periodontitis is fibrous form of periodontitis in the period of remission. For treatment of after filling acute attack, physiotherapeutic treatment or blokades with 1 % solution of hydrocortizone acetate are indicated.

Thus the problem of treatment of chronic periodontitis can be considered as solved. Unsuccessful treatment can take place only in cases of full or partial root canal obliteration or when it is impossible to prepare the canal that has been treated before.

Peculiarities of treatment of chronic periodontitis of multirooted teeth.

Successful treatment depends on permeability of root canals. If it is possible to widen canals with rod instruments and then to fill the canal to the apex or behind the apex, periapical tissues can be restored. Though teeth with non completely filled root canals may cause no pain, there is no improvement in periapical tissues, most often the focus of bone discharge increases.

Treatment of chronic periodontitis presupposes the same manipulations as for single rooted teeth. Better result can be achieved by medicines with anti-inflammatory & cleaning effect on the canal, endodontic intervention is necessary. Hardly permeable part of the canal is widened both with instruments & with chemicals. If it is impossible to prepare the canal, alternative methods of treatment are used (extraction, hemisection).

The result of treatment.

The result mainly depends on quality of obturation. If the canals is completely filled or if biologically active paste is reaches beyond the apical foramen, one can cxpect favourable results. As bone is restored slowly the results of treatment should be checked not earlier than after 6-9 months.

Sometimes it takes 2-3 years to restore bone with the diameter of resorption 1,5-2 cm.

Rehabilitation of periodontitis after treatment,

Rehabilitation of tissues of apical periodontitis is of great practical importance, because there can be no subjective manifestations of apical periodantal ligament diseases. Rehabilitation means normalization of function of the tooth, restoration of tissues surroundmg tooth apex, exclusion of pathogenic influence of periapical focus on the adjoining tissues and organism in general.

Clinical and radiographical examenation arc repeated in 3,6 & 12 months. If there are no complaints after 12 months and x-ray picture shows no pathologic changes in the area of root apex, there is no need in further observations.

If treatment of chronic granulating and granulomatous periodontitis gave no positive results, such as reduction of the focus of

destruction, after 12-18 months, it is necessary to remove periapical focus with the help of surgical methods of treatment (extraction of the tooth, apex resection etc.). If periapical chronic focus of inflammation is retained, it can cause and maintain chronic septic state and diseases conditioned by focus.

Conservative treatment can be unsuccessful in the following cases.

1) Root canal isn't fully filled.
2) Apical focus of chronic inflammation has communication with periodontal poket.

8. Alternative methods of periodontitis treatment.

There are the following conservative - surgical methods of periodontitis treatment: apex resection, coronoradicular separation, hemisection, amputation of root and replantation.

On the first stage of treatment, all permeable canals are treated endodontically, and then the roots become subjected to surgical intervention.

Apex resection means cutting off the affected root apex and removing pathologically changed tissues adjoined to the apex. Apex resection followed by curettage is indicated in the following cases;

- chronic granulating and granulomatous periodontitis, when it is impossible to remove chronic inflammatory process in periodontal ligament by conservative method;
- incomplete filling of root canal because of anatomic obstacle (curve, narrowing, denticles, obliteration of root canal);
- a part of broken instrument in the canal as a result of endodontic treatment;
- root fracture in the apical part;
- perforation of the wall of the root in its apical third.
- damage of root apex as a result of extraction of neighbouring unerrupted tooth.
- odontogenic cyst.
- excess filling material behind the apex, when lesion is formed in that area or when non arrested painful syndrome appears.

- biopsy for differential diagnosis of jaws tumors of odontogenic origin.

The most frequent indication for apex resection is granuloma or granulating process or periapical cyst. The contraindications to apex resection are:

- acute periodontitis or acute stage of chronic periodontitis, acute osteomyelitis
- when roots are situated near the walls of maxillary sinus or mandibular canal;
- the atrophy of alveolar process more than the half of the root length;
- significant destruction of crown and the adjoining subgingival part of the root as a result of caries, when it is impossible to restore the root with filling materials and core inlays:
- general diseases (cardio-vascular pathology, blood diseases, insulin dependent sugar diabetes, organic changes in kidneys etc.)

Apex resection is done as a rule in incisors, canines and premolars of the upper jaw and in canines of the lower jaw. This operation is more selectively done for treatment of upper molars and lower premolars.

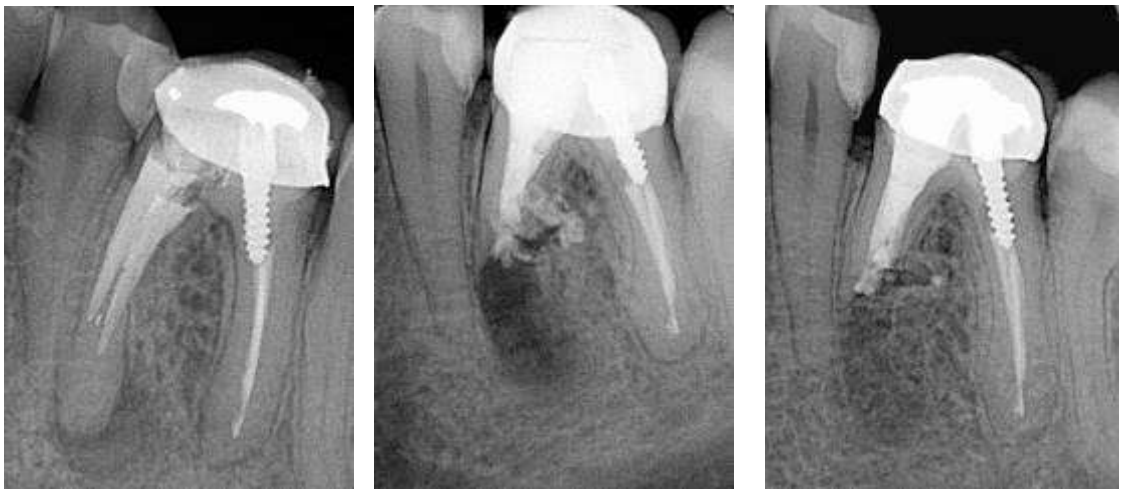


Fig.39 Radicular cyst 4,6; resection of mesial root.

Coronoradicular separation is done tor treatment of lower molars. Coronoradicular separation is the dissection of a tooth into two parts in the area of bifurcation followed by curettage in this area and

covering each segment of the tooth with crowns. The indications for coronoradicular separation are:

1. the affected periodontium in the area of bifurcation (2-3 degree) with lysis of apex or the radicular septum;
2. perforation of fundus of the tooth as a result of destructive process:
3. the presence of additional canals that join the cavity of the tooth with periodontal ligament in the area of bifurcation.

The operation is contraindicated in cases when bifurcation is close to the root apex, thus operation will remove major part of tissues and it will cause functional uselessness of the remaining parts of the tooth.

Hemisection is done on lower molars and upper premolars. Hemisection means extraction of a root together with the crown adjoining it. Thus a part of the tooth on alveolar crest is retained, the fragment retains proprioception and fulfils its function when included to prosthodontic construction. The place of amputation doesn't necesserily coincide with anatomic neck of the root, it should be done on the level of termination of affected area in periodontium.

Indications to hemisection and amputation of root in multirooted teeth are:

1. when bony pockets are longer than 3/4 of root in the area of one of the roots in a multirooted tooth,
2. reduction of bone in the area of bifurcation and exposure of one of the roots,
3. root fractures, vertical split of a tooth.
4. perforation in the area of bifurcation or wall of the root canal with the discharge of alveolus.
5. when a multirooted tooth with chronic periodontitis has one root with impermeable canals.
6. when one of the roots have large carious lesions.
7. in such cases when retention of the affected root can result in distribution of pathologic process to the neighboring teeth inspite of endodontic treatment.
8. when the tooth is used as support for bridge and when there is a significant bone discharge in one of the roots.
9. the presence of root cyst in which 1/3 of the root is situated.

10. when it is impossible to do apex resection because of anatomic obstacles.

Such operations can be done in multirooted teeth if their roots are separated and the remaining part of the tooth can be used to support prosthetic after endodontic treatment.

The containdications are:

- it is impossible to use the remaining part of the tooth for bridge or claps prosthetics because due to its functional unacceptability.
- acute and inflammatiry processes in oral cavity and periodontal ligament.
- general condition of a patient' s health.
- poor oral hygiene.

9. Periapical focus of chronic inflammation. Rehabilitation.

Stomatogenic persistent sepsis or stomatogenic focal infection is such a condition of the organism, when a disease of its system or an organ is in a pathogenic connection with a chronic or sometimes latent inflammatory process, taking place in the oral cavity.

Development of chronic focal ifection is promoted by the specific structure of dental tissues, large number of dead spaces, which create condition for pathogenic microflora to stay for a long time latent, food debris, moisture, favorable temperature. Development of study about chronic focal infection has made topics of immunity and infection, con-elation of the local and general quite urgent.

Septic reaction of the organism - is a special type of reaction, its mechanism may be influenced by many diseases, various microflora, endotoxins, products of inflammatory focal cellular elements disintegration. The role of microbes as an infections agent is significant. But final outcome depends upon the microorganism's ability to localize and neutralize the causative agent's activity, to paralyze its virulence.

Depending upon the source of infection chroniosepsis may be: odontogenic, otogenic, urogenic, umbilical, wound and so on.

The first data about the connection of dental diseases and various organs diseases can be referred to the ancient times. So, Hippocrate pointed out at the significance of the oral cavity recovering in joints diseases. In 1884-1885 Russian scientists M.S. Kocharkovsky, A.I. Kudrjashov supplied clinical and experimental data about the connection of dental diseases with rheumatic carditis, having pointed out the idea of the organism's integrity. In the beginning of the 20th century English and German scientists detected the influence of odontogenic foci of infection on the condition of the internal organs. In 1910 English scientist Gentor worked out a conception about chronic focal infection of the oral cavity. It was a surgical conception. As a result of it pulpless teeth were extracted. American scientists, Rozenov in particular, considered that in oral sepsis streptopneumococcal microbes change takes place, which under certain conditions may be virulent for the organism, that pathogenic microorganisms possess organic trophism. According to their theory, bacteria from a tooth with necrotizing pulp, getting into the blood stream, do not cause phenomena of general sepsis, but affect separate organs and organism's systems, to which they have affinity. But it was a bold, not sufficiently substantiated and that's why erroneous clinical conclusion, which caused mass extraction of pupless teeth. The theory of American scientists was based on the results of experiments, which were carried out methodologically in the wrong way, that's why their results were invalid. The theory of oral sepsis, worked out by English and American scientists, was severely criticized by European scientists at conference in Viesbaden in 1930. The question of oral cavity’s focal infection was first raised in Russia by professor S.S. Steriopulo in 1925. Professor Y.S. Pekker, who worked with him, pointed out accessability and easiness of chronic foci removal in the oral cavity. Both scientists confirmed the pathogenic role of chronic infectious foci in the teeth. But they doubted the simplified mechanism of bacteria getting from odontogenic foci to other organs. In 1928 B.N. Mogilnitsky first assumed pneumona of intoxication, issuing from the mouth cavity without generalization of the process, which is liquidated after stopping the local phenomena action.

M.M. Priselkov's (1933), I.G. Lukomsky's (1933), D.A. Antin's (1938) investigations not only revealed the bacteriological conception of American and English scientists, but substantiated their own

notions about the mechanism of stomatogenic local foci influence in the development of chronic odontogenic intoxication.

M.M Priselkov substantiated allergic mechanism of odontogenic sepsis, I.G. Lukomsky -neuro-toxic conception, D.A. Antin, referring to fundamental works by A.D. Speransky about the role of neurotrophic mechanism in the genesis of pathologic processes of the microorganisms, substantiated the conception of odontogenic oral sepsis pathogenesis. In that theory neurodystrophic process plays the main part, which appeares as a result of local irritation of initial focus neural elements. Development of a chronic disease is caused by nervous centers irritation and intoxication. The role and significance of sensitization during a stomatogenic infectious focus was confirmed by T.A. Vasiliev in 1933. In pathogenesis of chronic stomatogenic intoxication an allergic condition plays the main part, which occurs in chronic streptostaphylococcic intoxication (A.M. Rebreeva, 1962). Amino-like substances, formed at protein disintegration in a pathologic focus, cytotoxins are antigens. They change the organism's sensibility, causing sensitization. Organism's autoallergization is considered to be also very important, concerning focal infection, which is often observed in chronic apical granulating periodontitis.

I.G. Lukomsky (1955) pointed out three groups of clinically determined connections of an odontogenic focus with other organism's lesion. According to their activity centers of infection are divided into active, subjective and involutional.

Clinical symptoms of chronic odontogenic infection are various and nonspecific. To subjective symptoms headache, dizziness, vertigo, a heavy sensation in the head, fatigue, tremor and hyperhidrosis belong. Lymphadenitis in submental lymph nodes belong to objective data. For forming a diagnosis it is very important to know changes in peripheral blood morphologic content. The latter gives a notion about a degree of the local inflammatory process and about defensive forces of the organism (lymphomonocytosis, leukopenia, eosinophil ia, secondary anemia, erythrocyte sedimentation rate is 40 mm an hour and more). There may be changes in protein blood fractions (increase in globulin fraction, which is typical of an allergic reaction). Methods of determination of capillary circulation disturbance in an inflammatory focus:

1) methods of capillaroscopy, which detect increased capillaries, permeability (A.M. Nesterov, T.D. Zelensky, R.A. Karabaheva, (1953), T.E. Pertchikova, L.V.Ivleva, V.P. Kaznacheev, Ferent, (1957), Seizov (1956).
2) Histamine probe (cutaneus and conjunctival), based on change in vegetative nervous system reactivity;
3) Stendal's and Gelen's electrotest (1955) - based on functional disturbances and morphologic changes in neurovascular apparatus of mucous membrane and skin, situated directly above the place of chronic stomatogenic focus;
4) Determination of adrenaline susceptibility.

Adrenaline experiment is based on the fact, that at the presence of a chronic focus the patient's adrenaline susceptibility increases. Adrenaline injections (lml 0,1 % solution) increase arterial pressure, make pulse more frequent. After the focus elimination signs of increased adrenotrophism are not observed. (LA. Miisahovich). This test can be performed only among patients, who do not have cardiovascular diseases, chronic nephritis and so on.

Unfortunately each of these tests does not give reliable results, that's why 2-3 tests are usually carried out, adding sometimes insufficient clinical symptomatology.

Treatment of patients with a chronic odontogenic focal infection, caused by chronic periapical inflammation (chronic granulating periodontitis, chronic granulomatous periodontitis, cystogranuloma, radicular cyst of a jaw) must be strictly individual. The patient must be thoroughly examined according to the scheme: teeth with necrotized pulp are clinically determined, teeth under artifical crowns are examined; Roots of each separate tooth, suspected on the primary infectious focus presence, are roentgenologically investigated; parodontal recesses are detected; regional lymph nodes are investigated; blood analysis is carried out; histamin test, electrotest is carried out.

At determination of chronic stomatogenic infection and intoxication foci radical elimination of pathogenic periapical foci must be viewed as etiopathogenic and symptomatic treatment of systemic somatic diseases.

Russian scientists think it is possible to preserve single-rooted teeth with periapical focus of inflammation. In multirooted teeth

combined methods of chronic periodontitis treatment must be performed, such as hemisection, root's amputation, coronal-radicular separation, root apex resection, dental reimplantation.

Alongside with local treatment of teeth, one may also perform general treatment, directed at organs' and tissues' defense of organism's and its immunobiological forces resistance. Speaking about prophylaxis of chronic focal infection and intoxication it must be pointed out, that it includes caries prevention, oral cavity sanation at rheumatism, nephritis, endocarditis, polyarthritis and so on.

Works of Russian and foreign scientists on chronic odontogenic focal infection issues made stomatology closer to other disciplines. Stomatologists have drawn attention of doctors of other specialities to the change in organism's reactivity under the influence of chronic dental foci.

Their study of the problem asserts, that prophylaxis and treatment of internal organs diseases must be connected with oral cavity sanation.

Chronic odontogenic infection and intoxication must be differentiated with tuberculosis, diabetes, nephritis and other chronic diseases, having specific reactions and symptoms, compared with chronic sepsis.

10. Pitfalls and complications of periodontitis treatment.

Periodontitis treatment plays an impotent part in stomatological practice, that is why the question about diagnostic pitfalls and complications is very important in stomatology.

I. Diagnostic pitfalls appear when marginal and apical periodontitis, acute and exacerbated chronic periodontitis are not strictly differentiated. Roentgenologic investigation is very significant for final forming of a diagnosis. Pattern of a bone with its disturbances, widening of a periodontal fissure are characteristic sings of exacerbated chronic process. One must be very cautious, when examining teeth of the upper jaw, as an inflammatory process in the maxillary sinus may give symptomatology of periodontitis.

Examining periodontium, one may make a mistake, if only one dominating symptom acquires paramount importance. One must remember, that at diseases of alveolar appendix, intradental septa, soft

tissues of adjacent teeth, trifacial neuralgia, adjacent teeth pulp inflammation, symptoms of periodontium's lesion may appear.

One of the mistakes is wrong determination of the degree of the bone's lesion, since based on that the choice of method and success of treatment depends.

One must not make a mistake in determination of a latent carious cavity. One must assert to what extent apical periodontitis is a nidus of latent infection.

In diagnostics crucial moments are right accounting of the patient's general condition, estimation of local symptoms, complex of diagnostic investigation.

At treatment of chronic periodontitis it is a common mistake to start treatment without proper examination, especially without X-ray study of teeth and alveolar appendix.

II. Complications during carrying out of anesthesia can be of a local and a general character.

1. Breaking of an injection needle during carrying out of anesthesia is uncommon. For prophylaxis of this complication it is necessary to observe rules of maintenance of needles, to check their fastening in a cannula of a syringe.
2. Formation of hematoma is a result of a damage of a vessel with a needle. If a significant bleeding appears at site of an introduction of a needle it is recommended to press this area of tissue with a finger for several minutes. In three days it is possible to use warm applications. It is possible to prevent damage of vessels, moving a needle forward after a stream of an anesthetic solution.
3. Damage of a nerve trunk with an injection needle is possible. Paresthesia, desensitization, painful sensations of different intensity, which can last from 2-3 days to several months. Physiotherapeutic procedures, analgetics (per os), usage of vitamins of B group accelerate recovery. It is possible to prevent a damage of a nerve trunk, moving a needle further after a stream of an anesthetic. Getting of an anesthetic solution into blood flow can be observed in case of penetration of a needle into lumen of a vessel. As a result a high concentration

of the anesthetic becomes in blood, it can cause poisoning, to prevent complications it is recommended to pull piston of the syringe slightly toward oneself before introduction of an anesthetic. If blood stream appears in a syringe it is necessary to take the needle out partially and move it inside the tissues only after a stream of the solution.

4. Postinjection pains and edema appear if anesthesia is carried out roughly and quickly, as a result there can be a damage of periosteum. Pains and edema can be caused by inaccuracy in making an anesthetic solution or termination of its keeping time. For decongestion and pain relief physiotherapy is prescribed, analgesics and antihistamines (suprastin, pipolphen) are used. Observance of rules of carrying out of anesthesia injection prevents appearance of postinjection pains and edema.
5. Contraction of jaws (contracture) appears as a result of a damage of a muscle with a needle at failure of the anesthesia technique. More often internal pterygoid muscle is damaged during carrying out of mandibular anesthesia. Without any therapeutic measures contraction of a jaw stops in 2-3 days. If an infection appears and an abscess develops it is necessary to open it.
6. Paresis of branches of facial nerve can be observed when an anesthetic solution blocks some of its branches.
7. Necrosis of tissues can appear on hard palate after a rapid introduction of a solution under mucous membrane and periosteum under great pressure, as well as after an intraligament anesthesia.
8. A mistaken introduction of solutions which are not used for anesthesia. There also can be necrosis of tissues when ammonia spirit, solution of calcium chloride, etc. are introduced instead of an anesthetic solution by mistake. In this case it is necessary to make dissection of the tissues, to carry out an abundant infiltration of surrounding tissues with 0.25 - 0.5 % Novocaine solution, draining, to provide care for the wound.
9. Idiosyncrasy to local anesthetics is evident by appearance of red spots on skin, skin itch, sensation of heat, sweating, paleness of skin of face, sometimes by syncopal state. In order to suppress such reactions of an organism it is necessary to introduce

intravenously or subcutaneously 1-2 ml of 1% Dimedrol solution and 1-2 ml of 2.5 % pipolphen solution or 1-2 ml of 2% Suprastin solution mixed with 10 ml of 10 % solution of calcium chloride. The patient should be laid on a couch. A continuous control of a general state of the patient, pulse rate, respiration and arterial pressure is necessary. In case of an aggravation of the state it is necessary to send for a brigade of experts in resuscitation, since in case of hypersensitization of an organism to an anesthetic the first manifestation of the reaction can change to collapse or anaphylactic shock. Improvement of the state and paleness of red skin spots indicate a beginning of a reverse development of a perverted reaction of the organism. In this case in an hour the patient can go home. The patient should be warned that in future usage of the anesthetic to which there was the reaction is contraindicated. A note about intolerance of this anesthetic must be made in the case history on the first page.

Clinical picture of anaphylactic shock, which is characterized by an acute onset: skin redness and itch are changed by paleness of the skin with manifestation of cyanosis. The patient feels constraint in the chest, pain which can spread to the region of the abdominal cavity. The arterial pressure falls to 70 Hg mm, the pulse becomes weak, heartbeats become infrequent, loss of consciousness, cramps, heavy breathing are possible. An acute anaphylactic shock can end with a death after several minutes.

First aid in case of anaphylactic shock consists in an immediate introduction of 0.5 ml of 0.1 % adrenaline solution at the site where the anesthetic solution was injected to decrease its absorbability. Simultaneously the same quantity of adrenaline is introduced intravenously. To increase reactivity of organism it is necessary to prescribe glucocorticoids (1 - 2 ml of 3% prednisolone solution intravenously or subcutaneously),. 50 ml of 5% glucose solution, isotonic solution of sodium chloride are introduced by means of intravenous drip-feed. In combination with glucose solution 10 ml of 2.4 % aminophylline solution, 2ml of 2.5 % pipolphen solution and 2 ml of 2 % Dimedrol solution are introduced. By indications Corglyconum, strophanthine are used. If a terminal state develops it is necessary to start external cardiac massage, artificial ventilation or

introduction of adrenaline intracardially. After normalization of the arterial pressure, heavy breathing and regaining of consciousness the patient becomes transportable and must be taken to a department of intensive care and resuscitation in a special ambulance car.

Carefully obtained anamnesis by the doctor and data about intolerance of the patient to different drugs, which will be used for treatment, should have a significant place in prevention of anaphylactic shock.

III. Complications during processing of root canals

Perforation of a wall of a carious cavity more often happens close to the neck of a tooth. On vestibular and oral surfaces of a cavity perforation is detected visually. On approximal walls it is evident by a mild pain in the gum or if there is blood in the carious cavity. Perforation of a wall of a carious cavity happens if overhanging edges are not removed before. To eliminate the complications it is necessary to fill a cavity, which is to be filled.

Necessary conditions for prophylaxis of perforation of a wall of a carious cavity: a good visibility and an accurate orientation in respect to each of its walls.

Breaking off of a wall of a carious cavity is characterized by a defect of a crown part of a tooth. An immediate cause of a breaking off of a wall of a carious cavity is usually lever-like movements of instruments: an angle probe, an excavator. To eliminate a defect, related to a breaking off, it is necessary to form the cavity and fill it with an additional supporting ground or restoration of the defect with an insert.

Prophylaxis of the mistake consists of the exclusion of lever-like movements with an instrument and a complete excision of enamel, not having support on dentin.

Damage of adjacent teeth with a bur is observed not often. Adjacent teeth can be damaged with a bur during preparation of a carious cavity and its moving out on masticatory surface (palatine, lingual). Damage of adjacent teeth is provided by their overcrowding. Range of interventions, aimed at elimination of this complication, depends on degree of enamel damage. If superficial layers are damaged, when the formed defect doesn't have clear edges, it is sufficient to do only grinding of enamel edges and to carry out

remineralizing therapy. In such cases it is preferable to cover the tooth with a fluorine varnish. If a formed defect is within enamel with clear edges or if a defect is formed as a result of a trauma reaching enamel-dentinal junction a cavity is formed and filled. To prevent damages of adjacent teeth with a bur it is necessary to move out the semi-axis on a masticatory (or palatine, lingual) surface in all cases during preparation of approximal defects. Preparation must be started with formation of an additional ground after introduction of a metal matrix into an interdental space; it protects enamel of an adjacent tooth from damage.

Damage of a gingival edge can happen during an operative treatment of carious cavities localized on approximal surfaces and close to a dental neck. Sign of damage is bleeding, which is especially evident in a damaged inflammatory gum. In any case it must be stopped. Usually it is enough to treat the gum with 3 % solution of hydrogen peroxide and to press the gum with a cotton tampoon for several minutes. To prevent damage of gum during an operative treatment of a carious cavity it is necessary before an intervention to "press away" the edge of the gum or a gingival papilla touching the edge of the carious cavity with a tight cotton tampoon. In case of "growing-in" of a gum into a carious cavity its diathermocoagulation is carried out. 5% solution of aminocapronic acid, 0.1 % solution of adrenalin, caprophen, oxicylodex have a good hemostatic effect. Traumas of soft tissues surrounding a tooth do not always allow the filling of carious cavity during the first visit. If a dentist is not sure about a complete arrest of bleeding, i.e. about dryness of the cavity, it is advisable to postpone filling for the second visit in order to prevent falling out of the filling because of getting of blood, lymph into the carious cavity.

Right processing of root canals is of great importance. Insufficient opening of apical fissure in acute periodontitis treatment is a mistake. One should not forward the instrument to periodontal fissures too fast. It may cause infection of root apex and periodontal trauma. Poor mechanical cleaning of root canals may also be a mistake in perodontitis treatment. Leaving infected dentin in this case will prevent sterilization and creating hermetic filling. One may make

mistakes at medicational treatment of root canals, using strong drugs irritating periapical tissues and inactivating other drugs impact.

One must not continue conservative treatment of the tooth, when it must be extracted according to all indications.

A serious complication may be needles, pulp extractors, burs breaking in canal.

Underestimation of pulpal chamber and root canal topography may also have bad consequences. In this case perforation of the pulp chamber's floor and walls or canal walls may take place, that's why one must forward a needle very carefully. Insufficient filling of root canal or accumulation of excessive amount of material at root apex may be a mistake too. One may make a wrong choice of filling material for root canals. The material will gradually stain dental tissues.

It is a mistake to perform many visits for treatment or to prolong treatment too much. While working with instruments for root canal treatment, none of them should be left in the tooth without fixation. The patient may incidentally swallow it or it may get into the patient's respiratory tracts.

SUPPLEMENT

Situational task 1

The patient complains of pains in 11, appearing at biting on it, mobility of 11. In anamnesis, there occurred a trauma 24 hours ago, that caused the pains. Before, the tooth never pained and been treated. It is objectively found out, that crown 11 is not damaged, percussion 11 is painful, palpation of the mucous membrane in 11 and 12 is slightly painful, reaction on the cold absent.

Your diagnosis. Your (doctor's) tactics? What supplementary methods of examination must be carried out?

Situational task 2

After treatment of 16 by the methods of necrotizing pulp (acute diffuse pulpitis) in 48 hours (in two days) after arsenic paste application the patient suffers from pain, which appears at biting on the tooth and also painful sensation in the intradental space.

At examination: bandage on 16 is preserved, horizontal and vertical percussion of 16 is painful. The gingival papilla between 16 and 17 has dark staining, painful at palpation. Form a diagnosis. Your tactics.

Situational task 3

The patient,reffered to the dentist after objective examination, had a diagnosis of acute purulent periodontitis 15. Your tactics.

Situational task 4

The patient visited the dentist, pursuing an objective examination of a fistula in gingival 33, a deep carious cavity in 33, communicating with pulp chamber, probing, painful reaction to the cold were detected. What preliminary methods of examination must be carried out?

Situational task 5

A 30-year-old patient complained of falling of a filling from the tooth, which had been treated 3 years ago. During that time he sometimes experienced painful sensation in 26 at food intake.

Objectively: There is a carious cavity on mesial-masticatory surface in 26, not communicating with the cavity of the tooth. The floor is dense, pigmented, percussion is painful.

There is a cicatrix (0,1 x 0,1 cm) in the projection of medial root apex 26 on the vestibular surface. The symptom of vasoporesis (vasoslit) is positive. Submaxillary lymph nodes are not palpated.

Ennumerate diseases,which are quite probable for the symptomatology.

Name a preliminary diagnosis.

Choose from the task data, specific for the supposed diseases.

Indicate the results of supplementary methods of investigation, confirming the supposed diagnosis.

Ennumerate objects of medical influence in the supposed disease.

Situational task 6

A 53-year-old patient has no painful sensations. Last year tooth 11 ached, there was swelling, the doctor made only a cut. There is a carious cavity on medial-approximal surface 11, communicationg with the cavity of the tooth, probing, percussion are painful. Roentgenogram shows that the root canal is not filled, near apex 11 there is a bone tissue resorption focus with rounded margins of 0,5 cm size. Diagnosis: chronic granulomatous periodontitis 11. Your plan of treatment. Define a diagnosis.

Situational task 7

A 37-year-old patient complains of the upper lip deformation. Tooth 21 is intact, the crown has changed its color. There was a trauma earlier. Roentgenogram shows a bone resorption focus with rounded margins of 2 cm size in the projection of root apex 21. Diagnosis: radicular cyst of the upper jaw in 21. What is the doctor's tactics? Substantiate the plan of treatment.

Situational task 8

Patient B. complains on the presence of a cavity and insignificant painful sensation at biting on the 11-th tooth, presence of a fistula on the gingiva in root apex 11.

Objective: tooth 11 has a carious cavity of the IV-th class (Black), a fistula on the gingiva. Percussion is slightly painful,

probing is painless. Diagnosis: chronic granulating periodontitis 11. Can the tooth be filled during one visit? If it can, then with what filling material would it be better to fill the root canal?

Situational task 9

A 19-year-old patient complains on staining tooth 21. In anamnesis there was a trauma of frontal 12, 11, 21, 22 dental groups. The teeth are intact. Percussion is painful. The mucous membrane above 21 has cyanosis staining. Roentgenogram shows a bone resorption focus with rounded margins of 0,5 x 0,5 cm size in root apex 21. Your diagnosis. Your plan of treatment.

Situational task 10

A 35-year-old patient has no complaints. There is a carious cavity on the mesial-approximaly surface 23. There is a fistula on gingiva surfasce 23. Percussion of the tooth is painless. Roentgenogram shows a wide root canals a bone resoption focus with obscure margins of 0,5 cm size in the apex.

Diagnosis: granulating periodontitis 23. Choose a method of treatment. Substantiate it.

Situational task 11

A 47-year-old patient experiences painful sensations in 22 at food intake. In anamnesis the patient has rheumatism. Tooth 22 was treated earlier, (pulpitis). There is a filling on approximal-lingual surface 22. Percussion 22 is painful. Roentgenogram shows, that the canal is filled on 2/3 of its length, there is a bone resoption focus of uneven incorrect form of 0,5 cm size in the apex. Your diagnosis. Your plan of treatment.

Situational task 12

The patient refers to the doctor, pursuing an objective of mouth sanation. After analizing anamnesis a diagnosis is formed: chronic granulating periodontitis 46. In anamnesis it is pointed out, that the patient has polyarthritis, the patency of medial canals is poor. What methods of treatment can be used in this case to attain rehabilitation?

Situational task 13

A 20-year-old student complains of long pulsating pains in the tooth on the low jaw to the left, which irradiate to the ear, temple. Malaise, headache, sleep disturbances are marked. The tooth has been aching during 3 days. It used to hurt earlier, too. A week ago the patient endured acute respiratory disease (ARD).

Objectively: the mouth is half open, tooth 24 is mobile, there is a deep carious cavity on the masticatory surface, not communicating with the dental cavity. Percussion is painful. Roentgenogramm shows thinness (rarefaction) of bone tissue with obscure margins of 0,2 x 0,3 cm size, in root apex 24.Ennumerate diseases, concerning the symptomatology.Name a probable diagnosis. Choose all the essential data from the task, confirming the diagnosis. Indicate medical measures, necessary to be carried out at the first visit.

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