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DEPARTMENT OF THERAPEUTIC DENTISTRY

Educational and methodical textbook
for 3rd year students in the discipline «Therapeutic dentistry».

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Educational and methodical textbook are developed for studying the program in the discipline «Therapeutic Dentistry» by the 3rd year students of the dental faculty. These guidelines include methodological developments for conducting practical classes in conjunction with control tasks and a list of recommended educational and methodological literature, which are aimed at deepening students' knowledge of the diagnosis and treatment of diseases of hard dental tissues of various etiologies (carious and non-carious genesis), as well as the study of generally accepted and modern classifications of pathologies of hard dental tissues, their etiology, pathogenesis and clinical manifestations; approaches to the diagnosis of hard tissue pathologies of teeth using clinical and additional methods of examination of patients with odontopathologies; principles of treatment and prevention of the above diseases and their complications.

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INTRODUCTION

Despite the progressive development of modern dentistry, endodontic diseases remain quite relevant and account for about 40% of all dental diseases. Inflammation of the pulp occurs in accordance with the general patterns of development of the pathological process in other structures of the body.

The nature of the inflammation is determined by different levels of body reactivity and is characterized mainly by manifestations of exudation, alteration or proliferation. The inflammatory process in the pulp occurs in response to stimuli entering the tooth cavity. The etiological factors may include microorganisms and their toxins, chemicals, toxic substances, temperature, mechanical and other irritants.

Periodontitis is a disease that occurs in the periodontal tissues under the influence of various factors and is manifested by local inflammation, which can lead to damage to the ligamentous apparatus of the tooth and even tooth loss. The clinical picture of periodontitis is quite different, so it is necessary to study the main causes of this disease. The factors that can cause periodontal disease are quite diverse, and their nature is essential for the onset and development of the pathological process.

The study of the mechanisms of occurrence and course of inflammatory changes in the pulp, taking into account the state of the macroorganism, neuro-reflex activity and interaction of the body with the environment, creates the basis for understanding the essence of the development and course of inflammation of the tooth pulp and the theoretical justification for choosing a method of treatment of pulpitis and periodontitis at different stages of the clinical course.

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METHODICAL DEVELOPMENT №1

TOPIC: Organisation of dental care in Ukraine.

Sanitary and hygienic requirements for the dental office:

1. The area of the room
2. The height of the room
3. The width of the room
4. Requirements for:
- Walls of the office;
- Ceiling of the office;
- The floor of the office;
- Doors and windows of the office.
5. Lighting of the office:
- Natural;
- Artificial.
6. Microclimate:
- Air conditioning;
- Ventilation;
- Heating;
- Quartzing;
7. Distance between seats and their placement
8. Bathroom facilities

Dental office equipment:

I. Basic:

1. Dental unit:
   - Toolbox;
   - Control unit;
   - Hydraulic unit;
   - Lighting unit;
   - Dental chair;
   - Compressor.

2 Mobile chair with backrest for the doctor.

3 Chair for an assistant.

4 Dental table - mobile or stationary.

II. Additional:

1. Photopolymer lamp.
2. Scaler.
3. Diathermocoagulator.
4. Apex locator.
5. Endomotor and endonuclease.
6. Radiovisiograph.

III. Equipment for sterilisation and disinfection:

1. Dry oven for sterilising instruments.
2. Table with sets of sterile instruments.
3. Quartz lamp.
4. Fume hood.

IV. Equipment for hand treatment and pre-sterilisation:

1. Hand wash basin for washing hands.

2. Containers with disinfectant solution for soaking instruments with a cabinet for containers with disinfectant solutions.

V. Equipment for the work of a nurse:

1. A table for keeping records.
2. A chair.
3. Computer.
4. A safe for storing documents.

VI. Medical furniture:

1. The doctor's desk.
2. Cabinet for storing medicines of group A and B.
3. Cabinet for storing instruments, bandages and sealing material.

ORGANISATION OF THE DENTAL SERVICE

The foundations of state dental care in Ukraine were defined in April 1920 by the Order "On the State Organisation of Dental Care in the Ukrainian Republic", which legalised it as publicly accessible and free of charge with a preventive focus.

The first centralised management body for the dental service in Ukraine was the dental section of the Medical Medicine Department, established in April 1920 within the structure of the People's Commissariat of Health, with similar sections in provincial health departments.

In the early 20s, the search for optimal forms of organising dental care within the framework of an outpatient service was underway. Dentists were trained at odontology faculties established at medical institutes (Kyiv, 1920) and universities (Kharkiv, 1921), at odontology departments and dental schools. In many cities of Ukraine, private dental schools were transformed into public ones.

After the organisation of medical faculties into independent medical institutes, the departments of odontology were renamed into the departments of dentistry. A. I. Yevdokimov, I. G. Lukomskyi, E. M. Gofung, D. A. Epstein, Z. B. Pyriatynskyi invested a lot of effort and knowledge in the development of higher dental education.

The publication of a special journal, which is now published under the name "Dentistry", was important for the expansion of scientific knowledge.

In the 1920s, more than 400 dental outpatient clinics, more than 40 dental laboratories, 6 children's clinics in Odesa and a central school clinic in Mykolaiv were established on the basis of nationalised dental offices. In the early 1930s, there were 4 types of dental institutions in the country: independent dental outpatient clinics (factory and school), dental offices at dispensaries and hospitals, private dental offices and dental prosthetic
institutions. Since 1930, odontology faculties in Ukraine have been separated into independent dental institutes (in Kyiv and Kharkiv).

Currently, there are the Ukrainian Medical Dental Academy, the Research Institute of Dentistry in Odesa, and a number of dental faculties in Ukraine.

In the course of training, great importance is attached to improving the basic knowledge of dentists, especially in the context of possible specialisation in dentistry, in the so-called mini-specialties (periodontics, odontology and hygiene) within therapeutic dentistry, and material science in orthopaedic dentistry.

The formation of a new type of specialist involves improving the targeted training of dentists through the system of postgraduate training through internships, creating a "specialisation model".

Dental care (outpatient) is provided in Ukraine:

- independent dental clinics (republican, regional, city, district);
- dental departments within territorial polyclinics of cities, central district hospitals and primary health care centres at industrial enterprises;
- in dental offices of hospitals, dispensaries, antenatal clinics, educational institutions;
- -independent children's polyclinics;
- self-supporting polyclinics.

Admission to dental polyclinics and departments is carried out in the field of therapeutic, orthopaedic and surgical dentistry.

The modern dental clinic includes departments and offices:

- Therapeutic dentistry with a room for the treatment of periodontal and oral mucosa diseases;
- Department (office) of Surgical Dentistry with an operating unit;
- Orthopedic department with a dental prosthetic laboratory and a foundry;
- Mobile dental care and dental disease prevention department;
- X-ray rooms.

The dental clinic's examination room plays an important role in regulating the flow of patients.

Specialised outpatient dental care is one of the most widespread types of medical services provided to the population. The proportion of dental morbidity in the total morbidity of the population by referral reaches more than 16%. The number of people seeking dental care is second only to visits to general practitioners, with 90% of the population suffering from caries and 40% from periodontal disease.

In-patient dental care is provided in dental departments or specialised beds in republican, regional, municipal, district, and clinics of research institutes, medical universities, and institutes and institutes for the improvement of doctors.

Planning and management of the work of all institutions is entrusted to chief specialists.

The chief specialist is appointed from among the chief doctors of republican, regional and municipal dental clinics. The chief dentist, through the management of healthcare institutions and local healthcare authorities, must provide organisational, methodological and scientific guidance to the work of dental institutions.

Independent dental clinics

Regardless of the location (republican, oblast, city, district), according to the number of medical positions, they can be of 5 categories:

non-category - more than 40 positions;

- Category 1 - 30 - 40 positions;
- Category 2 - 25 - 30 positions;
- Category 3 - 20 - 25 positions;
- Category 4 - 15 - 20 positions;
- Category 5 - 10 - 15 positions.

The number of medical staff is calculated based on the population, assuming that each resident has 2.2 visits per year. For every 10,000 adults, 4.0 dentist positions (therapist + surgeon) and 1 prosthetist position are allocated for 4-5 dentist positions.

For every 10 thousand children, 4.5 dentist positions are allocated.

Currently, the number of dentists in Ukraine is 4.0 per 10 thousand people.

Structure of a dental clinic

The polyclinic includes special departments:
1 - therapeutic dentistry;
2 - surgical dentistry;
3 - orthopaedic dentistry;
4 - paediatric dentistry (where there is no children's dental clinic).
In addition, the polyclinic includes the following rooms:
1 - functional diagnostics;
2 - paradontology;
3 - anaesthesia;
4 - physiotherapy;
5 - radiological.

The polyclinic is managed by dental offices or departments at general polyclinics, dispensaries, schools, and the Ministry of Health. The polyclinic also includes
1 - dental laboratory;
2 - clinical laboratory;
3 - organisational and methodological office;
4 - registration office;
5 - administrative and economic part.

The dentist's office is equipped with a dental chair,

a universal dental or turbine drill with a set of dental instruments (spatula, mirror, tweezers, probes, etc.), instruments for filling and removing dental plaque, burs, pulp extractors, root needles, root canal fillers, filling material and medicines.

Doctors work in two shifts on a rolling schedule.

The district therapeutic principle of dental care has been developed. This takes into account the incidence of dental diseases, the age composition of the population, and the availability of medical staff.

One anaesthetist position is established for every 20 dental medical positions. Dental departments of hospitals and healthcare facilities

Dental departments are established where there are no independent dental clinics. They provide differentiated treatment. A department is created if there are 8 or more dentists. One of them is appointed head of the department.

Independent dental offices are set up in hospitals, polyclinics, antenatal clinics, medical centres, health posts, and schools.

Most often, dental care is provided to the public in a dental office. In hospitals with more than 250 beds, one dentist position is allocated and one additional position for every 300 beds above this. If a hospital has fewer than 250 beds, but has a dental office, 0.5 dentist positions are allocated.

Tuberculosis hospitals are allocated 0.5 positions for 75 to 200 beds. If tuberculosis hospitals have more than 200 beds, one dentist position is allocated. In paediatric tuberculosis hospitals, even if there are 50 beds, 0.5 positions of dentist are allocated.

In general polyclinics, if the number of dentists is less than 8, dental offices are created, but they must provide differentiated reception.

Dental offices are set up in medical and sanitary units and health centres if there are 1500 or more employees at all industrial enterprises and construction sites.

Chemical industry enterprises are allocated 0.75 positions per 1,000 employees.

Schools and universities have a dental office if there are 800 or more students.

In antenatal clinics, one position per 100,000 people is allocated, and 0.5 positions per 50,000 people. More than one position in an antenatal clinic is not allocated.

Functions of a dental clinic.

A dental polyclinic is the main specialised medical and preventive care institution that provides highly qualified and specialised care for diseases of the oral cavity and teeth.

The main functions of dental clinics are:

1) organisation and provision of specialised dental care to various population groups;

2) implementing the dispensary method in the activities of dentists to prevent diseases of the oral cavity and teeth;

3) organising and carrying out oral cavity sanitation both in a planned manner and when the population addresses dental institutions;
4) development and improvement of comprehensive medical check-ups in cooperation with district (shop) therapists, as well as paediatricians, obstetricians-gynecologists, ENT and other specialists of general and children's polyclinics, antenatal clinics, and dispensaries;
5) development of narrow-profile types of dental care and their approximation to the population;
6) improvement of treatment and diagnostic methods: physiotherapeutic, X-ray, biochemical, cytological, functional diagnostic, etc;
7) promotion of hygienic and medical knowledge in the field of dentistry among the population;
8) accounting and reporting dental documentation.

Medical examination of dental patients is a system of measures aimed at early detection of diseases of the teeth and other organs and tissues of the oral cavity and maxillofacial region; elimination of the causes of these diseases; quantitative examination and treatment of patients registered for medical examination and preservation of their working capacity.

The selection of dental patients for dispensary supervision should be carried out by dentists of all specialties, regardless of the place of work and the nature of the appointment.

Patients subject to dispensary supervision are referred to district and workshop dentists.

The following patients are subject to dispensary supervision by dentists with multiple progressive dental caries, fluorosis, periodontal disease of I-III degree, chronic gingivitis and stomatitis, cheilitis, glossalgia, odontogenic neuralgia of the trigeminal and facial nerves, chronic osteomyelitis of the facial bones, chronic sinusitis, chronic inflammation of the salivary glands, precancerous diseases of the jaws and cavity malignant neoplasms of the jaws and oral cavity, congenital clefts of the maxillofacial region, dentoalveolar anomalies, congenital and acquired deformities of the jaws.

A plan of treatment and preventive measures is drawn up for each patient in the clinic, which includes:

1) an in-depth study of the patient's working and living conditions and a detailed clinical examination;
2) outpatient (or inpatient) treatment: oral cavity sanitation, medication, surgery, physiotherapy, etc;
3) referral of a patient for consultation with doctors of another speciality (ENT 1, etc.)
4) compliance of the patient with personal prevention measures, work, rest, nutrition, and rational employment;

5) analysis of the effectiveness of dental patients' medical examination.
For each patient, along with the outpatient card, a control card of the dispensary patient is filled out (form Nº 030/y).

The main medical and preventive measures of the dispensary are: scheduled oral cavity sanitation, which is carried out from the age of 3 throughout life, primarily in organised teams.

Key performance indicators of dentists in the outpatient service:

Patient admission rates:

- general dentists - 3 persons per hour,
- surgeons - 5 persons per hour,
- orthopedists - 2 persons per hour,
- mixed reception - 3.2 persons per hour.
- Qualitative indicators of a dentist-therapist
- Prevention of dental caries.
- Scheduled sanitation of employees of leading professions with hazardous working conditions.
- Sanitation among schoolchildren and preschoolers.
- Sanitation of pregnant and lactating women.
- Sanitation of patients with sore throats and gastrointestinal diseases. Percentage of visits by rural residents.
- Share of primary visits.
- Share of sanitised patients among all primary patients.
- Percentage of patients in need of oral cavity sanitation identified from among those examined as part of routine sanitation.
- Percentage of sanitised patients among those identified as part of the routine sanitation.

A unit of work of a dentist is considered to be a completed work (applying a filling within 20 minutes, taking anamnesis, making a diagnosis, recording in the medical history, washing hands, processing and shaping the tooth cavity and applying a filling).

- The period of preservation of the filling is at least 2 years.
- Treatment of pulpitis - in 2 visits.
- Treatment of periodontitis - in 3 visits.

IV. Control questions for the class topic:

1.Organisation of therapeutic dental care in Ukraine.
2.Types and structure of dental clinics.
3.Functions of the dental clinic.
4.The main indicators of the work of a dentist - therapist.
5.Sanitary and hygienic requirements for the organisation of a dental office.
6.Equipment of the dental office.
7.Safety rules when working in a dental office.
8.Organisation of the workplace of a dentist - therapist.

Lesson №2

TOPIC: Equipment of the dentist's workplace: universal dental units and chairs. Safety precautions. Ethics and deontology in dentistry. Iatrogenic diseases.

Basic equipment

Traditionally, all dental equipment in an office is divided into the main equipment, without which it is impossible to perform medical procedures, and auxiliary equipment, which is necessary for specific manipulations.

The dental unit is a key element of the dentist's workplace. It provides the dentist with the conditions necessary for the efficient and technological performance of the main types of dental treatment.

Universal dental units

A modern dental unit is a complex set of electrical, mechanical and hydraulic elements that convert external energy into the energy of dental instruments and is designed to provide the necessary conditions for dental treatment.

Dental units can be classified:

1) by the way they are located in the office:
- stationary (fixed to the floor of the office);
- portable (in which the dental unit does not have a rigid connection to the chair);

2) special purpose:
- laser (for beam preparation);
- for endodontic work;
- for periodontal manipulations;

3) for professional hygiene:
- removal of dental plaque;
- bleaching with soda under pressure;

4) electric: for the installation of electric micromotors (brush and brushless) and piezoelectric scalers.

5) by the number of staff:
- only for the doctor;
- for the simultaneous work of a doctor and his/her assistant, i.e. the so-called "four-handed" principle.

According to modern views, ergonomic requirements are best met by the work of a general dentist with an assistant in a four-handed position with the patient lying horizontally, due to the reduction of unwanted movements of the working team and the reduction of the time required to perform almost all medical procedures.

Unlike a nurse, a doctor's assistant works directly in the oral cavity. Their duties include

- professional hygiene and cleaning of teeth before restoration;

- Participation in the identification of colour and shades;
- Assisting in the restoration of teeth;
- control over the cleanliness of the surgical field, evacuation of oral fluid and aerosols;
- operation of a polymerisation lamp and protection of personnel from polymerisation light;
- polishing of teeth undergoing restoration.

In addition, the assistant performs the usual duties of a dental nurse.

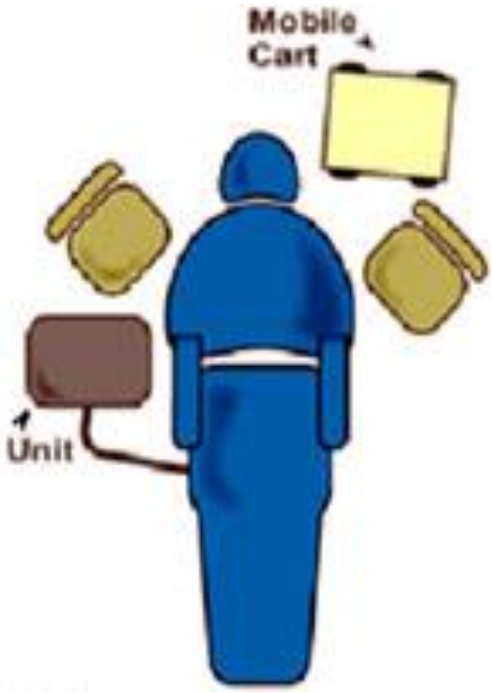
When choosing equipment, the main focus should be on the assistant's workplace, which will save the dentist from unnecessary movements. Access to handpieces and other dynamic instruments should be focused primarily on the assistant and only then on the doctor. The dentist's workstation should be to the right of the patient, and the assistant's workstation should be to the left.

It is also desirable that the positioning of the handpieces corresponds to the frequency of their use. For example, a turbine handpiece should be located closer to the doctor than a mechanical handpiece; a high-speed vacuum cleaner and a water/air gun should be located closer to the assistant than a saliva ejector. In order to reduce movements, all instruments should be within the reach of the assistant for quick transfer to the doctor.

Most dental units used by dentists are equipped with a back table and a side table. Figures 1-4 show schematic examples of four common types of dental unit design.

Installation with side feed

This design has been popular for decades. In fact, many dental schools use this type of unit, equipped with a fixed worktable, due to the fact that students usually have to work without an assistant. This style of setup requires the dentist to remove and replace handpieces, which in turn causes attention to shift from the working field to this procedure, forcing the dentist to turn his or her body to pick up the instrument and then refocus back to the working field. This can lead to physical fatigue and eye strain.



The side-feed design separates the assistant from the instruments and the ability to change handpieces or burs, which requires more unnecessary movements on the part of the clinician and therefore a loss of efficiency.

Installation with rear feed

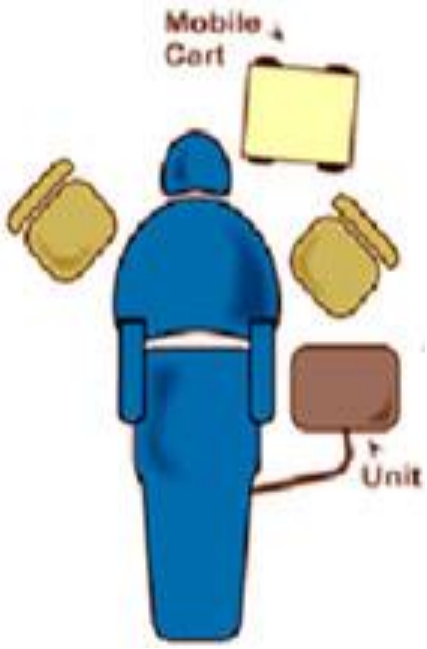
With this design, the dentist is forced to remove and replace the handpieces himself. This leads to constant turning, eye fatigue due to distraction from the working field. At the same time, there is often a moment when, in order to start working, it is necessary to transfer the handpiece from one hand to the other, the working hand. The units themselves are mounted in a fixed position, which does not allow adjusting it to change the working position to a more comfortable one for either the doctor or the assistant. The vacuum cleaner and water/air gun are fixed directly to the assistant's workspace. However, as the vacuum cleaner and gun are located at the back, this forces the assistant to bend over to reach them. When a movable table is used with a rear-feed unit, it can obstruct access to the sink, vacuum cleaner or water/air gun. For the assistant, this design will cause constant stress and fatigue, and limit their efficiency. However, despite ergonomic drawbacks, rear-feed units remain popular because they fit perfectly into the design of the room and hide the unit from the patient's view.

Split installation/tabletop

The concept of separating the unit and table separates the machine into the dentist's part and the assistant's movable table with vacuum and water/air gun. As with side-feed units, this design requires the dentist to change handpieces and makes them inaccessible to the assistant, thus reducing productivity. The assistant can only work with a vacuum cleaner and water/air gun that are integrated into the mobile table and is unable to hand over handpieces to the dentist and change burs. Very often, mobile tables of this design are not adapted for storage of auxiliary instruments and adequate space for storage of dental materials.

The split-level design of the unit can limit the assistant's workspace and forces them to place ancillary instruments in trays on a fixed table. This position requires additional movements to change the required instruments and materials stored in this way, and also opens the way for cross-infection of instruments stored in trays.

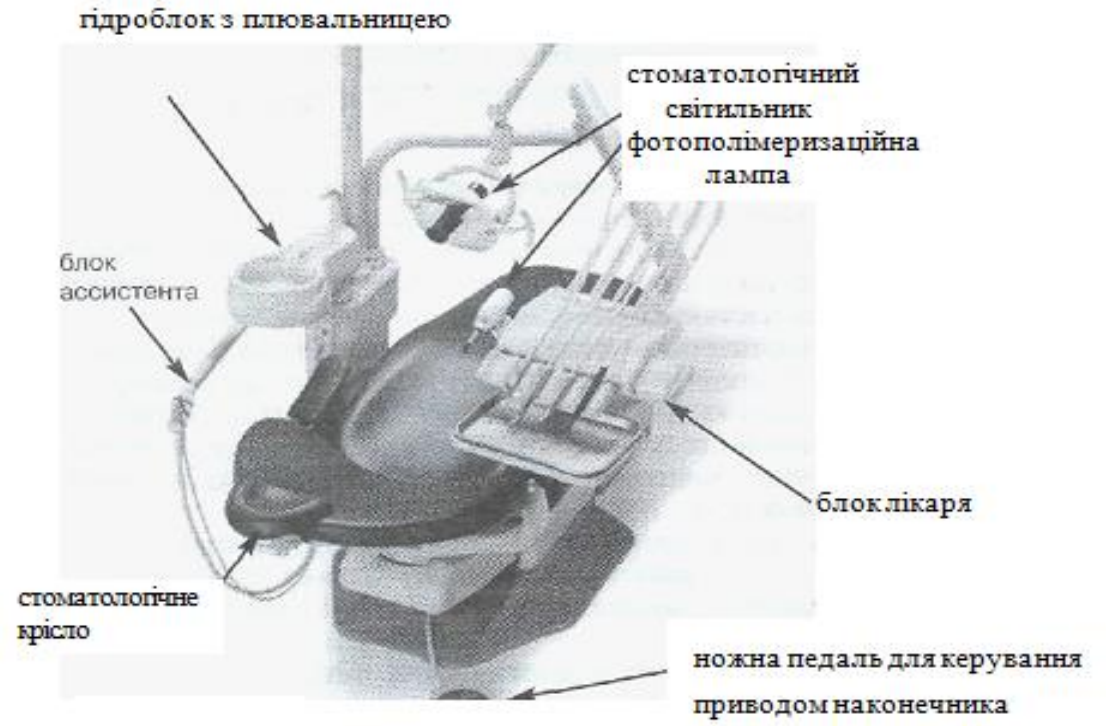
Transtorax type unit with chest table



The chest table setup offers the best ergonomic compromise between the movements required and the time spent. With the table positioned on top of the patient's chest, the assistant can easily change handpieces and hand them over to the dentist, saving the dentist from having to switch their gaze from the working field. Repeatedly switching a close-focused gaze from a brightly lit working field to a dimmer space at a distant distance forces the doctor's eyes to adjust to changes in lighting and distance, which can lead to eye fatigue and subsequent headaches. And a mobile table with a mobile top that slides over the sitting assistant's lap serves as the main source of materials and instruments, conveniently located within the assistant's reach. There are no hoses to restrict the assistant's mobility. This type of setup is specially designed for the most efficient four-handed operation.

The universal dental unit is equipped with the following units:

- electric and air drive for handpiece operation;
- modular dental unit has 2-3 hoses for micromotor and turbine handpieces;
- equipped with an air and water cooling system for the drills;
- connected to the water supply and sewerage system;
- pusher (water-air gun);
- equipped with an aspiration system (saliva ejector, vacuum cleaner), etc;
- a hydraulic unit with a spittoon;
- special dental lamp "Reflector";
- oil-free compressor;
- dental chair with automatic control.



In addition, the machines can be equipped with additional devices and equipment:

- system for monitoring the working field through the tip;
- built-in lamp for light curing materials with a wavelength of 450 - 500 nanometres;
- ultrasonic scaler for removal of dental plaque;
- diathermocoagulator;
- computer
- radiovisiograph;
- intraoral video camera;
- endodontic microscope.

Safety precautions in the organisation of work in dental offices

Safety in dentistry is aimed at preventing injuries to patients and medical staff during treatment.

Each dental department (office) must have occupational health and safety (OHS) instructions in place.

Regular OHS briefings shall be conducted. Responsibility for compliance with the requirements of occupational health and safety lies with the chief physician and heads of departments.

Dental offices (therapeutic, surgical, orthopedic, children's, orthodontic) must have 14 m2 of space for one dental chair and 7 m2 for each additional chair. If the additional chair has a universal installation, the area is increased to 10 m2 In therapeutic and surgical rooms, no more than three chairs should be placed, and in surgical rooms - no more than two chairs with the mandatory separation of

doctors' workplaces by opaque partitions up to 1.5 m high, a nurse's workplace should be equipped.

The walls of dental offices should be smooth, without cracks. All corners and joints of walls, ceilings and floors should be rounded, without cornices and decorations. The walls of surgical rooms and the sterilisation room shall be lined to a height of at least 1.8 m, in the operating room - with glazed tiles. Above the panel, the walls are painted with oil or water-based paints.

Adverse factors in the work of dental professionals are noise and vibration caused by the use of new dental equipment - high-speed drills and turbines, which generate a high noise level of 75-100 dBA. This leads to a decrease in hearing acuity and can cause deafness. It has been established that high noise levels lead not only to changes in the hearing organ, but also in the central nervous system, cardiovascular and other systems.Правила техніки безпеки

1. Medical staff wearing special clothing and footwear:

- Dental gown - protects clothing from contamination and the patient from contamination from clothing.
- Medical cap - protects the hair from damage during the movement of the dental unit and prevents during the movement of the dental unit and prevents clogging with turbine aerosol.
- Medical mask - protects the dentist from inhalation of microorganisms exhaled by the patient and turbine aerosol.
- Dental goggles - transparent goggles that cover the eyes, including the sides. They protect the eyes from aerosol, dust and blood drops. Instead, a dental shield can be used - a transparent plastic sheet that covers the entire face.
- Dental gloves. They differ from general medical gloves in that they have a special relief on the fingertips to prevent the instrument from slipping.
- Changeable footwear - with a smooth surface, easy to clean; wearing shoes only inside the medical facility.
- Compliance with the rules of work with flammable, strongly acting and poisonous substances.

2. Compliance with the anti-epidemic regime. A physician who strictly adheres to safety rules reduces the risk of harm to health not only to his/her own health, but also to the health of patients and colleagues.

The role of the doctor in the occurrence of iatrogenic diseases

The term "iatrogeny" was coined in 1925 after the publication of the work of the German psychiatrist Bumke (O. S. E. Bumke) "The physician as a cause of mental disorders". Since then, specialists in various clinical professions have been actively researching this concept and trying to formulate its definition.

According to one of them, iatrogenic diseases (from the Greek iatros - doctor and gennao - to create, produce) are various pathological processes associated with preventive, diagnostic, therapeutic and resuscitation measures.

Thus, it is any harm caused to a patient by a healthcare professional without malice.

According to this definition, iatrogenies can be divided into eight groups:

1. Iatrogenies associated with preventive measures - vaccinations and their complications, violation of the epidemiological regime - including infectious diseases caused by them.
2. Iatrogenies associated with diagnostic examinations due to unsafe research methods, technical errors, equipment failure.
3. Iatrogenies associated with medicinal, surgical and other treatment methods: intolerance, erroneous use, wrong choice of treatment method, anaesthesia hazards.
4. Iatrogenies associated with resuscitation (rib fracture during closed heart massage, etc.); complications caused by catheterisation and puncture of veins and arteries.
5. Iatrogenies associated with changes in the patient's psyche due to unethical actions of healthcare professionals: careless words in the presence of the patient; disrespectful attitude towards the patient.
6. Iatrogenies associated with shortcomings in the organisation of medical care and patient care - lack of beds in the hospital, long walks to the examination rooms.
7. Iatrogenies associated with inactivity or failure to provide medical care for one reason or another.
8. Iatrogenic pseudo-illnesses, including "computer pseudo-illnesses", associated with the establishment of an incorrect diagnosis and the conduct of inappropriate treatment. This can be observed during mass examinations, as a result of diagnostic errors in the equipment.

However, if we accept this classification and definition of iatrogenic diseases, then any pathology that occurs as a result of a doctor's action can be attributed to them. Therefore, it is not justified to expand the content of the concept of "iatrogenic diseases". After all, some actions of healthcare professionals that have negative consequences cannot be avoided, such as injuries during surgery.

On the other hand, complications caused by incorrect diagnosis and treatment fall into the category of medical errors or medical offences as failure to provide medical care.

The occurrence of iatrogenic diseases depends not only on the behaviour of the doctor, but also on the patient: his or her gender, age, emotionality, and intellectual development. Depending on the peculiarities of the psyche, the type of nervous activity, different patients react differently to the actions or some words of the doctor. For example, women are more likely to have iatrogenies.

Among age groups, adolescents, menopausal women, and older people are more likely to be affected. The public's awareness of diseases and the prevalence of diseases at a certain time play a major role in the occurrence of iatrogenic

diseases. For example, at the beginning of the twentieth century, there were many people who considered themselves to have tuberculosis or syphilis. Today, people often "look for" cancer and HIV/AIDS.

The main method of treatment for iatrogenic diseases is psychotherapy, which can be supplemented with appropriate symptomatic and sedative treatment. In no case should you tell a patient that they do not have a disease and do not need treatment. You need to take an active part in their treatment. Sometimes it is necessary to involve a psychologist or psychiatrist in treatment.

Lesson № 3
TOPIC: Scheme of examination of a dental patient. Subjective examination: complaints, medical and life history. Characteristics of pain syndrome in various dental lesions

Diagnosis and treatment planning are common elements in all disciplines of dentistry. In evaluating a patient, the clinician evaluates information from the history and clinical findings; this information may suggest a clinical diagnosis. The purpose of a diagnosis is to determine what problem the patient is having, and why the patient is having that problem. Ultimately, this will directly relate to what treatment, if any, will be necessary.

The process of making a diagnosis can be divided into five stages:

• The patient tells the clinician why the patient is seeking advice.

• The clinician questions the patient about the symptoms and history that led to the visit.

• The clinician performs objective clinical tests.

• The clinician correlates the objective findings with the subjective details and creates a tentative differential diagnosis.

• The clinician formulates a definitive diagnosis. This information is accumulated by means of an organized and systematic approach that requires considerable clinical judgment. Questioning, listening, test-ing, interpreting and finally answering the ultimate question of why will lead to an accurate diagnosis and in turn result in a more successful treatment plan. An initial historical profile should identify the patient; establish the chief com-plaint; refiect the dental history; document drug allergies or other adverse drug effects; identify medications, vitamins, dietary supplements, or special diets; and provide a record of past and present illness, major hospitalizations, and a review of major organ systems. The historical profile shall be reviewed with the patient at each subsequent appointment and any new information obtained should be documented in the progress notes.

Patient Identification: The basic biographical data should include the patient's name, age, sex, ethnic extraction, marital status, occupation and place of residence. The date of the evaluation also must be recorded. Not only are these items essential for patient identification but also they may provide invaluable background information for the differential diagnosis of certain conditions, or identify patients in a high — risk category for a variety of diseases.

CHIEF COMPLAINT. Upon arrival for a dental consultation, the patient should complete a thorough registration that includes information pertaining to medical and dental history. This should be signed and dated by the patient, as well as initialed by the clinician as verification that all of the submitted information has been reviewed The clinician may find dental pathosis, but it may not be the pathologic condition that mediates the patient's chief complaint. Investigating these complaints may indicate that the patient's concerns are secondary to a medical condition or possibly a result of recent dental treatment.

Occasionally, the chief complaint is simply that another clinician correctly or incorrectly advised the patient that he or she had a dental problem, with the patient not necessarily having any symptoms. There-fore, the clinician must pay close attention to the actual expressed complaint; determine the chronology of events that led up to this complaint and question the patient as to any other pertinent issues, including medical and dental history.

For future reference and in order to ascertain a correct diagnosis, the patient's chief complaint should be properly documented, using the patient's own words.

Character of the Symptoms: The most common complaint causing a person to seek the services of a healthcare provider is pain.

Determine its character. Is it sharp or dull? Is it pain or is it merely discomfort? Does it appear suddenly and disappear quick-ly, or does it gradually increase in intensity and subside slowly? A lesion should be inspect-ed. Is it white, red, pigmented, ulcerative, vesicular, bullous, exophytic, or a combination of these various characteristics? Admittedly, this observation is part of the examination, not the history, but there are at least two good reasons for doing it at this point in time. First, it establishes the dentist's concern for the patient's problems, and second, it may suggest additional questions to be asked during the history - taking process.

MEDICAL HISTORY. A fundamental principle in establishing a diagnosis is gathering information relevant to the disease process. The clinician must complete the database before beginning the interpretive and decision making process. The database begins with the patient's medical history. Obtaining a comprehensive written medical history is mandatory and should precede the examination and treatment of all patients.

The medical history provides information regarding the patient's overall health and susceptibility to disease and indicates the potential for adverse reactions to treatment procedures. Information regarding current medications, allergies, and diseases, as well as the patient's emotional and psychologic status, can be assessed as it relates to the clinical prob-lem. This information is important in diagnosis because the patient may have a systemic disease with oral manifestations. Moreover, a systemic disease may present initially as an oral lesion.

The clinician is responsible for taking a proper medical history from every patient who presents for treatment. It is imperative that vital signs be gathered at each treatment visit for any patient with a history of major medical problems. The temperature of patients presenting with subjective fever or any signs or symptoms of a dental infection should be taken.

The clinician should evaluate a patient's response to the health questionnaire from two perspectives:

(1) those medical conditions and current medications that will necessitate altering the manner in which dental care will be provided and,

(2) those medical conditions that may have oral manifestations or mimic dental pathosis. Patients with the following medical conditions may require either a modification in the manner in which the dental care will be delivered or a modification in the dental treatment plan: cardiovascular, gastrointestinal, haematologic, neurolog-ic and other diseases. Several medical conditions have oral manifestations, which must be carefully considered when attempting to arrive at an accurate dental diagnosis.

Many of the oral soft tissue changes that occur are more related to the medications used to treat the medical condition than the medical condition itself. If at the completion of a thorough dental examination, the subjective, objective, clinical testing, and radiographic findings do not result in a diagnosis with an obvious dental etiology, then consideration must be given that an existing medical problem could be the true etiology. In such instances a consultation with the patient's physician is always appropriate.

DENTAL HISTORY. The taking of a dental history allows the clinician to build rapport with the patient and is often more important than the examination and testing proce-dures. The dental history almost always contributes to the establishment of a diagnosis. The dental history should include the chief complaint and a history of the present illness if the patient has signs and/or symptoms of disease. The clinician should question the patient regarding the inception, location, type, frequency, intensity, duration, and cause of any pain or discomfort to develop a differential and definitive diagnosis. The process of information gathering may provide the clinician with a tentative diagnosis and guide the examination and testing process.

Pain is a complex physiologic and psychologic phenomenon and often cannot be used to differentiate endodontic problems from non-endodontic pathosis. Identifying the source of a patient's pain may be routine or complex. Inflammation and pain in the dental pulp are often difficult to localize and may be referred to a tooth in the opposing quadrant or to the preauricular region. A history of previous pain from a symptomatic tooth is also an important finding.

The chronology of events that lead up to the chief complaint is recorded as the dental history. This information will help guide the clinician as to which diagnostic tests are to be performed. The history should include any past and present symp-toms, as well as any procedures or trauma that might have evoked the chief com-plaint.

Proper documentation is imperative. It may be helpful to use a premade form to record the pertinent information obtained during the dental history interview and diagnostic examination. The dialogue between the patient and the clinician should encompass all of the details pertinent to the events that led up to the chief complaint. The conversation should be directed by the clinician in order to produce a clear and concise narrative that chronologically depicts all of the necessary information about the patient's symptoms and the development of these symptoms. After starting the interview and determining the nature of the chief

complaint, the clinician continues the conversation by documenting the sequence of events that promulgated the request for an evaluation.

The dental history is divided into *five basic directions of questioning: localization, commencement, intensity, provocation and the duration of pain.*

With the dental history interview complete, the clinician has a better understanding of the patient's chief complaint and can concentrate on making an objective diagnostic evaluation, although the subjective (and artistic) phase of making a diagnosis is not yet complete and will continue after the more objective testing and scientific phase of the investigatory process.

Anamnesis Morbi. A number of questions should be considered. How long has the condition associated with the patient's chief complaint been present? Has the problem developed slowly or rapidly? Some conditions have a sudden onset, but others begin slowly and insidiously. Have the symptoms become worse or better? Are they better at times and worse at other times? The oral healthcare provider should document a history of allergic drug reactions and other adverse drug effects and investigate whether drugs or medications are being taken. Many patients habitually take drugs for minor complaints, a practice that should be documented carefully. Patients often do not recognize nonprescription medications as drugs and, therefore, do not mention the habitual use of aspi-rin, decongestants, antihistamines, vitamins, and many other over - the - counter medications. Inquire about dietary supplements or special diets the patient may be on. Immunosuppressant therapy may place a patient in the high - risk category for many viral, fungal, and bacterial infections and de novo malignancies.

Anamnesis Vitae. Family History. The dentist should inquire about the patient's general health, as perceived by the patient and summarize past and present medical conditions. A clinician must record any hereditary or developmental abnormalities.

Previous operations, injuries, accidents and hospitalizations should be recorded, as well as comments about anaesthesia, drug reactions, blood transfu-sions, or transmissible diseases. In addition to hereditary conditions, acquired infectious diseases may be transmitted from one family member to another, some requiring only casual contact, while others are transmitted only through repeated, intimate encounters (sometimes associated with child abuse).

The personal habits of patients may reveal important clues to diagnosis. Excessive use of tobacco and alcohol may produce symptoms whose significance is lost without knowledge of a patient's smoking and drinking habits. Information about educational, social, religious, and economic background and feelings of achievement or frustration can provide important insight into understanding the patient as a person.

Review of Organ Systems. The chief complaint and the medical, family, and social histories of the patient should guide the clinician to investigate areas of special concern. All signs and symptoms related to specific organ systems should be recorded. The status of organ systems may suggest the presence of

concomitant systemic conditions, contribute to the diagnostic process and influence projected treatment protocols and prognosis.

Lesson № 4

TOPIC: Clinical examination methods, their importance for the diagnosis of diseases of the oral cavity: examination (external, of the patient's face, vestibule, oral cavity, dentition). Examination of the disease site

EXTRAORAL EXAMINATION. Basic diagnostic protocol suggests that a clinician observe patients as they enter the operatory. Signs of physical limitations may be present, as well as signs of facial asymmetries that result from facial swelling. Visual and palpation examinations of the face and neck are warranted to determine whether swelling is present. Palpation allows the clinician to determine whether the swelling is localized or diffuse, firm or fluctuant.

These latter findings will play a significant role in determining the appropriate treatment. Extraoral facial swelling of odontogenic origin typically is the result of endodontic etiology because diffuse facial swelling resulting from a periodontal abscess is rare. Swellings of non-odontogenic origin must always be considered in the differential diagnosis especially if an obvious dental etiology is not found. Sinus tracts of odontogenic origin may also open through the skin of the face.

These openings in the skin will generally close once the offending tooth is treated and healing occurs. A scar is more likely to be visible on the skin surface in the area of the sinus tract stoma than on the oral mucosal tissues. The term fistula is often inappropriately used to describe this type of drainage. The fistula, by defini-tion, is actually an abnormal communication between two internal organs or a pathway between two epithelium-lined surfaces.

The extraoral examination may give the clinician insight as to which intraoral areas may need a more focused evaluation. Extraoral swelling, localized lymphade-nopathy, or an extraoral sinus tract should provoke a more detailed assessment of related and proximal intraoral structures. Palpation of the cervical and submandibular lymph nodes is an integral part of the examination protocol. If the nodes are found to be firm and tender along with facial swelling and an elevated temperature, there is a high probability that an infection is present.

INTRAORAL EXAMINATION. Visual inspection of the soft tissues should include an assessment of colour, con-tour, and consistency.

Localized redness, edema, swelling, or a sinus tract can indicate inflammatory disease.

Examination of the hard structures may reveal clinical findings such as developmental defects, caries, abrasion, attrition, erosion, defective restorations, fractured cusps, cracked teeth, and tooth discolouration. Intraoral swellings should be visualized and palpated to determine whether they are diffuse or localized and whether they are firm or fluctuant.

These swellings may be present in the attached gingiva, alveolar mucosa, mucobuccal fold, palate, or sublingual tissues. Other testing methods are required to determine whether the etiology is endodontic, periodontics, or a combination of these two or whether it is of a nonodontogenic origin.

EXAMINATION OF THE ORAL CAVITY. Dentists should have a special interest in the physical examination of the oral cavity since the mouth is the anatomical area of the body for which they are the ultimate authority. Therefore, the organization of this section is more detailed and provides greater emphasis on possible findings and interpretation of data.

Basic instrumentation for the oral examination includes a *good light source, a mouth mirror, an explorer, a periodontal probe, dry gauze sponges, and an air syringe.* The need for specialized instrumentation and additional diagnostic procedures will vary with the findings and differential diagnoses developed.

Examine the Teeth. Note the number, size, shape and colour of teeth, There are such terminology employed in the literature around caries diagnosis (which should imply a human professional summation of all available data), lesion detection (which implies some objective method of determining whether or not disease is pres- and lesion assessment (which aims to characterise or monitor a lesion, once it has been detected.

Direct Dentin Stimulation. This is probably the most accurate and, in many cases, the best pulp vitality test.

Exposed dentin may be scratched with an explorer; however, the absence of a response is not as indicative as the presence of a response. Caries are probed deeply with an explorer to non-carious dentin, sudden, sharp sensation indicates that the pulp contains vital tissue.

Visual-tactile examination. Visual changes of the dental structure resulting from the demineralization process can be visually observed during caries develop-ment, such as an increase in opacity and roughness of the enamel.

Visual examination has been widely used in dental clinics for detecting carious lesions on all surfaces. This method is based on the use of a dental mirror,

a sharp probe and a 3-in-l syringe and requires good lighting and a clean/dry tooth surface.

The examination is based primarily on subjective interpretation of surface characteristics, such as integrity, texture, translucency/opacity, location and colour. However, tactile examination of dental caries has been criticized because of the possibility of transferring cariogenic microorganisms from one site to another, leading to the fear of further spread of the disease in the same oral cavity. More-over, use of an explorer can cause irreversible damages to the iatrogenic and demineralized tooth structure.

Visual and tactile methods typically go hand in hand, because most dentists use dental probes and other tools to examine teeth during the clinical examination. The first visual indication of caries in enamel is generally small white lesions where demineralization has occurred under the dental plaque. However, greyish lesions also can be seen at the level of dentin, and in more advanced stages, caries can appear as open cavities on enamel, dentin layers, or all the way to the pulp.

Other tools used in visual-tactile examination may include magnifying devices to look at teeth, or orthodontic elastic separators to separate teeth over the course of 2 to 3 days for a closer look between teeth prone to caries lesions. Fibre-optic transillumination is also sometimes used. This is a method by which visible light is emitted through the tooth using an intense light source. If the transmitted light reveals a shadow, this may indicate a carious lesion. Vision aids: Combination visual inspection of lesions with binocular magnification, radiographs and probes.

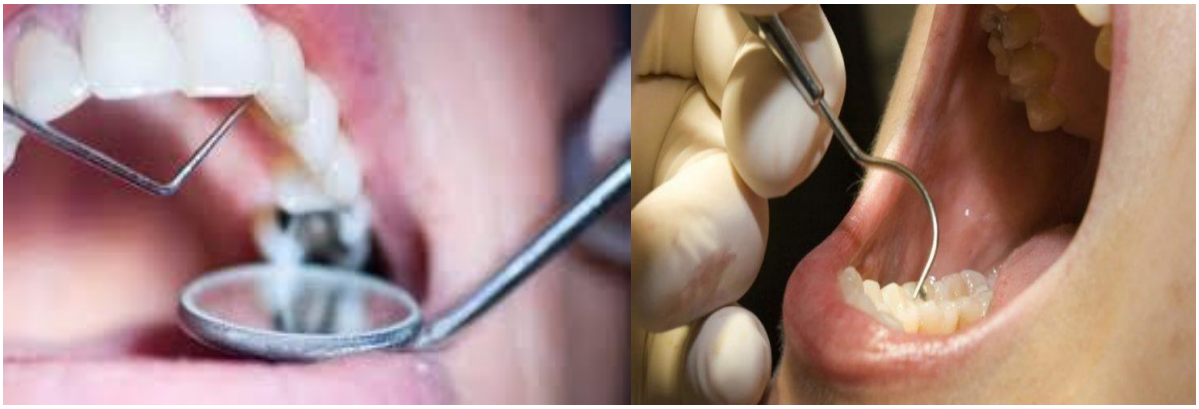
All combinations shared the low sensitivity whilst the combination of bitewing radiographs and visual inspection significantly improved the sensitivity. The traditional method of detecting caries signs is by visual inspection of dental surfaces, with the aid of a bright light and dental mirror if necessary to see teeth from all angles. Reflecting light onto the mouth mirror also can be done to search for dark shadows that could indicate dentin lesions. The coronal carious lesion starts as a clinically undetectable subsurface demineralization. With further progression, it will (eventually) become clinically detectable, and then can be classified according to type, localization, size, depth, and shape.

The visual method, a combination of light, mirror, and the probe for detailed examination of every tooth surface, is by far the most commonly applied method in general practice worldwide.

Probing. The probe didn't add to the sensitivity of vision, and even decreased the sensitivity of visual diagnosis (0.6 versus 0.65). In an «in vitro» validation study of the sharp probe to detect fissure caries, sensitivities were found for caries stages at initial and deep stages.

Clinical examination was quite variable between practitioners owing to the size and shape of the explorer tip, the force applied, and the judgment of the examiner. This examination uses a fine, pointed probe to test for surface defects in the enamel. If the probe remains stuck when probing the fissures, this is a sure

sign of dentin softening. While the use of a dental probe continues to be controversial, it is extremely helpful when used correctly and judiciously.



A dental probe can be used to remove plaque that may be covering, and when the blunt side of the probe is used, it can help remove bio-film to check for signs of demineralization and to assess the surface roughness of a lesion. Studies show that gentle probing does not disrupt the surface integrity of non-cavitated lesions, while vigorous poking can cause irreversible damage to the surface of a developing lesion.

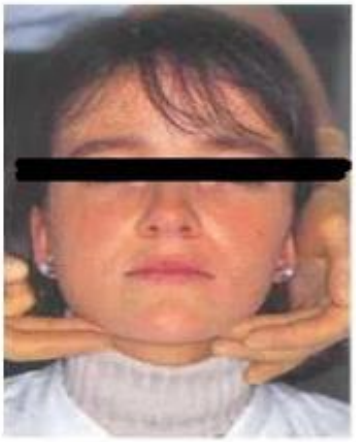
A probe is unnecessary if visual inspection detects a cavity. During a visual-tactile examination, the dentist will also use a syringe or drying tool to blast air on to the tooth, which makes it easier to see some lesions. If in doubt, or to confirm the visual assessment, the probe can be used gently across a tooth surface to confirm the presence of a cavity apparently confined to the enamel.

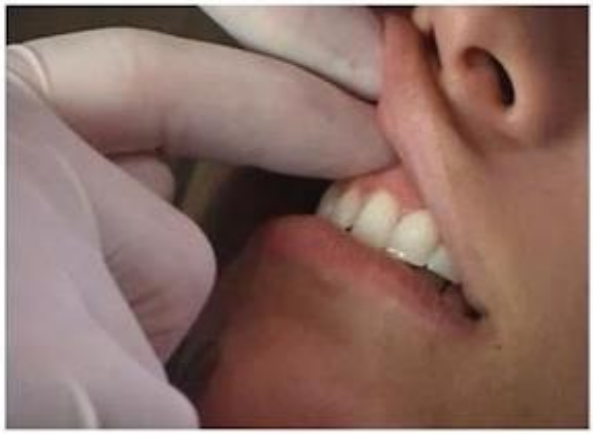
This is achieved by sliding the ball end along the suspect pit or fissure and a limited discontinuity is detected if the ball drops into the surface of the enamel cavity/discontinuity. The same dragging motion used to clean the fissure using a blunt probe conveys to the dentist information about the surface roughness around the walls of lesions and an idea if the base is soft or hard. Further results had showed arrested lesions became smooth and lost surface frostiness especially with regard to root caries. In cavitated lesions, the information, which could be gained, is enormous.

The highest increase in the tools performances was noticed for the probe in cavitat-ed lesions. Even for non-cavitated, the probe is still accepted for the removal of plaque from the fissures that might obscure cavities and to improve the access of direct vision. Care must be applied whilst using the probe not to produce damage by disrupting the continuity of the surface. Probing of root caries lesions with a sharp explorer using controlled, modest pressure, however, may create surface defects that prevent complete remineralization of the lesion.

The technique of temporary elective tooth separation as an aid to diagnosis of caries in proximal smooth surfaces is now regaining popularity, albeit with less traumatic methods that seem acceptable to most patients and dentists. This

method permits a more definite assessment of whether there is a detectable proximal enamel lesion by using radiography.





Tooth separation can be used as a method for examination of a suspicious area on the approximal surface. With this technique an orthodontic elastic separator can be applied for 2-3 days around the contact areas of approximal surfaces, facilitating the clinical and probing assessments. However, this method might create some discomfort and requires an extra visit. Studies have shown that tooth separation have detected more non-cavitated enamel lesions than visual-tactile examination without separation or bitewing examination.

The presence of cavitation (loss of surface integrity) associated with a root caries lesion does not necessarily imply lesion activity. Non-cavitated (early) root caries lesions almost universally are considered to be active. A cavitated lesion, however, may be either active or arrested. Lesion activity has been linked to lesion depth, but this clinical observation has not been verified. The texture of a root caries lesion also has been linked to lesion activity.

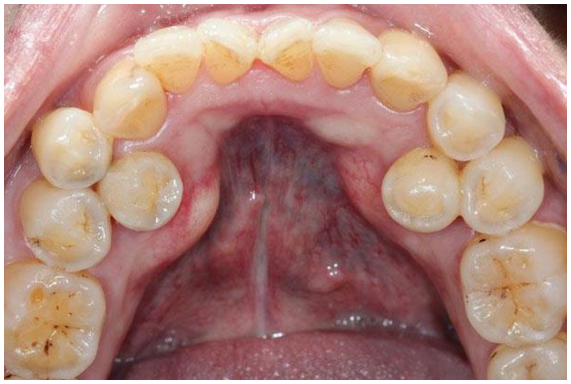
Active lesions have been described as soft or leathery compared to arrested lesions that have a hard texture. There is supporting laboratory evidence from a study that used microbiological indicators for lesion activity that «soft» or «leathery» lesions on root surfaces are more heavily infected with bacteria than are «hard» root surfaces.

Given the paucity and generally low level of the scientific evidence, the ICDAS Coordinating Committee, recommends that the following clinical criteria be used for the detection and classification of root caries:

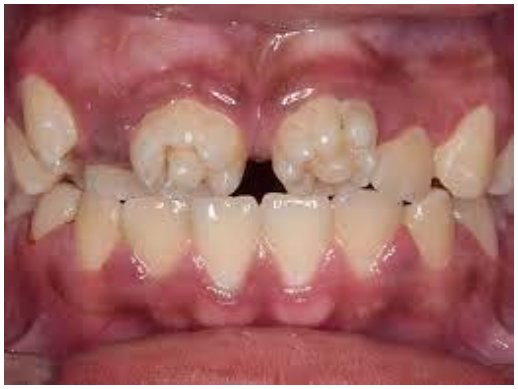
1. Colour (light/dark brown, black);
2. Texture (smooth, rough);
3 Appearance (shiny or glossy, matte or non-glossy);
4. Perception on gentle probing (soft, leathery, hard).
5. Cavitation (loss of anatomical contour).

Additionally, the outline of the lesion and its location on the root surface are uSeful in detecting root caries lesions. Root caries appears as a distinct, clearly demarcated circular or linear discolouration at the cementoenamel junction (CEJ) or wholly on the root surface. The colour of the lesion can also be used to make

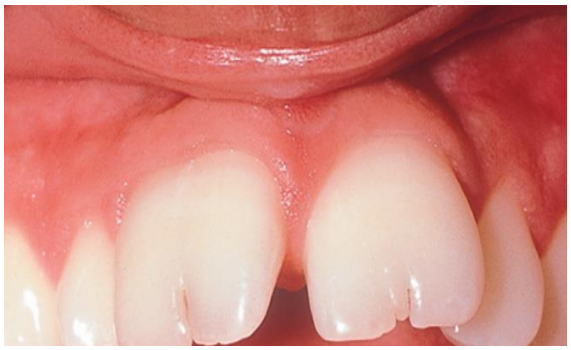
the distinction between arrested and active, with arrested lesions acquiring internal brown pigmentation and surface stain, while active lesions retain their white appearance.



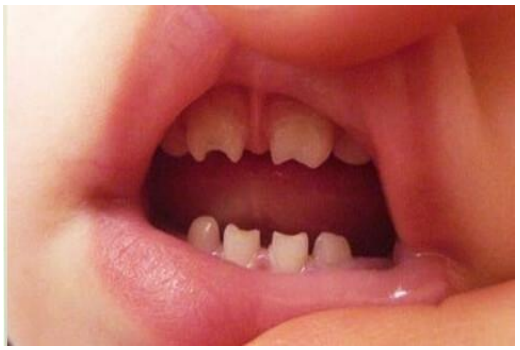
1. *Extra teeth.*



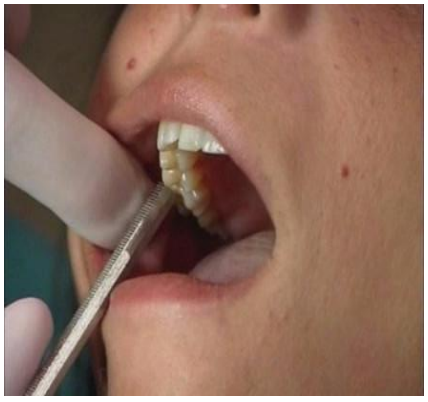
2. *Hatchinson's teeth*

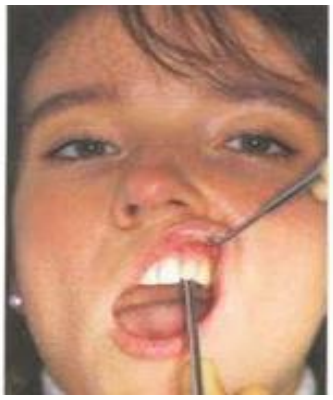


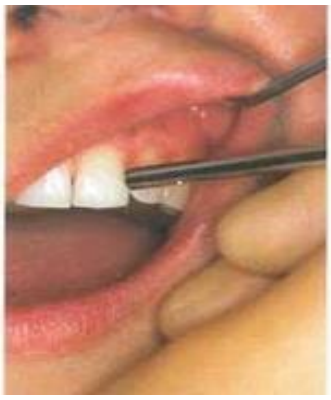
3. *Macrodentia*

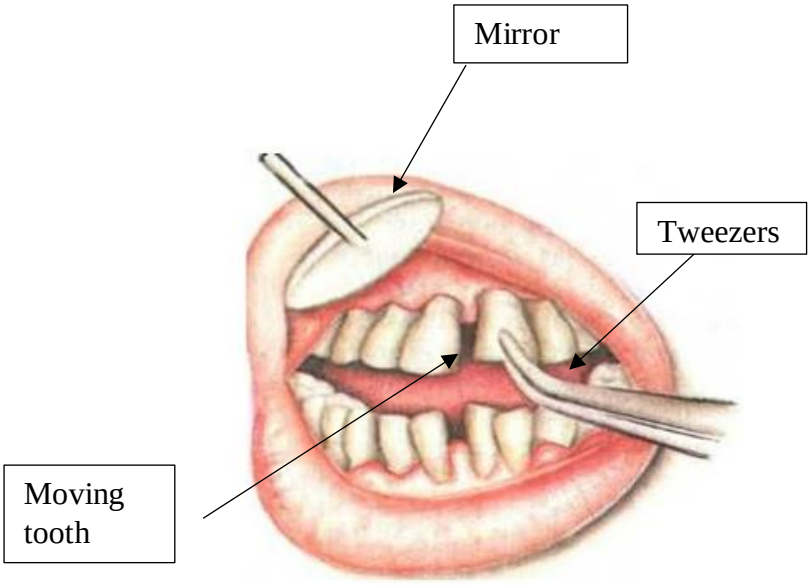


4. *Furnie's teeth*

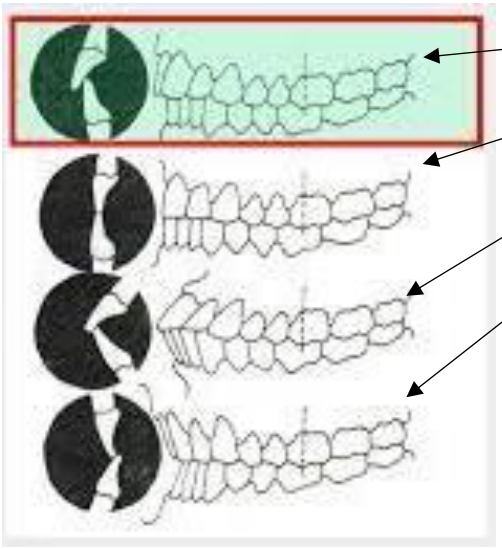








Physiological bite:

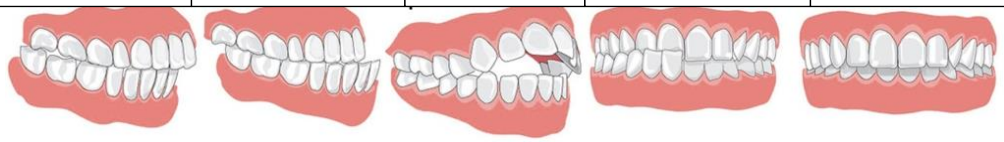


- orthognatic;
- straight;
- biprognatic;
- opistognatic.

Pathological bite:

- distal;
- medial;
- deep;
- open;
- crossed.

Distal	Medial	Open	Crossed	Deep

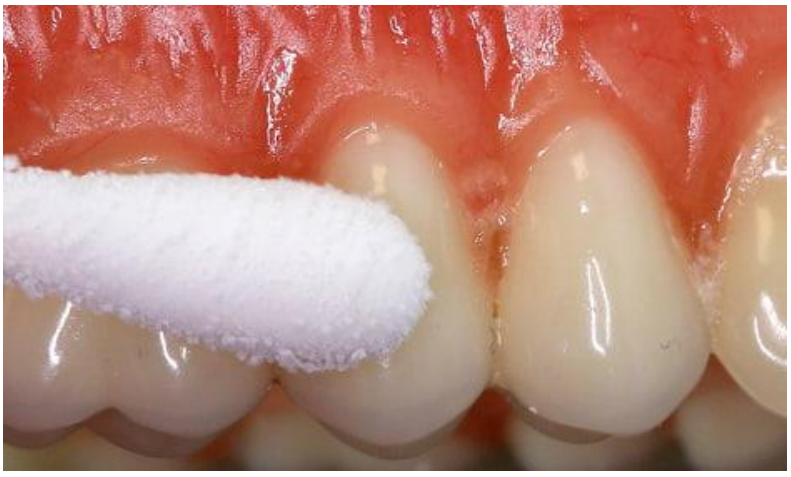


Lesson №5
TOPIC: Auxiliary methods of examination of the patient: thermodiagnostics, EOD, use of caries detectors: methodology, interpretation of results.

At the caries spots located in fissures there can be difficulties at their diagnostics and differentiation with other pathological conditions, especially with children. It is linked with accumulation of food remnants in fissures, pigments which can change colour of surface fissures. In such cases it is hard visually to reveal a whitish or some pigmented caries spot in the enamel. Using another diagnostic method to reveal the clinical caries signs at probing - softening, sensitiveness of surface of enamel. In children these signs may be very subjective, that is caused by insufficient mineralization of enamel in fissures. In such cases it is had to use the additional methods of caries investigation.

To reveal caries it is possible to use, except for mechanical, (probing) and also other irritants (thermal, chemical). On the fissures surface placed cold (5—10° C) or hot (60-70° C) water or gutta-percha warmed-up to soft consistency. In intact enamel surface the heating of a fissure within the limits of 10-60° C does not cause a characteristic for a caries pain.

Chemical irritants can be used, such as organic solutions (lactic, apple) acids; and inorganic (hydrochloric, carbolic) acids; for solutions of sucrose, glucose, alcohol and ether. Intensity and frequency of feeling pain almost does not rely on the use of an irritant, but is dependent mainly on its con-centration, age of patient (in elderly patients the reaction on the pain feeling low) and organism resistance.



The method of electro diagnostic of initial forms of caries was founded (Novik I. O., 1951; N.A. Kodola, 1956). By the special device the conductivity is measured between the surfaces of affected by a pathological process (caries) or suspicious on its presence and intact enamel areas.

A tooth is isolated, dry out, an active electrode is placed on the suspicious area of enamel surface and the passive is placed in the patient hand. If enamel

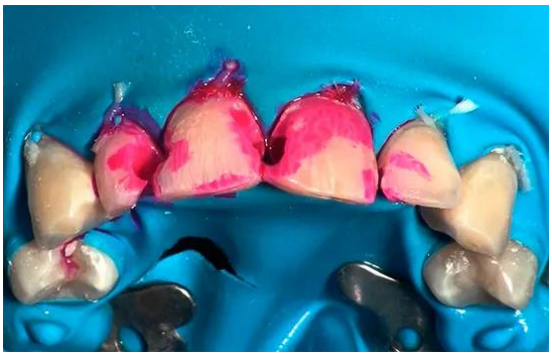
surface is intact, the conductivity is practically equal to the zero because of large electric resistance of intact enamel. At caries presence the condostics arises Approximately on the same principle based the method of electro diagnostics of caries offered by V.K. Leontyev (1983).

The very effective diagnostic method of acute incipience caries is usage of the reactions with *different dyes.* They are based on properties of dye to penetrate into the demineralized enamel and, thus, dyeing a caries spot, while in an intact enamel it cannot penetrate.

It is used a 2% water solution of methyl blue, a 0.1% water solution of methyl red, carmine, Congo red and others. A tooth is isolated from saliva by cotton rolls, dry out and on its surface with cotton pellet place a dye. In a few minutes the remnants of dye wash off by water and at presence of carious spot the surface of enamel gets the colour of dye (dark blue, rose and others). Another method is the use the solution of nitric acid silver, in a caries spot it is restored to metallic silver under influencing of present in her products of protein disintegration.

A spot here acquires the black colour. More rapid renewal of silver in this reaction can be attained by placed on the treated by nitric acid silver surface of enamel the repairer, for example a 4% solution of hydroquinone. In a carious spot the quantity of mineral components (apatites) of enamel diminishes because of processes of demineralization. It causes changes of conductivity of this hard tooth

tissues, namely to its increase, because electric resistance of such area of enamel diminishes.



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Lesson №6

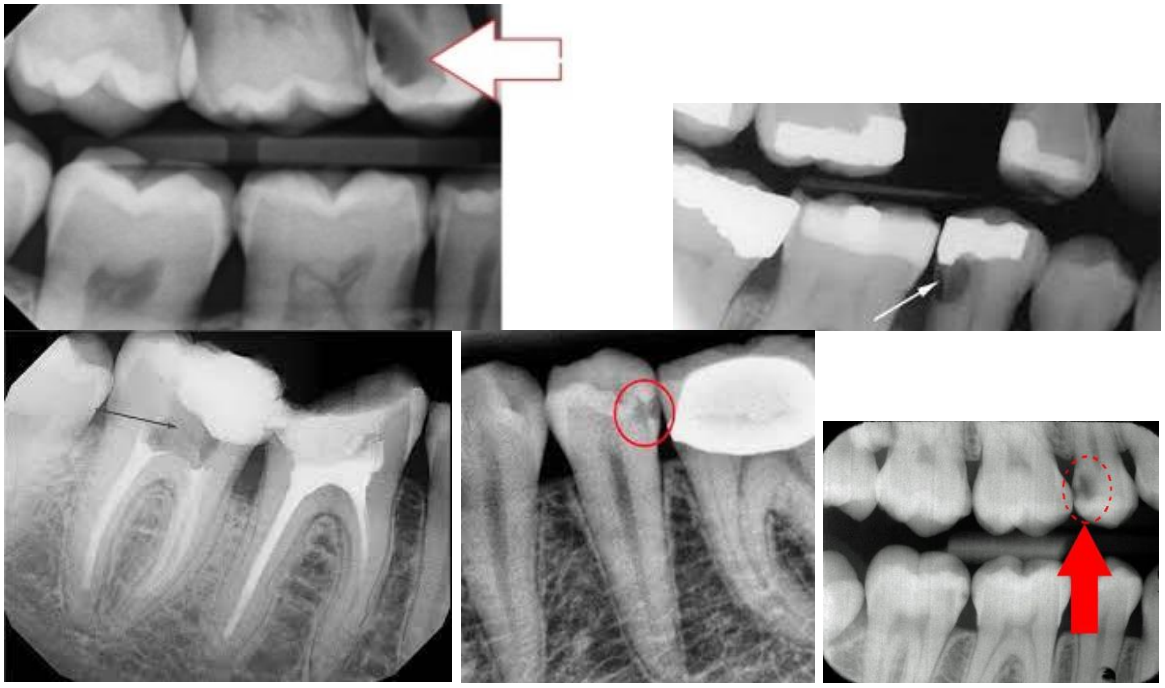
TOPIC: X-ray, luminescence and transillumination diagnostics. Indications, features of the procedure. Diagnostic tests: with anaesthesia and for dissection. Methods of determining trigger zones and examination of the exit points of the peripheral branches of the trigeminal nerve. Laboratory methods of examination. Interpretation of results.

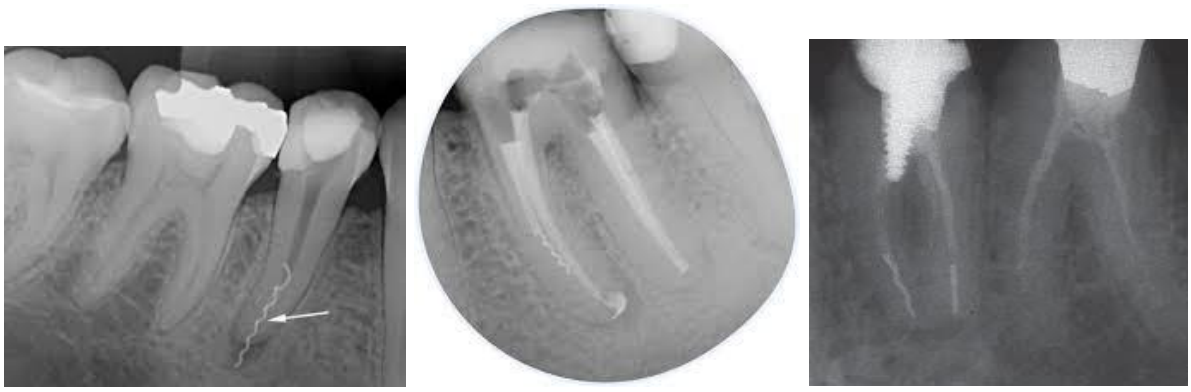
Diagnostic radiography is an integral part of the clinical process. It is predicated on a careful correlation of patient history and clinical findings. Radiographs should be ordered in those instances in which the clinician anticipates that the expected obtained will contribute materially to the proper diagnosis, treatment, and prevention of disease.

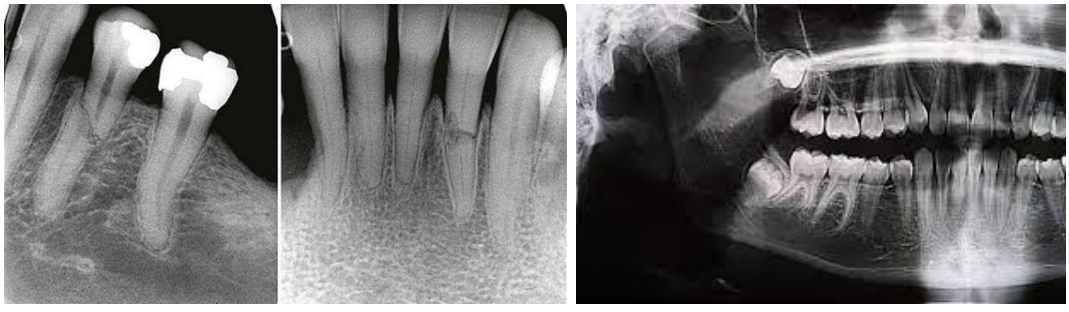
The standard X-ray image in caries diagnostics is an imaging of the bite wing or of the individual tooth. This is done to search specifically for caries near the contact points between the teeth (approximal caries) and dentin caries created in the depth of the fissures. For standard two-dimensional radiography, clinicians basically project X-radia-tion through an object and capture the image on a recording medium - either X-ray film or a digital sensor. On an X-ray image, caries appears as a dark spot.

X-rays are less absorbed in dental areas with mineral loss or defects and thus blacken the film. Radiographic examination of the hard tissues can often provide valuable information regarding caries and existing restorations, calcifications, internal and external resorptions, tooth and pulpal morphology, root fractures, the relationship of anatomic structures, and the architecture of the osse-ous tissues. In general, when endodontic pathosis appears in radiography, it appears as bone loss in the area of the periapex.

The infection in the pulpal space transgresses through the pulp canal space and into the adjacent alveolar bone. The pathosis may present merely as a widening or break in the lamina dura - the most consistent radiographic finding when a tooth is non vital - or it may present as a radiolucent area at the apex of the root or in the alveolar bone adjacent to the exit of a lateral or furcation canal. Bitewing Radiography: The process of creating radiograph images of the posterior teeth, with the specific objective of identifying carious lesions on the proximal surfaces that may be inaccessible to visual and tactile examination. Less mineralized tissues permit more x-rays to pass through (radiolucency) and therefore create greater levels of exposure to radiographic film or a digital transducer. The viewing of a digital radiographic image on a high-resolution monitor allows for rapid and easy interpretation for both the clinician and the patient. The image appears almost instantly, with no potential for image distortion from improper chemical processing, because there is none. The clinician can zoom in to different areas on the x-ray image, digitally enhance the image in order to better visualize certain anatomic structures, and in some cases the image can even be colourized, which is a useful tool for patient education.

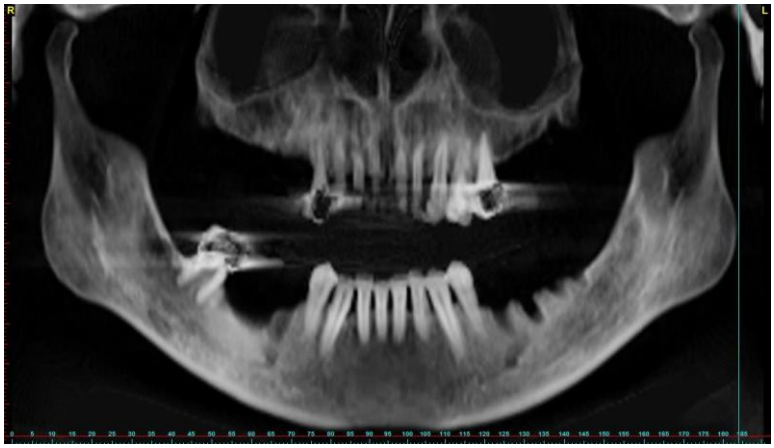




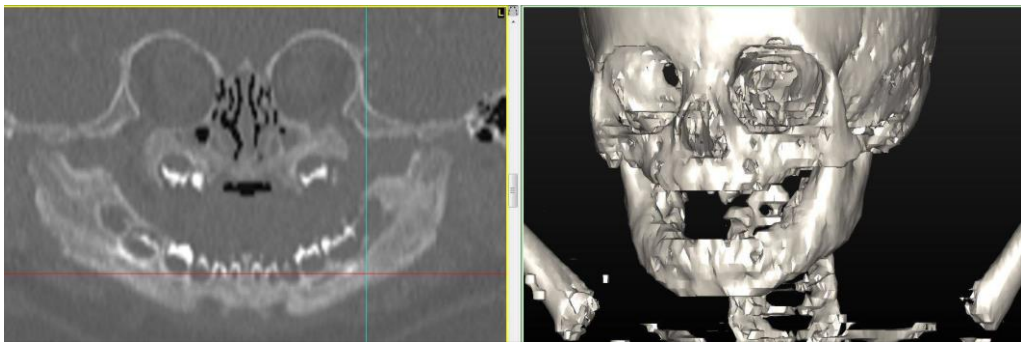


1. Tooth fracture *2. Retained teeth*

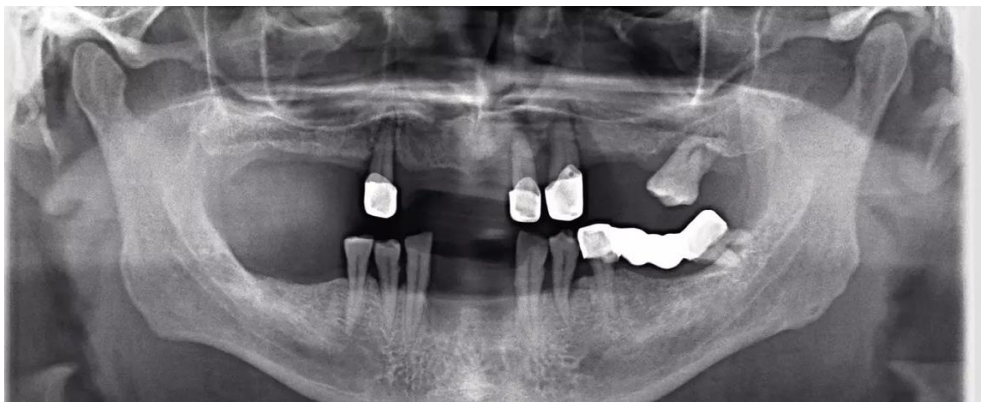
Atrophy — reduction in bone volume due to bone resorption.



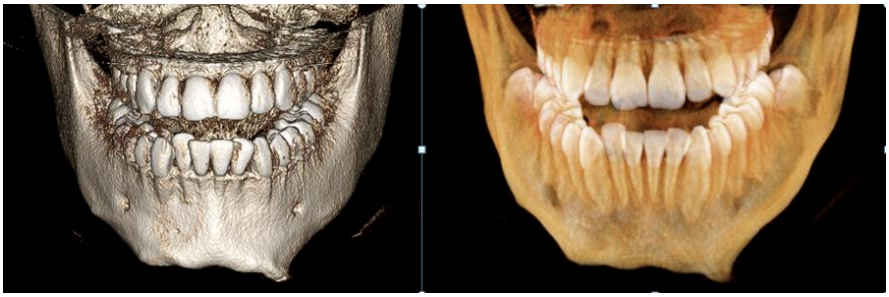
Hyperostosis — volumetric bone augmentation.



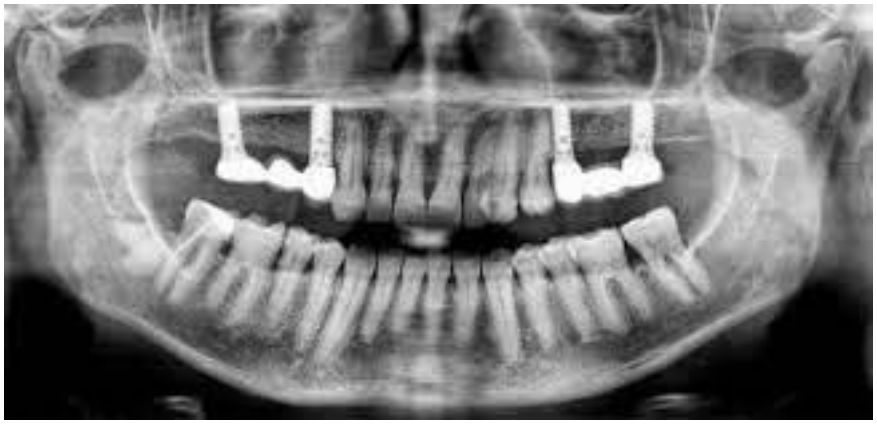
Destruction — destruction, change of bone structure with its subsequent replacement by another pathologically altered tissue. A distinction is made between focal (in the form of one or more foci of lightening of flat bones) and diffuse destruction. Complete resorption of the bone and disappearance of all its elements is called osteolysis.



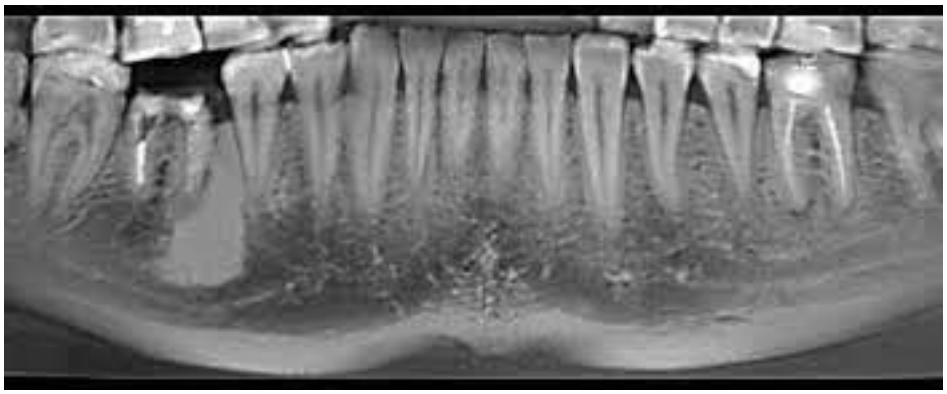
Deformation — violation of the anatomical shape of the bone.



Osteoporosis — is a dystrophic process that results in a decrease in the thickness of bone plates. It is characterised by increased radiological transparency of bone tissue, can be focal and diffuse.



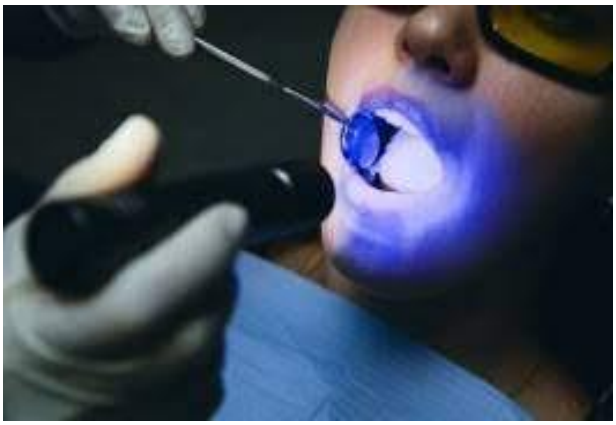
Osteosclerosis — bone compaction: the spongy substance becomes compact; it can be focal and diffuse. The combination of these radiological symptoms creates a diverse radiological picture of the disease.

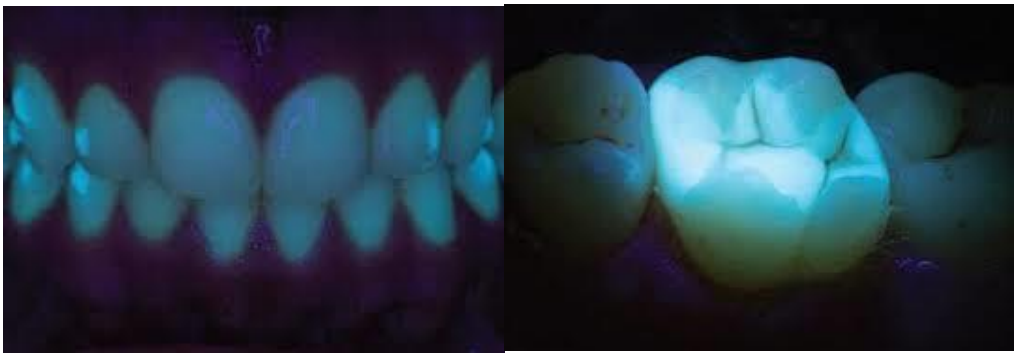


It is possible to apply the method of luminescent diagnostics, which is based on the phenomenon of fluorescence of intact hard tooth tissues under action on them of ultraviolet rays. The special ultraviolet lamps are used for this purpose. In the black-out apartment the surface of tooth is lighted by a lamp in the distance by 20-30 sm. By fluorescence intact dental enamel shines light-green or bluish light, while areas affected by caries areas do not radiate light. The histological features at an initial caries are characterized development of different degree of enamel demineralization. At the enamel section the body of caries lesion has the triangle appearance with the basis turned of to enamel sur-face. At the study in the polarized light depending on the structure changes in the enamel lesion distinguish a few areas.

The most demineralized is sub superficial lesion layer, which is covered by the mineralized superficial enamel layer. This interesting phenomenon is explained by the remineralization processes of carious lesion by the mineral components of saliva, if an oral fluid is unable to provide remineralization of the demineralized enamel area the rapid development of caries lesion occurs. The surtace texture of an incipient lesion is unaltered and is undetectable by tactile examination with an explorer.

A more advanced lesion develops a rough surface that is softer than the unaffected, normal enamel. Softened chalky enamel that can be chipped away with an explorer is a sign of active caries. In the interior layers of white caries spot there are the changes both enamel rods surfaces and prisms themselves. In separate areas the crystals of apatites are destroyed, their orientation in rods changes, in formed micro spaces there are atypical crystals.

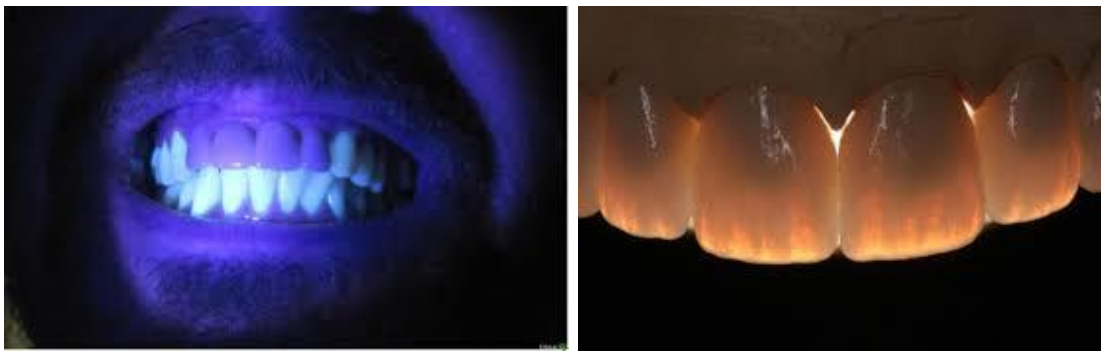




Fragmentation of crystals, formation of homogeneous fine-grained substance and disappearances of visible border, between prisms is marked in future. Non-cavitated enamel lesions retain most of the original crystalline framework of the enamel rods and the etched crystallites serve as nucleating agents for remin-cralization.

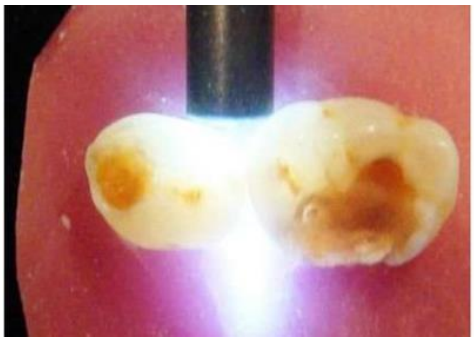
Calcium and phosphate ions from saliva can then penetrate the enamel surface and precipitate on the highly reactive crystalline surfaces in the enamel lesion. Remineralized (arrested) lesions can be observed clinically as intact, but dis-colored, usually brown or black spots. Depending on the degree of the demineralization G. Gustafson (1975) distinguishes five zones. Most deeply in the enamel there is placed hyper mineralization zone with disappearance enamel structural components.

In the second zone there is reduction of its hardness because of partial dissolution of enamel minerals, in the third - increase of mineralization. In a sub superficial fourth demineralization zone the minerals are almost fully washed. In a superficial fifth zone there can be comPlete disintegration, however it during long time remains enough mineralized and undamaged, even when caries spread on all thickness of enamel.



One diagnostic aid in the anterior tooth area is the transillumination of contact points between incisors. A polymerisation lamp can be used as a light source. In contrast to healthy enamel, caries will appear darker. • *Transillumination/dye staining.* The use of a fibre-optic light is an excellent

method of examining teeth for coronal cracks and vertical root fractures. The tooth or root should be examined in the presence of minimal background lighting. The fibre optic light is then placed on the varied surfaces of the coronal tooth structure or on the root after flap reflection. Fracture lines can be visually detected when light fails to traverse the fracture line. The fractured segment near the light appears brighter than the segment away from the light. Application of dyes to the tooth can also demonstrate fractures as the dye penetrates the fracture line. An ancillary technique is the application of dye to the internal surfaces of a cavity preparation or access opening; the clinician leaves the dye in place for a week before reexamining the tooth.



LABORATORY METHODS. In physical evaluation, clinical laboratory procedures may provide the final clue essential to confirm a diagnosis. They may also lead to the early detection of disorders with vague signs and symptoms contribute to the discovery of significantly, unexpected conditions, or provide a baseline against which response to or the safety of a therapeutic intervention may be measured. Consequently, in some situations, clinical laboratory information may be essential prior to the initiation of therapy. In other instances, it may be an important component of a diagnostic or therapeutic follow — up evaluation. Prior to ordering laboratory procedures, the clinician should elicit a careful medical history, perform a thorough physical examination, evaluate radiographic studies, and then request the tests from the laboratory that are most likely to either confirm or exclude the provisional diagnosis.

MUTANT STREPTOCOCCI IN SALIVA. In toddlers (1-2 years) the presence of mutant streptococci as the sole predictor for caries during the following 2—3 years had low accuracy (either low sensitivity combined with high specificity, or vice versa).

SALIVARY LACTOBACILLUS COUNT. In a large number of studies on both toddlers and preschool children, the presence of lactobacilli in saliva has been tested as the sole predictor or in models. The accuracy in predicting the development of caries lesions is low.

Lesson №7
TOPIC: Medical documentation of a therapeutic appointment
Medical record of a dental patient (f.043/ O) is a medical, scientific and legal document.

Medical record (medical history) of a dental patient

The first section of the card - the passport part - contains basic data about the patient: surname, name and patronymic; age, sex, place of residence, occupation, address. This part of the card is filled in at the reception desk during the initial visit to the doctor.

Later, the doctor enters data from the interview (complaints, medical and life history), examination, clinical and additional methods of objective examination. A special section contains data on the general examination of the patient and his or her oral cavity.

Special schemes have been developed for the convenience of recording the results of the oral examination. Today, there are several recording schemes. The most commonly used is the designation of the dentition in the form of a dental

8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

formula:

Permanent teeth are marked with Arabic numerals, and temporary (milk) teeth are marked with Roman numerals.

V	IV	III	II	I	I	II	III	IV	V
V	IV	III	II	I	I	II	III	IV	V

The numbers above the horizontal line represent the upper teeth and below it the lower teeth. The vertical line divides the formula in the middle, which corresponds to the midline of the dentition. The numbers on the left indicate the teeth of the right half of the jaw, and the numbers on the right indicate the teeth of the left half. In other words, the doctor looks at the patient through this formula, superimposing it on the patient's dentition.

Conventionally, each tooth of the permanent and temporary bites has its own numerical designation. Permanent teeth are designated by Arabic numerals from 1 to 8:

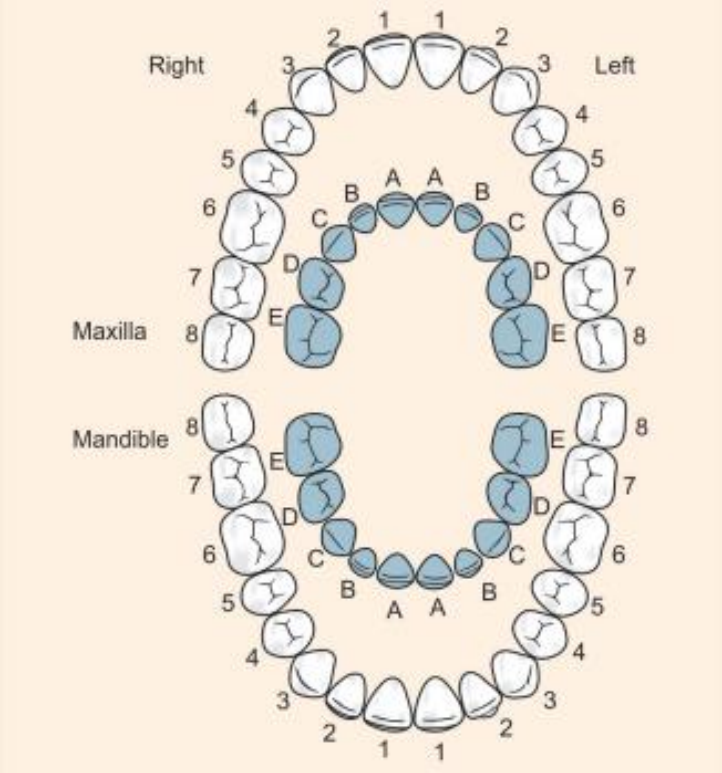
1 - central incisor,
2 - lateral (lateral) incisor,
3 - canine,
4 - first premolar (first small angular tooth),
5 - second premolar (second small angular tooth),
6 - first molar (first large angular tooth),
7 - second molar,
8 - third molar.

Temporary (milk) teeth are designated by Roman numerals from I to V:

I - central incisor,
II - lateral (lateral) incisor,
III - canine,
IV - first molar,
V - second molar.

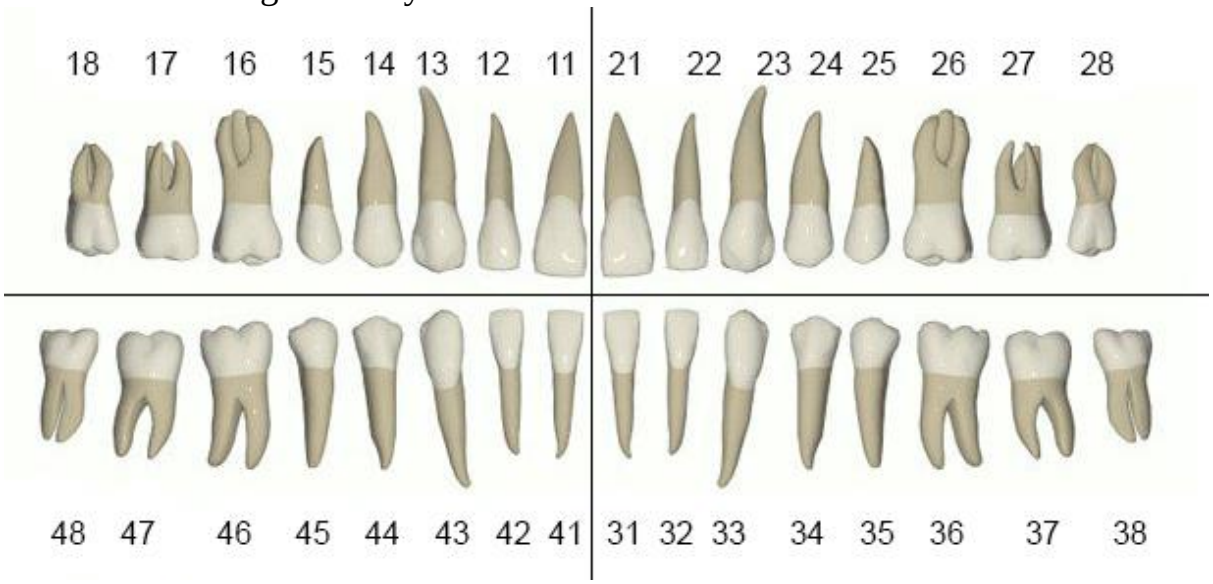
When writing down the formula for the teeth of an alternating bite, Arabic and Roman numerals are added to it in accordance with the location of the permanent and temporary teeth. To indicate that an individual tooth belongs to one side of the jaw or the other, a combination of vertical and horizontal angled lines is used, together with the number corresponding to the tooth. The digit above the horizontal line indicates the tooth of the upper jaw, and the digit below it indicates the tooth of the lower jaw. A vertical line on the left indicates a tooth in the left half of the jaw, and a vertical line on the right indicates a tooth in the right half of the jaw. For example, |_1 means the upper left central incisor, 2_| means the upper right lateral (lateral) incisor, |`4 means the lower left first premolar, 6`| means the lower right first molar, |_IV means the upper left first temporary (milk) molar, etc.

To unify the recording of the dental formula, the WHO (World Health Organization) and FDI (International Federation of Dentists) proposed a two-digit system of designation of teeth depending on their location on one side or the other of the upper or lower jaw.

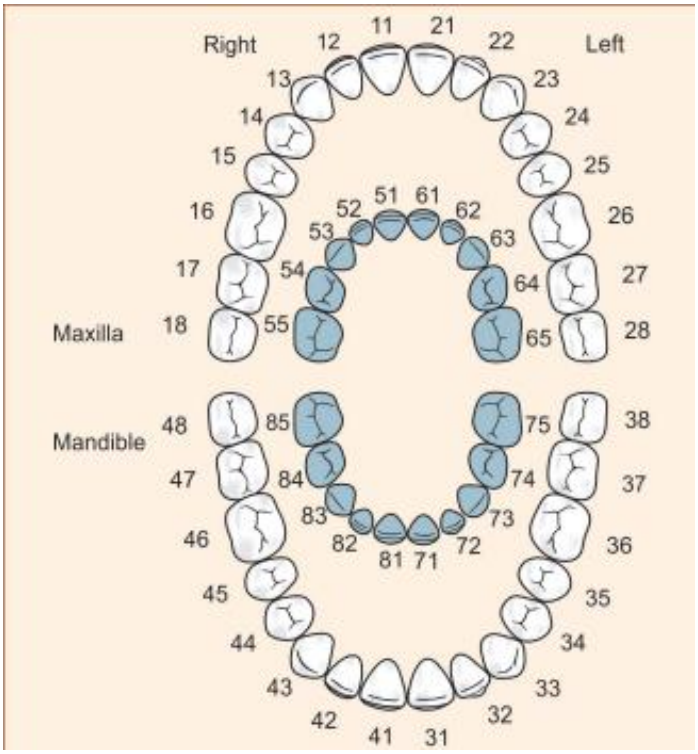


In this system, each tooth is labelled with two Arabic numerals, the first of which indicates the quadrant of the corresponding half of the jaw and the second the serial number of the tooth in that quadrant. Both jaws of the patient are divided into four quadrants, which have their own numbers. The designation begins with the upper jaw on the right, which has number 1. Then they move clockwise and mark the corresponding halves of the jaws: the upper left jaw with the number 2, the lower left jaw with the number 3 and the lower right jaw with the number 4.

The serial number of each tooth is indicated by the generally accepted system (see above) from the central incisor (1) to the third molar (8). The dental formula according to this system looks like this:



For temporary teeth in this system, instead of Roman numerals, Arabic numerals are also used, but unlike the permanent bite, the jaw quadrants have numbers from the 5th (right half of the upper jaw) to the 8th (right half of the lower jaw). According to this system, the dental formula for a temporary bite is as follows:



Accordingly, the designation of teeth according to this system is as follows: 21 - upper left central incisor, 46 - lower right first molar, 62 - upper left lateral (lateral) temporary incisor, etc. The advantage of the new system of dental formula expression over the conventional one is the possibility of easy transfer of notations to a computer and further processing of information.

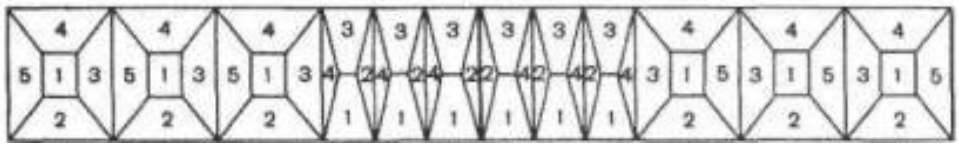
A specific variant of the digital tooth marking system is the system developed by the American Association of Dentists. According to this system, all permanent bite teeth are designated by numbers from 1 to 32: the teeth of the right half of the upper jaw, starting with the third molar, have numbers from 1 to 8; on the left half, the count continues - the first incisor has the number 9 and then up to the third molar - the number 16.

The teeth of the lower jaw begin with the third left molar, number 17, and continue to number 32, the third right molar. The sequence of counting is similar for temporary teeth, but they are designated by letters of the Latin alphabet. Thus, the formula of teeth according to this system looks like this:

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17

A	B	C	D	E	F	G	H	I	J
T	S	R	Q	P	O	N	M	L	K

Dental formula diagrams have also been developed, which show all the surfaces of the tooth crowns as if in an expanded form. They can be used to accurately mark the location of a carious cavity, filling or other pathological lesion on each individual tooth.



In this formula, the front teeth have 4 surfaces, and the premolars and molars have 5 surfaces. For the correct interpretation of the data in this formula, the following conventions have been adopted for tooth surfaces. For anterior teeth (incisors and canines): 1 - vestibular (gingival, labial) surface; 2 - medial (median); 3 - lingual (palatal) and 4 - distal. On premolars and molars: 1 - masticatory; 2 - vestibular (gingival, buccal); 3 - medial; 4 - lingual (palatal); 5 - distal surface.

Alphabetic designations are used to schematically record diseases and dental conditions in the dental formula:

C - caries,

P - pulpitis,

Pt - periodontitis,

P - filling,

K - artificial crown,

H - artificial tooth of a bridge prosthesis,

X - tooth root,

O - missing tooth,

T - trauma (tooth crown),

H - hypoplasia,

F - fluorosis.

The degree of tooth mobility is indicated by Roman numerals - I, II or III.

Sometimes carious cavities are designated by Latin letters and classified according to their location on different tooth surfaces. For this purpose, the following letter designations of different tooth surfaces are accepted:

O - occlusal (chewing);

I - cutting (incisal) edge;

M - medial contact;

D - distal contact;

F - vestibular (gingival, frontal, labial);

C - buccal (buccal);

L - lingual (palatal, lingual).

Combined carious cavities located simultaneously on several tooth surfaces are indicated by a combination of several letters, for example:

MO - medial occlusal cavity, i.e. a cavity located on the chewing and medial contact surfaces of the tooth crown;

M O D - medial occlusal-distal cavity, i.e. located on the chewing, medial and distal contact surfaces of the tooth crown;

MI - medial invasive cavity, i.e. located on the medial surface and cutting edge of the anterior tooth crowns;

FO - vestibular occlusal cavity, i.e. located on the vestibular (gingival) and chewing surfaces of the crowns of the posterior teeth (premolars and molars);

LO - lingual-occlusal cavity, i.e. located on the lingual and chewing surfaces of the crowns of the teeth.

Cavities located on the other upper surfaces of the tooth crowns are designated in the same way.

After indicating the condition of the teeth on the dental formula, the dentist notes the nature of the bite (physiological or pathological), the condition of the periodontal tissues and oral mucosa. The doctor describes in detail the local condition of the lesion - carious cavity, non-carious defect, traumatic lesion, etc. Then notes the data of the clinical examination (examination, percussion, palpation, etc.). If additional examinations were performed (e.g., X-ray, electro-odontodiagnostic, laboratory), the data obtained are also recorded in the card.

After the interview, examination, and additional tests, the doctor determines the diagnosis of the disease and enters it in a special column. In some complicated cases, it is permissible, but it must be noted in the card, to make a preliminary diagnosis. In this case, the final diagnosis, indicating the exact date of its establishment, is entered after a full range of diagnostic examinations or even preliminary, trial treatment.

A special section of the medical record contains a treatment plan developed on the basis of the diagnosis and the patient's individual characteristics. The availability of such a plan allows for a complete comprehensive treatment, taking into account the smallest details. In addition, it allows treatment to be carried out even in unforeseen cases of illness or in the absence of the doctor in question.

The stages of treatment (each visit to the doctor) are recorded in a special section called the Treatment Diary. It indicates the date of the visit, the patient's condition and a short list of the medical treatments and surgical interventions performed. The name of the doctor who performed the treatment must be indicated. This section of the card is filled in by the doctor immediately after the visit.

In the section "Epicrisis", after the treatment, the result of the treatment and recommendations to the patient are briefly described. If no treatment is planned in the near future, the medical record is kept in the registry or in the archive of the medical institution.

Recently, it has become increasingly common in medical institutions to enter all information about patients and the course of their treatment into a computer. This allows you to store all the necessary data for many years, and if necessary, they can be easily retrieved from the computer's memory (for prompt use by the doctor, etc.).

Documentation of the patient-dentist relationship has always been a hot topic among dentists.

All documents can be divided into mandatory and optional.

Mandatory documents are:

- Informed voluntary consent in the form of N003-6/o (the most important document);
- medical record in the form N043/o and N043-1/o;
- other forms of primary accounting documentation (forms N037/o, N037-1/o, N037-2/o, N039-3/o, N039-4/o);
- maintenance of these forms is provided for by sub-clause 22, clause 13 of the Licensing regulations on medical practice and by the orders of the Ministry of Healthcare of Ukraine N110 dated 14.02.12 and N435 dated 29.05.13;
- internal regulations (subclause 1 of clause 12 of the Licensing regulations on medical practice);
- statement (act) of patient's refusal from treatment (Article 43 of the Law of Ukraine "Fundamentals of the Legislation of Ukraine on Healthcare").

Optional:

- contract for the provision of dental services/public contract;
- patient's health questionnaire (if the patient needs to be administered a medicine, a questionnaire should be filled out (Order of the Ministry of Health No. 916 of 30.12.15));
- special informed consent of the patient;
- patient's treatment plan;
- consent to photo, audio or video recording;
- recommendations of the dentist;
- guarantees for dental services;
- an act of dental services rendered;
- act of refund to the patient;

Lesson № 8

TOPIC: Oral hygiene and its importance in the comprehensive prevention of diseases of the oral cavity and the body as a whole. Definition of GI according to Fedorov-Volodkina and Green-Vermillion (standard and simplified methods), oral hygiene efficiency index.

Preventive *oral hygiene* is a system of methods used to care for the oral cavity, as well as means aimed at preventing the development of pathological processes in the oral cavity.

Oral hygiene has existed since ancient times. There are descriptions of various tools used for this purpose: toothpicks, wooden sticks, tongue scrapers. The works of Hippocrates mention rinsing to eliminate bad breath. Resins, ground cocoa beans, white clay, talcum powder, pumice, coral and corundum powder, etc. were used to clean teeth.

The level of oral hygiene is inextricably linked to the intensity of dental pathology. In the oral cavity of a modern person, favourable conditions are created for the active development of microorganisms, whose waste products cause pathological processes in periodontal tissues and tooth enamel.

The most common dental diseases are caries, inflammatory and dystrophic periodontal disorders, diseases of the oral mucosa and salivary glands, and anomalies of the dentition. And while the latter are not directly related to poor hygiene, other dental pathologies, according to the results of many studies, are necessarily associated with a negligent attitude to hygiene.

Poor oral hygiene affects various body systems through bacterial infections and inflammatory processes that spread through the circulatory system. Here are some of the organ systems that can be affected by poor oral hygiene:

1. Cardiovascular system: Bacterial infections and inflammatory processes that start in the oral cavity enter the circulatory system and cause inflammatory reactions in the arteries. This can lead to a risk of heart disease, such as endocarditis (inflammation of the inner lining of the heart).
2. Respiratory system: Inflammatory processes in the oral cavity spread to the upper respiratory tract and lungs, leading to inflammation and other diseases of the respiratory system.

Inflammatory processes that occur due to poor oral hygiene can affect different parts of the respiratory system.

Here are some examples:

Gingivitis and periodontitis: Inflammatory gum disease (gingivitis) and periodontitis (an inflammatory process that affects the tissues that support the teeth) can lead to the spread of inflammation to the upper respiratory tract. The inflammatory reactions can spread through the mucous membranes and aerosols produced during chewing and speaking and cause inflammation in the nasal cavities and larynx.

Pharyngitis: Inflammation in the upper mouth can lead to pharyngitis, an inflammation of the back of the throat. This can cause discomfort and sore throat, cough and other respiratory symptoms.

Bronchitis and pneumonia: Inflammatory reactions can spread further down the airways and cause inflammation of the bronchi (bronchitis) or even the lungs (pneumonia). This can cause severe coughing, chest pain, shortness of breath and other unpleasant symptoms.

General impact on the immune system: Inflammatory processes that develop in the mouth due to poor hygiene can also affect the body's overall immunity. A deteriorating immune system can make you more vulnerable to respiratory diseases.

3. Periodontium (The support system for the teeth/tissue around the tooth): Inflammation of the gums and supporting tissues can lead to damage to the bones that support the teeth. This can lead to tooth loss and other problems with the structure of the mouth.

4. Digestive system: The initial stage of food digestion takes place in the mouth under the influence of saliva and enzymes. Inflammatory processes can affect this process and cause problems with the gastrointestinal tract.

5. General condition of the body: The general inflammatory state of the body caused by inflammatory processes in the oral cavity can affect the overall health, immunity and overall resistance to disease.

Hygiene items include toothbrushes, dental floss, tongue cleaners, oral irrigators, dental brushes, interdental stimulators, and items for the care of removable orthopaedic and orthodontic structures.

Toothbrushes

A toothbrush is a device for brushing teeth and gum massage. It is used with toothpaste. Modern toothbrushes come in a variety of shapes. Their working surface consists of synthetic or natural fibres of different sizes and stiffness.

Toothbrushes are divided into:

1.By the type of bristles:

- with natural bristles
- with synthetic smooth bristles
- with synthetic microtextured bristles

2. By the stiffness of the bristles:

- very soft
- soft
- medium hardness
- hard
- very hard

3.By the number of rows of bristles:

- four-row
- three-row
- two-row (sulcular - for cleaning the gingival sulcus)
- single-bunch (used when it is impossible to clean with a large head brush)

4.By the size of the working part:

- children's
- teenagers
- adults
- special purpose
- single beam

5.By the method of actuation:

- manual
- automatic (electric)

6.By purpose:

- standard
- orthodontic (with a middle recess for the orthodontic arch)
- special purpose (after surgical interventions)

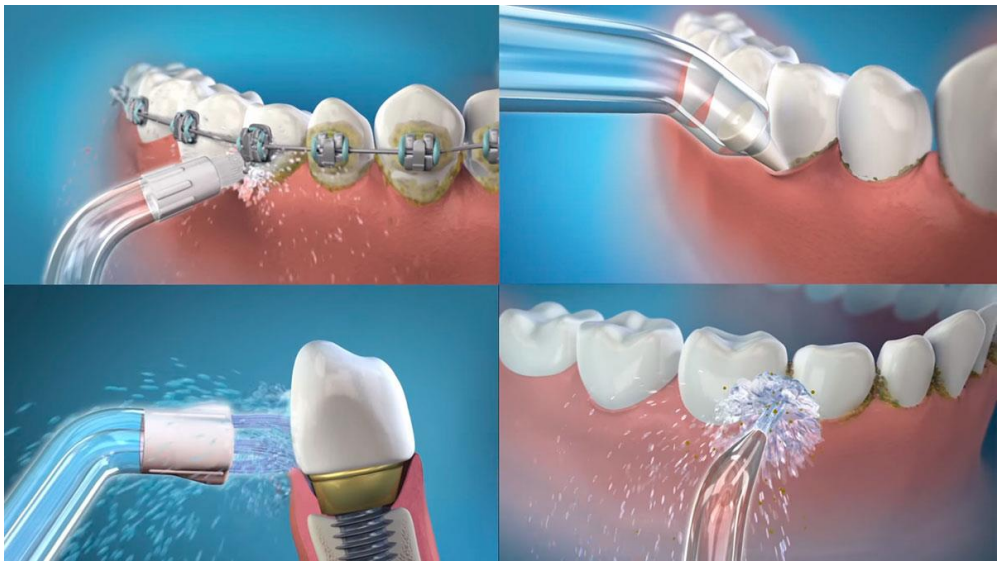
Tools for cleaning the back of the tongue

The most popular hygiene items are spoons and tongue scrapers. They are made of plastic with a rounded tip that does not cause a gag reflex. Scrapers have soft bristles at the end for more effective cleaning. A regular toothbrush, an electric toothbrush, or an irrigator with a special nozzle are also suitable. The main thing to remember is that tongue cleaning is an addition to oral hygiene, but not its main stage.

Oral irrigators (electric hydromassagers)

They have the form of nozzles that deliver a jet of liquid under pressure, which is pre-poured into a reservoir. The force of the liquid flow is adjustable.

The jet can be centred ("jet" mode) or sprayed ("shower" mode), continuous or pulsating. When operating in the "jet" mode, food debris and partially soft plaque are washed away from the surfaces of the teeth, interdental spaces, gums, tongue, and SOPD under high pressure; when operating in the "shower" mode, the gums, SOPD, and tongue are massaged, resulting in normalised peripheral blood circulation. Effective for periodontal and OSA diseases.



Dental floss

Designed for cleaning interdental spaces. The floss is effective in narrow, difficult-to-clean interdental spaces, crowded teeth, periodontal disease, fixed orthopedic and orthodontic structures in the oral cavity, implants, and apical fillings.

Interdental brushes

Designed for cleaning wide interdental spaces, spaces under fixed orthodontic arches, areas under the flushing parts of bridges and spaces between implants and dentures, as well as exposed bifurcations and trifurcations of teeth.

Methods of brushing your teeth

The standard method of brushing your teeth. The dentition is conventionally divided into several segments (3 segments on each jaw): two segments for molars and premolars, one segment for canines and incisors. Teeth and gums cleaning starts with the upper right large angular teeth and continues towards the upper left molars. The teeth are cleaned sequentially on the upper and then on the lower jaw.

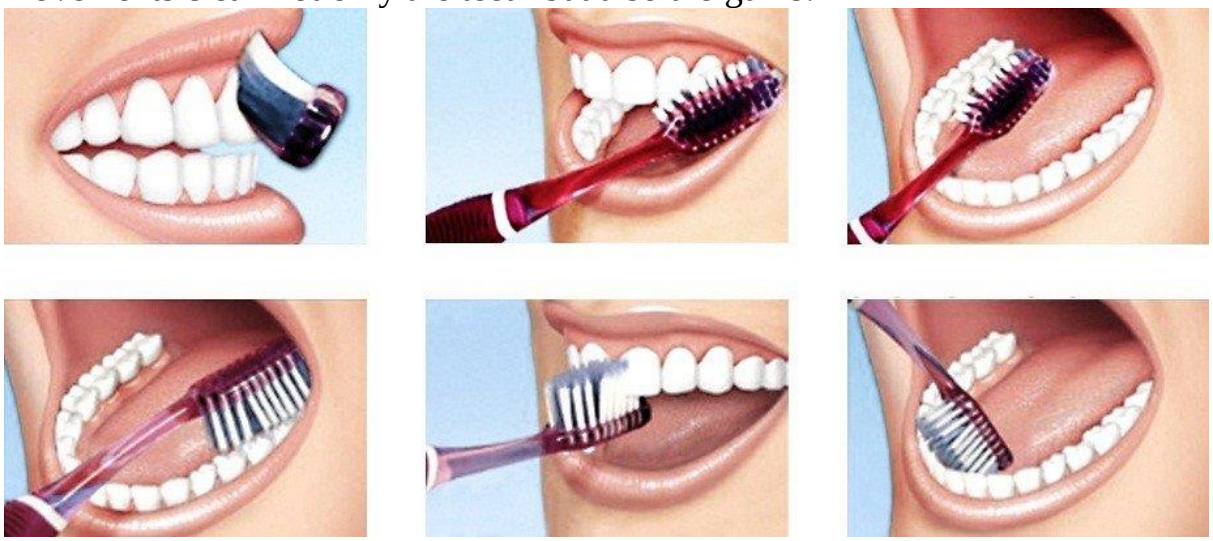
When brushing the buccal surfaces of the large and small angular teeth of the upper and lower jaw, the brush bristles should be directed at an acute angle to the tooth surface.

"Using a downward sweeping motion for the upper jaw and an upward sweeping motion for the lower jaw, plaque is removed from the gums at the same time. Then make several horizontal (back and forth) movements, and finish brushing in a circular motion, with pressure applied to the brush when it passes from the gums and rubs against the tooth. Then a counter semicircle is performed without pressure to return the brush to its original position. After that, the toothbrush is moved forward one segment and the entire combination of movements is repeated. The chewing surfaces of the large and small angular teeth of the upper and lower jaws are cleaned with brush bristles directed perpendicular to the occlusal plane (penetrating deep into the fissures and interdental spaces) with horizontal movements of the brush head (without changing its position) back and forth.

The palatal surfaces of the large and small angular teeth of the upper jaw and the lingual teeth of the lower jaw are cleaned in the same way as the buccal surfaces. When brushing the buccal surfaces of the upper and lower anterior teeth, use the same movements as when brushing the buccal surfaces of molars and premolars.

When brushing the palatal surfaces of the upper and lingual surfaces of the lower anterior teeth, hold the brush handle parallel to the occlusal plane, with two or three bundles of bristles covering the teeth and gums. Horizontal movements are performed. Then, the position of the brush is changed so that the brush head

is directed perpendicular to the occlusal plane of the teeth, and the bristles are at an acute angle to them and reach part of the gum. Up and down brushing movements clean not only the teeth but also the gums.



The method of brush rotation. The brush bristles are placed on the gum mucosa. The brush is moved in a rotating motion to the crown of the tooth. This operation is performed 10-12 times in each segment of the dentition.

Leonard's method. The toothbrush is placed perpendicular to the surface of the teeth, and vertical movements are made only in the direction from the gums to the tooth crown: on the upper jaw from top to bottom, on the lower jaw from bottom to top. Such movements avoid damage or refraction of the gums. The vestibular surfaces of the teeth are cleaned with the teeth closed, the chewing surfaces are cleaned by moving the brush back and forth.

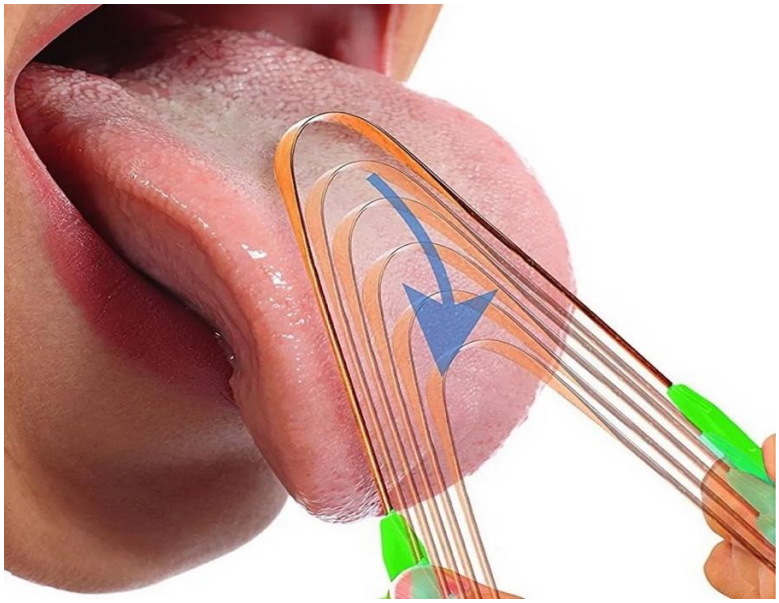
Reite method. The brush bristles are placed parallel to the tooth axis, with their free ends touching the gum margin. When brushing, evacuation movements are performed from the gums to the tooth crown. At the end of the movement, the bristles are set at right angles to the tooth axis. The process is repeated. Lingual surfaces are cleaned in the same way, chewing surfaces - by moving the brush back and forth.

Bass method. The bristles of the brush should be at an angle of 45° to the tooth axis, with the ends of the bristles partially falling into the gingival sulcus and interdental spaces. When brushing, make vibrating movements back and forth without moving the tips of the bristles. A number of mistakes are possible here: incorrect positioning of the toothbrush head to the surface of the teeth and gums, incorrect brushing motion, i.e., the transition to horizontal movements. Internal surfaces are cleaned in the same way. Chewing surfaces, as with other methods, are cleaned by moving the toothbrush back and forth.

Brushing the tongue. The plaque on the tongue is a combination of a thin epithelial film that has not had time to renew, food debris and microorganisms. Therefore, dysbiosis may result in an unpleasant taste or breath.

It should be cleaned twice a day after brushing your teeth. Proper tongue cleaning begins at the root of the tongue, where the largest number of bacteria

accumulates. Use gentle pressure to move from the root of the tongue to its tip, and then work the side surface, right and left. Finally, rinse your mouth with water or mouthwash.



One of the indicators of oral hygiene is the hygiene index. It is an assessment of plaque and calculus, as well as the general condition of teeth and gums. Standard clinical and simplified methods of determining the hygiene index allow you to get an objective overview of the condition of your teeth and suggest the need for additional procedures/measures/manipulations.

Standard clinical method for determining the hygiene index:

This methodology is based on a detailed examination of the condition of the teeth and gums using clinical examination indicators. It includes an assessment of plaque and calculus on the teeth, the degree of gum disease, and the general condition of the teeth. The technique can use special instruments to examine the teeth and measure hygiene indicators. The hygiene index is calculated based on the results of this examination.

Simplified method of determining the hygiene index:

This method involves using fewer observational indicators to assess hygiene status. It may include assessing the presence of plaque on specific teeth or groups of teeth, without the detailed examination required by the standard clinical method. This technique can be used to quickly assess the hygiene status in some cases, for example, during a routine examination.

Hygiene indices

To objectively assess the hygienic state of the oral cavity, indices are used, which can be divided into 4 groups:

1 - indices that assess the area of plaque;

2 - indices assessing the thickness of plaque;

3 - indices that estimate the mass of plaque;

4 - indices that assess the physical, chemical, microbiological parameters of plaque.

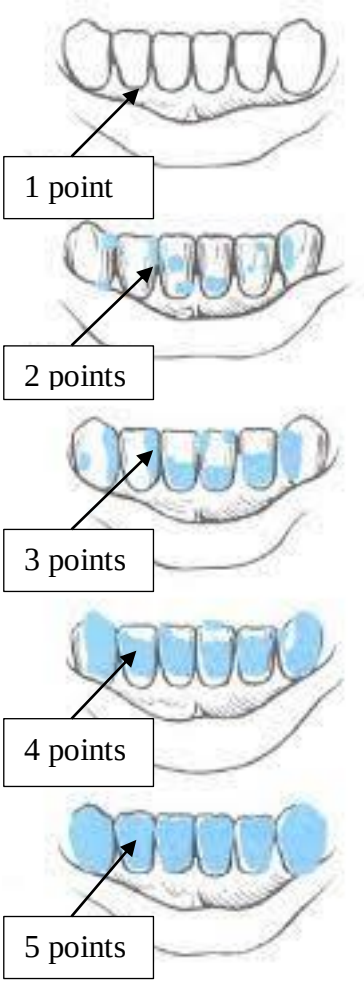
In the clinic, the indices belonging to groups 1 and 2 are more often used, due to their sufficient information content and ease of implementation.

Hygiene performance indices, such as the Fedorov-Volodkin and Green Vermillion indices, and the Silence Lowe index, make it possible to assess hygiene results and correct the home (individual) care strategy.

The Fedorov-Volodkina Hygiene Index (HI) (1970)

To determine the Fedorov-Volodkina oral hygiene index, the vestibular surfaces of the 6 anterior teeth of the lower jaw are lubricated with a solution of iodine and potassium iodide (crystalline iodine 1 g, potassium iodide 2 g, distilled water 40 ml). Quantitative assessment is carried out on a five-point scale:

- staining the entire surface of the crown - 5 points;
- 1/4 of the surface - 4 points
- 1/2 of the surface - 3 points
- 3/4 of the surface - 2 points
- no staining - 1 point.



The average value of the index is calculated by the formula:

GI = the sum of all teeth indicators/6

Assessment of the hygienic condition of the oral cavity:

o 1.1-1.5 points - good;

o 1.6-2.0 points - satisfactory;

o 2.1-2.5 points - unsatisfactory;

o 2.6-3.4 points - poor;

o 3.5-5.0 points - very poor.

The qualitative assessment of the hygiene index is based on a three-point system:

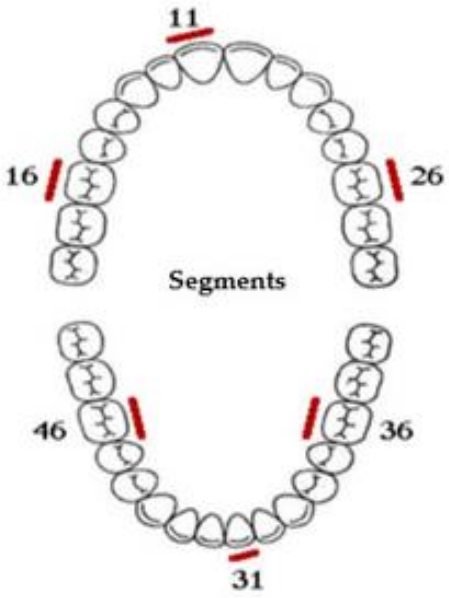
o 1 point - no staining;

o 2 points - weak staining of the tooth crown surface;

o 3 points - intense staining.

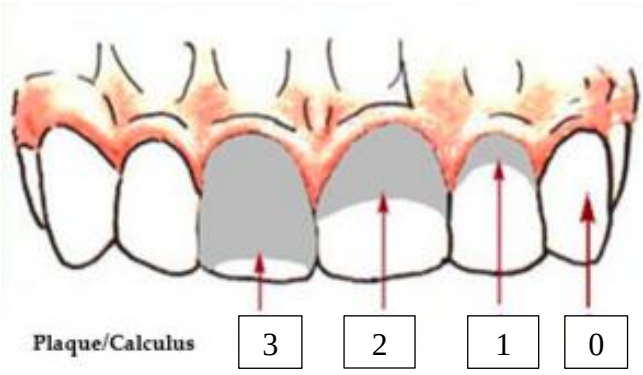
Normally, the hygiene index should not exceed 1.

A simplified index of oral hygiene. To do this, determine the presence of plaque and tartar on the buccal surface of 11, 16, 26, 31 teeth, lingual surface of 36, 46 teeth after staining them with Schiller-Pisarev solution.



Hygiene index ONI-S Green-Vermillion (1964)

- On all surfaces, plaque is first determined and then tartar. The following scores are used:
-
- 0 - no plaque;
- 1 - plaque covers no more than 1/3 of the tooth surface;
- 2 - plaque covers 1/3 to 2/3 of the tooth surface;
- 3 - plaque covers more than 2/3 of the tooth surface.



The plaque index (PI) is determined by the formula:

PLI = Sum of 6 teeth/6

A score of 3 indicates unsatisfactory oral hygiene, and 0 indicates good oral hygiene.

The tartar index (TI) is assessed in the same way as plaque:

- 0 - no calculus;
- 1 - supragingival calculus on 1/3 of the tooth surface;
- 2 - supragingival calculus on 1/2 of the crown surface or some areas of supragingival calculus;
- 3 - supragingival calculus covers more than the tooth surface, subgingival calculus encircles the neck of the tooth.

CPI = Sum of 6 teeth/6

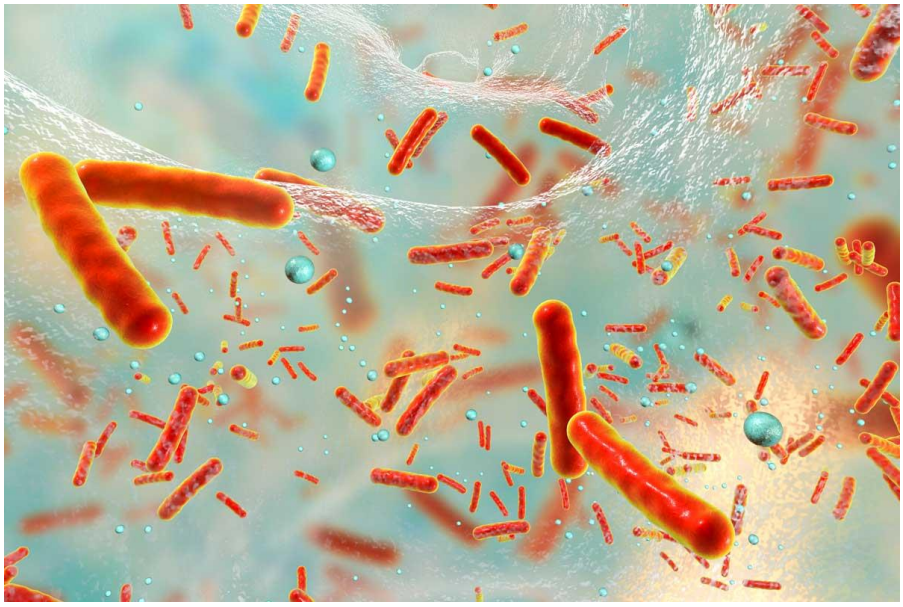
Simplified hygiene index = HI + HPI.

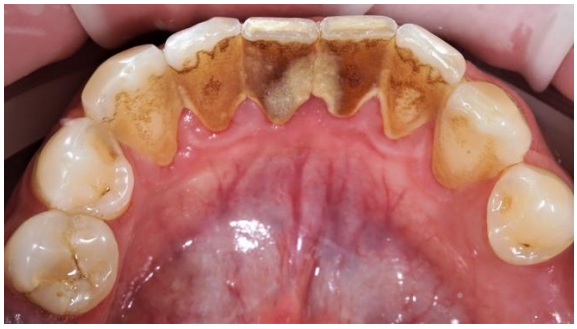
Lesson № 9
TOPIC: Plaque and tartar. Mechanism of formation. Structure, physical properties, chemical composition, microflora. Methods of removal. Carrying out professional oral hygiene. Evaluation of the effectiveness of plaque and tartar removal.

The presence of plaque on the labial surfaces of the front teeth of toddlers (1-2 years) has been tested as a predictor of the development of caries lesions during the following 2-3 years, but the accuracy is poor (sensitivity 26%, specificity 88%). Children aged 1-3 years who brush their teeth with fluoride toothpaste at least once a day have a greater chance of remaining free of caries at age 3 than those with poor oral hygiene. However, as only one study on this topic was includ-ed, it is not possible to draw any conclusions about the frequency of tooth brushing as a predictor.

Sensitivity test. A further diagnostic aid is a sensitivity test. The detection of increased dental sensitivity can be an indication of hidden caries. The simplest way to perform this test is with cold spray, during which cotton wool is sprayed with a special, rapidly evaporating agent. Stronger pain can be triggered especially in teeth with acute pul-pitis but also in teeth with broad pulpal cavities (e.g. in young people). A further option is electrical pulpal irritation with special devices. If sensitivity cannot be trig-gered, this indicates a devital tooth. Direct dentinal stimulation (test cavity): The test cavity is an invasive procedure that is often used to ensure that a negative response to previous pulp tests was accurate. Because this test is invasive and requires removal of tooth structure and/ or restorative materials, it is used primarily to exclude false negative results. The test can be used in clinical cases in which a tooth does not respond to cold testing and EPT but lacks a distinct etiology for necrosis. In such cases direct dentinal stimulation can be used to reveal necrosis or establish vitality. Direct dentinal stimulation involves removing enamel or restorative materials using a high-speed handpiece without local anaesthesia. If the tooth is vital, the patient will experience a sharp, painful response when dentin is reached. Clinicians must caution patients that they will feel the sensations of vibration and pressure so that they can interpret the test correctly.

Pulp testing involves attempting to make a determination of the responsiveness of pulpal sensory neurons. The tests involve thermal or electrical stimulation of a tooth in order to obtain a subjective response from the patient (i.e. to determine whether the pulpal nerves are functional), or the tests may involve a more objective approach using devices that objectively detect the integrity of the pulpal vasculature.





Professional oral hygiene is a set of measures aimed at cleaning teeth, for which various techniques can be used. Almost every clinic can offer the following four:

- Air flow, or the so-called pearl brushing. The doctor directs a stream of air with water and crystals of a special powder mixture under high pressure onto the tooth. The procedure is available even if you have orthodontic structures (dentures, braces), crowns and implants, veneers and lumineers.
- Laser. It is a completely non-contact procedure, so it is suitable for teeth with high sensitivity. Each tooth is cleaned individually by directing a laser beam at it. In fact, a laser is a concentrated effect of sunlight.
- Ultrasound. The doctor uses a dental scaler (scaler) - a device with a tip that emits ultrasonic vibrations. They affect the enamel surface, gently chipping off tartar. The deposit is destroyed as if inside the tooth and then washed off with water.
- Mechanical. The simplest type that is suitable for both adults and children. It is performed using a special brush attachment for a dental drill and paste. In this way, the doctor can remove soft plaque from each tooth, clean the tooth enamel from dark spots, making it visually lighter. This method is considered the most gentle, this professional cleaning does not cause tooth sensitivity, but it is ineffective for interdental spaces.

Indications for professional oral hygiene.

According to WHO statistics, about 92% of people do not know how to brush their teeth properly. As a result, by the age of 30, more than 60% of patients start using dentures. In this case, professional dental cleaning comes to the rescue. This is a preventive procedure for maintaining oral health performed by a dentist. It allows you to:

- remove plaque (both soft and hard) and prevent its deposition for some time;
- restore the teeth to their natural colour, which the enamel has lost as a result of smoking and contact with food dyes (i.e., in fact, whitening)
- strengthen the enamel, i.e. reduce or eliminate tooth sensitivity;
- reduce the likelihood of developing dental diseases by at least 50%;
- restore fresh breath.

General situations in which a patient should limit himself to home oral hygiene and cannot afford professional care are as follows

- Pregnancy and breastfeeding;
- very high sensitivity of the enamel;
- the presence of bronchitis or bronchial asthma;
- severe infectious diseases;
- cardiovascular pathologies;
- mental disorders, etc.

How the Airflow method works.

An overpressure is created in the chamber of the Air flow device, where powder granules of a certain size are located. As a result, the mixture formed from water, air and powder flies out of the nozzle. This water-air mixture and powder

are exposed to the tooth and surface cleaning takes place. The waste ingredients, along with plaque, are captured and removed by a dental vacuum cleaner.

There are several types of powder used in Air flow technology.

- Sodium bicarbonate (baking soda) with a certain particle size and flavouring. A classic option that has been used in Air flow technology for a long time. It has the ability to clean well, but in some patients it can cause temporary gum irritation.
- Calcium carbonate with regular shaped particles. It cleans teeth well and gently, polishes perfectly and does not irritate the oral mucosa.
- Glycine, an amino acid that is highly soluble in water. The powder has a pleasant taste, is low-abrasive, and polishes the surface of teeth very gently. Air flow with glycine can be used more than twice a year.

Air flow does not remove tartar and subgingival plaque, for this purpose an ultrasonic scaler is used. Air flow only brightens and restores the natural colour of the teeth by removing plaque and tartar.

Ultrasonic teeth cleaning allows you to remove all dental plaque in 1-3 sessions. The procedure is performed using a special ultrasonic scaler in the dentist's office. It is absolutely painless and takes no more than an hour per visit.

Ultrasonic tartar removal is used as an independent procedure and as an initial stage of the general sanitation of the oral cavity.

If the patient has no complaints, except for dark plaque on the teeth, it is possible to limit the removal of dental plaque and appropriate recommendations. In cases where additional treatment is required, professional ultrasonic teeth cleaning is an appropriate procedure.

Indications for ultrasonic teeth cleaning include

- the presence of mineralised and unmineralised dental plaque on the teeth, which cannot be removed on their own;
- sanitation (complete treatment) of the mouth;
- periodontal diseases.

Contraindications:

- presence of orthopedic structures and implants in the oral cavity;

- presence of infectious, viral diseases (HIV, TB, hepatitis);
- respiratory infections;
- cardiac arrhythmia;
- hyperesthesia of the teeth (hypersensitivity);
- patient complaints of chronic bronchitis, endocarditis or asthma.

Some dentists still practice calculus removal with an iron. But this procedure can hardly be called "professional cleaning". Manipulations with hand-held dental instruments resemble rough chipping, which results in

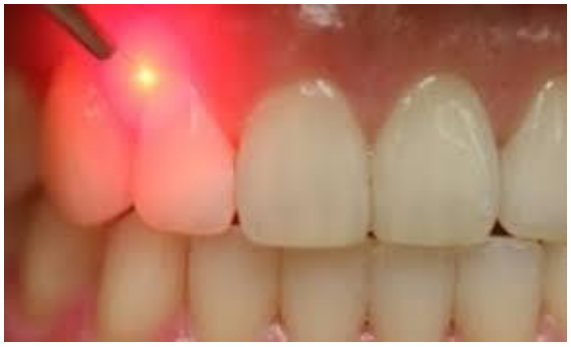
- scratches
- cracks
- Remnants of dental plaque;
- injured gingival margin.

Ultrasonic teeth cleaning helps to avoid such troubles. The scaler removes mineralised dental plaque even in the most inaccessible places. Since ultrasound is the main acting factor, there is no damage to the teeth or gums from the instrument.

Laser teeth cleaning

Professional laser cleaning helps to effectively remove soft plaque and tartar, and is quick and painless. The beam is directed at the tooth surface, the laser evaporates moisture from dental plaque, as a result of which it becomes brittle and easily washed away under a stream of water.

The use of a laser device requires special knowledge from a specialist, and not every clinic has such a device, which is why the price for this service is quite high.



MODULE 2: «LESIONS OF THE HARD TISSUES OF THE TEETH. CARIES AND NON-CARIOUS LESIONS OF THE TEETH»

Lesoon №1

TOPIC: Tooth caries. Definition of the concept. Statistical indicators of of tooth caries. Classification of caries. The concept of structural and functional resistance of hard tissues of the tooth. Determination of enamel resistance (TER test).

When the three essential parameters for dental caries - cariogenic organisms, susceptible teeth and a suitable local substrate — exist in an individual for a considerable time, then dental caries may develop. Caries causes damage by demineralization and dissolution of tooth structure, resulting from (1) a highly localized drop in the pH at the plaque-tooth interface and (2) tooth demineralization. The local pH drop occurs as the result of plaque metabolism, but only plaque communities with high concentrations of MS and lactobacilli can produce a sufficiently low pH to cause demineralization of teeth. A single exposure of sucrose solution to a cariogenic plaque results in rapid metabolism of the nutrients to organic acids. The organic acids (primarily lactic acid) dissociate to lower the local pH. Single events of lowered pH are not sufficient to produce significant changes in the mineral content of the surfaces of the teeth. However, many episodes of long-duration demineralization (lowered pH), occurring over long periods of time, will produce the characteristic lesions of caries. Frequent sucrose exposure is the single most important factor in maintaining a pH depression at the tooth surface, often resulting in demineralization. The output (production) of acid from caries-active plaques is twice that of car-ies-inactive plaques per milligram wet weight of plaque. The production of acid from a caries-active plaque can overcome the buffering capacity of salivary bicarbonate available at the tooth-plaque interface, causing the local pH to fall. Once the pH falls below 5.5, tooth mineral is dissolved. At lower pH values, such as 3.0 or 4.0, the surface of enamel is etched and roughened. At a pH of 5.0, the surface remains intact while the subsurface mineral is lost. Cavitation of the surface occurs when the subsurface demineralization is so extensive that the tooth structure surface collapses. Cavitation of enamel is not reversible and is usually associated with the acceleration in the process of cari-ous destruction of the tooth. It occurs when a series of demineralization (pH drop) and remineralization (salivary ions) episodes are dominated by the demineralization process.

There are three distinctly different clinical sites for caries initiation:

(1) the recesses of developmental pits and fissures of enamel, which is the most susceptible site;

(2) smooth enamel surfaces that shelter plaque;

(3) the root surface

CLASSIFICATION OF CARIES The clinical features of caries are various enough: from the chalky white spot on the surface of enamel to the expressed destruction of hard tooth tissues. These numerous forms of caries, per se, are only consistently changing each other (in absence of treatment) stages of tooth destruction. The progress of caries lesion leads to the destruction of all bulk hard tooth tissues, perforation of pulp chamber and development of inflammation of pulp (pulpitis) or periodontal ligament (apical periodontitis). Therefore pulpitis and apical periodontitis, other tissues of maxillofacial region, which develop as a result of caries process named complications of caries. A caries may affect either one tooth or almost simultaneously several and even a row of teeth (numerous caries), then in such cases that is a basis to speak about carious illness. There is definite conformity with the law in localization of caries lesions. These are frequently affecting fissures, pits on the occlusal and contact surfaces of molars and premolars (bicuspids). On frontal teeth caries is localized more frequent on vestibular surfaces in neck areas. Characteristic in a caries development is the retention areas where food residues and microorganisms are most often accumu-lated. On the basis of clinical features and patterns, dental caries may be classified according to three basic factors: - Morphology, i.e. according to anatomical site of lesions. - Dynamics, i.e. according to severity and rate of progression of lesions. - Chronology, i.e. according to age patterns at which lesions predominate.

Classification Based on Morphology (Anatomical Site of Lesion). Occlusal (Pit and Fissure) and Smooth Surface Caries. Caries lesions can be classified according to their anatomical site. There is nothing chemically special about these sites; they simply reflect the location of a metabolically active biofilm. The most common and simplest classification of dental caries is based on relative susceptibility of surfaces of teeth.

The different surfaces of a tooth may be divided into two morphological types. Type I refers to pit, fissure and occlusal surfaces and type II refers to smooth surfaces of which there are two variations, interproximal and cervical or gingival. Pit and fissure caries are limited to the occlusal surfaces of molars and bicuspids, the buccal pits of molars, and lingual surfaces of maxillary anterior teeth (caries fis-sus or caries occlusus). These irregular surfaces are inherently more prone to dental caries due to their mechanical characteristics which result in poor self-cleansing features. Occlusal caries usually occur early in life before smooth surface lesions appear. Carious lesions located on surfaces other than pits and fissures are classified as type II, smooth surface lesions. Smooth surface lesions may be further subdivided as interproximal, occurring at mesial or distal contact points, or cervical, occurring on buccal or lingual surfaces near the dentin-enamel junction. The caries localization on the contact surfaces (mesial or distal contact points) of teeth is named the caries of contact surfaces or interproximal caries (caries approximatus s. contactus). Smooth-surface lesions may start on

enamel (enamel caries) or on the exposed root cementum and dentin (root caries, caries cervicalis).

At the further progression of caries lesion on these areas (in particular case in baby teeth of weaken, diseased chil-dren) it formed specific localization of caries cavity: along the all neck of the tooth. Sometimes practically all neck of tooth is affected by caries like a ring. Such localization of caries named circular caries (caries anularis).

Primary caries is used to differentiate lesions on unrestored surfaces from those that develop adjacent to a filling, which are commonly referred to as recurrent or secondary caries. These two terms are synonyms. It is a carious lesion that develops at the interface of a restoration and the cavosurface of the enamel. Recurrent lesions may indicate an unusual susceptibility to caries attack, a poor cavity preparation, a defective restoration or a combination of these factors. Residual caries is demineral-ized tissue that has been left behind before a filling is placed.

Caries lesions may also be classified according to their activity. This is a very important concept and one that impinges directly on management, although the clinical distinction between active and arrested lesions is sometimes difficult. A lesion considered to be progressive would be described as an active caries lesion. In contrast, a lesion that may have formed years previously and then stopped further progression is referred to as an arrested or inactive caries lesion.

There is clinical evidence that incipient and even more advanced carious lesions may become arrested if there is a significant shift in oral environmental conditions from those that predispose to those that tend to slow the caries process. A clinical feature of arrested caries involving dentin is the marked brown pigmentation and induration of the lesion The first sign of a caries lesion on enamel that can be detected with the naked eye is often called a white-spot lesion. This appearance has also been described as an early, initial or incipient lesion. These terms are meant to say something about the stage of lesion development.

However, a white-spot lesion may have been present for many years in an arrested state and to describe such a lesion as early would be inaccurate. A dictionary definition of incipient is «beginning; nascent stage», in other words, an initial lesion appears as a white, opaque change (a white spot), but any white-spot lesion is not incipient. The terms remineralized or chronic lesions are sometimes used to signify arrested lesions, but the term remineralization should be used with caution. The distinction between active and arrested lesions may not be totally straightforward. Thus, there will be a continuum of transient changes from active to arrested, and vice versa. A lesion (or part of a lesion) may be rapidly progressing, slowly progressing or not progressing at all. This will depend entirely on the ecological balance in the biofilm covering the site and the environmental challenge.

CLASSIFICATION BASED ON SEVERITY AND RATE OF CARIES PROGRESSION. Dental caries may be classified according to the severity and

rapidity of attack. The severity may be very mild to very severe or rampant (acute). Different teeth and surfaces are involved depending upon the severity of the caries challenge. In mild caries only the most vulnerable teeth and surfaces are attacked such as the occlusal surface of first permanent molars. In moderate caries the occlusal surfaces of other posterior teeth are involved as well as interproximal surfaces. In rampant caries surfaces of anterior teeth which are relatively less frequently attacked become carious. Rampant caries is a common and important manifestation of the disease in children and some adults and merits a further discussion.

Rampant (acute) Caries: One of the most distressful clinical conditions for both patient and practitioner is rampant caries in which there occurs a sudden, rapid and almost uncontrollable destruction of teeth. For an acute caries characteristic is considerable demineralization of hard tooth tissues, relatively rapid progressing of caries lesion. The rampant (acute) caries has typical rapid (within the limits of a few weeks) course with rapid development demineralization of hard tooth tissues and caries lesions (caries defect of tooth crown).

Demineralization spread all over large areas of tooth surfaces - enamel and dentin. The affected enamel has a white chalky like colour, has not characteristic enamel translucency and becomes like a white spot.

These demineralized (friable) enamel margins easily broken during mastication of preparation of caries cavity by instruments (burs, excavators). The progression of caries lead to its spread out of the enamelodentinal junction results in development caries lesion in dentin.

Dentin has less mineralization than enamel so developing caries lesion in dentin formed more quickly. Thus a relatively large area of dentine is affected, in spite of an apparently small lesion in the occlusal surface with narrow opening and overhang margins.

Affected dentin is demineralized and softened, had cartilage like consistence and not practically changed in a colour (yellow or yel-low-brown in colour). Such quickly caries progression during rampant caries, lead to considerable destruction of hard tooth tissues and spreading pathologic process into pulp and periodontal membrane. In some cases it is multiple active carious lesions occurring in the same patient.

Rampant caries also involves surfaces of teeth that are ordinarily relatively caries-free. A caries increment of 10 or more new carious lesions over a period of about a year is characteristic of a rampant caries attack. Proximal and cervical surfaces of anterior teeth, including the mandibular incisors which are relatively caries-free, may be affected.

Patients with rampart caries can be classified according to the assumed causality, e.g. bottle or nursing caries, baby caries, early childhood caries, radiation caries or drug-induced caries. Subjectively the rampant caries course is

characterized by more expressed pain caused food irritants (sucrose or another chemical, thermal irritant).

Typically are multiple caries lesions. Such caries course often depended on low resistance of organism, systemic and concomitant diseases, and nutritional factors. Among them there are some physiologic conditions (adolescence, pregnancy), systemic exhausting diseases (tuberculosis, inflammatory processes in some inner organs), blood diseases, endocrine disorders and others. Some role play age of patient, anatomical features of different teeth (temporary or permanent dentition) and so on. A chronic caries develops for years, softening of hard tooth tissues expressed in less degree. The state of organism and constitutional factors, carried and concomitant diseases, unfavorable conditions of development in child's age, social conditions and others influence on a different course of caries. These features cause the necessity of a different approach to their treatment. The chronic caries course has typical slow development caries lesion.

A pathologic process usually lasts for years; subjective pain feeling as a result of different irritants action is insignificant. The areas of demineralization of hard tooth tissues do not occupy such a considerable area as at the rampant caries course. As a result of penetration of food pigments into softened hard tooth tissues, the enamel and dentin become brown or black in colour. The typical for acute caries course overhang enamel margins are broken and formed wide caries cavity opening.

The softened and pigmented hard tooth tissues have more dense consistency, than at the acute caries course. The chronic caries course more often develops in practically healthy people with relatively insignif- ¡cant violations of metabolic processes, nutritional disorders and so on. On such basis of satisfactory general organism condition the numerous caries lesions developed very rarely, more typically are single caries lesion. The long existence of caries cavity, lead to developing in pulp some defensive formations (for example, the secondary dentin).

Firstly, there is a zone of sclerosis walling off the lesion from the surrounding normal dentine. This zone of sclerosis is often referred to as the translucent zone, which is a defense reaction on the part of the pulp-dentinal unit. A further defense reaction can be seen some distance from the lesion. A region of reactionary, or reparative, dentin is laid down on the pulpal aspect of the lesion. Therefore the caries complication (pulpitis, apical periodontitis) developed considerably rarely. Acute and chronic caries course may change one another - under some conditions chronic caries course may changes rampant caries course. And, vice versa, at the improvement of the general organism status, character of nutrition acute caries may become chronic caries with slow course, or it may be arrested following several active phases. Such caries named arrested caries - caries stataria (Latin).

The more often such caries localized on occlusal surface which sustained attrition during mastication. When the general status of a patient's organism

becomes considerably worse this development is so-called caries acutissima. This pathologic process is characterized by the very rapid course of caries process (during two-five weeks), which quickly developes the caries lesions. Such caries course accompanies numerous caries lesions especially in all teeth with one or two cavities in the tooth crown and this caries course is named caries florida. Sometimes caries begins to develop in a filled tooth, affecting hard tooth tissues around the filling or restorations. In these cases it is named caries recidiva s. secundaria.

Regardless of clinical course caries can affected single or numerous teeth. In the last case caries, as a rule, has the rampant course and named numerous (multiple) caries or systemic caries. In pedodontics distinguish compensative, sub compensative and decompensative caries form (after T.F. Vinogradova, 1978).

Hidden caries is a term used to describe lesions in dentin that are missed on a visual examination but which are large and demineralized enough to be detected radiographically. It should be noted that whether a lesion is actually hidden from vision depends on how carefully the area has been cleaned and dried and Whether an appropriate clinical examination has been performed. In other words, to talk about hidden caries implies an insufficient clinical examination.

Caries process can affected different hard tooth tissues therefore in obedience to International classification of diseases there is also anatomic classification of car-ies. According this classification distinguish enamel caries (caries enameli), dentin caries (caries dentini, cementum caries (caries cementi), arrested caries, odontoclasia, another caries, indefinite caries.

Caries destruction of hard tooth tissues in case of treatment absence may lead to complete destruction of enamel and dentin. In such cases the microorganisms and its toxins penetrate from a carious cavity in pulp and, even periodontal ligament, causing its inflammation development - pulpitis and apical periodontitis. These pathologic processes so named complications of caries. Depending on the degree of this penetration caries process without complications named caries simplex s. incom-plicata and with these complications - caries complicata (with pulpitis and apical periodontitis).

Classification of tooth preparations according to the anatomic areas involved as well as by the associated type of treatment was presented by G. V. Black and is designated *as Class I, Class II, Class III, Class IV, and Class V*. Since Black's original classification, an additional class has been added, *Class VI.* This classification has very practical value for operative treatment of caries by preparation and further filling (restoration) of carious cavities.

Class I Restorations: All pit-and-fissure restorations are Class I, and they are assigned to three groups, as follows: Restorations on Occlusal Surface of Premolars and Molars; Restorations on Occlusal Two Thirds of the Facial and Lingual Surfaces of Molars; Restorations on Lingual Surface of Maxillary Incisors.

Class II Restorations: Restorations on the proximal surfaces of posterior teeth are Class II: the proximo-occlusal (MO) conventional preparation, the distocclu-sal preparation (DO) and an MOD preparation has similar walls, line angles, and point angles.

Class III Restorations: Restorations on the proximal surfaces of anterior teeth that do not involve the incisal angle are Class III. Walls, line angles, and point angles of a representative conventional tooth preparation. Note that the faciolingual line angle at the incisal is termed the incisal line angle; likewise, the faciolingual incisal point angle is termed the axioincisal point angle.

Class IV Restorations: Restorations on the proximal surfaces of anterior teeth that do involve the incisal edge are Class IV.

Class V Restorations: Restorations on the gingival third of the facial or lingual surfaces of all teeth (except pit-and-fissure lesions) are Class V. For posterior teeth the incisal (i) becomes occlusal (o).

Class VI Restorations: Restorations on the incisal edge of anterior teeth or the occlusal cusp heights of posterior teeth are Class VI.

A general anatomical classification of caries penetration into dental tissues includes:

(1) initial surface caries confined to the enamel,

(2) penetration of enamel,

(3) penetration of the dentinoenamel junction,

(4) early penetration into dentin,

(5) advanced penetration into the dentin,

(6) penetration to or into the pulp of the tooth. In addition to the above classification several other clinical patterns have been described which relate to the degree and rate of progression of caries. In a clinic topographical classification of caries depending on the depth of destruction of hard tooth tissues is most often used.

In 1948 I.G. Lukomskiy offered to distinguish two basic clinical features of caries - carious spot and carious cavity.

Depending on the depth of caries lesion of enamel and dentine the caries was divided on a superficial, middle and deep caries.

Depending on the caries course distinguish acute (rampant) and chronic caries. Therefore in classification distinguish incipi- ent caries (carious spot), superficial caries, middle and deep caries; acute or chronic course.

Incipient caries (caries incipiens): The characteristic feature is development demineralization on the enamel surface. The early carious lesion on visible smooth surfaces of teeth is clinically manifested as a white, opaque region, which is best demonstrated when the area is air-dried. There is not caries cavity in enamel.

Superficial caries (caries superficialis) - there is carious defect in enamel; den-tinoenamel junction is not destroyed by caries process.

Middle caries (caries media) - the caries cavity formed in the dentin: in a mantle dentin. This layer of dentin juxtaposed to the enamel and converted into the initial laver of dentin from the basement membrane.

Deep caries (caries profunda) - the caries cavity formed in the dentin: in a cir-cumpulpal dentin. This layer of dentin localized very close to pulp. The caries cavity may be divided from the pulp chamber only very thin partition or only the layer of secondary dentin.

CLASSIFICATION OF CARIES

I. Clinical
1. Incipient caries (acute, chronic).
2. Superficial caries (acute, chronic)
3. Middle caries (acute, chronic).
4. Deep caries (acute, chronic) or another variant:
1. Initial lesion
2. Extensive lesion
3. Moderate lesion
4. Severe lesion
II. Classification based on localization:
1. Occlusal (pit and fissure) caries.
2. Contact (interproximal) caries.
3. Root (cervical) caries.
4. Circular (caries anularis) caries.
III. Classification based on severity and rate of caries progression.
1. Rampant (acute) caries.
2. Chronic caries.
3. Caries acutissima.
4. Caries florida.
5. Recurrent (caries recidiva s. secundaria) caries.
IV. Classification based on number of lesion.
1. Single lesion.
2. Numerous lesions.
Anatomic classification (WHO).
1. Enamel caries.
2. Dentine caries.
3. Cementum caries.
4. Arrested caries.
5. Odontoclasia.
6. Another.
7. Indefinite.
VI. Classification based on the presence of complications.
1. Caries simple (uncomplicated).
2. Complicated caries.
VII. Classification based on severity

1. Very mild caries.
2. Mild caries.
3. Moderate caries.
4. Severe caries.
5. Very severe caries.

CLINICAL FEATURE OF CARIES.

Incipient caries: Patients complain at the presence of spots (white, chalky white, opaque or pigmented), rarely on feeling of insignificant sensitiveness, soreness of the mouth from different irritants mainly chemical (sour, sweet). The development of spots is linked with disorders of mineral composition of enamel, its dis-mineralization and demineralization. A process clinically begins from the loss the natural enamel translucency which is characteristic for intact enamel. The limited enamel area becomes opaque, chalky white or brown colour. The last is caused by penetration of pigments (food, tobacco, pigmented microflora) in the demineralized (hypo mineralized) area of enamel during prolonged development of pathological process. At the acute (rampant) course on the limited areas of dental enamel appeared the opaque, deprived of natural transparency, chalky white colour spots. At the first time spots are small, but, gradually increased in sizes. Frequently it is located on occlusal surfaces in retentive points: pits and fissures of occlusal surfaces of teeth, cervical areas. In children it often localized on a vestibular surface and cervical areas. For the best revealing of caries spots it is recommended to remove from the spot surface debris and dry up the crown of the tooth: intact enamel saves their natural transparency and brilliance, while the surface of caries spot loses transparency and becomes opaque. During probing a roughness, insignificant pliability and sick-liness of their surface, can be revealed.

At the caries spots located in fissures there can be difficulties at their diagnostics and differentiation with other pathological conditions, especially with children. It is linked with accumulation of food remnants in fissures, pigments which can change colour of surface fissures. In such cases it is hard visually to reveal a whitish or some pigmented caries spot in the enamel.

Using another diagnostic method to reveal the clinical caries signs at probing - softening, sensitiveness of surface of enamel. In children these signs may be very subjective, that is caused by insufficient mineralization of enamel in fissures. In such cases it is had to use the additional methods of caries investigation. To reveal caries it is possible to use, except for mechanical, (probing) and also other irritants (thermal, chemical). On the fissures surface placed cold (5—10° C) or hot (60-70° C) water or gutta-percha warmed-up to soft consistency. In intact enamel surface the heating of a fissure within the limits of 10-60° C does not cause a characteristic for a caries pain.

Chemical irritants can be used, such as organic solutions (lactic, apple) acids; and inorganic (hydrochloric, carbolic) acids; for solutions of sucrose, glucose, alcohol and ether. Intensity and frequency of feeling pain almost does not rely on the use of an irritant, but is dependent mainly on its con-centration,

age of patient (in elderly patients the reaction on the pain feeling low) and organism resistance.

The very effective diagnostic method of acute incipience caries is usage of the reactions with different dyes. They are based on properties of dye to penetrate into the demineralized enamel and, thus, dyeing a caries spot, while in an intact enamel it cannot penetrate. It is used a 2% water solution of methyl blue, a 0.1% water solution of methyl red, carmine, Congo red and others. A tooth is isolated from saliva by cotton rolls, dry out and on its surface with cotton pellet place a dye. In a few minutes the remnants of dye wash off by water and at presence of carious spot the surface of enamel gets the colour of dye (dark blue, rose and others). Another method is the use the solution of nitric acid silver, in a caries spot it is restored to metallic silver under influencing of present in her products of protein disintegration. A spot here acquires the black colour. More rapid renewal of silver in this reaction can be attained by placed on the treated by nitric acid silver surface of enamel the repairer, for example a 4% solution of hydroquinone. In a carious spot the quantity of mineral components (apatites) of enamel diminishes because of processes of demineralization. It causes changes of conductivity of this hard tooth tissues, namely to its increase, because electric resistance of such area of enamel diminishes. The method of electro diagnostic of initial forms of caries was founded (Novik I. O., 1951; N.A. Kodola, 1956).

By the special device the conductivity is measured between the surfaces of affected by a pathological process (caries) or suspicious on its presence and intact enamel areas. A tooth is isolated, dry out, an active electrode is placed on the suspicious area of enamel surface and the passive is placed in the patient hand. If enamel surface is intact, the conductivity is practically equal to the zero because of large electric resistance of intact enamel. At caries presence the condostics arises Approximately on the same principle based the method of electro diagnostics of caries offered by V.K. Leontyev (1983).

Very useful method for diagnostic caries lesions is transillumination. It is possible to apply the method of luminescent diagnostics, which is based on the phenomenon of fluorescence of intact hard tooth tissues under action on them of ultraviolet rays. The special ultraviolet lamps are used for this purpose. In the black-out apartment the surface of tooth is lighted by a lamp in the distance by 20-30 sm. By fluorescence intact dental enamel shines light-green or bluish light, while areas affected by caries areas do not radiate light. The histological features at an initial caries are characterized development of different degree of enamel demineralization. At the enamel section the body of caries lesion has the triangle appearance with the basis turned of to enamel surface. At the study in the polarized light depending on the structure changes in the enamel lesion distinguish a few areas. The most demineralized is sub superficial lesion layer, which is covered by the mineralized superficial enamel layer. This interesting phenomenon is explained by the remineralization processes of carious lesion by the mineral components of saliva, if an oral fluid is unable to provide

remineralization of the demineralized enamel area the rapid development of caries lesion occurs.

The surtace texture of an incipient lesion is unaltered and is undetectable by tactile examination with an explorer. A more advanced lesion develops a rough surface that is softer than the unaffected, normal enamel. Softened chalky enamel that can be chipped away with an explorer is a sign of active caries. In the interior layers of white caries spot there are the changes both enamel rods surfaces and prisms themselves. In separate areas the crystals of apatites are destroyed, their orientation in rods changes, in formed micro spaces there are atypical crystals. Fragmentation of crystals, formation of homogeneous fine-grained substance and disappearances of visible border, between prisms is marked in future. Non-cavitated enamel lesions retain most of the original crystalline framework of the enamel rods and the etched crystallites serve as nucleating agents for remin-cralization. Calcium and phosphate ions from saliva can then penetrate the enamel surface and precipitate on the highly reactive crystalline surfaces in the enamel lesion. Remineralized (arrested) lesions can be observed clinically as intact, but dis-colored, usually brown or black spots. Depending on the degree of the demineralization G. Gustafson (1975) distinguishes five zones. Most deeply in the enamel there is placed hyper mineralization zone with disappearance enamel structural components. In the second zone there is reduction of its hardness because of partial dissolution of enamel minerals, in the third - increase of mineralization. In a sub superficial fourth demineralization zone the minerals are almost fully washed. In a superficial fifth zone there can be comPlete disintegration, however it during long time remains enough mineralized and undamaged, even when caries spread on all thickness of enamel.

At a polarization microscopy five zones (S.N. Onishenko, 1968; V.P. Zenovskiy, 1970) distinguish:

1) superficial,
2) sub superficial,
3) central — body of caries lesion,
4) intermediate and
5) internal — translucent zone of enamel.

These zones are characterized by a different degree of transparency, therefore still named as superficial, dark (body of lesion) and semi-translucent zones (A. Darling, 1959)

There were the four regularly observed zones in a sectioned incipient lesion: (1) the translucent zone, (2) the dark zone, (3) the body of the lesion, and (4) the surface zone.

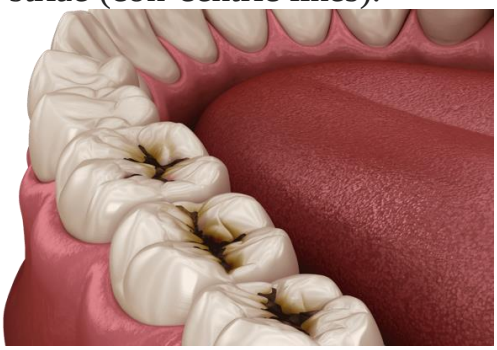
Zone 1: Translucent Zone. The deepest zone is the translucent zone and represents the advancing front of the enamel lesion. In this zone, the pores or voids form along the enamel prism (rod) boundaries, presumably because of the ease of hydrogen ion penetration during the carious process. The pore volume of the translucent zone of enamel caries is 1%, 10 times greater than normal enamel.

Zone 2: Dark Zone. The next deepest zone is known as the dark zone because it does not transmit polarized light. This light blockage is caused by the presence of many tiny pores too small to absorb quinoline. These smaller air- or vapor-filled pores make the region opaque. The total pore volume is 2% to 4%.

Zone 3: Body of the Lesion. The body of the lesion is the largest portion of the incipient lesion while in a demineralizing phase. It has the largest pore volume, varying from 5% at the periphery to 25% at the center. The Retzius striae are well marked in the body of the lesion, indicating preferential mineral dissolution along these areas of relatively higher porosity.

Zone 4: Surface Zone. The surface zone is relatively unaffected by the caries attack. It has a lower pore volume than the body of the lesion (less than 5%) and a radiopacity comparable to unaffected adjacent enamel. The surface of normal enamel is hyper mineralized by contact with saliva and has a greater concentration of fluoride ion than the immediately subjacent enamel. As the enamel lesion progresses, conical-shaped defects in the surface zone can be seen by SEM.

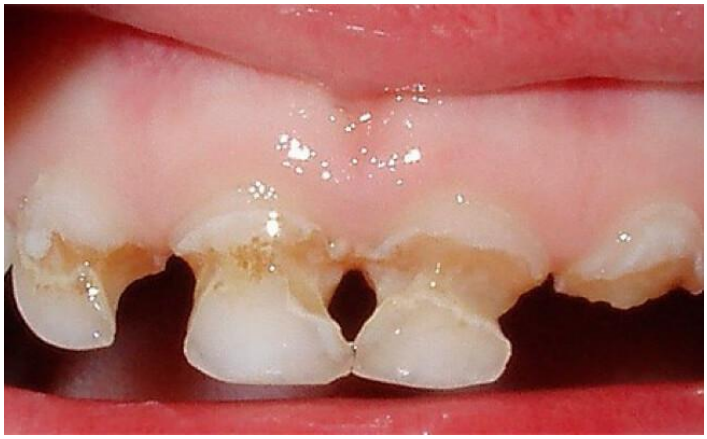
Also was revealed, that in the caries enamel lesion (carious spots) the volume of micro spaces is increased. If in an intact enamel they make approximately 0,2% from the general volume of this tissue, in a carious spot the volume of micro spaces increases to 0,8% in a superficial zone and till about 16% in sub superficial and central zones. There is decline of enamel hardness which is most distinct in the central area of carious lesion. Initial demineralization, namely the decline of calcium content in a superficial layer of white carious spot takes place on the so-called Retzius striae (con-centric lines).



Occlusal caries

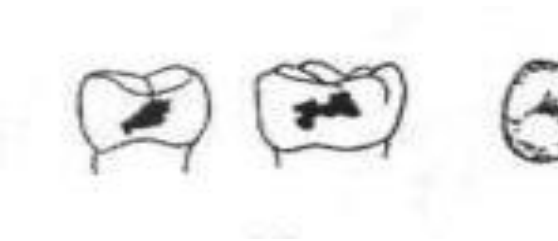
Contact caries

Cervical caries



Class I includes carious cavities located in natural fissures and pits on the occlusal (chewing), buccal and lingual (palatal) surfaces of molars and premolars and the lingual (palatal) surface of incisors;

Class II - carious cavities on the contact surfaces of molars and premolars;



Class III - located on the contact surfaces of incisors and

canines;

Class IV - carious cavities of class III with a violation of the integrity of the angle of the cutting edge;

Class V - located in the neck area of all tooth groups.

Lesson №2
TOPIC: Modern ideas about the etiology and pathogenesis of caries.

When the three essential parameters for dental caries - cariogenic organisms, susceptible teeth and a suitable local substrate — exist in an individual for a considerable time, then dental caries may develop.

Caries causes damage by demineralization and dissolution of tooth structure, resulting from (1) a highly localized drop in the pH at the plaque-tooth interface and (2) tooth demineralization. The local pH drop occurs as the result of plaque metabolism, but only plaque communities with high concentrations of MS and lactobacilli can produce a sufficiently low pH to cause demineralization of teeth.

A single exposure of sucrose solution to a cariogenic plaque results in rapid metabolism of the nutrients to organic acids. The organic acids (primarily lactic acid) dissociate to lower the local pH. Single events of lowered pH are not sufficient to produce significant changes in the mineral content of the surfaces of the teeth. However, many episodes of long-duration demineralization (lowered pH), occurring over long periods of time, will produce the characteristic lesions of caries. Frequent sucrose exposure is the single most important factor in maintaining a pH depression at the tooth surface, often resulting in demineralization.

The output (production) of acid from caries-active plaques is twice that of car-ies-inactive plaques per milligram wet weight of plaque. The production of acid from a caries-active plaque can overcome the buffering capacity of salivary bicarbonate available at the tooth-plaque interface, causing the local pH to fall. Once the pH falls below 5.5, tooth mineral is dissolved. At lower pH values, such as 3.0 or 4.0, the surface of enamel is etched and roughened. At a pH of 5.0, the surface remains intact while the subsurface mineral is lost.

Cavitation of the surface occurs when the subsurface demineralization is so extensive that the tooth structure surface collapses. Cavitation of enamel is not reversible and is usually associated with the acceleration in the process of carious destruction of the tooth. It occurs when a series of demineralization (pH drop) and remineralization (salivary ions) episodes are dominated by the demineralization process.

There are three distinctly different clinical sites for caries initiation:

(1) the recesses of developmental pits and fissures of enamel, which is the most susceptible site;

(2) smooth enamel surfaces that shelter plaque;

(3) the root surface.

Lesson№3

TOPIC: Acute and chronic caries in the spot stage

Pathomorphology, clinic, diagnosis, differential diagnosis, treatment methods.

Actually the formation of *brown pigment in a spot* is connected with the accumulation in spot of amino acid such as tyrosine with further its transformation into pigment melanin. Under a white caries spot does not still revealed changes of dentinoenamel junction, under pigmented spots such changes is revealed. The reactive changes were revealed in dentin: the narrowing of dentinal tubules, appearances of layer of translucent (sclerotic) dentin; the odontoblasts changing in the pulp areas according to the caries lesion. Electron microscopic studies of den-tine with an incipient caries allowed to revealed two phases of pathological process development in spot. At the first phase there are the rough changes of odontoblastic processes, destruction of collagen fibres of dentine matrix.

Disorganization of odontoblasts layer, pathological changes of nervous fibres and vessels were revealed in pulp. At the second phase, on a background of further progress of these changes, the destroying of form and dimension of dentin apatites crystals developed, in its tubules begins the deposition of mineral salts. Superficial caries: In some period of time in the center of caries spot the superficial layer of enamel loses the integrity and in enamel defect appears.

The first sign of a caries lesion on enamel that can be detected with the naked eye is often called a white-spot lesion. This appearance has also been described as an early, initial or incipient lesion. These terms are meant to say something about the stage of lesion development. However, a white-spot lesion may have been present for many years in an arrested state and to describe such a lesion as early would be inaccurate.

A dictionary definition of incipient is «beginning; nascent stage», in other words, an initial lesion appears as a white, opaque change (a white spot), but any white-spot lesion is not incipient. The terms remineralized or chronic lesions are sometimes used to signify arrested lesions, but the term remineralization should be used with caution. The distinction between active and arrested lesions may not be totally straightforward. Thus, there will be a continuum of transient changes from active to arrested, and vice versa. A lesion (or part of a lesion) may be rapidly progressing, slowly progressing or not progressing at all. This will depend entirely on the ecological balance in the biofilm covering the site and the environmental challenge.

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At the acute (rampant) course on the limited areas of dental enamel appeared the opaque, deprived of natural transparency, chalky white colour spots. At the first time spots are small, but, gradually increased in sizes. Frequently it is located on occlusal surfaces in retentive points: pits and fissures of occlusal surfaces of teeth, cervical areas. In children it often localized on a vestibular surface and cervical areas. For the best revealing of caries spots it is recommended to remove from the spot surface debris and dry up the crown of the tooth: intact enamel saves their natural transparency and brilliance, while the surface of caries spot loses transparency and becomes opaque. During probing a roughness, insignificant pliability and sick-liness of their surface, can be revealed.

Incipient caries (caries incipiens): The characteristic feature is development demineralization on the enamel surface. The early carious lesion on visible smooth surfaces of teeth is clinically manifested as a white, opaque region, which is best demonstrated when the area is air-dried. There is not caries cavity in enamel.

The surtace texture of an incipient lesion is unaltered and is undetectable by tactile examination with an explorer. A more advanced lesion develops a rough surface that is softer than the unaffected, normal enamel. Softened chalky enamel that can be chipped away with an explorer is a sign of active caries. In the interior layers of white caries spot there are the changes both enamel rods surfaces and prisms themselves. In separate areas the crystals of apatites are destroyed, their orientation in rods changes, in formed micro spaces there are atypical crystals. Fragmentation of crystals, formation of homogeneous fine-grained substance and disappearances of visible border, between prisms is marked in future. Non-cavitated enamel lesions retain most of the original crystalline framework of the enamel rods and the etched crystallites serve as nucleating agents for remin-cralization. Calcium and phosphate ions from saliva can then penetrate the enamel surface and precipitate on the highly reactive crystalline surfaces in the enamel lesion.

In case of incipient caries it is possible to speak about demineralization affected hard tooth tissues. A white carious spot can fully disappear.

BACKGROUNDS OF REMINERALIZATION THERAPY

1. Ability of restoration the level of hard tooth tissues mineralization during incip- ience caries.

2. Phenomenon of enamel permeability for mineral matters.

3. Dynamic balance processes of demineralization and remineralization in enamel.

4. Ability of fluoride for restoration enamel mineral structure.

5. Phenomenon of «enamel maturation» in children. in favour to primary atraumatic restorative treatment or minimal invasive treatment with the subsequent control of surfaces affected by caries. Neither of the varieties of restoration treatment of dental caries may be fully «curing».

Destroyed by a caries hard tooth tissues (and adjoining areas of healthy enamel) are not substituted for by formed again enamel and dentin. Besides, there is no existing restorative material capable during all life to protect hard tooth tissues from further destructive caries processes.

Tooth restoration is only symptomatic treatment which does not eliminate the etiologic factors of dental caries. Therefore the prevention of development caries lesions (prophylactic measures) is the basic principle of caries treatment, rather than the necessary medical treatment (remineralization therapy) and, in the last turn, as the forced measure of restoration the caries cavity with restorative materials, conducted along with the measures of the second caries prophylaxis.

Thus, now there are two main methods of local caries treatment:

1) caries treatment without preparation and restoration - remineralization therapy,

2) operative caries treatment by the operative preparation of demineralized hard tooth tissues with the subsequent restoration of carious cavity. The choice of treatment method depends of the stage of caries development, activity of caries (rampant or chronic), localization of carious cavity, age and general condition of patient. Preventive treatment methods are designed to limit tooth demineralization caused by cariogenic bacteria, thereby preventing cavitated lesions.

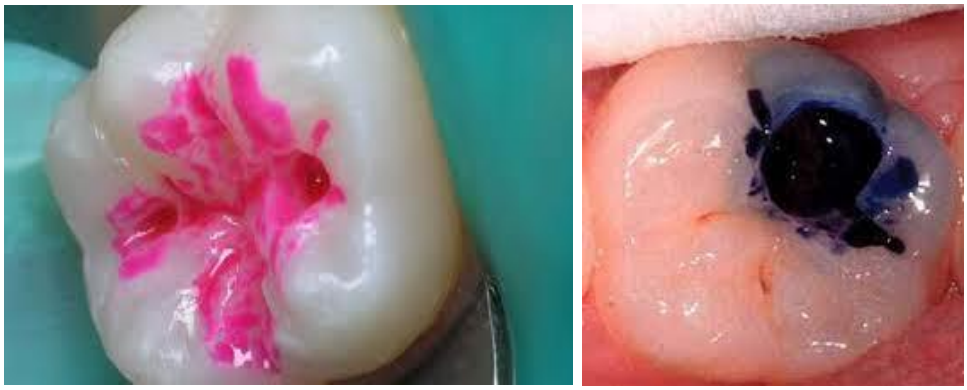
They include:

(1) limiting pathogen growth and metabolism,

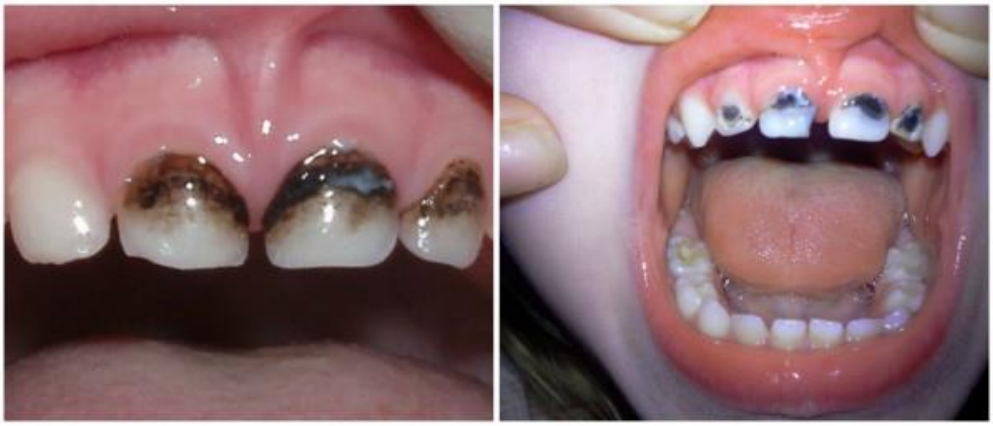
(2) increasing the resistance of the tooth surface to demineralization. Disease caries control concerns influencing biofilm formation and growth, or modifying the dissolution kinetics of the apatites, or both.

The following may have a role to play:

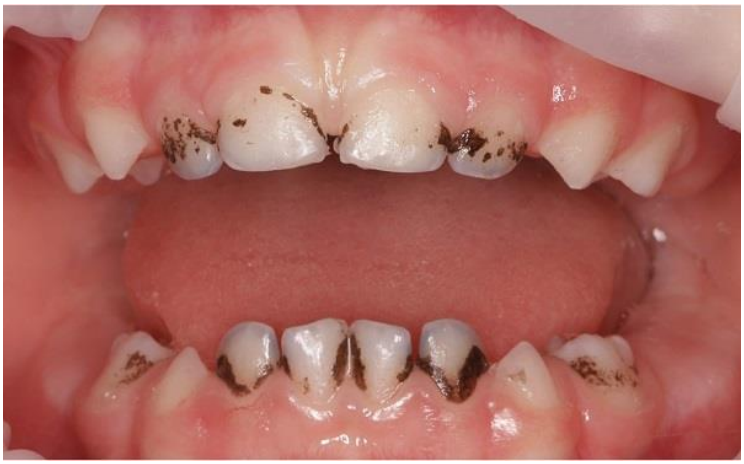
- mechanical/chemical removal of plaque (oral hygiene)
- chemical (antimicrobial) modification of plaque
- use of fluorides
- dietary composition
- salivary composition and stimulation.



If there is a carious spot, it is the colour of the dye (blue, pink, etc.). Another method has been proposed: instead of dyes, a silver nitrate solution is used, which is reduced to metallic silver in the carious spot under the influence of protein breakdown products present in it.



In this case, the stain becomes black. A faster recovery of silver in this reaction can be achieved by applying a 4% hydroquinone solution to the silver nitrate-treated enamel surface.



Pristley's plaque

Treatment of caries with the Icon system

Icon caries treatment is a non-invasive technique that allows to cure early caries and preserve tooth tissue without treating it with a dental drill.

The system is a set of special gels and a liquid polymeric substance. This German innovation, which allows treating caries without a drill, quickly became popular and in demand. The use of Icon in paediatric dentistry can be compared to a real magic wand, which makes caries disappear without pain and tears.

The useful elements of the product literally saturate and fill the tooth tissues, preventing the carious process from developing.

Lesson №4
TOPIC: Acute and chronic superficial caries: pathomorphology, clinic, diagnosis, differential diagnosis, treatment.

Superficial caries (caries superficialis) - there is carious defect in enamel; den-tinoenamel junction is not destroyed by caries process.

Superficial caries: In some period of time in the center of caries spot the superficial layer of enamel loses the integrity and in enamel defect appears. In an acute superficial caries the patients complains of insignificant pain, more frequent on feeling of soreness of the mouth and affected tooth, which caused by chemical irritants and quickly disappeared after stopping of irritant action. Sometimes there can be short-term pain from thermal and mechanical irritants, more frequent in a place of caries lesion. At the examination of tooth in the area of chalky white colour spot revealed of defeat of a shallow enamel defect (cavity) is determined, placed within the enamel borders. The enamel wall of lesion is softened, yellow-grey colour by and some sensible at probing. Sometimes may be only rough surface, but after removing softened enamel surface the lesion (cavity) was found.

A chronic superficial caries has course mainly without the pain feeling. Rarely may be insignificant pains from chemical irritants which at once disappeared after their removal. On the surface of enamel revealed small enamel lesion (cavi-ty), with enough dense yellow-brown or brown colour enamel walls. A cavity has wide, exposed, without overhanging margins entrance opening. Probing of carious defect is practically painless. When superficial caries located in fissures the margins of lesions remained undamaged.

Superficial caries is diagnosed on such basis:

a) patient complaints of short-term pain feeling mainly from chemical irritants; pain disappeared after stopping of irritant action;

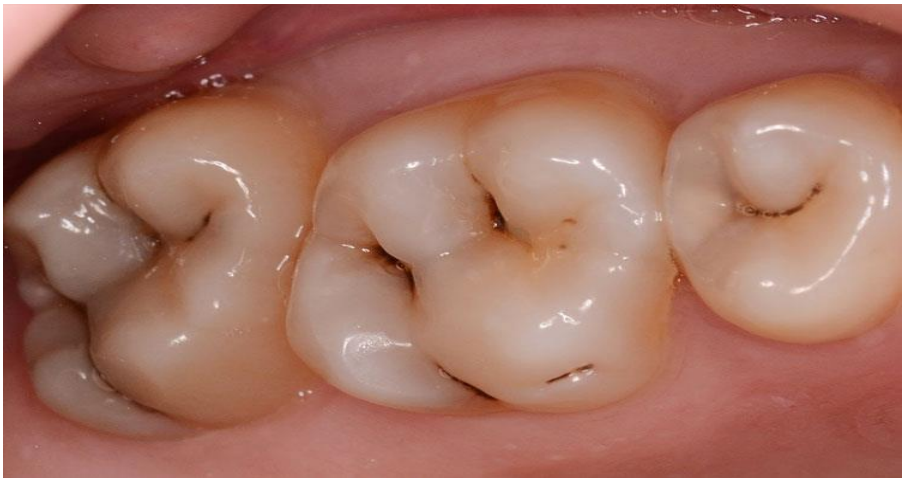
b) revealing of shallow carious cavity, located within the limits of enam-el, or of fissures pigmentation on occlusal surface, in which by probing the softened demineralized enamel is revealed:

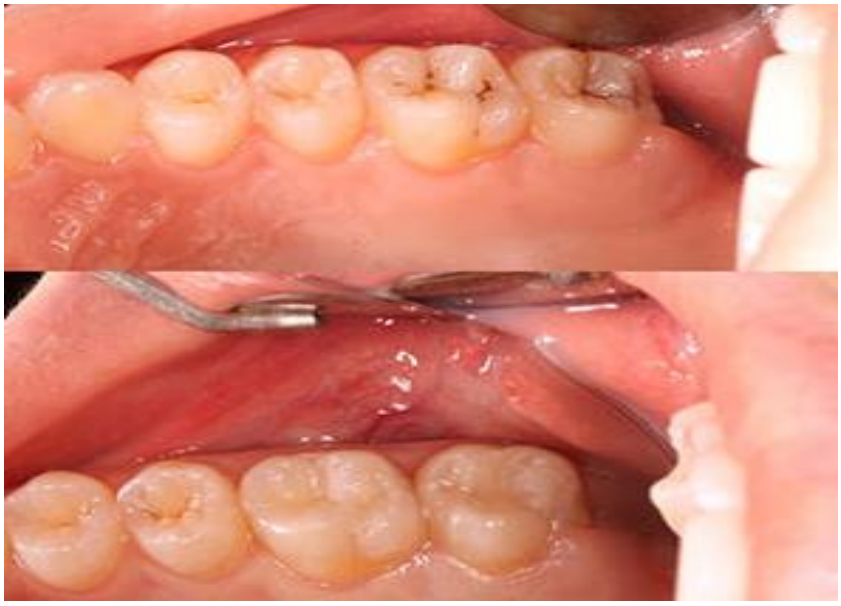
c) painful preparation of hard tooth tissues especially at the dentinoenamel junction.

Histopathology: The main features consist of complete destruction of all enamel rods (prisms) in the body of lesion. Around it there is disintegration peripheral areas of enamel prisms, increase of inter-prism distances with the further involving in the pathological process of dentinoenamel junction. Thus in enamel formed a different sizes cone-shaped lesion. The apex of lesion reaches a dentine in which also the process of demineralization and initial destruction begins.

All these changes are more expressed at the acute caries course and less at chronic, in the last case the widening inter-prism distances contain the large amount of pigments (mainly brown). In the areas of dentine, which located closer to the caries enamel lesion the characteristic changes are also revealed. Directly

near dentinoenamel junction den-tinal tubules are widening and filled microorganisms. In deeper dentin layers the tubules are narrowed, sclerotic; the dentin matrix hyper mineralized forming zone translucent or sclerotic dentin. In pulp according to localization of caries cavity odontoblasts are deformed, reduction in quantity, the subodontoblastic plexus capillaries are widening.





Lesson №5
TOPIC: Acute and chronic medium caries: pathomorphology, clinic, diagnosis, differential diagnosis, treatment.

Middle caries (caries media): After destruction by the pathological process of dentinoenamel junction caries begins quickly to spread in a dentine. As a middle caries (caries media) mean such pathological condition, when a caries cavity located in mantle dentin. The patient with acute middle caries often complains of feeling pain. More frequent the pain has weak intensity and appeared only at action of irritants: chemical, thermal, mechanical. On the tooth surface there is a chalky white colours caries spot with the enamel defect in a center. Examination of the cavity is difficult because of narrow entrance opening. A cavity usually has a depth 1.5-2 mm, is filled by food residue and softened dentin. Complete examination of carious cavity is possible only after removing by the instruments (burs, excavators) of overhanging chalky white colour enamel margins. Cavity is most wide near dentinoenamel junction and gradually narrows towards pulp. The softened dentine which covers a cavity, has grey-white or yellow colour, rarely is it pigmented.

The degree of dentine softening depend of activity of caries process: at acute (rampant) caries the hard tooth tissues are most softened like a cartilage, at chronic course it may be harder and pig-mented. Probing of carious cavity is practically painless except dentinoenamel junction. A chronic middle caries has practically little clinical symptoms. In some case may be weak pain which appeared because of action of chemical, rarely thermal and mechanical irritants and is at once stopped after their removal. At the examining a caries cavity with the enough wide entrance opening is revealed, it located in mantle dentin, depth of cavity is 1.5-2 mm depending on the surface of tooth. The caries cavity is covered with enough dense pigmented dentin; floor and walls of cavity are painless at probing. During electric pulp testing (electro odonto-diagnostic method) the pulp reacts on strength of current 6—12 mkA.

Histopathology: The development of caries leads to some path histological changes in hard tooth tissues and pulp. In an enamel it have practically the same character, as at incipience caries with addition the lesion with the walls consist of broken and accordingly changed enamel prisms. More various are the histological features in a dentine and distinguish such zones.

Zones of Dentinal Caries (J.B. Summit et al., 2001).

Caries advancement in dentin proceeds through three changes:

(1) weak organic acids demineralize the dentin;

(2) the organic material of the dentin, particularly collagen, degenerates and dis-solves; and

(3) the loss of structural integrity is followed by invasion of bacteria. Five different zones have been described in carious dentin. The zones are most clearly distinguished in slowly advancing lesions.

In rapidly progressing caries, the difference between the zones becomes less distinct.

1. Zone of disintegration. It is characterized by the complete loss of any dentin structure, its almost complete demineralization and softening.

The areas of dentin closer to periphery of lesion are practically destroyed, brown or rather yellow-brown colour. There is considerable accumulation of microorganisms in this dentin, at the chronic caries course - also and pigments. The depth of this destruction zone is different depending on duration and caries: it is enough considerable at acute caries and less at chronic course.

2. Zone of demineralization. It is located deeper than previous zone; in it a dentin still saves its structure, but is considerably changed. Dentinal tubules are unevenly extended on comparison with normal. At caries first changes the processes of odontoblasts and not diameter of dentinal tubules. The tubules broaden unevenly forming cavities, which contain great numbers microorganisms, mainly streptococci. The same bacteria also accumulated in tubules, infected the products of disintegration odontoblasts processes and penetrated into peritubular dentin. There is considerable reduction of quantity of mineral matters revealed in the of dentine matrix.

In the demineralization zone in the direction of pulp there are revealed more frequently the areas of sound dentine with the right structures. These areas are lighter on tooth section. In these areas dentinal tubules are narrower, some of them obliterated and do not contain bacteria. Towards pulp these areas connected together in the continuous layer of so called transparent dentin.

3. Zone of transparent (sclerotic) dentin. In this area a dentin completely saves the tubular structure, but its organic matrix heavy mineralized and therefore den-tinal tubules are narrowed. Mineral salts are settled down in dentinal tubules and dentin becomes a homogeneous structure. These areas become more transparent on teeth section than surrounding hard tooth tissues.

The high mineralization of dentinal tubules diminishes a difference between the indexes of light refraction of separate structural elements of dentin - obliterated dentinal tubules and organic matrix, therefore a dentin becomes optically homogeneous. A transparent den-tine frequently meets at a chronic caries and is almost absent at acute caries. The clinical observations and experimental researches proved a transparent dentine as a protective structure which represents resistance of organism to the pathologic process - caries. A transparent dentin which appears at a chronic caries is characterized by more high micro hardiness (R. G. Sinitsin, 1970).

On comparison with normal a transparent dentine is characterized in one case by the increase, and in other - reduction of general degree of mineralization. Thus, its homogeneity does not rely on absolute contents of mineral components, and is connected only with reduction of differences between the light refraction indexes of different dentin structures.

4. Zone of the visible unchanged dentin (it can be sometimes almost absent). In it dentin has practically unchanged, dental tubules correctly oriented and contains the odontoblasts processes. The dentin organic matrix also practically unchanged and contained the usual quantity of mineral components. In those case, very strong stimuli (microflora and others), which influence on hard tooth tissues and pulp exceeds their protective possibilities; the protective layer of transparent dentine does not appear. As a result of such irritant action on the odontoblasts some tooth area; their processes in dentinal tubules are undergoing disintegration and the tubules filled by air and bacteria. On tooth section such empty tubules are black painted and known as «dead tracts». Clinically at probing of such areas there are practically complete absence of sensitiveness.

5. Zone of the secondary (reparative) dentin. As a defence reaction on caries development the cellular elements of pulp - odontoblasts — react by increasing of dentinogenesis - formation of the secondary dentin. It produced directed against a specific area in places closer to caries cavity and served as protective barrier for pulp. A new formed dentin has an enough irregular structure: there are areas which contain dentinal tubules (regular dentine) and present areas without tubules (irregu-lar dentine). Secondary dentin is more mineralized than primary dentine, and that is more resistance to caries development. Formation of the secondary dentine is the defence pulp reaction. **There is another systematic of these zones.**

Zone 1: Normal dentin. The deepest area is normal dentin, which has tubules with odontoblastic processes that are smooth, and no crystals are in the lumens. The intertubular dentin has normal cross-banded collagen and normal dense apatite crystals. No bacteria are in the tubules. Stimulation of the dentin (e.g., by osmotic gradient from applied sucrose or salt], a bur, a dragging instrument, or desiccation from heat or air), produces a sharp pain.

Zone 2: Sub transparent dentin. Next is the sub transparent layer, which is a zone of demineralization of the intertubular dentin and initial formation of very fine crystals in the tubule lumen at the advancing front. Damage to the odontoblastic process is evident; however, no bacteria are found in the zone. Stimulation of the dentin produces pain, and the dentin is capable of remineralization.

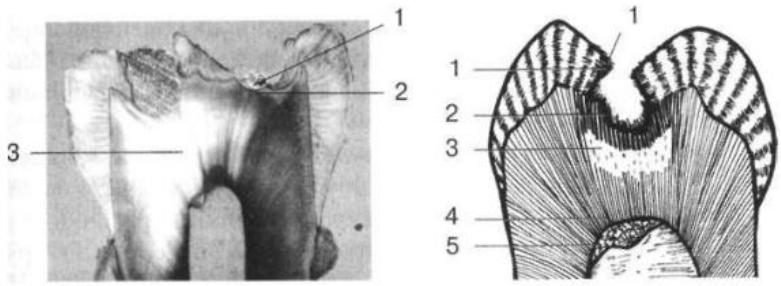
Zone 3: Transparent dentin. The transparent layer is a zone of carious dentin that is softer than normal dentin and shows further loss of mineral from the inter-tubular dentin and many large crystals in the lumen of the dentinal tubules. Stimulation of this region produces pain. No bacteria are present.

Although organic acids attack both the mineral and organic content of the dentin, the collagen cross-linking remains intact in this zone. The intact collagen can serve as a template for reminer-alization of the intertubular dentin, and thus this region remains capable of self-re-pair provided the pulp remains vital.

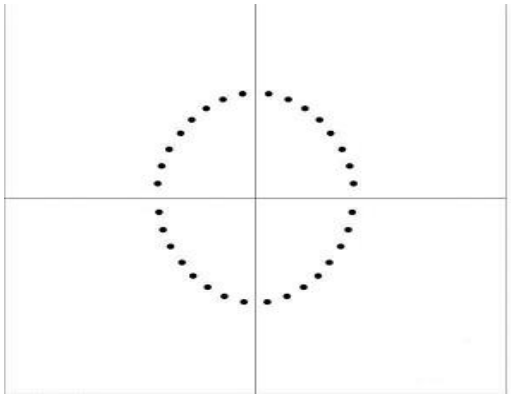
Zone 4: Turbid dentin. Turbid dentin is the zone of bacterial invasion and is marked by widening and distortion of the dentinal tubules, which are filled with

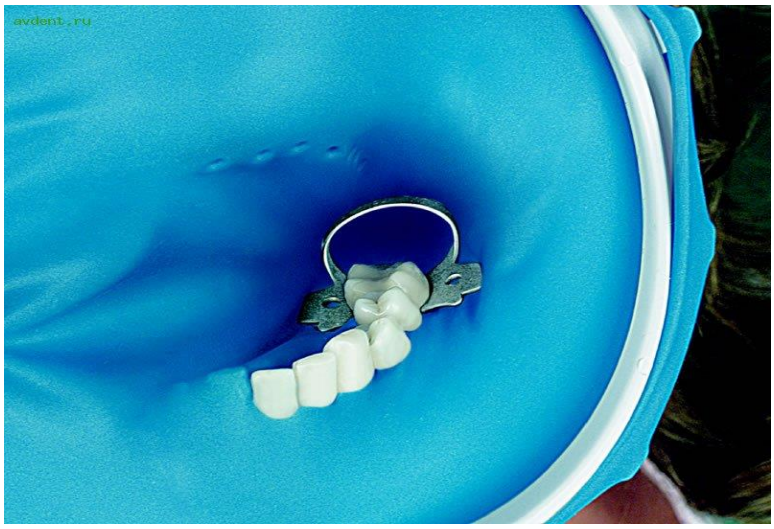
bac-teria. There is very little mineral present and the collagen in this zone is irreversibly denatured. The dentin in this zone will not self-repair. This zone cannot be re-min-eralized and must be removed before restoration.

Zone 5: Infected dentin. The outermost zone, infected dentin, consists of decomposed dentin that is teeming with bacteria. There is no recognizable structure to the dentin and collagen and mineral seem to be absent. Great numbers of bacteria are dispersed in this granular material. Removal of infected dentin is essential to sound, successful restorative procedures as well as prevention of spreading the infection. All these areas are best distinguished at the chronic course of middle caries. At acute caries it prevailed that the processes of destruction and demineralization in all dentin depth up to the pulp. Also there is not formation transparent and secondary dentin, therefore proper zones on tooth sections are absent. The substantial changes of different pulp components are revealed: the intercellular matter becomes transparent, widening distances between separate collagen fibres. Odontoblasts lose their specific structure, their cytoplasm becomes homogeneous and electron dense, which evidenced of the odontoblasts damage. Blood vessels widened, there are breaks of capillaries walls, penetration leucocytes and erythrocytes into pulp tissue. There is disorganization of odontoblastic er, destruction of nervous fibres and further development of inflammatory changes in pulp. These changes depend of the caries course: they are most expressed at acute course.

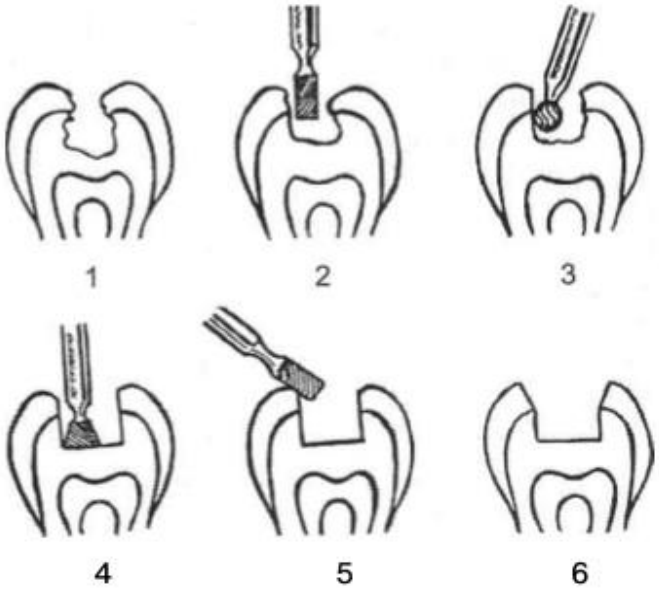


Chronic middle caries





Rubber dam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Lesson №6
TOPIC: Acute and chronic deep caries: pathomorphology, clinic, diagnosis, intra- and extra-syndromic differential diagnosis.

Deep caries (caries profunda): It is characterized by formation of caries cavity which affected almost all layers of dentine practically to pulp and located in cir-cumpulpal dentin. Patients with acute deep caries complain of causal pain which arises up because of action of thermal, mechanical, chemical irritants and disappears after their removal. Inserting into the carious cavity a cotton pellet with hot (no more than 50 degrees C) or cold water, and also either, as a rule, is accompanied by the sharp pain reaction, nevertheless, pain disappears after the removal of the irritant from the cavity. Carious cavity is located within the limits of cir-cumpulpal dentin with the overhanging margins of enamel. Enamel around the entrance of the cavity softened chalky white colour.

A caries cavity is filled with softened dentin, grey-whitish or yellow colour. At probing it revealed a painful area at the floor of the cavity and dentinoenamel junction. Frequently it is the places of projection of pulp horns, which directly react on irritants; nevertheless, perforation of carious cavity is absent. At acute deep caries, probing of caries cavity floor must be made very carefully. In the points of pulp horns projection of the dentinal wall is very thin, a dentine is softened and may be easy pierce by a probe and injures the pulp. It is accompanied by a sharp pain and the appearance in the carious cavity a drop of blood.

At the chronic deep caries the complaints on pain can be absent or an insignif-icant, brief pain after thermal, chemical and mechanical irritants is revealed. Defect of hard tooth tissues located in limits of circumpulpal dentin, large enough in size and occupies a considerable part of tooth crown. Cavity is wide opened outside (the overhanging edges of enamel are broken off because of their fragility). Hereupon the transversal sizes of cavity exceed its depth. A wall and floor of carious cavity is filled to enough dense, pigmented dentin but without sclerotic brilliance.

Pigmentation of its walls and floor has an enough wide spectrum - from yellow-brown to brown and even almost black colour. Probing of walls and floor of cavity is painless, because of development under them well expressed areas of transparent and secondary dentin. Surface of carious dentine is rough when probing and it is heavy enough to excavate. Development of such cavity lasts for years. Along with the traditional methods of examination in caries diagnostics, use of an electric method - electro testing of nervous receptors of pulp and periodontal ligament - electro odonto-diagnostic. It was revealed that intact tooth pulp and also teeth with caries reacts on strength of current within the limits of 2-6 mkA. In case of a deep caries degenerative changes in pulp are more expressed, so it reacts on 15-20 mkA. After the correct medical treatment the excitability of pulp is gradually restored.

Decline of excitability within the limits of 15-60 mkA point at the prevailing inflammatory process crown pulp, 60-100 mkA - diffuse inflammation in all (crown and root) pulp. Decline of excitability more than 100 mkA, point at death of pulp and reaction on the electric current nervous receptors of periodontal ligament.

Histopathology: The pathohistological features in hard tooth tissues are practically the same as at a middle caries. Sometimes, because of small thickness of dentinal partition between a carious cavity and pulp, not all zones of lesion can be expressed. At the acute caries course there is the predominance of the demineralization processes. Common reduction of the cellular elements number, hyperemia and edema of pulp, perivascular infiltrates, and increase nervous fibres thickness are revealed in pulp. Except of the acute and chronic caries course in a clinical conditions there is distinguished the most rampant caries - caries acutissima and arrested (stationary caries or caries stationary).

Caries acutissima often arises up in a child's age weakened by different systemic children's' diseases. In adults it can arise as a complication after surgical removing of salivary glands and because of the absence a selection of saliva, the xerostomia («dry mouth») develops. Thus the processes of remin-eralization of the hard tooth tissues by saliva are seriously violated and results in the caries development. It is characterized by the very rapid (within the limits of 2-5 weeks) development of carious lesions. The numerous lesions developed on most teeth with formation of a few cavities on the one tooth crown.

Enamel on the areas of lesion heavy demineralized, chalky white colour, a dentine is softened to cartilage consistency, its layers easily removed by excavator. The carious lesions developed very quickly from the incipience lesion to deep cavity and almost develop caries complications - inflammation of pulp (pulpitis) and periodontal ligament (apical periodontitis). Histopathology: The pathohistological features of this caries form are characterized by the predominance of the demineralization processes with the destruction of hard tooth tissues. The defence reactive zones (sclerotic, secondary dentin) are practically absent. The clinical variant of such caries course is known as caries florida.

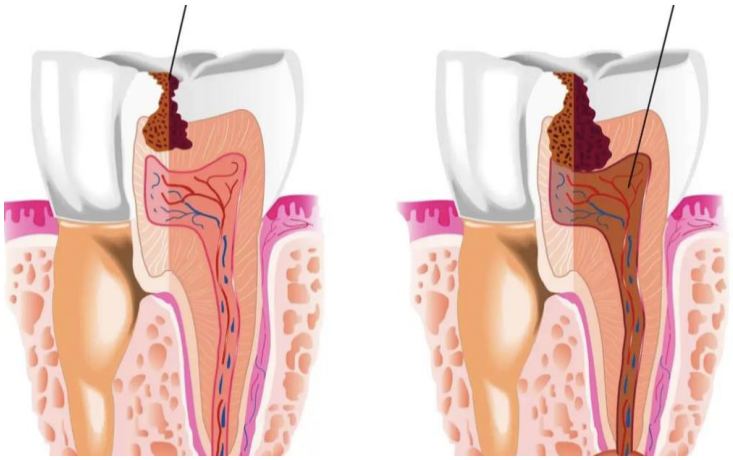
Arrested (stationary) caries or halted caries is considered as variant of chronic caries development. At favorable conditions and sufficient organism resistance the caries development stopped and carious cavity does not spread deep into hard tooth tissues. In hard tooth tissues this caries form may be consider as an original «scar», that indicates on the caries process, because of the hard tooth tissues are not regenerated. In case of incipient caries it is possible to speak about demineralization affected hard tooth tissues.

A white carious spot can fully disappeared; brown (pig-mented) carious spots because of the expressed pigmentation are remained pigmented on enamel even after stopping of the caries process. Initial and the subsequent stages of arrested caries development are characterized by the intensive (dark-brown)

dentine colour in the lesion. The pain feeling are fully absent because of high enamel and dentine mineralization in carious cavity. The affected hard tooth tissues had practically the same or and even more high degree of mineralization of hard tooth tissues. In some cases protective mineralization is so expressed, that a dentin becomes glassy and had considerable barrier properties. Even in a deep carious cavity is not feeling of pain in action of any (chemical, mechanical, thermal and that similar) irritants. The high level of mineralization of the affected hard tooth tissues results in such a state, that when because of unfavorable conditions begin, there is further caries progress so new lesions appear on other surfaces of the tooth and not at the place of high mineralized arrested (stationary) caries.

A stationary caries is frequently the result of the original development of chronic caries. The defects of hard tooth tissues have the wide entrance opening, which exposed a saucer like form. At very deep carious cavities their floor can already be within limits of the secondary dentine, that it is placed below than level of vault pulp chambers. It is explained by very slow development of caries, which destroyed not only primary dentine and spread within the limits of the secondary dentine.

Because of such chronic caries course the pulp has enough time to form the considerable protective layer of the secondary dentin. There is not the perforation of pulp cham-ber, infection of pulp and pulpitis development. The caries is characterized by causal pain, which arises only because of the action of different (chemical, thermal, mechanical) irritants and disappears at once after stopping of irritant action. Unlike pulpitis pain it is localized and never is there an irradiation of pain into other places of maxillary-facial regions. The disease duration in case of acute course can be within the limits of a few months, while at chronic - months and for years.



METHODOLOGICAL RESEARCH №7
TOPIC: One-session and two-session methods of treatment of acute deep caries. Therapeutic pastes: groups, properties, methods of use.

Medical treatment of caries consists of a row of measures of both general and local character depending on the stage of development of pathological process and character of its course.

When a pathological process spreads to enamelodentinal junction, strikes a dentine and appears carious cavity, conservative (remineralization) therapy can not result in success. It is connected with such condition that hard tooth tissues do not possess property to regenerate the primary form in the area of caries lesion. Therefore for local medical treatment of carious cavities their preparation is used, with the subsequent restoration of cavity and renewal of anatomic form of tooth by filling material. How justly mark B. Helvig, Y. Klimek, T. Attin 1999) it is presently observed the tendency to refuse of principle «only restoration effectively protects from caries» in favour to primary atraumatic restorative treatment or minimal invasive treatment with the subsequent control of surfaces affected by caries. Neither of the varieties of restoration treatment of dental caries may be fully «curing». Destroyed by a caries hard tooth tissues (and adjoining areas of healthy enamel) are not substituted for by formed again enamel and dentin. Besides, there is no existing restorative material capable during all life to protect hard tooth tissues from further destructive caries processes. Tooth restoration is only symptomatic treatment which does not eliminate the etiologic factors of dental caries.

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Destroyed by a caries hard tooth tissues (and adjoining areas of healthy enamel) are not substituted for by formed again enamel and dentin. Besides, there is no existing restorative material capable during all life to protect hard tooth tissues from further destructive caries processes. Tooth restoration is only symptomatic treatment which does not eliminate the etiologic factors of dental caries. Therefore the prevention of development caries lesions (prophylactic measures) is the basic principle of caries treatment, rather than the necessary medical treatment (remineralization therapy) and, in the last turn, as the forced measure of restoration the caries cavity with restorative materials, conducted along with the measures of the second caries prophylaxis.

Thus, now there are two main methods of local caries treatment: 1) caries treatment without preparation and restoration - remineralization therapy, and 2) operative caries treatment by the operative preparation of demineralized hard tooth tissues with the subsequent restoration of carious cavity. The choice of treatment method depends of the stage of caries development, activity of caries (rampant or chronic), localization of carious cavity, age and general condition of patient.

Preventive treatment methods are designed to limit tooth demineralization caused by cariogenic bacteria, thereby preventing cavitated lesions.

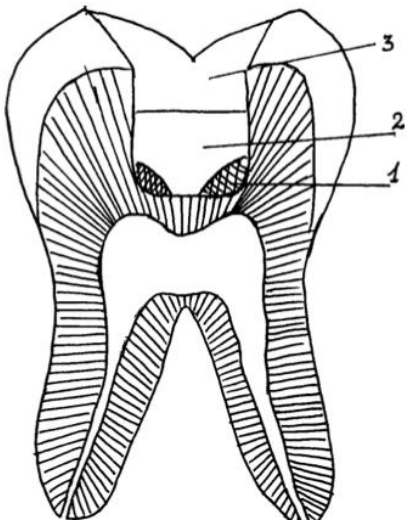
They include:

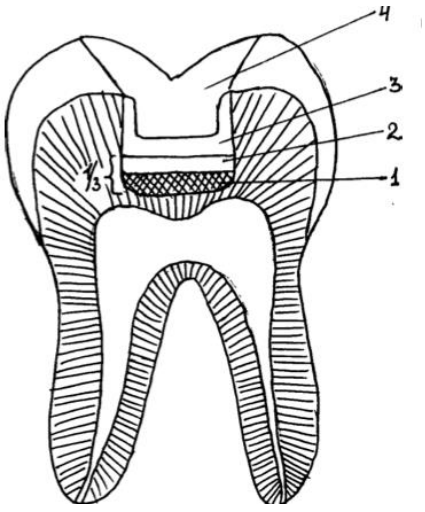
(1) limiting pathogen growth and metabolism

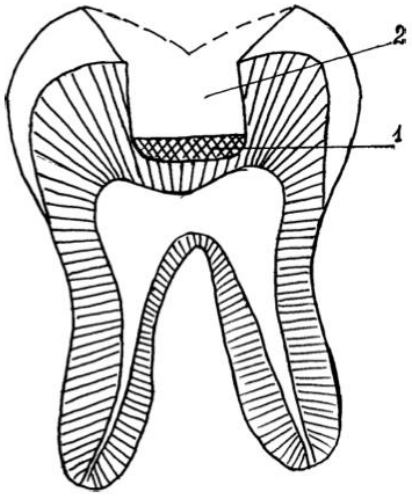
(2) increasing the resistance of the tooth surface to demineralization. Disease caries control concerns influencing biofilm formation and growth, or modifying the dissolution kinetics of the apatites, or both.

The following may have a role to play:

- mechanical/chemical removal of plaque (oral hygiene)
- chemical (antimicrobial) modification of plaque
- use of fluorides
- dietary composition
- salivary composition and stimulation.



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Lesson № 8
TOPIC: Prevention of caries. The importance of individual and social prevention. Means of prevention. Organisation of prevention of dental caries in pregnant women, conscripts, workers in certain industries. Evaluation of effectiveness.

The prevention of development caries lesions (prophylactic measures) is the basic principle of caries treatment, rather than the necessary medical treatment (remineralization therapy) and, in the last turn, as the forced measure of restoration the caries cavity with restorative materials, conducted along with the measures of the second caries prophylaxis.

Similar properties of hard tooth tissues, and also property of enamel in the physiological condition for mineralization after of teeth eruption (maturation of enam-el) were served as pre-conditions for creation of artificial sources of addition to hard tooth tissues macro - and microelements with the purpose of medical treatment and prophylaxis of caries. On this basis were developed different methods of patho-genetic medical treatment of early stages of tooth (incipience caries) by remineral-ization its hard tissues.

Preventive treatment methods are designed to limit tooth demineralization caused by cariogenic bacteria, thereby preventing cavitated lesions.

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(1) limiting pathogen growth and metabolism

(2) increasing the resistance of the tooth surface to demineralization. Disease caries control concerns influencing biofilm formation and growth, or modifying the dissolution kinetics of the apatites, or both.

The following may have a role to play:

• mechanical/chemical removal of plaque (oral hygiene)
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• use of fluorides
• dietary composition
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Pit-and-fissure sealants provide a safe and effective method of preventing caries. Sealants are most effective in children when they are applied to the pits and fissures of permanent posterior teeth immediately upon eruption of the clinical crowns. Adults also can benefit from the use of sealants if the individual experiences a change in caries susceptibility because of a change in their diet or medical condition.

Fluoride treatment is capable of rendering tooth surfaces more acid resistant and in some circumstances also may arrest active caries. Sealants were designed as a preventive measure, yet studies have shown that deliberately

sealing active carious lesions effectively arrests the caries progress by cutting off the nutrient supply to the pathogenic plaque trapped under the sealant. Pits and fissures typically result from an incomplete coalescence of enamel and are particularly prone to caries.

These areas are insufficiently mineralized (as compared with cusps) after tooth eruption. Pits and fissures are the retentive places for accumulation food remnants which can lead to caries development. The fissures sealing hinder these retention and demineralization of enamel by means of microorganism's acid. From the other hand during treatment of incipience.

Regardless of age, caries risk of an individual should be the major factor for selecting teeth for sealant application. Sealants may be indicated for either preventive or therapeutic uses, depending on the patient's caries risk, tooth morphology, or presence of incipient enamel caries. Clinical studies also show that sealants can be applied even over small, cavi-tated lesions, with no subsequent progression of caries. However, it is recommended that sealants be used for the prevention of caries rather than for the treatment of existing carious lesions. Therefore a recent bitewing radiograph should be made and evaluated before sealant placement, to ensure no dentinal caries is evident.

Only caries-free pits and fissures or incipient lesions in enamel not extending to the den-tinoenamel junction (DEJ) currently are recommended for treatment with pit-and-fissure sealants. Clinical Technique: Because materials and techniques vary, it is important to follow the manufacturer's instructions for the sealant material being used. A standard method for applying sealants to posterior teeth is presented. The tooth is isolated by a rubber dam (or cotton rolls). The isolation of the area is critical to the success of the sealant.

Because sealant placement in younger patients is more common, the molar teeth are often not fully erupted, and therefore isolation is difficult. If proper isolation cannot be obtained, the bond of the sealant material to the occlusal surface will be compromised, resulting in either loss of the sealant or recurrent caries under the seal-ant. The area is cleaned with slurry of pumice on a bristle brush. Bristles reach into faulty areas better than a rubber prophy cup, which tends to burnish debris and pumice into the pits and fissures. The tooth is rinsed thoroughly while the explorer tip is used carefully to help remove residual pumice or additional debris. After the area is dried, a liquid acid etchant (35% to 50% phosphoric acid) may be placed on the occlusal surface with a small sponge, brush, or applicator tip for 30 seconds.

Gel etchants, traditionally used for most restorative procedures, may have less ability to effectively penetrate into the pits and fissures. Next, the tooth is rinsed with water for 20 seconds while the area is evacuated, and then dried of all visible moisture. The properly acid-etched enamel surface has a lightly frosted appearance. Fluoride-rich, resistant enamel may need to be etched lon-ger. Any brown stains that originally may have been in the pits/fissures may still be present and should be allowed to remain. The self-cured sealant is mixed and applied with

a small applicator provided in the sealant kit. The sealant is gently teased place, to avoid entrapping air, and it should slightly overfill all pits and fissures. Some operators prefer light-cured sealants, which also work well. After polymerization of the sealant, the rubber dam is removed, and the occlusion is evaluated using articulating paper.

If necessary, a round 12-bladed carbide finishing bur or white stone is used to remove the excess. The surface usually does not require further polishing. Deep penetration into dentin of components forming the composite adhesive system and reliable hermetic sealing of dentinal tubules served by foundation for the empiric use of the adhesive systems at medical treatment of the hyperesthesia of enamel and dentine. Besides the diminishing of the hyperesthesia this preparation protect hard tooth tissues from the abrasion. The manufacturer proposed special preparation - «Seal&Protect» («Dentsply»). It is the mixture of methyl methacrylate (MMA) monomers an acetone basis, contains nano fillers and antibacterial remedy — triclosan.

Lesson №9

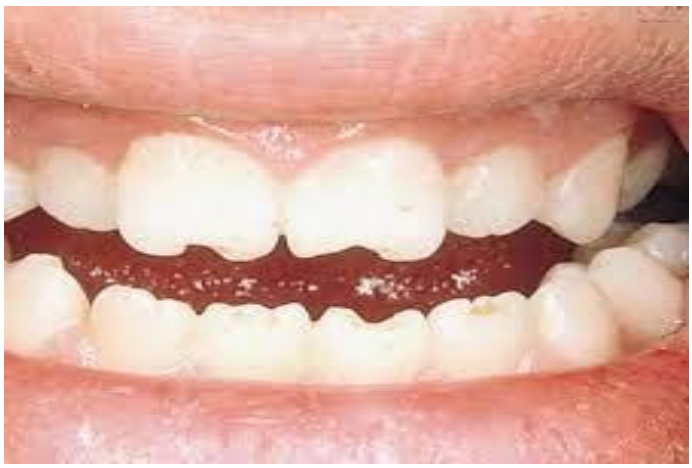
TOPIC: Non-carious lesions of the teeth.

Classification, pathomorphology, clinic and diagnostics of non-carious lesions occurring before the eruption of teeth: hypo-, hyperplasia, endemic fluorosis. Classification of dental fluorosis according to A.K. Nikolishin.

Non-carious lesions of the teeth

In addition to caries, the most common dental disease, there is another pathology of hard tissues - non-carious lesions of the teeth.

Non-carious tissue lesions occur without softening of tissues and without the participation of microorganisms. At the heart of these processes is a violation of the mineralization of the hard tissues of the teeth under the influence of external or internal factors. These pathologies occur in approximately 25% of patients, but seek their help for dental care no more than 5%, because non-carious lesions of the teeth, as a rule, do not cause pain or other subjective sensations, and often only worsen the appearance. However, if left untreated, a number of complications can occur, including early tooth loss.



Dental enamel hypoplasia is an enamel defect characterized by thin or absent enamel. In some cases, the defect occurs on only part of a tooth's surface, resulting in pits or grooves in the tooth's enamel. In other cases, an entire tooth may have an overly thin layer of dental enamel or may have no enamel at all.

Enamel hypoplasia is a *developmental* enamel defect, meaning that is already present at the time the affected tooth first erupts from the gums. In contrast, enamel *wear*, such as dental abrasion and erosion, occurs after a tooth has erupted.

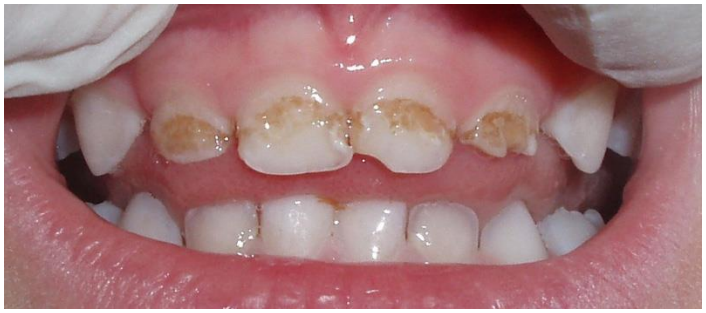
Enamel hypoplasia occurs when the special cells that produce dental enamel are disturbed during a particular stage of enamel formation (the matrix

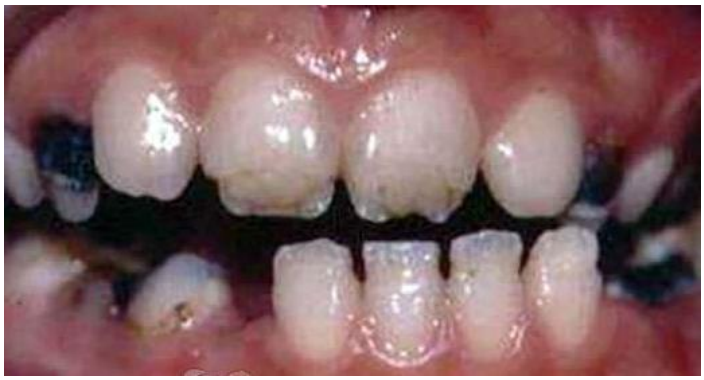
formation stage). A wide variety of factors can potentially cause such a disturbance, including both genetic and environmental factors.

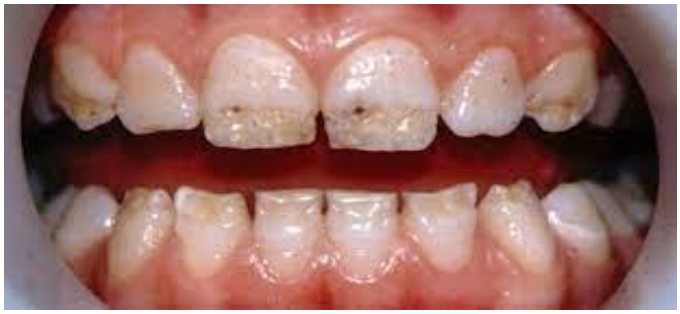
The hereditary factors that lead to enamel hypoplasia in children consist primarily of relatively rare genetic disorders, such as amelogenesis imperfecta and Ellis van-Creveld syndrome.

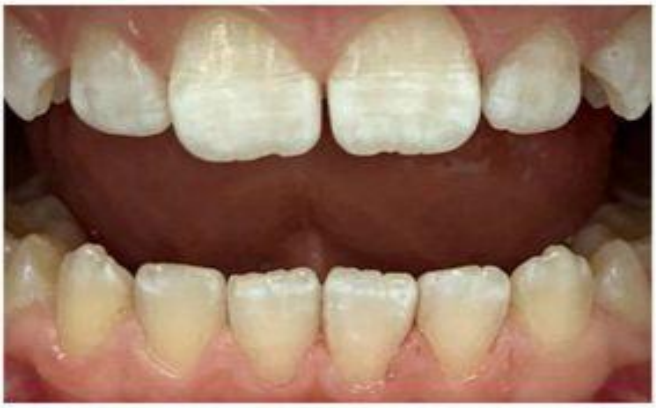
Current research suggests that environmental factors that may increase the risk of enamel hypoplasia in children include the following:

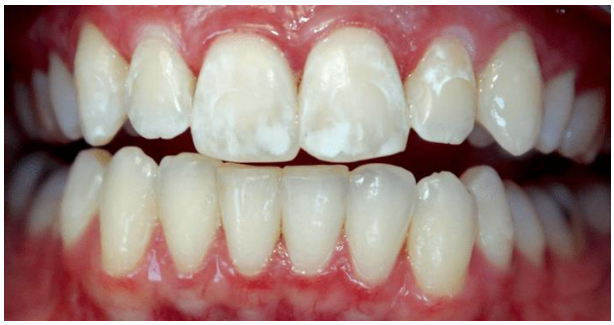
- Premature birth
- Low birth weight
- Malnutrition, including vitamin D deficiency rickets
- Hypoparathyroidism
- Diabetes
- Gestational diabetes in the child's mother
- Viral and bacterial infections, including congenital syphilis
- Inflammation
- Dental trauma
- Ingestion of large amounts of fluoride

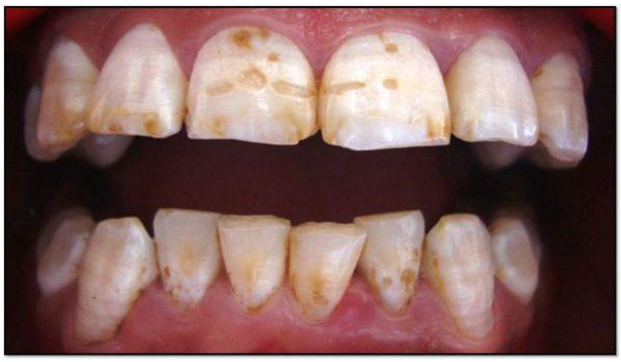


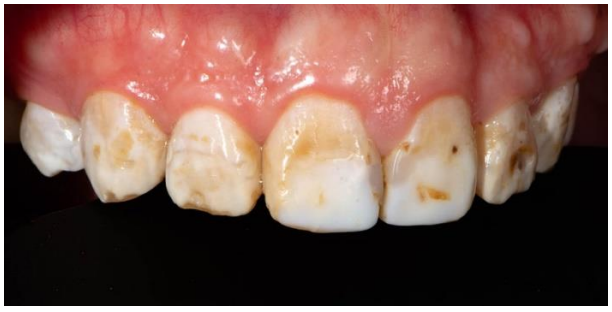


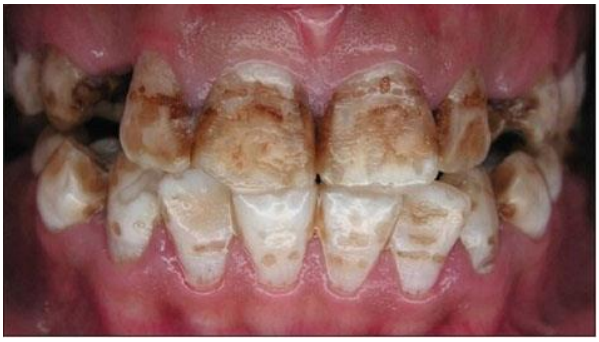












Fluorosis in different forms

Lesson №10

TOPIC: Non-carious lesions of the teeth occurring after eruption. Pathomorphology, clinic, diagnosis and treatment of enamel erosion, wedge-shaped defects. Traumatic and chemical damage. Hyperesthesia of the hard tissues of the teeth. Elimination of hyperesthesia with the help of modern desensitizers: composition, properties, methods of use

Symptoms of tooth enamel erosion can vary. They often include:

- increased sensitivity to taste, textures, and temperature
- cracks and chips
- discoloration
- indentations known as cups on the surface of your teeth

Patient may have significant enamel erosion if they experience:

- pain
- high sensitivity when exposed to cold, hot, acidic, and spicy food and drink
- discoloration in teeth

Over time, enamel erosion can lead to complications, such as:

- yellow, stained teeth
- overly sensitive teeth
- rough edges on teeth
- shiny spots on teeth
- increased tooth decay
- gradual wearing of enamel, leading to clear, slightly translucent teeth
- fractured teeth

Causes of enamel erosion

One of the main causes of enamel erosion are acids found in the foods and liquids patient consumes. Saliva constantly neutralizes acid in a mouth to protect teeth.

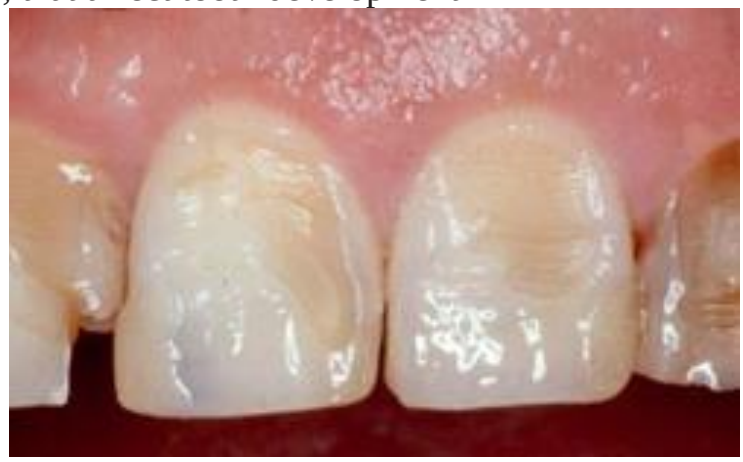
Enamel erosion can be caused by what patients eat, particularly:

- sugary foods, such as ice cream, syrups, and caramel
- starchy foods, such as white breads
- acidic foods, such as apples, citrus fruits, berries, and rhubarb
- fruit drinks and juices
- sodas, which typically contain damaging citric acid and phosphoric acid in addition to sugar
- excess vitamin C, found in citrus fruits

Other causes of enamel erosion include:

- teeth grinding
- chronic acid reflux, also known as gastroesophageal reflux disease (GERD)
- low salivary flow, also known as xerostomia, which is a symptom of conditions like diabetes

- regular use of certain medications, such as antihistamines and aspirin
- eating disorders like bulimia, which disrupts the digestive system and exposes teeth to stomach acid
- genetic disorders, including amelogenesis imperfecta or enamel hypoplasia, that affect tooth development

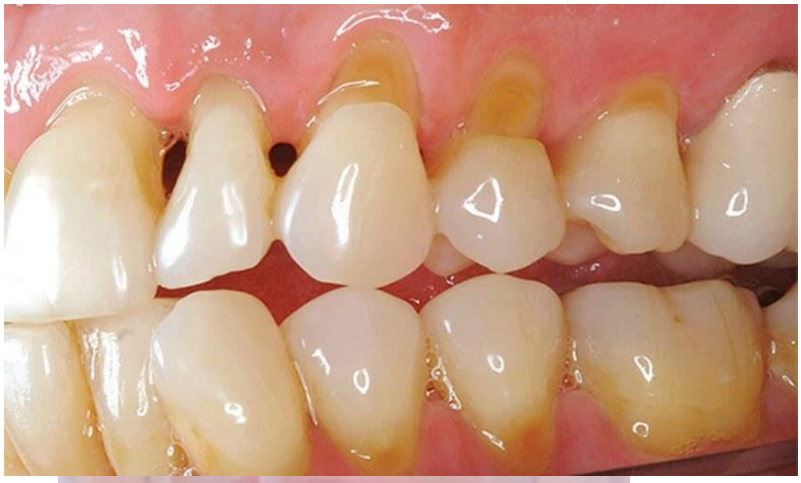


Wedge-shaped defects - a type of damage to dental tissues located near the walls of the teeth, on the cheek and lip surfaces. The defect is wedge-shaped with the base to the neck of the tooth and the tip to the cutting edge or chewing surface. Wedge-shaped defect, as a rule, does not bother the patient: pain is rare (only briefly from thermal and chemical stimuli), the tooth cavity is not affected and does not open, the defects slowly deepen, softening is not defined (this defect is different from caries) .

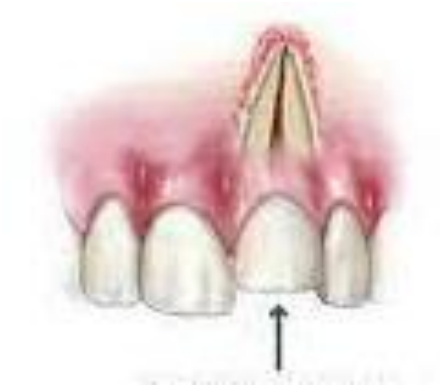
The causes of the wedge-shaped defect are not fully established. There is a view that it occurs under the influence of uneven load on the teeth, due to malocclusion.

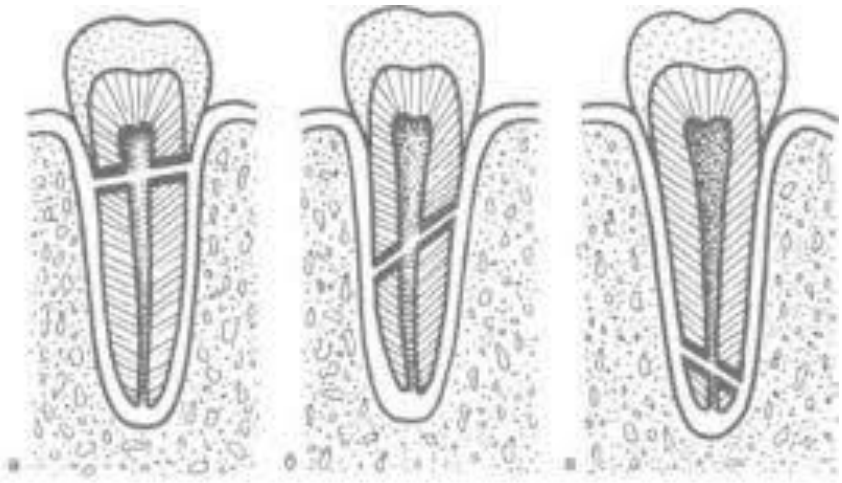
It is sometimes believed that because the wedge-shaped defect begins after the exposure of the tooth wall, it is one of the manifestations of periodontal disease. There is evidence of the role of endocrine disorders, diseases of the central nervous system and gastrointestinal tract in the occurrence of a wedge-shaped defect.

Treatment of wedge-shaped defect is aimed at strengthening the hard tissues of the teeth through the use of remineralizing therapy (application of calcium, phosphorus, fluoride, fluoride varnish, fluorogel, etc.).



Dental trauma refers to trauma (injury) to the teeth and/or periodontium (gums, periodontal ligament, alveolar bone), and nearby soft tissues such as the lips, tongue, etc. The study of dental trauma is called dental traumatology.





Dental injuries

Dental injuries include:

- Enamel infraction
- Enamel fracture
- Enamel-dentine fracture
- Enamel-dentine fracture involving pulp exposure
- Root fracture of tooth

p.

Lesson № 11

TOPIC: Errors and complications in the diagnosis and treatment of caries. Secondary caries of depulpated and non-depulpated teeth: cause, clinic, diagnosis, differential diagnosis. Treatment and prevention.

During treatment of dental caries, doctor performs a variety of manipulations, not very thorough or improper performance of which can lead to some kind of complications.These errors can occur both during the actual surgical treatment, preparation of carious cavities and on the stages of carious cavity filling and at a different times after sealing. It is therefore advisable to divide them into complications arising during the preparation of carious cavities and during filling of carious cavity, and the complications that arise after treatment of caries.

Errors and complications arising during carious cavity preparation are:

1. Insufficient carious cavity preparation may lead to secondary caries, thus progressing of caries process and possible development of pulpitis or filling loss.
2. Perforation of the carious cavity bottom or carious cavity wall and fracture of the carious cavity wall may happen due to not proper fixed hand of clinical thus leading to such complications. Perforation of carious cavity floor may happen in the case of acute deep dental caries, when bottom is softened and thin layer of demineralised dentine separates carious cavity from tooth cavity.
3. Injury of adjacent tooth crown by bur may happen when visible control of operative field is not provided.
4. Injury of gingival margin by bur may happen during preparation of carious cavities that goes deep under the gums or good vision of operative field was not provided.

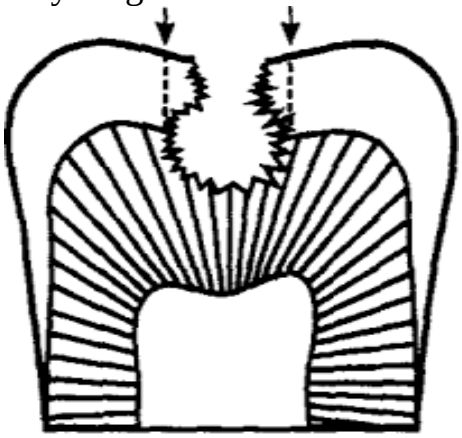
Errors and complications arising during carious cavity filing are:

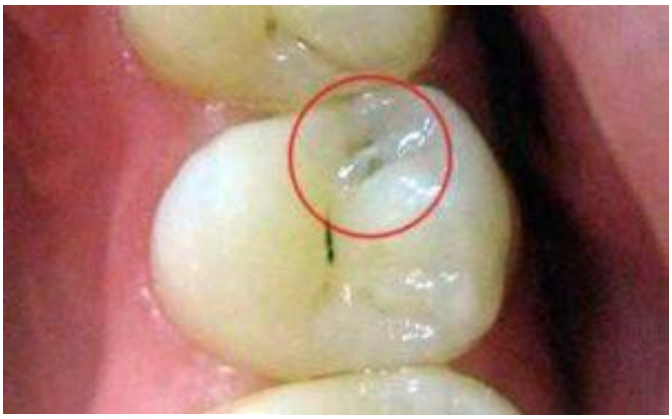
1. Absence of a contact point, hanging edges of a filling and placement of a single filling in adjacent carious cavities will lead to inflammation papilla, thus causing pain to the patient and development of periodontal diseases. That is why during restoration of proximal cavities it is necessary to use matrix holder and matrices in order to restore contact point, thus preventing these complications.
2. Formation of high occlussion usually happen when filling is not adjusted to the bite , when high spots are left, this will lead to development of apical periodontitis in future, such tooth will change its color to grey shades and will be painful while biting.

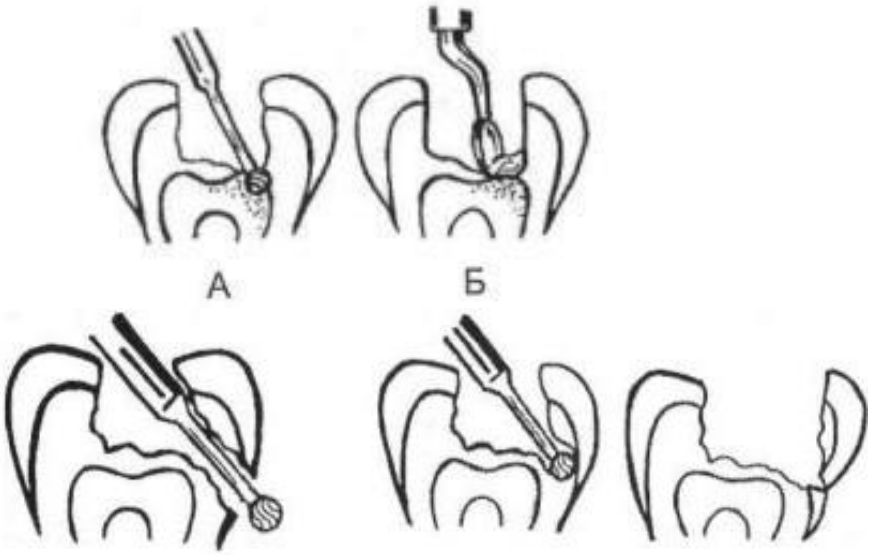
Errors and complications arising after dental caries treatment are:

1. Inflammation or necrosis of the pulp.
2. Inflammation of an intradental papila or papilitis.
3. Acute or chronic course of an apical periodontitis.
4. Color change of the tooth crown.
5. Displacement, fracture and loss of filling.
6. Inadequate color of filling to the color of tooth anamel.

The main task of therapeutic dentistry is to prevent dental caries and its complications – pulpitis, periodontitis, papilitis. Knowledge of possible mistakes and errors during carious cavity preparation and filling material placement will prevent young clinician from the complications arising as the result of mistakes.







Lesson №12

TOPIC: Writing an extended card for an outpatient with various forms of caries and non-carious lesions.

Instructions

for filling in the form of primary accounting documentation No. 043/o "Medical record of a dental patient"

1. This Instruction defines the procedure for filling out the primary record form No. 043/o "Medical record of a dental patient" (hereinafter referred to as form No. 043/o).

2. Form No. 043/o is filled in by responsible persons of outpatient health care institutions that provide dental care to the population: dental clinics, dental departments and offices of outpatient clinics, polyclinics, hospitals, dispensaries, research institutes, higher education institutions of III-IV accreditation levels, hospitals for disabled veterans, women's clinics, health care facilities regardless of subordination and form of ownership.

3. The patient's passport data (surname, name, patronymic, gender, place of residence, year of birth) is filled in by a nurse or registrar.

4. The diagnosis and other sections of form No 043/o are filled in directly by the attending physician. Depending on the complaints and the initial clinical diagnosis, the doctor must refer the patient for laboratory tests, X-rays, and the conclusions of specialists of the relevant profile, including general somatic ones, with the submission of an extract from the dental patient's card, tests and other medical documentation. The said documentation shall be entered or pasted into the form No. 043/o in the section "Data of X-ray examinations, laboratory tests".

4.1 Further clarification of the diagnosis, expansion or even replacement is allowed with the obligatory indication of the date. The diagnosis should be detailed, only dental diseases should be described.

5. In line 6, "Complaints", the patient or relatives should write down the complaints that most accurately reflect the patient's condition in relation to the dental disease. 6. In line 7 "Past and concomitant diseases", data on past and concomitant diseases shall be indicated according to the patient's words, as well as data confirmed by

by specialists from other departments of the healthcare facility. It is necessary to indicate whether the patient is registered with a dispensary and for what disease.

7. In line 8 "Development of the current disease" indicate: the time of the first symptoms of the disease, what the patient associates them with, the nature of the disease and previous treatment and its effectiveness.

8. In line 9 "Objective examination data, external examination, dental condition", the external examination data is described, indicating the condition of the skin, bone skeleton of the face, red lip border, etc. This line indicates the results of palpation of the temporomandibular joint, submandibular, parotid salivary glands. Recording of oral cavity examination data begins with determination of the condition of the hard tissues of the teeth and periodontal tissue. The first row

above and below the teeth arranged schematically is reserved for entering data on the condition of the crown part of the tooth with conventional designations, including the presence of various denture designs.
8.1. The numerator indicates the state at the time of the examination, the denominator - the state after treatment. Above and below the second row of schematically depicted teeth, the data of the objective examination of the periodontal state, its norm (N), the degree of atrophy - 1/4, 1/2, 3/4 and the degree of tooth mobility - I, II, III are entered.
8.2. Under the table of schematically arranged teeth, additional data on the teeth, bone tissues of the alveolar processes (changes in their shape, position, etc.) are reflected in writing.
9. In line 10 "Occlusion", the type of relationship of the dentition in the normal, abnormal, pathological state, as well as the nature of the relationship of the alveolar processes of the jaws in the absence of antagonistic teeth or their complete absence, focusing on their relationship in a state of relative rest, is noted.
10. In line 11, "State of oral hygiene, condition of the oral mucosa, gums, alveolar processes and palate. GI and PMA indices" describes the state of the oral mucosa according to visual examination, hygiene index (hereinafter referred to as GI) and papillary marginal alveolar index (hereinafter referred to as PMA), which is an indicator for assessing the manifestations of gingivitis and periodontal index (PI), aimed at detecting advanced forms of pathology.
11. In line 12 "Data of X-ray examinations, laboratory tests", the conclusions of X-ray examinations and laboratory tests should be indicated.
12. In line 13 "Colour according to the Vita scale" indicate the correspondence of the colour range of the applied material to the colour of the patient's dental crowns.
13. In line 14 "Date of training in oral hygiene skills", indicate the date when a conversation was held on proper toothbrushing and other oral hygiene skills.
14. In line 15, "Date of oral hygiene control", enter the date after the assessment of the hygienic state of the oral cavity.
15. Section 16, "Doctor's diary," indicates all cases of patient visits to the doctor, draws up an examination plan, a patient treatment plan with notes on the consultative opinions of related specialists. It is completed with an epicrisis, a brief description of the results of treatment and practical measures recommended by the doctor. After the treatment, the doctor who performed the treatment and the head of the department sign the epicrisis, and after the completion of certain stages of treatment, the doctor who directly treats the patient signs it.
16. In a dental polyclinic, department or office, there shall be one form No. 043/o per patient.
17. Form No. 043/o is signed and dated by the physician who is in charge of the patient's dispensary supervision.
18. If Form No 043/o is kept in electronic format, it must include all the information contained in the approved paper form.

19. Form No 043/o shall be kept in the registry of the health care facility. 20. The storage period of Form No 043/o is 5 years.

METHODOLOGICAL RESEARCH №1

TOPIC: PULPITIS. ETIOLOGY, PATHOGENESIS, CLASSIFICATION (Y.M. GOFUNG, KMI (1964), ICD-10), THEIR POSITIVE FEATURES AND DISADVANTAGES. ACUTE TRAUMATIC PULPITIS: PATHOMORPHOLOGY, CLINIC, DIAGNOSTICS

In the 1960s a number of investigations revealed a lack of a correlation between clinical signs and symptoms and the actual histological status of the pulp. Since the histological diagnosis of a pulp is impossible to determine without removing it and submitting it for histological examination, a clinical classification system was devel-oped. This system was based on the patient's symptoms and the results of clinical tests. A clinical classification of this sort is not meant to list every possible variation of inflammation, ulceration, proliferation, calcification, degeneration of the pulp, or attachment apparatus (M. H. Smulson, 1974; S. Cohen, R.C. Burns, 2002).

Clinical classification:
Pulpal disease

1. Within normal limits
2. Reversible pulpitis.
3. Irreversible pulpitis
4. Asymptomatic irreversible pulpitis
5. Hyperplastic pulpitis
6. Internal resorption
7. Symptomatic irreversible pulpitis
8. Necrosis Periapical disease

- Acute apical periodontitis
- Acute periradicular abscess
- Chronic apical periodontitis

Pulp Pain: Pulpalgia, that is, pulp pain, can be classified into three different categories: hyper reactive, acute, and chronic.

Histologically, pulpitis, which leads to pulpalgia, is classified as reversible or irreversible. One hopes that pulp with reversible pulpitis can be saved. Pulps suffering irreversible pulpitis cannot be saved. The vital teeth can have one of the following presentations: Normal: The teeth are asymptomatic with no objective pathosis.

Reversible pulpitis: There is a reversible sensitivity to cold and/or osmotic changes (i.e., sweet, salty, and sour).

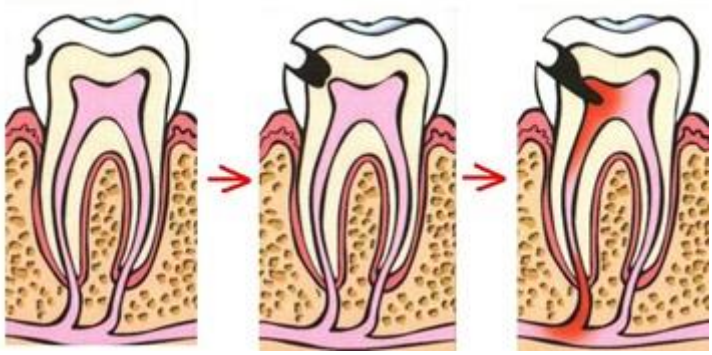
Irreversible pulpitis: The sensitivity to temperature changes is more intense and with a longer duration. International classification pulp diseases (WHO, 1998):

1.1. Pulpal: - abscess - polyp
1.2. Pulpitis: - acute - chronic (hypertrophic, ulcerative) - purulent.
1.3. Pulp necrosis. Pulp gangrene.
1.4. Pulp degeneration Denticles.
1.5. Pulpal: - petrificates, - stones.
1.6. Anomal formation hard tissue in pulp. Secondary or irregular dentin.

In former Soviet Union one of widespread classifications was the classification by Y. M. Gofung. It supposed that the base of different clinical features of pulpitis was pathologic process of inflammation. Clinical features correspond at different stages of inflammation from acute to chronic course. All form of pulpitis was divided in to two groups:

I. Acute pulpitis: 1) partial; 2) total; 3) purulent.

II. Chronic pulpitis: 1) simple; 2) hypertrophic; 3) gangrenous. On the basis of this classification it developed a classification for the National medical university (former Kiev medical institute).
There are distinguishes in the next pulpitis forms:
I. Acute pulpitis (Inflammatio pulpae acuta):
1) Pulp hyperemia (hyperemia pulpae);
2) Acute circumscription pulpitis (Pulpitis acuta circumscripta);
3) Acute diffusion pulpitis (Pulpitis acuta diffusa)
4) Acute purulent pulpitis (Pulpitis acuta purulenta)
5) Acute traumatic pulpitis (Pulpitis acuta traumatica)
II. Chronic pulpitis (Inflammatio pulpae chronica):
1) Chronic fibrous pulpitis (Pulpitis chronica fibrosa);
2) Chronic hypertrophic pulpitis (Pulpitis chronica hypertrophica);
3) Chronic gangrenous pulpitis (Pulpitis chronica gangrenosa);
4) Chronic concremental pulpitis (Pulpitis chronica concrementosa)
III. Exacerbative chronic pulpitis (Pulpitis chronica exacerbata).
IV. Pulpitis complicated apical periodontitis (Pulpitis complicatus periodontitis).
Clinical features.
Chief complaints and chief clinical features: The main characteristic symptom of acute pulp inflammation is spontaneous (i.e. unprovoked), intermittent, or continuous paroxysms of pain. Sudden temperature changes (usually cold) elicit prolonged episodes of pain (i.e. pain that lingers after the thermal stimulus is removed). The pain attack arises up suddenly, regardless of external irritants, sometimes it is provoked by chemical, thermal and mechanical irritants. It is characterized by the spontaneous pain and development of acute pain attack, referred from one arch to the other and along the branches of n. trigeminus.



Pic.1. Schematic image of pulpitis

The pain differs from that of a hyper reactive pulp in that it is not just a short, uncomfortable sensation but an extended pain. Moreover, the pain does not necessarily resolve when the irritant is removed, but the tooth may go on aching for minutes or hours, or days for that matter. Pain may start spontaneously from such a simple act as lying down. This alone accounts for the seeming prevalence of toothache at night.
Some patients report that the pulp aches each evening, when they are tired. Others say that leaning over to tie a shoe or going up or down stairs - any act that raises the cephalic blood pressure - will start the pain. The list of inciting irritants would not be complete without mentioning hot food or drink, sucking on the cavity, and biting food into the cavity. Most pain, however, is started by eating, usually something cold. The patient can tell which side is involved and frequently whether pain is in the maxilla or the mandible. This may not be absolute, however, for the pain may be referred from one arch to the other.

Patients have reported with aching of a maxillary molar when the maxillary lateral incisor has been found to be the offender. The patient may insist that a mandibular molar is aching, whereas examination reveals that a maxillary molar is the offender. Characteristically, that pain lingers after the external irritants is removed. Usually there is the presence of tooth affected with caries process. Character, duration and intensity of pain also depend of the common state of organism, levels of organism resistance and the state of the nervous system of patient. Pain attack arising up spontaneously without a visible reason is the characteristic sign of sharp pulp inflammation. Duration of pain attack depend of irritant force, prevalence and character of inflammatory process in pulp. At pulp inflammation the pain always arises as pain attacks with short painless intervals. A pain attack may be short duration with long painless intervals or long-term with short intermission peri-od. The pain character may be different duration, intensive, pulsatile, unbearable. The pain character depends on pulp area affected by inflammation: the more diffuse inflammation the more duration of pain attack.

These characteristic features are the basis for clinical classification of pulpitis. Clinically, therefore, pulpal pathosis in most instances is diagnosed during routine dental examinations and not as a result of episodes of symptomatic pulpitis. Thus, it is not possible to determine the type and severity of pulpal damage by the absence or presence of clinical symptoms. Histopathological and clinical nomenclature has, therefore, been combined, resulting in a number of descriptions and diag-noses. The diagnosis acute serous pulpitis, for example, has traditionally meant that the patient had pain (acute), that the pulp is inflamed (pulpitis), and that as yet no abscesses have formed in the pulp (serous).

Clinically, it has been assumed that this condition is present when a tooth is especially sensitive to cold and when the pain persists for some time after the cold stimulus is removed. Similarly, acute suppurative pulpitis meant that an abscess has formed in the pulp. Clinically, heat would supposedly increase the pain in a tooth with this diagnosis and cold would cause relief. Four features of pulpal pain are especially important: the intensity of the pain, its duration, whether it occurs after stimulation (provoked) or spontaneously (unpro-voked), and whether it occurs repeatedly. Thus, severe, irreversible inflammation should be suspected when a patient has intense and continuous pain. Similarly, spontaneous pain usually indicates the presence of severe and irreversible pulp pathosis. Anamnestic information about repeated attacks of pain over a long period of time will also give reason to suspect serious pulp damage.

Pic.2. Trauma of the tooth (crown fracture)

Acute traumatic pulpitis (Pulpitis acuta traumatica): At acute traumatic pulpitis depending on the character of traumatic factor there are three basic clinical forms distinguished: 1. Accidently opening the pulp. The main cause of this form of acute pulpitis is careless

preparation of caries cavity, which results in perforation of pulp chamber with insignificant pulp trauma by rotary instrument (burs). Often enough it occurred during acute caries coarse preparation of carious cavity or removal of leather decal-cinated dentin during excavation and as a result appeared a blooding point perfora-tion. Through this perforation dentist may see the rose colour pulp. The probing of pulp is very painful and it is not recommend. Pathohistological features. It is characteristic for the acute course of deep caries.

There are sign of reactive changes of the pulp, dilation of vessels located near caries cavity and perforation.

Accidentally pulp wounding. The main cause of this form of acute pulpitis is penetration of instrument (burs, excavator) into the pulp chamber and pulp wound- ing. In these cases usually pulp tissue microbial contamination from caries dentin is occurred. The first sign of wounding is acute pain in moment of trauma. On the caries cavity floor appeared area of wounded bleeding pulp. 2 - 3.

Opening the pulp at crown fracture. This clinical condition occurred as a result of unexpected acute trauma.

The clinical feature depend 3 of the fracture line: it can located in crown (at the crown equator), in cervical area etc. In such 1. cases the pulp tissue quickly contaminated by microorganisms. Acute intensive pain can arise from different irritants (e.g. heat, cold, air etc.). Pathohistological features. It is characteristic for the acute pulp inflammation and depends on the term of trauma and cause of inflammation.

METHODOLOGICAL RESEARCH №2

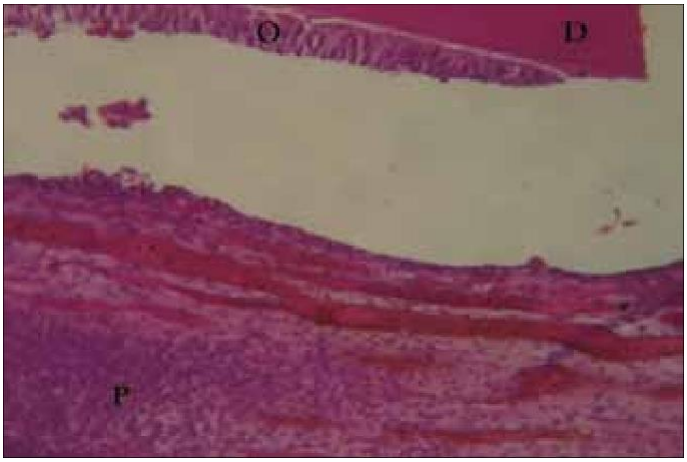
TOPIC: PULP HYPERAEMIA AND ACUTE PARTIAL PULPITIS: PATHOMORPHOLOGY, CLINIC, DIAGNOSIS, DIFFERENTIAL DIAGNOSTICS

a. **Pulp hyperemia (Hyperemia pulpae, incipient acute pulpalgia)**: All minor pulp sensations were once thought to be associated with hyperemia, an increased blood flow in the pulp. An increase in intrapulpal tissue pressure is produced only when heat is applied to the tooth, not when cold is applied. The increased pressure against the sensory nerve endings in the pulp might well produce the sensation associated with hyperemia. Quite possibly, this will explain why the pain appears to be of a different intensity and character with applications of cold or heat, the cold producing a sharp hypersensitivity response and the heat producing true transient hyperemia and a dull pain.

b. Pulp pain causes first a fall and then, when removed, a rise in intrapul-pal tissue pressure. Incipient acute pulpalgia should be completely reversible. It is characterized by mild discomfort such as that experienced following cavity or crown preparation. It may be gone by the next day. If one could study the cells of the pulp at this time, one would find a marginal increase in leukocytes and fluid pressure against the nerves that accompany the odontoblasts into the tubules. When pressure returns to normal, the discomfort disappears. Some patients may report slight discomfort from a carious lesion that has just broken through the enamel into the dentin Development of hyperemia is more frequent to manifest in sub acute pain, rarer pul-2 satile, pulsating.

c. Pain arises up spontaneously or as result of irritant action, pulp attacks shot duration 1-2 minutes with large painless intervals (intermission) up to 6-12-24 hours. Pain attacks more frequent and arises up at night. The affected tooth usually had deep caries cavity. The softened dentine which covers a cavity, has grey-white or yellow colour, rarely is it pigmented. The degree of dentine softening depend of activity of caries process: at acute (rampant) caries the hard tooth tissues are most softened like a cartilage, at chronic course it may be harder and pigmented. Probing of carious cavity is practically painless except denti-noenamel junction and floor of the cavity. Tooth usually responds to applications of cold: ice, carbon dioxide «ice» with pain 1-2 minutes duration. Pathohistological features.

d. The end result, whether induced by direct irritation or from the immune system, is the release of chemical mediators that initiate inflammation. This is a vascular response. The increase in the permeability of vessels nearest the site of injury and extravasations of fluid into the connective tissue spaces (edema) cause an elevation in local pressure. This edema alters or destroys the odontoblast layer. Chemical modification of the ground substance also occurs, as evidenced by an increased eosinophilia. Marked dilation of vessels leads to slowing of erythrocytes and the margination of leukocytes along the walls. The leukocytes then squeeze through the intracellular spaces of the vessel endothelia in response to chemotactic signals originating in the damaged tissue. This is called diapedesis.



e.

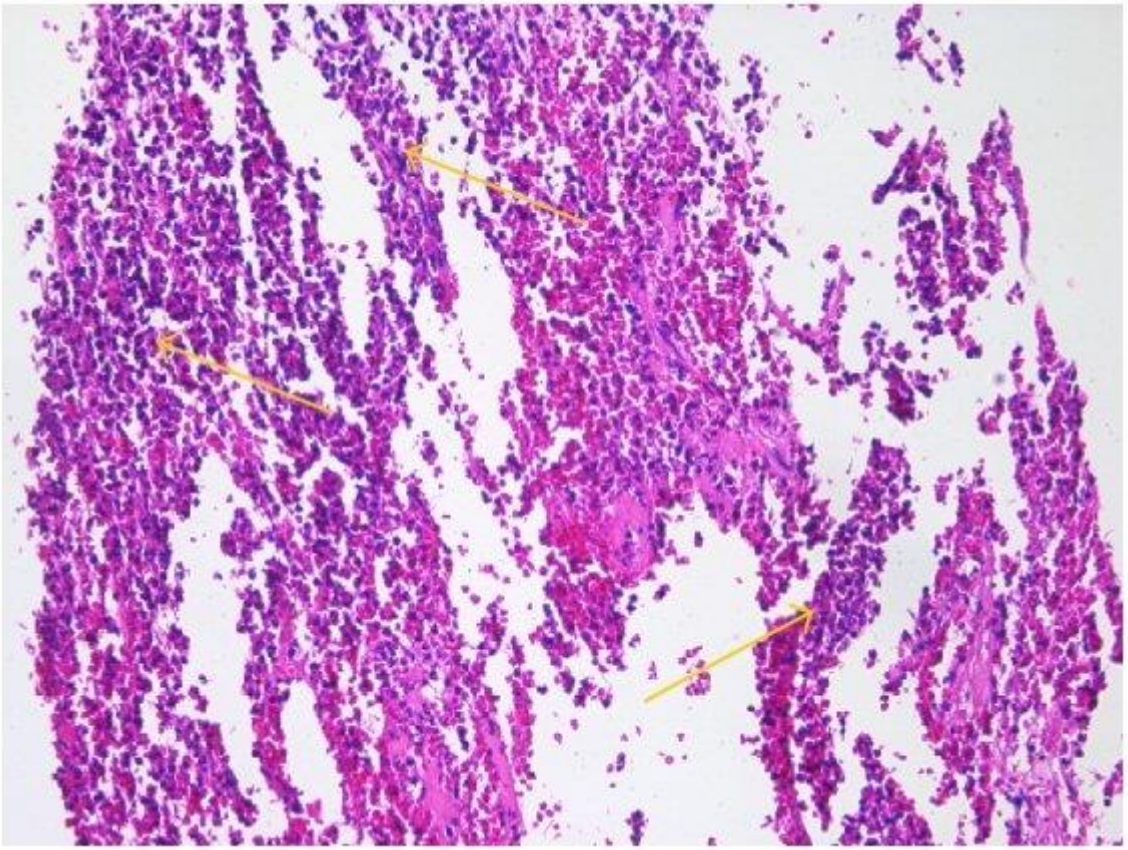
Pic.3. Microscopical image of pulp hyperaemia

Acute circumscription pulpitis (Pulpitis acuta circumscripta, a moderate acute pulpalgia): Moderate acute pulpalgia is a true but tolerable toothache, often described as «nagging» or «boring». In such cases inflammation of the pulp is pres-ent, so the pulp may be either reversible or irreversible. This is extended pain, often diffuse and hard to locate as it refers to other areas. The pain may start spontaneously or from a simple act such as lying down. Interestingly, cold may be the irritant that starts the pain, but hot food or drink and biting down on the cav- ity are more common. If the pain has been mild and has had a short duration then the pulpitis may be reversible.

This pulpitis is characterized by spontaneous (i.e., unprovoked), intermittent, or 4 continuous paroxysms of pain. The pain is frequently described as a «nagging» or a «bor-ing» pain, which may at first be localized but finally becomes diffuse or referred to anoth 5 er area. The pain may start spontaneously or 2 from a simple act such as lying down. Inter-estingly, cold may be the irritant that starts the pain, but hot food or drink and biting down on the cavity are more common. Pain attacks at first lasted 15-30 minutes, but with development of the inflammatory process in pulp its duration increase to 1-2 hours. Pain attacks increase and become more frequent at night.

Painless intervals usually last 2-3 hours and than decreased to more shot. Usually patients indicate on a causal caries tooth, but in some cases the pain may be referred from one arch to the other. If this pain has been mild and has had a short duration, then the pulpitis may be reversible. Examination revealed the tooth with caries cavity, frequently deep, rarer middle caries. The pain is diffuse, and two or three teeth may give similar responses to electric pulp testing. The walls and floor of cavity covered with soft demineralized den-tin; in some cases it can be more dense and pigmented. During probing the cavity floor is painful, especially in the areas located near the pulp horn. Pathohistological features. It is determined marked dilation of vessels leads to slowing of erythrocytes and the margination of leukocytes along the walls. In some areas located near the caries cavity there are hemorrhages into pulp tissue, which is saturated with serous exudate. There are accumulations of erythrocytes and leukocytes in the pulp near the caries cavity. With increasing of vascular wall permeability the inflammatory

infiltration increased. As a result the odontoblast layer altered or destroyed.



Pic.4. Microscopical image of acute partial pulpitis

METHODOLOGICAL RESEARCH №3

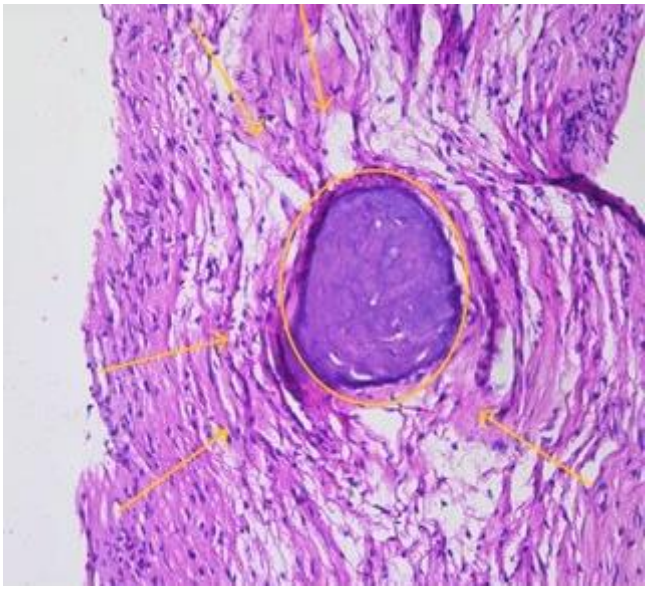
TOPIC: ACUTE GENERALISED PULPITIS: PATHOMORPHOLOGY, CLINIC, DIAGNOSTICS

Acute diffuse pulpitis (Pulpitis acuta diffusa): It is characterized by the spontaneous pain and development of acute pain attack, referred from one arch to the other and along the branches of n. trigeminus. Acute diffuse pulpitis usually is a result of further development of circumscription inflammation. The character of pain attack is like of neuralgic attacks: often described as «nag-ging» or «boring». This is extended pain, often diffuse and hard to locate as it refers to other areas. The pain may start spontaneously or from a simple act such as lying down. Interestingly, cold may be the irritant that starts the pain, but hot food or drink and 1 3 biting down on the cavity are more common. 2 If this pain has been mild and has had a short duration, then the pulpitis may be reversible. One-two days ago the durability of pain attack was 10-30 minutes and presently the attacks are lasting an hour.

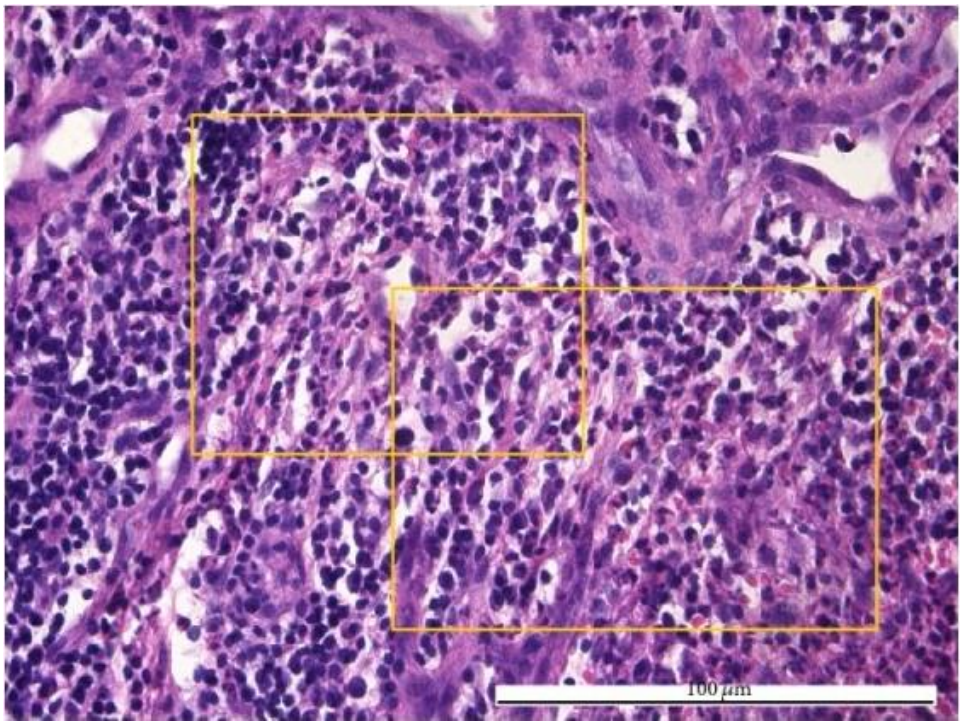
The durability of pain- 5 less intervals decreases up to 10-30 minutes. Pain attacks increase and become more fre- quent at night and at horizontal position of patient. Usually patients indicate on a causal caries tooth, but in some cases the pain may be referred from one arch to the other. This pulpitis is irreversible; the pulp must be sacrificed. Pinpointing the exact tooth involved in moderate pulpalgia is often difficult. Examination revealed the tooth with caries cavity, frequently deep, rarer middle caries. The pain is diffused and two or three teeth may give similar responses to electric pulp test-ing.

The walls and floor of cavity covered with soft demineralized dentin; in some cases it can be more dense and pigmented. During probing the cavity floor is painful. Percussion may reveal a slight difference in response between teeth. A warm rinse does not relieve the pain, and cold may make it worse. Thermal testing with cold should be attempted first. If pain response from the suspected tooth increases but then goes away, stop! Do not test other teeth, but wait for the rebound of pain that may occur.

Pathohistological features. It is determined marked dilation of vessels leads to slowing of erythrocytes and the margination of leukocytes along the walls. In all pulp (coronal and radicular) there are hemorrhages into pulp tissue, which is saturated with serous exudate. There are accumulations of erythrocytes and leukocytes in the pulp near the caries cavity. With increasing of vascular wall permeability the inflammatory infiltration increased. As a result the odontoblast layer altered or destroyed.



Pic.5. Microscopical image of acute diffuse pulpitis



Pic.6. Inflammatory infiltrate in pulp tissues

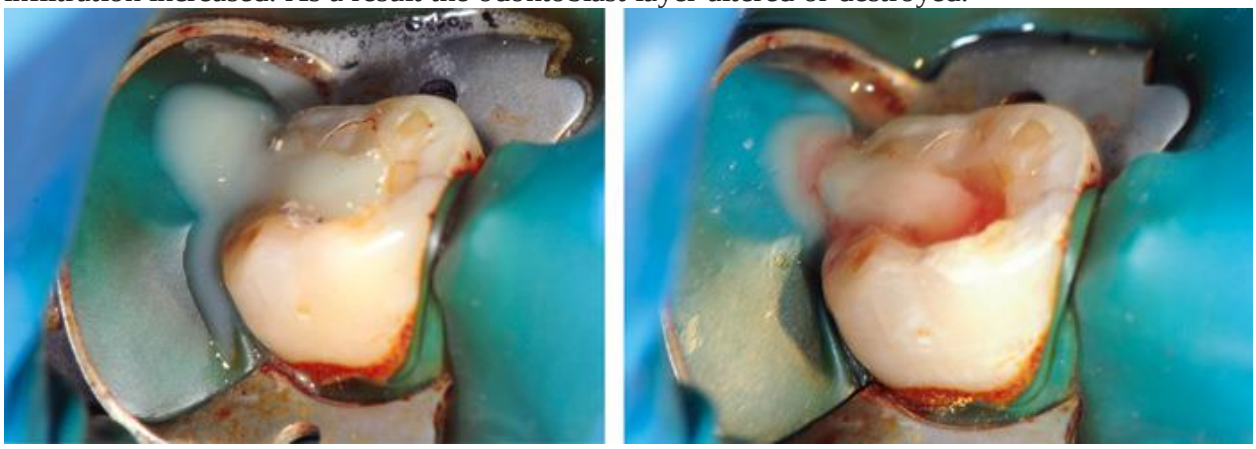
METHODOLOGICAL RESEARCH №4

TOPIC: ACUTE PURULENT PULPITIS: PATHOMORPHOLOGY, CLINIC, DIAGNOSTICS

Acute purulent (suppurative) pulpitis. Pulpitis acuta purulenta, advanced acute pulpalgia): Acute purulent pulpitis usually is a result of further development of diffuse pulp inflammation. It is characterized by the spontaneous pain and development Di acute pain attack, referred from one arch to the other and along the branches of n. trigeminus. The pain may start spontaneously or from a simple act such as ying down. Pain attack increases, pain become pulatile, continuous with remission only some minutes. At night pain attack becomes more intensive. The pain arises and increases as a result thermal irritants (the hot meals, temperature more than 37° C). The cold irritant relieves the pain. Examination revealed the tooth with caries cavity, frequently deep, rarer middle caries.

The walls and floor of cavity covered with soft demineralized dentin; in some cases it can be more dense and pigmented. Testing with heat 1 gives an immediate explosive response! Vertical percussion is painful. During probing the cavity floor is painful, the pulp chamber may easily be perforated with drop of pus or pus with blood appears. As usually such perforation relieves the pain attack. This is irreversible pulpitis!

Pathohistological features. It is determined marked dilation of vessels leads to slowing of erythrocytes and the margination of leukocytes along the walls. In all pulp (coronal and radicular) there are hemorrhages into pulp tissue, which is saturated with serous exudate. Histologically, one finds necrosis of the coronal pulp with vital remnants left. In the coronal pulp there are forming abscesses. There are accumulations of erythrocytes and leukocytes in the pulp near the caries cavity. With increasing of vascular wall permeability the inflammatory infiltration increased. As a result the odontoblast layer altered or destroyed.



Pic.7. Purulent exudate.

METHODOLOGICAL RESEARCH №5

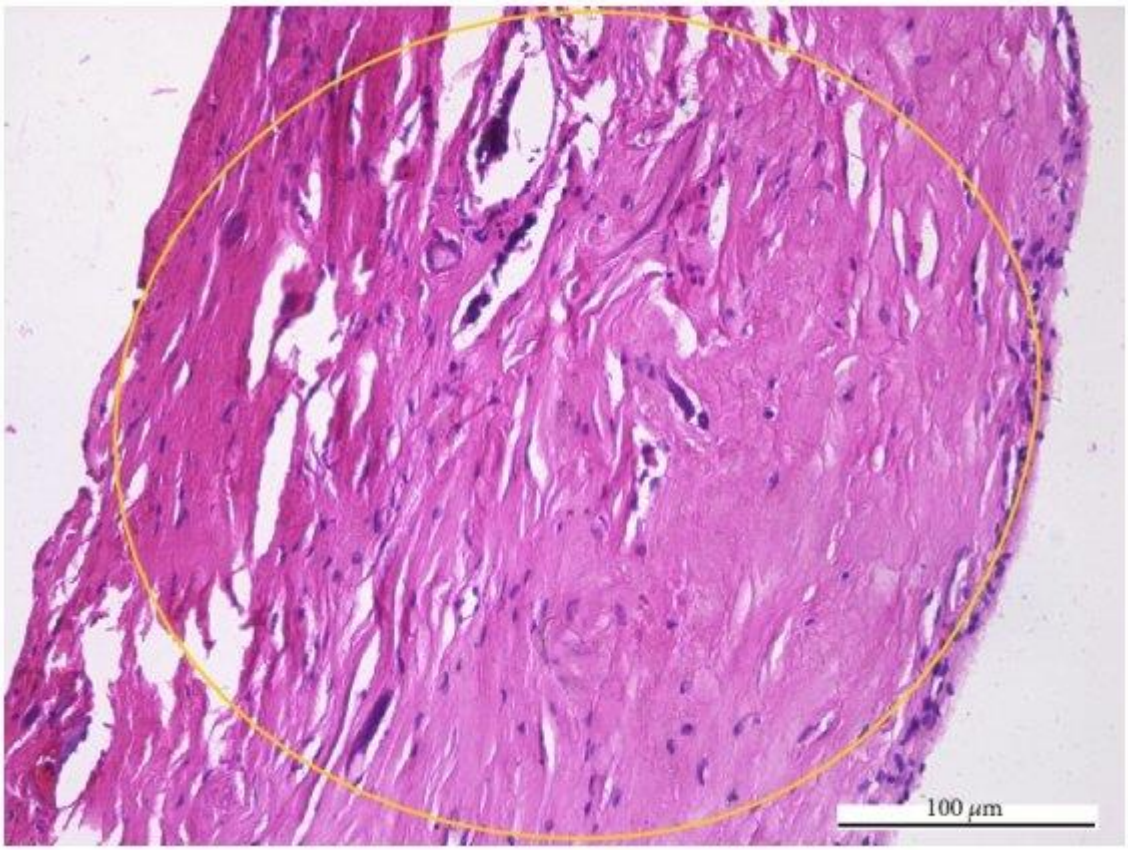
TOPIC: CHRONIC SIMPLE PULPITIS: PATHOMORPHOLOGY, CLINIC, DIAGNOSTICS

Chronic fibrous pulpitis (Pulpitis chronica fibrosa, chronic pulpalgia): Unlike the acute form of pulpitis at chronic fibrous pulpitis the patient feels heaviness in a tooth. Pain appears in reply to action of thermal, chemical and mechanical irri-tants, intensity of which is usually depend of location of caries cavity. At the opened pulp chamber of the tooth and central location of caries cavity «sucking» from a tooth can cause quickly passing aching pain. Unlike caries the pain at chronic fibrous pulpitis lasted 30-90 minutes after cessation irritant action. At chronic pulpitis the acute pain is absent (it may revealed during anamne-sis in past) and now patient felt heavy dull pain. It is often described as a «grum-ble», not severe but consistent discomfort. Patients have admitted withstanding the discomfort for weeks or even years, suppressing the pain with analgesics. On the other hand, they may not have had any overt symptoms that would alert them to seek a dentist. Finally, when the pulp starts to ache all night or flare up during an airplane flight, they come in for treatment.

The pain is mild enough and diffuses enough to complicate its location. Moreover, chronic pulpalgia often refers to other teeth or the opposing arch. Pain may be precipitated by biting down on an open cavity. Cold has little effect, but heat may increase the discomfort. The examination of tooth with caries cavity or fractured filling may be revealed. Cavity covered with soft demineralized dentin, which may be pigmented. The probing revealed perforation with gray-brown pulp tissue, which is painful during probing.

The patient usually reports no significant pain, 2 and tests reveal little or no pain on percussion. Pathohistological features. The prominent feature is the excrescence of fibrous connective tissue. The fibres of pulp are thickened. There is hyalinosis of collagen fibres of pulp of ground substance. The cell reaction manifested odonto-blasts layer vacuolization, intensification of cells proliferation. In root pulp there are fibrous and petrification of ground

substance.



Pic.8. Microscopical image of chronic fibrous pulpitis.

METHODOLOGICAL RESEARCH №6

TOPIC: CHRONIC HYPERTROPHIC PULPITIS: PATHOMORPHOLOGY, CLINIC, DIAGNOSTICS

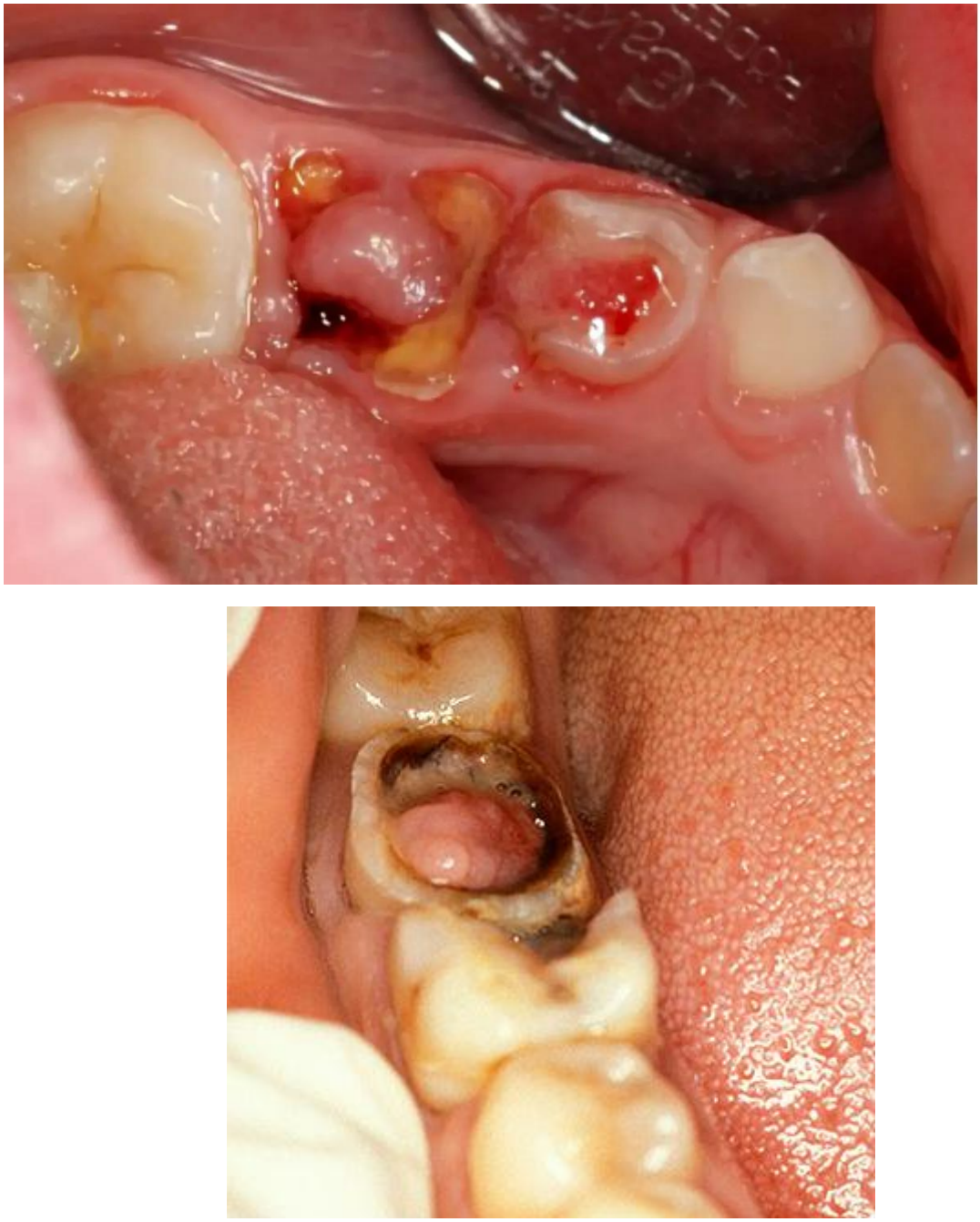
Chronic hypertrophic pulpitis (Pulpitis chron-ica hypertrophia, Hyperplastic pulpitis). Hyperplastic pulpitis (pulp polyp) is a form of irreversible pulpitis, which results from growth of chronically inflamed young pulp into occlusal surfaces. It is usually found in carious crowns of young patients. Ample vascularity of young pulp, adequate exposure for drainage, and tissue proliferation are associated with formation of hyperplastic pulpitis. This form of pulpitis often develops in children and persons of young age. From anamnesis the presence in the past the acute pain is revealed. Patient's complaint of pain and the appearance of blood from a carious cavity during chewing are as a result of the trauma of a food lump or at «sucking» from a tooth. Hyperplastic pulpitis is usually asymptomatic. It appears as a reddish cauliflow-er-like outgrowth of connective tissue into caries that has resulted in a large occlusal exposure.

Objectively in affected tooth there is a large carious cavity filled by fleshy like colour tumor like formation. This overgrowing pulp is bleed and little sensitive at its probing, but is painful in area of entrance of root channels. Outlining round a «polyp» by a probe (determination of «area of growth»), it is possible to make sure in its connection with pulp. There is dull pain a result of cold irritants. Rising out of the carious shell of the crown is a «mushroom» of living pulp tissue that is often firm and insensitive to the touch.

A redish, cauliflower-like growth of pulp tissue through and around a carious exposure is one variation of asymptomatic irreversible pulpitis. The proliferative nature of this pulpal' reaction, sometimes known as a spulp polyp», is attributed to a low-grade, chronic irritation of the pul and the generous vascularity characteristically found in young people. Occasionical is condition may cause mild, transient pain during mastication. The chronically inflamed young pulp, widely exposed by caries on its occlusal aspect, is the forerunner of this unique growth. Proliferative growth of inflamed connective tissue resembles a pyogenic granuloma of the gingiva.

Pathohistological features. The considerable changes developed in vessels: from one side, there is violation of anatomo-topographical architectonics of blood and lymphatic vesselS, with other — clear features of morphological changes characteristic for a chronic inflammatory process. The changed pulp is usually presented by young granulation tissue. Microscopically, the pulp polyp is a complex of new cap-illaries, proliferating fibroblasts, and inflammatory cells. Among tender connective tissue fibres there is a plenty of the young thin-walled capillaries. Support for the protruding mass is supplied by collagenous fibres rooted in the deeper pulp tissue of the chamber.

Sensory nerve elements are almost totally absent near the surface, in contrast to the rich innervations and exquisite sensitivity of an exposed pulp that is not hyperplastic. Before the lesion has grown to any extent, its surface layer consists of massed necrotic cells and leukocytes with chronic inflammatory cells (leucocytes) beneath forming a zone of variable width. As the tissue expands, it may acquire a stratified squamous epithelial cover that may form by a true cell graft. Cells of the oral mucosa floating free in the saliva may grow over the surface of the highly vascularized young connective tissue, or a direct migration of epithelial cells may occur from the gingi-va. Hyperplastic pulpitis is irreversible and therefore requires pulpectomy and root canal treatment or extraction.



Pic. 9-10. Chronic hypertrophic pulpitis.

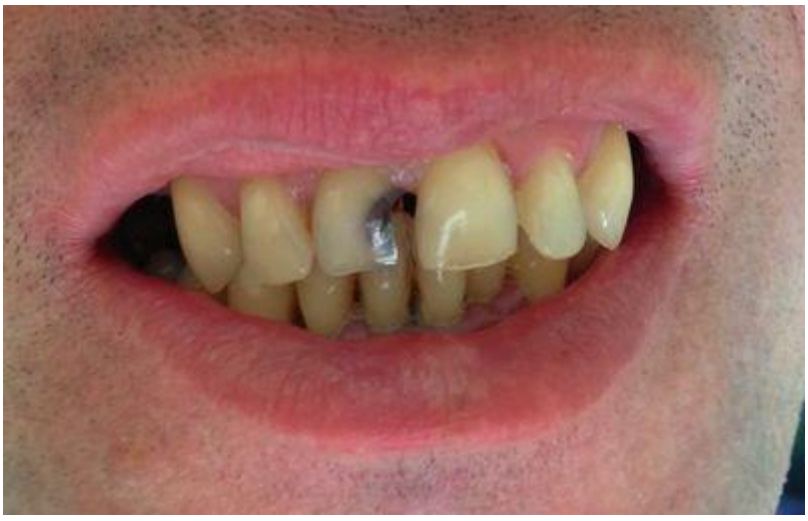
METHODOLOGICAL RESEARCH №7

TOPIC: CHRONIC GANGRENOUS PULPITIS: PATHOMORPHOLOGY, CLINIC, DIAGNOSTICS. PULP NECROSIS AND ATROPHY

Chronic gangrenous pulpitis (Pulpitis chronica gangraenosa, Pulp necrosis): This develops from acute purulent or chronic fibrous pulpitis as a result of penetration into pulp of putrid bacteria. Necrosis, the death of the pulp, actually refers to a histological condition resulting from an untreated irreversible pulpitis, a traumatic injury, or any event that causes long-term interruption of the blood supply to the pulp. As inflammation progresses, tissue continues to disintegrate in the center to form an increasing region of liquefaction necrosis.

Because of the lack of collateral circulation and the unyielding walls of the dentin, there is insufficient drainage of inflammatory fluids. These results in localized increases in tissue pressures, causing the destruction to progress unchecked until the entire pulp is necrotic. The rate of progress of liquefaction necrosis varies. Spontaneous pain is absent, when there is perforation of the caries cavity floor. The unpleasant feeling of expansion in a tooth is the permanent sign of gangrenous pulpitis. The pain usually slowly arises up under influence of thermal (hot) irritants and lasted short time. Spontaneous pain arises up and is observed then, when the pulp chamber is closed and the exudates cannot flow from the inflamed

Objectively in affected tooth there is a large carious cavity filled by softened dentin. The pulp chamber at most patients is opened and filled by the products of pulp disintegration with an unpleasant smell. The reaction on the superficial probing is absent. The deep probing is painful. The leathery dentin covering these lesions may be removed with a spoon excavator, often without anaesthesis unface ne no sis a dise comfort. The pulp lies revealed, covered with a ray sum of surface necross. crown discolouration may accompany pulp necrosis in anterior teeth, but this diagnostic sign is not reliable. Pathohistological features. Once bacteria have invaded the necrotic pulp, they release enzymes to break down the necrotic tissue for assimilation of the available nutrients; by the process of heterolysis, liquefaction (also called «wet gangrene») occurs. The coronal pulp is necrotic with plenty of anaerobic microorganism. Adjacent to the liquefaction necrosis is a zone of chronic inflamma-tion. Although the width of this zone may vary, generally it is rather narrow. This activity produc- es an abundance of by-products, which eventually leak into periradicular tissues, causing inflammatory and immunologic reactions. Odontoblasts had dystrophic changes; cellular composition of root pulp is poor with areas of hyalinosis. Peri-radicular inflammation would not be expected to develop until the pulp is nearly totally necrotic



Pic.11. Chronic gangrenous pulpitis.

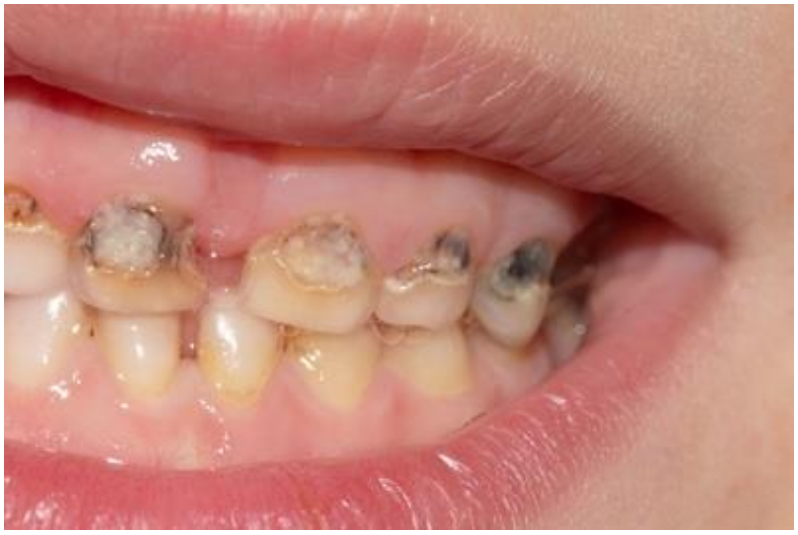


Рис.12. Chronic gangrenous pulpitis of temporary teeth.

METHODOLOGICAL RESEARCH №8

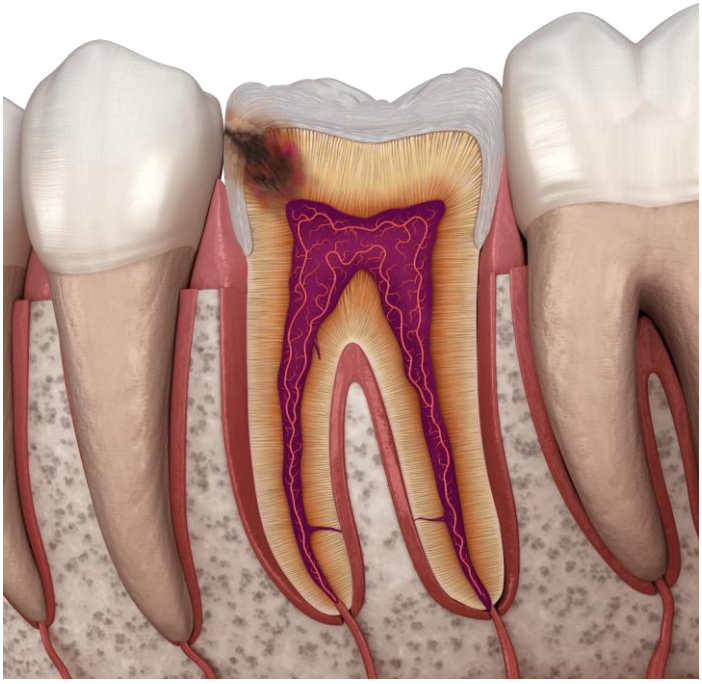
TOPIC: CALCULOUS AND ROOT PULPITIS: PATHOMORPHOLOGY, CLINIC, DIAGNOSTICS

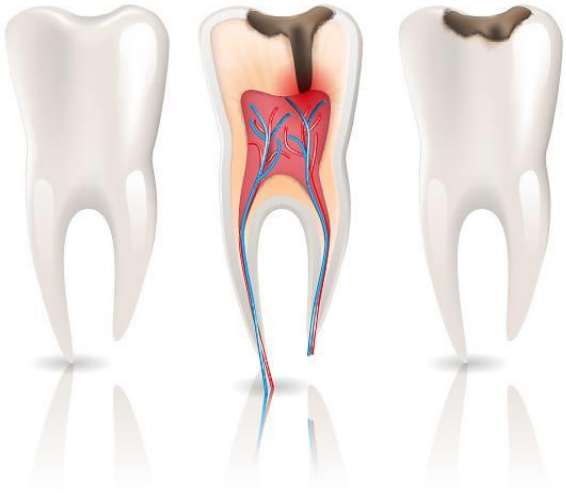
Chronic concremental pulpitis (Pulpitis chronica concrementosa): The causative factor of this form of pulpitis there are denticles - a calcified deposits in the dental pulp. Usually they are located in pulp chamber or root canals. It may be composed either of irregular dentine (true denticle) or an ectopic calcification of pulp tissue. Denticle most often formed in back located teeth (molars, premolars) at persons in age more 40 years. These formations cause the permanent irritation of nervous endings of pulp, resulting in chronic inflammation. Patients' complains of spontaneous pain and development of acute pain attack, referred from one arch to the other and along the branches of n. trigeminus.

The character of pain attack is like of neuralgic attacks. Pain attacks increase and become more frequent at night and during vibration. Clinical features resembled trigeminal neuralgia. Pain attack lasted 15-30 minutes. *Vertical percussion* is painful and may provoke the pain attack. Examination revealed the tooth with abrasion of occlusal surfaces, frequently in patients with periodontal diseases (in rarely case the tooth may be intact). In the main diagnostic procedure is radiographic examination which allow to revealed den-ticles in pulp chamber.

Pathohistological features. In pulp tissue denticles and petrifications are revealed.

Their localization, quantity, form and size, are various. In pulp tissue there are some sign of dystrophia: odontoblasts vacuolization, hyalinosis and areas of petrifac- tion with signs of chronic inflammation





Pic.13, 14. Schematic image of pulpitis.

METHODOLOGICAL RESEARCH №9
TOPIC: EXACERBATION OF CHRONIC PULPITIS: PATHOMORPHOLOGY, CLINIC, DIAGNOSTICS

Exacerbated chronic pulpitis (Pulpitis chronica exacerbata): Every form of chronic pulpitis can exacerbate. The frequently it is fibrous pulpitis more rarely - chronic gangrenous pulpitis. Exacerbated chronic pulpitis is characterized by the spontaneous pain and development of acute pain attack, referred from one arch to the other and along the branches of n. trigeminus. Pain attacks increase and become more frequent at night and at horizontal position of patient. The pain attack can provoke different irri-tants, frequently the cold.
Duration of pain attack may be different: pain attack at first lasted 15-30 minutes, but with development of the inflammatory process in pulp its duration increase to 1-2 hours. When inflammation develops in root pulp then the vertical percussion becomes painful.
Examination revealed the tooth with caries cavity, frequently deep, rarer middle caries. The state of caries cavity correspond some form of chronic pulpitis. For example, in case of exacerbated chronic fibrous pulpitis cavity covered with soft demin-eralized dentin, which may be pigmented. The probing revealed perforation with gray-brown pulp tissue, which is painful during probing. The patient usually reports no significant pain, and tests reveal little or no pain on percussion. But thermal (cold, hot) irritants may provoke pain attack.
Pathohistological features usually correspond with some forms of chronic pulpitis and with some sign of acute inflammation. It is determined marked dilation of vessels leads to slowing of erythrocytes and the margination of leukocytes along the walls. In all pulp (coronal and radicular) there are haemorrhages into pulp tissue, Which is saturated with serous or purulent exudate. The odontoblast layer altered or destroyed.
Pulpitis complicated with apical periodontitis (Pulpitis complicatus periodontitis). Pulpitis on the definite stage of the inflammation development can be complicated by different forms of periodontitis. More frequent complicated acute diffuse pulpitis. The clinical features combined the clinical signs of pulpitis and apical periodon-its For example there may be the signs of acute diffuse or purulent pulpitis and signs of periodontitis. The durability of pain attack was 10-30 minutes and attacks are lasting an hour. The durability of painless intervals decreases up 10 10-30 minutes. Pain attacks increase and become more frequent at night and at horizontal position of patient. Usually patients indicate on a causal caries tooth, but in some cases the pain may be referred from one arch to the other. These signs of pulpitis combined with signs of periodontitis. This may be aedema of the soft tissues of the face (lips, cheek), especially on the site of affected tooth. There is hyper-emia and edema in the area of tooth apex, sometimes hyperemia is present in the adjoining areas of gums. In some cases the periosteal abscess may develop. Horizontal and vertical percussion of tooth is very sensitive. There is tooth mobility in mesial, distal and vertical directions.
Examination revealed the tooth with caries cavity, frequently deep, rarer middle caries. The walls and floor of cavity covered with soft demineralized dentin; in some cases it can be more dense and pigmented. During probing the cavity floor is pain-ful, vertical percussion is painful. When chronic pulpitis complicated during probing the pulp chamber revealed perforation with gray-brown pulp tissue, which is painful during probing Pathohistological features usually correspond with some forms of acute or chronic pulpitis with some sign of acute inflammation in periodontal ligament.

METHODOLOGICAL RESEARCH №10

TOPIC: METHODS AND MEANS OF ANAESTHESIA IN PULPITIS. MEDICINAL PRODUCTS AND METHODS OF APPLICATION. PREMEDICATION

In no other area of dentistry is the management of pain of greater importance than in endodontics. All too often the patient in need of endodontic therapy has endured a prolonged period of ever-increasing discomfort before seeking dental care. The reasons for this discomfort are manifold; however, there is one simple explanation in the overwhelming majority of these patients. It is possible to achieve clinically elictive pulpal anaesthesia on all teeth, infected or not, in any area of the oral cavi-ty, with a very high degree of success and without inflicting any additional pain on the patient in the process.

The administration of inhalation sedation with nitrous oxide and oxygen carefully titrated, alleviates any fears of injections in the majority of needle-pho-bic dental patients. Continued administration during the endodontic procedure is entirely appropriate if the patient is at all apprehensive. When inhalation sedation is contraindicated (e.g., patient is a mouth breather, patient has a «cold» or upper respiratory infection, or sedation has proved ineffective in the past in eliminating the patient's fears), other techniques of conscious sedation should be considered. The safest and most effective, when used properly, is intravenous conscious sedation. Clinically effective pain control can be achieved in the vast majority of patients requiring endodontic therapy. When problems achieving pain control occur, it is usually at the initial visit, when a frightened patient, who has been hurting for some period of time, finally seeks relief from pain yet often times they are unable to manage the fears of dentistry. Through a combination of thoughtful caring for the patient, the use of conscious sedation, when indicated, and the effective administration of local anaesthesia, endodontic treatment can proceed in a more relaxed and pleasant environment for both the patient and dental staff.

Local anaesthetic techniques.

Mandibular Anaesthesia: To provide effective pulpal anaesthesia in the mandi-ble, one must administer the local anaesthetic drug at a site where the nerve is still accessible (e.g., before the nerve enters the mandibular foramen and into the man-dibular canal). Thus, one is limited to two injection sites. One site is the lingual aspect of the mandibular ramus, where three techniques may be used: the inferior alveolar (IA) nerve block (the traditional «mandibular block»); the Gow-Gates man-dibular nerve block (GGMNB), and the Akinosi-Vazirani closed-mouth mandibu-lar nerve block.

A second site of access to the mandibular nerve is available on the mandible, the mental foramen, located (usually) between the two premolars. Local anaesthetic administered at this site will provide profound pulpal anaesthesia of the premolar, canine and incisor teeth virtually 100% of the time, even when infection is present. On those occasions when these three mandibular nerve block injections fail to provide successful pulpal anesthesia, one of several supplemental techniques may be considered. These include the periodontal ligament (PDL) injection, intraosseous (IO) anaesthesia, and intrapulpal injection. The 10 technique has proved to be of tremendous benefit in endodontics, particularly as a means of providing anaesthesia to the «hot» mandibular molar.

Maxillary Anaesthesia: Although profound anaesthesia of maxillary teeth is normally easier to obtain, problems, if they occur, usually do so following the administration of an infiltration injection to a central incisor, canine, or molar. The apex of the central incisor may lie under the cartilage of the nose, making infiltration less effective (as well as more uncomfortable). Canines that have longer than usual roots may not be anesthetized when the anaesthetic is deposited below the apex (nee-dle is not inserted far enough).

Infiltration anesthesia of maxillary molars will fail in situations where the palatal root flares greatly toward the midline of the palate. Most local anesthetics infiltrated into the buccal fold

will not diffuse far enough toward the midline to provide adequate pulpal anaesthesia in this situation. Additional-ly, where periapical infection is present, the success rate of injected local anaes-thetics is diminished, sometimes considerably. Fortunately, maxillary anesthesia can readily be achieved through the administration of nerve blocks. Three nerve blocks, the posterior superior alve-olar (PSA), middle superior alveolar (MSA), and anterior superior alveolar (ASA, «infraorbital»), successfully provide pulpal anaesthesia to maxillary teeth, even in the presence of infection.

Supplemental Injection Techniques: Periodontal Ligament (PDL) Injection and Intraligamentary Injection (ILI). When pulpal anesthesia of a single tooth is required, the PDL injection should be considered. This is of special importance in the mandible, where nerve block anesthesia is the norm. In the maxilla, supraperi-osteal injection infiltrated above the apex of any tooth will provide successful pulpal anesthesia with a success rate of > 95%. Because of the thickness of the mandibular cortical plate of bone (in adults), infiltration techniques are doomed to failure. There-fore, although the PDL may be successfully administered to any tooth, its use is most often reserved for mandibular teeth, specifically mandibular molars.

Intraosseous (IO) Anaesthesia: In true IO anaesthesia, local anaesthetic is inject-ed directly into the bone surrounding the root of a tooth. Intrapulpal Anaesthesia. When the pulp chamber has been exposed and, because of exquisite sensitivity, treatment cannot proceed, intrapulpal anaesthe-sia should be considered. A small needle is inserted into the pulp chamber until resistance is encountered. The local anaesthetic must be injected under pressure. There will be a brief moment of intense discomfort as the injection is started, but anaesthesia usually supervenes almost immediately, and instrumentation can proceed painlessly. Clinically effective pain control can be achieved in the vast majority of patients requiring endodontic therapy. Through a combination of thoughtful caring for patient, the use of conscious sedation, when indicated, and the effective administration of local anaesthesia, endodontic treatment can proceed in a more relaxed and pleasant environment for both the patient and dental staff.

Table 1. Injectable Local Anesthetic Agents*			
Anesthetic Agent	**Agent/Formulation**	**Duration of Pulpal Anesthesia**	**Pregnancy Category****
Articaine *Brand Names:* Articadent Septocaine Ultracaine Zorcaine	4% articaine/1:100,000 epinephrine	Medium	C
	4% articaine/1:200,000 epinephrine	Medium	C
Bupivacaine *Brand Names:* Marcaine Sensorcaine Vivacaine	0.5% bupivacaine/1:200,000 epinephrine	Long	C
Lidocaine *Brand Names:* Xylocaine Lignospan Alphacaine Octocaine	2% lidocaine/1:100,000 epinephrine	Medium	B
	2% lidocaine/1:50,000 epinephrine	Medium	B
Mepivacaine *Brand Names:* Carbocaine Polocaine Scandonest	3% mepivacaine plain	Short	C
	2% mepivacaine/1:20,000 levonordefrin	Medium	C
Prilocaine *Brand Name:* Citanest	4% prilocaine plain	Short	B
	4% prilocaine/1:200,000 epinephrine	Medium	B

Pic.15. Local anesthetics in dentistry

METHODOLOGICAL RESEARCH №11

TOPIC: METHODS OF PULPITIS TREATMENT. REASONING FOR CHOOSING A METHOD OF TREATMENT OF PULPITIS DEPENDING ON THE FORM, PROGRESSION AND GENERAL CONDITION OF THE ORGANISM. METHOD OF PRESERVING THE PULP: INDICATIONS FOR USE. METHODS OF TREATMENT. MEDICINES, THEIR PRESCRIPTION. EFFECTIVENESS AND POSSIBLE COMPLICATIONS

ENDODONTIC PRETREATMENT. Root canal therapy does not necessarily begin with the placement of the rubber dam but with the restorative or periodontic procedures necessary to simplify its placement.

Rubber dam application. Rubber dam application is an essential prerequisite for providing nonsurgical endodontic treatment. For root canal treatment, rapid, simple, and effective methods of dam applications have been developed. In all but the most unusual circum-stances, the rubber dam can be placed in less than 1 minute. Although the modern endodontic approach to the use of the dam has changed, the importance and purposes of the dam remain the same:

1. It provides a dry, clean, and disinfected field.
2. It protects the patient from the possible aspiration or swallowing of tooth and filling debris, bacteria, necrotic pulp remnants, and instruments or operating materials.
3. It protects the patient from rotary and hand instruments, drugs, irrigating solu-tions, and the trauma of repeated manual manipulation of the oral soft tissues.
4. It is faster, more convenient, and less frustrating than the repeated changing of cotton rolls and/or saliva ejectors.

The rubber dam also provides a fluid seal from saliva from the working field. It has been recently shown in vivo that intraoral and extraoral microorganisms contaminating the root canal system will lead to eventual failure.

ENDODONTIC CAVITY PREPARATION.

Endodontic cavity preparation may be separated into two anatomic divisions:

(a) coronal preparation

(b) radicular preparation.

Actually, coronal prepa-lation is merely a means to an end, but to accurately prepare and properly fill the radicular pulp space, intra-coronal preparation must be correct in size, shape, and inclination. Caries and defective restorations remaining in an endodontic cavity preparation must be removed for three reasons: (1) to eliminate mechanically as many bacteria as possible from the interior of the tooth, (2) to eliminate the discoloured Loth structure, that may ultimately lead to staining of the crown, ane ase to alim-Inate the possibility of any bacteria-laden saliva leaking into the prepared cavity.

The last point is especially true of proximal or buccal caries that extend into the prepared cavity. Black's principles of cavity preparation - Outline Convenience, Retention, and Resistance Forms - may be applied. The entire length of the preparation is the full outline form. In turn, this outline may have to be modified for the sake of convenience to accommodate canal anatomy or curvature and/or instruments. For initial entrance through the enamel surface or through a restoration, the ideal cutting instrument is the round-end carbide fissure bur. As soon as the bulk of the overhanging dentin is removed from the roof of the chamber, the slower operating round burs are put aside, and, once again, the high-speed fissure bur is used to finish and slope the side walls in the visible portions of the preparation. Size and shape of endodontic coronal preparations related to size and shape of the pulp chamber. The outline form of the endodontic cavity must be correctly shaped and positioned to establish complete access for

instrumentation, from cavity margin to apica foramen. Moreover, external outline form evolves from the internal anatomy of the tooth established by the pulp.
To achieve optimal preparation, three factors of internal anatomy must be considered:
(1) the size of the pulp chamber,
(2) the shape of the pulp chamber,
(3) the number of individual root canals, their curvature, and their position. The finished outline form should accurately reflect the shape of the pulp chamber. For example, the floor of the pulp chamber in a molar tooth is usually triangular in shape, owing to the triangular position of the orifices of the canals. As another example, the coronal pulp of a maxillary premolar is flat mesiodistally but is elongated buccolingual.
Number, Position, and Curvature of Root Canals: The third factor regulating outline form is the number, position, and curvature or direction of the root canals. lo prepare each canal efficiently without interference, the cavity walls often have to be extended to allow an unstrained instrument approach to the apical foramen. When cavity walls are extended to improve instrumentation, the outline form is materially affected. This change is for convenience in preparation; hence, convenience form partly regulates the ultimate outline form. In endodontic therapy convenience form makes more convenient (and accurate) the preparation and filling of the root canal. Four important benefits are gained through convenience form modifications:
(1) unobstructed access to the canal orifice,
(2) direct access to the apical foramen,
(3) cavity expansion to accommodate filing techniques,
(4) complete authority over the enlarging instrumant.
Unobstructed Access to the Canal Orifice: In endodontic cavity preparations of al teth, enough tooth structure must be removed to allow instruments to be plas caily into the orifice of each canal without interference from overhanging with. The clinician must be able to see each orifie and casily reaca much the instrument points a must the other hand it is most importan that as much con structure be maintained as possible, MOD cavity preparations vaste soat-ches by more thana io, a as phe «loss of marginal ridge integrity was the great-et contribution to loss of tooth strength».
Direct Access to the Apical Foramen. To provide direct access to the apical foramen, enough tooth structure must be removed to allow the endodontic instruments freedom within the coronal cavity so they can extend down the canal in an unstrained position. This is especially true when the canal is severely curved or leaves the chamber at an obtuse angle. Infrequently, total de-cuspate is necessary.
Extension to Accommodate Filling Techniques: It is often necessary to expand the outline form to make certain filling techniques more convenient or practical. If a softened gutta-percha technique is used for filling, wherein rather rigid pluggers are used in a vertical thrust, then the outline form may have to be widely extended to accommodate these heavier instruments.
Complete Authority over the Enlarging Instrument. Direct access to the apical foramen It is imperative that the clinician maintain complete control over the root canal instrument. If the instrument is impinged at the canal orifice by tooth structure that should have been removed, the dentist will have lost control of the direction of the tip of the instrument, and the intervening tooth structure will dictate the control of the instrument. If, on the other hand, the tooth structure is removed around the orifice so that the instrument stands free in this area of the canal, the instrument will then be controlled by only two factors: the clinician's fingers on the handle of the instrument and the walls of the canal at the tip of the instrument. Nothing is to intervene between these two points

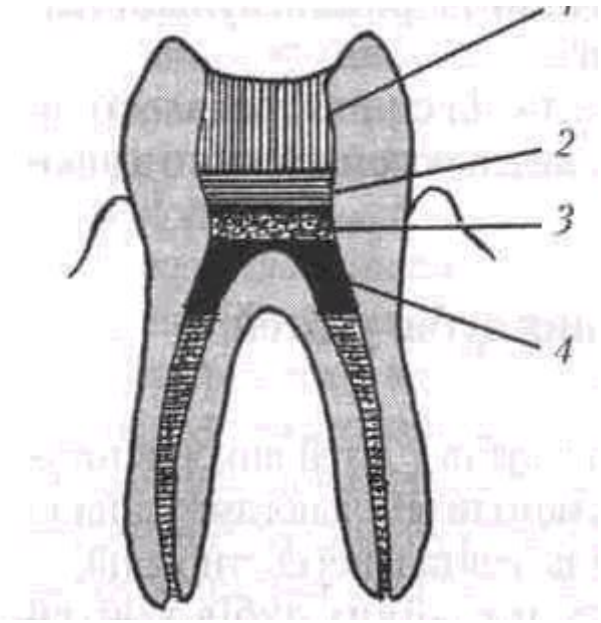
METHODOLOGICAL RESEARCH №12

TOPIC: VITAL AMPUTATION METHOD OF PULPITIS TREATMENT: INDICATIONS, TECHNIQUES, MEDICATIONS. EFFECTIVENESS AND POSSIBLE COMPLICATIONS

PULP AMPUTATION (PULPOTOMY). This method of pulpitis treatment consists of in removing part of pulp mainly coronal pulp. The vital pulp amputation is the partial pulp removing under anaesthesia. Indications: Pulpotomy is indicated in cases of irreversible pulpitis, mainly pulp hyperemia (in case of ineffective biological treatment), acute traumatic pulpitis (accidentally pulp wounded) and acute circumscription pulpitis in young patients. Also this method may be effective for treatment of chronic fibrous pulpitis, chronic hypertrophic pulpitis in young patients with incomplete root formation. Technique After careful antiseptic irrigation of oral cavity the dentist provide proper local anesthesia. The tooth isolate by cotton roll or rubber dam. The teeth surfaces wipe with 2% iodine solution, 1% chlorhexidine solution or 3 other antiseptics. The thorough preparation of caries cavity carried out.

During preparation the roof of the pulp chamber is best perforated with a round bur. This bur is 2 used to remove the roof of the pulp chamber from underneath to establish outline form. The outlines of caries preparation must coincide with outlines of pulp cham-ber. The pulp chamber should be frequently flushed with a sodium hypochlorite solution to remove debris. All of the tissue in the pulp chamber should be removed by the round bur or a sharp spoon excavator. The tissue is carefully curetted from the pulp horns and other ramifications of the chamber.

Failure to remove all tissue fragments from the pulp chamber may result in later discolouration of the tooth. At this point, the chamber should be irrigated well to remove blood and debris. It is very important to carefully control bleeding because the blood clot will hinder the dentin bridge formation. For the control of bleeding 3% hydrogen peroxide solution, 5% epsilon-amino-capric-acid (EACA) solution is used. After the control of bleeding and thorough irrigation of the cavity on the pulp stump medicament paste is placed. As usual the pastes with calcium hydroxide are used (see the pastes for the biological treatment of pulpitis). These pastes placed on the pulp stumps without any pressure and caries cavity hermetically sealed with temporary dressing. After 10-14 days when pain is absent the temporary dressing is changed on final restoration from composites or amalgam. Variant of vital pulpotomy in young patients with incompletely developed roots is called apexification. This procedure requires complete canal cleaning, shaping, removal of smear layer and disinfection before the apical placement of this material is accomplished using calcium hydroxide. The calcium hydroxide kills bacteria and creates an environment conducive for hard tissue formation. The material is left in place or changed every 3 to 6 months in an attempt to enhance the tissue response. During this time period the root hard tissue develops and the root formation is ended.



Pic.16. Schematic image of pulpotomy

METHODOLOGICAL RESEARCH №13

TOPIC: VITAL EXTIRPATION METHOD OF PULPITIS TREATMENT: INDICATIONS, TECHNIQUES, MEDICINES. EFFICACY AND POSSIBLE COMPLICATIONS. DIATHERMOCOAGULATION. INDICATIONS, TECHNIQUES

PULPECTOMY. The main treatment of pulp inflammation consists in removing a vital pulp from pulp chamber (pulp amputation) and root canal. This is termed pulp extirpation or pulpectomy. Total pulpectomy, extirpation of the pulp to or near the apical fora-men, is indicated when the root apex is fully formed and the foramen sufficiently closed to permit obturation with conventional filling materials. If the pulp must be removed from a tooth with an incompletely formed root and an open apex, partial pulpectomy (pulp amputation) is preferred. This technique leaves the apical portion of pulp intact with the hope that the remaining stump will encourage completion of the apex. In a number of instances, restorative and fixed prosthetic procedures require intentional extirpation. Technique The following are the steps in the performance of a well-executed pulpectomy:

1. Obtain regional anaesthesia.
2. Prepare a minimal coronal opening and, with a sharp explorer, test the pulp for depth of anaesthesia.
3. If necessary, intrapulpal injection of anaesthetic.
4. Complete the access cavity.
5. Excavate the coronal pulp (amputation).
6. Extirpate the radicular pulp.
7. Control bleeding and de-bride and shape the canal.
8. Place medication or the final filling.

Each of these steps must be completed carefully before the next is begun, and each requires some explanation.

The popular misconception that endodontic treatment invariably involves suffering will not be completely dispelled until all practitioners employ effective anaes-thesia techniques while completing procedures as potentially painful as pulpectomy. It is wise to anticipate that, in spite of apparently profound anaesthesia, an intra-ligamentary or intrapulpal injection may be required to obtain total anesthesia, particularly with an inflamed pulp. If the patient experiences pain during the inily, the anaesthetic can be forced into the pulp under pressure. Total anaesthesia follows immediately. Completion of the Access Preparation Coronal access must be adequate and complete to allow thorough excavation of the tissue from the pulp chamber. Because intrapulpal injection with 2% lidocaine or articaine with 1:50,000 epinephrine promotes excellent hemostasis, it can be used during the completion of the access cavity to prevent interference from haemorrhag-ing tissue. Excavation of the coronal pulp All of the tissue in the pulp chamber should be removed before extirpation of the radicular pulp is begun. All pulp tissue that has not been removed by the round bur should be eliminated with a sharp spoon excavator. The tissue is carefully curetted from the pulp horns and other ramifications of the chamber. Failure to remove all tissue fragments from the pulp chamber may result in later discoloration of the tooth. At this point, the chamber should be irrigated well to remove blood and debris.

Extirpation of Radicular Pulp. The instrument used for this procedure is determined by the size of the canal and/or the level at which the pulp is to be excised. Large Canal, Total Pulpectomy

If the canal is large enough to admit a barbed broach and a total pulpectomy is desired, the approach is as follows:

1. A pathway for the broach to follow is created by sliding a reamer, file, or pathfinder along the wall of the canal to the apical third. If the pulp is sensitive or bleeding, the anaesthetic syringe needle may be used as the «pathfinder». A drop of anaesthetic deposited near the apical foramen will stop the flow of blood and all pain sensations. At the same time, the needle displaces the pulp tissue and creates the desired pathway for a broach.
2. A broach, small enough not to bind in the canal, is passed to a point just short of the apex. The instrument is rotated slowly, to engage the fibrous tissue in the barbs of the broach, and then slowly withdrawn. Hopefully, the entire pulp will be removed with the broach. If not, the process is repeated. If the canal is large, it may be necessary to insert two or three broaches simultaneously to entwine the pulp on a sufficient number of barbs to ensure its intact removal.
3. If the pulp is not removed intact, small broaches are used to «scrub» the canal walls from the apex outward to remove adherent fragments. A word of caution: The barbed broach is a friable instrument and must never be locked into the canal. Handle with care!

Control of Bleeding and Debridement of Canal Incomplete pulpectomy will leave in the canal fragments of tissue that may remain vital if their blood supply is maintained through accessory foramina or along deep fissures in the canal walls. These remnants of the pulp may be a source of severe pain to the patient, who will return seeking relief as soon as the anaesthesia wears off. This is a desperately painful condition and requires immediate re-anaesthetization and extirpation of all tissue shreds. Any overlooked tissue will also interfere with proper obtu-ration during immediate filling procedures.

Persistent bleeding following extirpation is usually a sign that «tags» of pulp tissue remain. If the flow of blood is not stopped by scrubbing the canal walls with a broach, as described above, it may originate in the periradicular area. In these cases, it is best to dry the canal as much as possible after irrigating with anaesthetic. A dry cotton pellet is then sealed in until a subsequent appointment.

Placement of Medication or Root Canal Filling. If pulpectomy was necessitated by pulpitis resulting from operative or accidental trauma, or planned extirpation of a normal pulp for restorative purposes was done, cleaning and shaping and obturation of the canal can be completed immediately. If a delay is necessary, a drug of choice or dry cotton should be sealed in the chamber. The final canal filling should never be placed, however, unless all pulpal shreds are removed and haemorrhage has stopped. Immediate filling is contraindicated if the possibility of pulpal infection exists. Intracanal Medication Antibacterial agents such as calcium hydroxide are recommended for use in the root canal between appointments. While recognizing the fact that most irrigating agents destroy significant numbers of bacteria during canal debridement, it is still thought good form to further attempt canal sterilization between appointments.

Pic.17. Hand files.

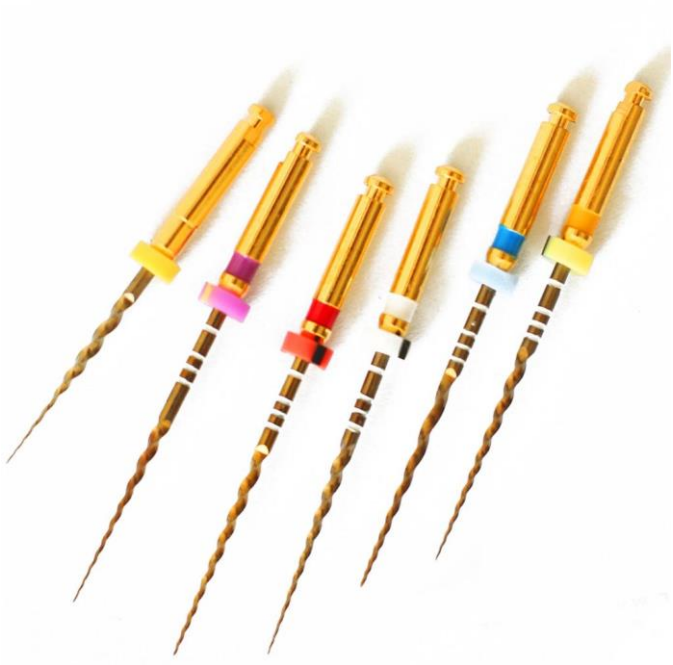


Рис.18. Mechanical files.

Рис.19. Schematical image of diathermcoagulation.

METHODOLOGICAL RESEARCH №14

TOPIC: DEVITALISATION OF THE PULP: INDICATIONS, METHODS, MEDICINES. DEVITALISATION OF PULP. INDICATIONS, STAGES OF TREATMENT, MODERN TECHNOLOGIES. POSSIBLE COMPLICATIONS AND THEIR ELIMINATION. EFFECTIVENESS OF THE METHOD.

The necrotic or «mummified» tissue remaining in the pulp cavity of a pulpless tooth has lost itsidentify as an organ; hence, its removal is called pulp cavity debridement. Indications Pulp «mummification» with either arsenic trioxide, formaldehyde, or other destructive compounds was at one time preferable to extirpation. With the advent of effective local anaesthetics, pulpectomy has become a relatively painless process and superseded «mummification», with its attendant hazards of bone necrosis and prolonged postoperative pain.

The necessary quantity of devitalizing paste with a probe (without pressure) to enter into a pre-cleaned from carious dentin and an open cavity. The amount of paste is determined individually. In the treatment of pulpitis of temporary teeth, as well as single rooted teeth in adults is enough to put the paste in an amount equal to the size of a millet grain.

For pulp devitalization of multi rooted teeth amount of paste must be doubled. After applying the paste it’s necessary to close hermetically the cavity by temporary filling material. The complete devitalization of the pulp takes place in 3-5 days. If the patient does not feel pain, permanent filling can be carried out within 24-48 hours after application of the paste.

If there is no direct contact with the pulp devitalization should be carried out in two stages. Direct contact may be achieved only after the second stage reducing the viability of the pulp. After devitalization and extirpation of the pulp needed instrumental and pharmacological root treatment. With increasing of pulpits pain after the imposition devitalizing paste (not opened pulp horn or paste imposed very tight) it is necessary to make infiltration anesthesia with lidocaine or another anesthetics.

METHODOLOGICAL RESEARCH №15

TOPIC: DEVITALISED AMPUTATION AND COMBINED METHODS OF PULPITIS TREATMENT. INDICATIONS FOR USE. METHODOLOGY: STAGES, MEDICINES. DISADVANTAGES AND POSSIBLE COMPLICATIONS

Placement of Medication or Root Canal Filling. If pulpectomy was necessitated by pulpitis resulting from operative or accidental trauma, or planned extirpation of a normal pulp for restorative purposes was done, cleaning and shaping and obturation of the canal can be completed immediately. If a delay is necessary, a drug of choice or dry cotton should be sealed in the chamber. The final canal filling should never be placed, however, unless all pulpal shreds are removed and haemorrhage has stopped. Immediate filling is contraindicated if the possibility of pulpal infection exists.

Intracanal Medication Antibacterial agents such as calcium hydroxide are recommended for use in the root canal between appointments. While recognizing the fact that most irrigating agents destroy significant numbers of bacteria during canal debridement, it is still thought good form to further attempt canal sterilization between appointments. Root canal preparation With the completion of the coronal access cavity, preparation of the radicular cavity may be started. Root canal preparation has two objectives: thorough debride-ment of the root canal system and the specific shaping of the root canal preparation to receive a specific type of filling. The first objective is achieved by skilful instrumentation coupled with liber-a irrigation. This double-pronged attack will eliminate most of the bacterial contaminants of the canal as well as the necrotic debris and dentin.

Cleaning and sanitizing the root canal have been likened to the removal of carious dentin in a restorative preparation - that is, enough of the dentin wall of the canal must be removed to eliminate the attached necrotic debris and, insofar as possible, the bacteria and debris found in the dentinal tubuli. Along with repeated irrigation, the instruments for debridement must be constantly cleaned. Sterile gauze square soaked in alcohol is used to wipe the instruments.

Over the years, two different approaches to root canal cleaning and shaping have emerged: the «step-back» and the «step-down» preparations.

The step-back preparation is based upon the traditional approach: beginning the preparation at the apex and working back up the coronal canal with larger and larger instruments.

The step-down preparation, often called «the crown-down approach», begins at the coronal and the preparation is advanced apically, using smaller and smaller instruments, finally terminating at the apical stop. Chemomechanical Debridement: The pulp chamber and root canals of untreated non-vital teeth are filled with a gelatinous mass of necrotic pulp remnants and tissue fluid. Essential to endodontic success is the careful removal of these rem-nants, microbes, and dentinal filings from the root canal system. The apical portion of the root canal is especially important because of its relationship to the perira-dicular tissue. Although instrumentation of the root canal is the primary method of canal debridement, irrigation is a critical adjunct. Irregularities in canal systems such as narrow isthmi and apical deltas prevent complete debridement by mechanical instrumentation alone. Irrigation serves as a physical flush to remove debris as well as serving as a bactericidal agent, tissue solvent, and lubricant. Furthermore, some irrigants are effective in eliminating the smear layer.

Root Canal Irrigants: A wide variety of irrigating agents are available. Sodium hypochlorite is one of the most widely used irrigating solutions. Household bleach such as Clorox contains 5.25% sodium hypochlorite. Some suggest that it be used at that concentration, whereas others suggest diluting it with water, and still others alternate it with other agents, such as ethylenediaminetetraacetic acid with cen-trimide (EDTAC) or chlorhexidine. By combining 5.0% sodium hypochlorite with EDTA, however, the bactericidal effect was considerably

enhanced. Sodium hypochlorite is an effective antimicrobial agent, serves as a lubricant during instrumenta-tion, and dissolves vital and no vital tissue.

Chlorhexidine gluconate is an effective antimicrobial agent, and its use as an endodontic irrigant has been well documented. It possesses a broad-spectrum anti-microbial substantive action and a relative absence of toxicity. The alternate use of sodium hypochlorite and chlorhexidine gluconate irrigants resulted in a greater reduction of microbial flora (84.6%) when compared with the individual use of sodium hypochlorite (59.4%) or chlorhexidine gluconate (70 %) alone. Regardless of the delivery system, the solution must be introduced slowly and the needle never wedged in the canal. Several types of plastic disposable syringes are available. The syringe is filled by immersing the hub into the solution while withdrawing the plunger. It is strongly recommended that the needle lie passively in the canal and not engage the walls. However, the closer the needle tip is placed to the apex, the greater the potential for damage to the periradicular tissues.

The determination of an accurate working length is one of the most critical steps of endodontic therapy. The cleaning, 0,5-1,0mm shaping, and obturation of the root canal system cannot be accomplished accurately unless the working length is determined precisely.

Working lengths defined in the endodontic Glossary as «the distance from a coronal reference point to the point at. Diagrammatic view of the periapex which canal preparation and obturation should terminate». Before cleaning and shaping are undertaken, the length of each canal must be established. This can be done radio-graphically or electronically.

One must first establish in one's own mind just where the preparation and obturation of the canal should terminate. It has long been suggested that the minor diameter at the cement dentinal junction is often the narrowest sie of the apical foramen, the apical constriction, and that this is where the apical stop should be established. Measurements have shown this site to be from 0.5 to 10 mm from the major diameter, the radiographic apex. So, if the full length of the canal is determined, 0.5 to 1.0 mm should be subtracted to stay within the confines of the canal and terminate at its narrowest point. This should be the working length. Some would argue that preparation and obturation should be ended at apical stop.

Determination of Working Length by Radiographic Methods: To establish the length of the tooth, a stainless steel reamer or file with an instrument stop on the shaft is needed. The exploring instrument size must be small enough to negotiate the total length of the canal but large enough not to be loose in the canal. Measure the tooth on the preoperative radiograph. Place the instrument in the canal until the stop is at the plane of reference unless pain is felt (if anesthesia has not been used). Expose, develop, and clear the radiograph. On the radiograph, measure the difference between the end of the instrument and the end of the root and add this amount to the original measured length the instrument extended into the tooth. If, through some oversight, the exploring instrument has gone beyond the apex, subtract this difference. From this adjusted length of tooth, subtract a 1.0 mm «safety factor» to conform to the apical termination of the root canal at the apical constriction.

Determination of Working Length by Electronics: The appliance «apex loca-tor» is commonly used. It is a simple direct current ohmmeter to measure a constant resistance of 6.5 kilo ohms between oral mucous membrane and the periodontium regardless of the size or shape of the teeth. One side of the apex locator's circuitry is connected to an endodontic instrument. The other side is connected to the patient's body, either by a contact to the patient's lip or by an electrode held in the patient's hand. The electrical circuit is complete when the endodontic instrument is advanced apically inside the root canal until it touches periodontal tissue. The display on the apex locator indicates that the apical area has been reached. Techniques of radicular cavity preparation:

Over the years, there has been a gradual change in the ideal configuration of a prepared root canal. After Schilder's classic description of «cleaning and shaping», the more accepted shape

for the finished canal has become a gradually increasing taper, with the smallest diameter at the apical constriction, terminating larger at the coronal orifice. Two approaches to debriding and shaping the canal have finally emerged: either starting at the apex with fine instruments and working one's way back up (or down) the canal with progressively larger instruments - the «step-back» or serial technique — or the opposite, starting at the cervical orifice with larger instruments and gradually progressing toward the apex with smaller and smaller instruments - the «step-down» technique, also called «crown-down» filing.

Step-Back Preparation: This preparation divided into two phases.

Phase I is the apical preparation starting at the apical constriction. Phase Il is the preparation of the remainder of the canal, gradually stepping back while increasing in size. The completion of the preparation is the Refining Phase IIA and IIB to produce the con-inuing taper from apex to cervical Prior to the introduction of nickel-titanium files, one of the first axioms of end-odontics has been to «always use a curved instrument in a curved canal».

The degree and direction of the curve are determined by the canal shadow in the radiograph.

Phase 1: To start Phase I instrumentation, it must be assumed that the canal has been explored with a fine pathfinder or instrument and that the working length has been established - that is, the apical constriction identified. The first active instrument to be inserted should be a fine (No. 08, 10, or 15) 0.02, tapered, stainless steel file, curved and coated with a lubricant, such as Gly-Oxide, R.C. Prep, File-Eze, Glyde, K-Y Jelly, or liquid soap.

The motion of the instrument is «watch winding», two or three quarter-turns clockwise-counter clockwise and then retraction. On removal, the instrument is wiped clean, recurved, re-lubricated and repositioned. «Watch winding, is then repeated. Remember that the instrument must be to full depth when the cutting action is made. This procedure is repeated until the instrument is loose in position. Then the next size file is used — length established, pre-curved, lubricated, and portion ing strokes an a rinding action and retraction are repeated.

Very short (10 mm filime a size 25ak also be used at the apex. By the time a size 25 K file has been used to full working length, Phase I is complate. The s his area on space back from the apical coking lens, put itcan o debris unless this astruments canal was large to begin with, as in a youngster. Theon, of course, larger instruments are used to start with. Using a num apical rese here as an example is not to imply that all canals should be shaped at the apical restriction only to size 23. Many, in fact most, canais should be enlarged beyont and 25 at the apical constriction in order to round, out the prepa-rion at this point and remove as much of the extraneous tissue, debris, and lateral canals as possible.

A size 25 file is used here as an example and as a danger point for beyond No. 25 lies danger!

Phase II. In a fine canal (and in this example), the step-back process begins with a No. 30 K-style file. Its working length is set I mm short of the full working length. It is pre-curved, lubricated, carried down the canal to the new shortened depth, watch wound, and retracted. The same process is repeated until the No. 30 is loose at this adjusted length. Recapitulation to full length with a No. 25 file follows to ensure patency to the constriction. This is followed by copious irrigation before the next curved instrument is introduced. In this case, it is a No. 35, again shortened by 1.0 mm from the No. 30 (2.0 mm from the apical No. 25). It is curved, lubricated, inserted, watch wound, and retracted followed by recapitulation and irrigation. Thus, the preparation steps back up the canal 1mm and one larger instrument at a time. When that portion of the canal is reached, usually the straight mid canal, where the instruments no longer fit tightly, then perimeter filing may begin, along with plenty of irrigation. It is at this point that Hedstroem files are most effective.

They are much more aggressive rasps than the K-files. The canal is shaped into the continuous taper so conducive to optimum obturation. Care must be taken to recapitulate between each instrument with the original No. 25 file along with ample irrigation. This mid canal area is the region where reshaping can also be done with power-driven instruments: Gates-Glidden drills,

starting with the smaller drills and gradually increasing in size to No. 4, 5, or 6. Proper continuing taper is developed to finish Phase IIA preparation. Refining Phase IIB is a return to a size No. 25 (or the last apical instrument used, smoothing all around the walls with vertical push-pull strokes, to perfect the taper from the apical constriction to the cervical canal orifice. In this case, a Sale-ended, non-cutting-tip Hedstroem file is the most efficient. It produces a good deal of dentin chips, however, that must be broken up at the apex with a cutting-tip K file and then flushed out with abundant sodium hypochlorite. This completes the chemomechanical step-back preparation of the continuing taper canal. It is now ready to be filled or medicated and sealed at the corone cavity until the next appointment. If it is to be filled, the smear layer should first be remove.

Step-Down Technique - «Crown-Down Less Pressure Preparation» in which Gates-Glidden drills and larger files are first used in the coronal two-thirds of the canals and then progressively smaller files are used from the «crown down» until the desired length is reached. This has become known as the step-down or crown-down technique of cleaning and shaping. A primary purpose of this technique is to minimize or eliminate the amount of necrotic debris that could be extruded through the apical foramen during instru-mentation. This would help prevent post-treatment discomfort, incomplete cleans-ing, and difficulty in achieving a biocompatible seal at the apical constriction. Also emphasized the importance of removing all pulp remnants before shaping begins to ensure that this tissue does not «pile up» at the constriction and impede full cleaning and shaping to that point. In this method, the access cavity is filled with sodium hypochlorite, and the first instrument is introduced into the canal. One should start with a wider (0.04 or 0.06 taper) instrument or a Gates-Glidden drill to free up the canal so that a fine instrument may reach the mid- and apical canal.

This would be the beginning of step-down preparation. The initial penetrating instrument is a small, curved, stainless steel K file, exploring to the apical constriction and establishing working length. To ensure this pen-etration, one may have to enlarge the coronal third of the canal with progressively smaller Gates- Glidden drills or with instruments of larger taper such as the.04 or the. 06 instruments. At this point, and in the presence of sodium hypochlorite and/or a lubricant such as Glyde, step-down cleaning and shaping begins with K-Flex, Tri-ple-Flex, or Safety Hedstrum (Sybron Endo/Kerr; Orange, Calif.) instruments in 0.02, 0.04, or 0.06 taper configurations depending on the canal size to begin with. Starting with a No. 50 instrument (for example) and working down the canal to, say, a size No. 15, and the instruments are used in a watch-winding motion until the apical constriction (or working length) is reached. When resistance is met to further penetration, the next smallest size is used.

Irrigation should follow the use of each instrument and recapitulation after every other instrument. To properly enlarge the apical third, and to round out ovoid shape and lateral canal orifices, a reverse order of instruments may be used starting with a No. 20 (for example) and enlarging this region to a No. 40 or 50 (for example). The tapered shape can be improved by stepping back up the canal with ever larger instruments, bearing in mind all the time the importance of lubrica-tion, irrigation, and recapitulation. At this point, the canal should be ready for smear layer removal, drying, and either medication or obturation. Over the past few years, the movement toward using rotary nickel-titani-um instruments for root canal preparation has resulted in a multitude of instrumentation systems in the marketplace. The manufacture of variably tapered and «Gates-Glidden-like», flexible nickel-titanium instruments, for use in gear-reduc-tion, slow-speed handpieces, either air driven or electric, has enabled the skilled clinician to deliver predictable canal shapes with enhanced speed and increased efficiency.

Root canal preparation is considered finished, if a root canal corresponds with such requirements: - it is fully free from an infectious dentin; - has a tapered shape from cervical orifice of the root canal to apex; - it is enough enlarged; - has the formed apically support; - dry, clean, sterile. Obturation of root canal Nearly 60% of the failures in the endodontic treatment were apparently caused by incomplete obliteration of the radicular space. Periradicular inflammation is presumed to persist under the influence of any oxious substance. Bacteria certainly play a major role in the production of toxic products in the root canal.
However, in the absence of bacteria, degraded serum per se may well assume the role of the primary tissue irritant. The persistence of perira-dicular inflammation, in the absence of bacterial infection, might thus be attributed to the continuing apical percolation of serum and its breakdown products. It is apparent that the preliminary objectives of operative endodontics are total debridement of the pulpal space, development of a fluid-tight seal at the apical fora-men, and total obliteration of the root canal. The anatomic limits of the pulp space are the dentinocemental junction apical-ly, and the pulp chamber coronal. The root canal is ready to be filled when the canal is cleaned and shaped to an optimum size and dryness. Dry canals may be obtained with absorbent points except in cases of apical periodontitis or apical cyst, in which «weeping» into the canal persists. The materials used to fill root canals have been legion, running the gamut from gold to feathers.
Grossman grouped acceptable filling materials into plastics, sol-ids, cements, and pastes. He also delineated 10 requirements for an ideal root canal filling material that apply equally to metals, plastics, and cements: (see chapter 10 «Apical Periodontitis» - «Obturation of root canal»). Cements, plastics, and pastes The cements, which have a wide dentist's acceptance, are primarily zinc oxide-eugenol (ZOE) cements, the polyketones, and epoxy. The pastes currently in worldwide vogue are chlorapercha and eucapercha, as well as the iodoform pastes, which include both the rapidly absorbable and the slowly absorbable types. Despite their disadvantages, pastes are applicable in certain cases. The plastics show prom-ise, as do the calcium phosphate products. At present the methods most frequently used in filling root canals involve the use of solid-core points that are inserted in conjunction with cementing materials. Gutta-percha and silver per se are not considered adequate filling material unless they are cemented in place in the canal.

METHODOLOGICAL RESEARCH №16

TOPIC: SEALANTS AND FILLERS FOR ROOT CANAL FILLING IN CASE OF PULPITIS: GROUPS, PROPERTIES, INDICATIONS FOR USE, FILLING TECHNIQUES. METHODS OF ROOT CANAL FILLING: METHODS OF CENTRAL PIN, VERTICAL CONDENSATION OF GUTTA-PERCHA, COLD AND WARM LATERAL CONDENSATION OF GUTTA-PERCHA. MISTAKES AND COMPLICATIONS

The sealers are to form a fluid-tight seal at the apex by filling the minor interstices between the solid material and the wall of the canal, and also by filling patent accessory canals and multiple foramina. Dye-immersion studies have shown the necessity of cementation, without which dye penetrates back into the canal after compaction; this occurs with all known solid-core root canal-filling techniques.

Gutta-percha is by far the most universally used solid-core root canal filling material and may be classified as a plastic. To date, modern plastics have been disappointing as solid-core endodontic filling materials. Chemically pure gutta-percha (or balata) exists in two distinctly different crystalline forms (alpha and beta) that can be converted into each other.

The alpha form comes directly from the tree. Most commercial gutta-percha, however, is the beta crystalline form. There are few differences in physical properties between the two forms, merely a difference in the crystalline lattice depending on the annealing and/ or drawing process used when manufacturing the final product. Traditionally, the beta form of gutta-percha was used to manufacture endodontic gutta-per-cha points to achieve an improved stability and hardness and reduce stickiness. Although techniques of gutta-percha placement involving heating in the root canal caused reversible physical changes, no apparent changes in chemical composition take place. For endodontic usage the gutta-percha supplied by manufacturers in form of gutta-percha filling points which contain about 20% of its chemical composition of gutta-percha, whereas the 60 to 75% of the composition is zinc oxide filler.

The remaining constituents are wax or resin to make the point more pliable and/ or compactable and metal salts to lend radiopacity. Gutta-percha points (or cones) are supplied in two shapes. The traditional form is cone shaped to conform to the perceived shape of the root canal. The other shape of gutta-percha points is standardized to the same size and shape as the standardized (ISO) endodontic instruments. These points are available in the standardized.02 taper as well as in increased taper sizes (.04,.06, etc) to correspond to the newer tapered instrument sizes. Colour coding the numbered points to match ISO instrument colour has become routine. Methods of obturating the root canal space Today, most root canals are being filled with gutta-percha and sealers. The methods vary by the direction of the compaction (lateral or vertical) and/or the temperature of the gutta-percha; either cold or warm (plasticized). These are the two basic procedures: lateral compaction of cold gutta-percha or vertical compaction of warmed gutta-percha.

Other methods are variations of warmed gutta-percha. The main methods are listed as follows:

I. Solid Core Gutta-Percha with Sealants

A. Cold gutta-percha points

1. Lateral compaction

2. Variations of lateral compaction

B. Canal-warmed gutta-percha

1. Vertical compaction

C. Thermoplasticized gutta-percha

1. Solid-core carrier insertion

a. Thermafil

METHODOLOGICAL RESEARCH №17

TOPIC: PERIODONTITIS: ETIOLOGY, PATHOGENESIS, CLASSIFICATION (I.G. LUKOMSKYI, M.A. GROSHYKOV, S.A. WEINDRUKH, ICD-10). PATHOLOGICAL ANATOMY OF ACUTE AND CHRONIC PERIODONTITIS

Table 18 etiology of periodontitis

infectious	**uninfectious**
1. Mixed anaerobic and aerobic microflora 2. Aerobes in a-Streptococcus-Streptococcus Neisseria Staphilococcus albus Candida 3. Anaerobes B.perfingens V. V. mesentericus subtitis Str. putridus 4. Bacteroids Veilonella Fuzobakteries	1. Acute Traumatic household trauma. Deep extirpation of the pulp. Injury endodontic instruments. Chronic trauma (fillings, crowns). 2. Chemical Application of toxic arsenic preparations. Application paraformu. The use of antiseptics. The use of endodontic materials. 3. Allergic medications (iodine, formalin, antibiotics, etc.). Filling materials
Pathways of infection 1. A root canal 2. Marginal way 3. through hematogenous way 4. Contact way (in case of osteomyelitis, sinusitis)	

2. Marginal way. Often it is implemented in patients with periodontal disease. In the case of generalized or localized periodontitis broadcast broken tooth plate cortical alveolus, periodontal pocket is formed, from which the infection enters the periodontalnyy space and it causes inflammation of tissues.

3. Contact way. In patients with osteomyelitis or sinusitis infection with pathological focus moves to the contact teeth are located.

4. Hematogenous or lymphogenous way. In this way the infection gets in periodontal patients with infectious for sickness.

To be infectious and so-called periods of perifocal dontyt, which was described IG Lukomski yet in 1955 he develops in patients with acute and chronic pulpitis, pulp gangrene and other diseases when infection is at the root canal tooth and there is focus of the pathological process. With this focus in the periapical space penetrated individual bacteria and their toxins, causing an inflammatory reaction of periodontal tissues. After removal of the fire (eg removing infected pulp canal sterilization) inflammation periodically disappears. This inflammatory reaction periodontalnyh tissues in the tooth root apex area serves as a barrier that obstacles further penetration of the infection.

Noninfectious periodontitis caused by the influence of periodic various local and general factors.

1. The most common cause of periodontitis is a traumatic factors, mainly mechanical trauma - acute or chronic.
2. The second large group is chemical-toxic periodontitis.
3. Inflammation of Periodontal may cause allergic factors.

As noted, the traumatic factors that cause periodontitis include various injuries. Thus, acute trauma (shock, drop, etc..) Causing an acute traumatic injury. In such cases often suffer front teeth. Pathological changes vary depending on the strength and direction of the traumatic factor. In the case of small injuries may be breaks apical periodontal tissues, causing inflammatory reaction, and later it can pass and the pulp is alive.

Severe periodontal damage may result in not only the development of periodontitis, but also damage the pulp. Wang infected pulp complicates inflammation in periodontal.

Chronic trauma. With a weak, but the Permanent injury (eg, patients with abnormality of the bite, an artificial crown, denture or mostopodibnym seal that overstates the bite) forces traumatic focus in the area of the root apex. This injury causes gradual resorption of alveolar bone and the body also gradually compensates by its produkuvan ¬ tion of granulation or fibrous tissue in the region of injury.

In addition, periodontitis may occur in smokers, smoking his pipe, in the musicians that played on wind instruments, in individuals who have bad habits teeth bite off the end, biting a pencil and generally in all cases, when you create a constant pressure on land top of the tooth root. In this case the crown of the tooth can remain intact, although for a long in the process can lead to periodontal destruction pulpal tissue.

An individual case of periodontitis should be further characterised using a simple matrix of four steps (see: Periodontitis: clinical decision tree for staging and grading, part of this toolkit) that describes the stage and grade of the disease. There are four stages and three grades. Staging relies on the standard dimensions of the severity and extent of periodontitis at presentation but adds the complexity of managing the individual patient. The information derived from assessing the stage of periodontitis should be supplemented by information on the inherent biological grade of the disease.

This relies on three sets of parameters:

1. The rate of periodontitis progression;
2. Recognised risk factors for periodontitis progression;
3. The risk of an individual’s case affecting their systemic health. Within this classification framework, staging is largely dependent upon the severity of disease at presentation and on the complexity of disease management, while grading provides supplemental information about biological features of the disease. These features include a history-based analysis of the rate of periodontitis progression, assessment of the risk for further progression, analysis of possible poor outcomes of treatment, and assessment of the risk that the disease or its treatment may negatively affect the patient’s general health.

There are two dimensions in the process of assessing the stage of periodontitis in a patient: severity and complexity.

Severity: The primary goal is to classify the severity and extent of destroyed and damaged tissue caused by periodontitis. This is done by measuring CAL by clinical probing and bone loss by radiographic examination. These measurements must include the number of teeth whose loss can be attributed to periodontitis.

Complexity: The secondary goal is to determine the complexity involved in controlling the disease and managing the long-term function and aesthetics of the patient's dentition. Scoring the stages: The severity score is based primarily on interdental attachment loss attributable to periodontitis (CAL) and marginal bone loss. It is assigned based on the worst-affected tooth. The complexity score is based on the complexity of treating the case. It considers factors including the presence of deep probing depths, vertical defects, furcation involvement, tooth hypermobility, drifting and/or flaring of teeth, tooth loss, ridge deficiency, and loss of masticatory function.

Grading a periodontitis patient involves estimating the future risk of periodontitis progression and the likely responsiveness to standard therapeutic principles. This estimate guides the intensity of therapy and secondary prevention after therapy. Grading adds another dimension and allows the rate of progression to be considered, using direct and indirect evidence. Direct evidence is based on the available longitudinal observation: for example, in the form of older diagnostic-quality radiographs. Indirect evidence is based on the assessment of bone loss at the worst-affected tooth in the dentition as a function of age (measured as radiographic bone loss in percentage of root length divided by the age of the subject). The periodontitis grade can then be modified by the presence of risk factors.

Clinicians should approach grading by assuming a moderate rate of progression (grade B) and look for direct and indirect measures of whether there is a higher disease progression that would justify the application of grade C. Grade A is applied once the disease is arrested. If the patient has risk factors that have been associated with greater disease progression or lesser responsiveness to bacterial-reduction therapies, the grade score should be raised independently of the primary criterion represented by the rate of progression. For example, a case could be characterised by moderate attachment loss (stage II), where the assumption of a moderate rate of progression (grade B) is modified by the presence of poorly controlled Type-2 diabetes, which is a risk factor that could shift the grade definition to rapid progression (grade C).

METHODOLOGICAL RESEARCH №18

TOPIC: ACUTE DRUG-INDUCED AND TRAUMATIC APICAL PERIODONTITIS: ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSIS. ACUTE SEROUS PERIODONTITIS OF INFECTIOUS GENESIS: ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSTICS

Traumatic factors that cause periodontitis include various traumas. For example, an acute trauma (blow, fall, heavy object, etc.) causes acute traumatic injury. In such cases, the front teeth are most often affected. Pathological changes vary depending on the strength and direction of the traumatic factor. In the case of minor injuries, there may be tears in the apical periodontal tissue, which causes an inflammatory reaction, which may later resolve and the pulp remains alive.

Severe damage to the periodontium can cause not only the development of periodontitis, but also damage to the pulp. Infected pulp complicates the inflammatory process in the periodontium.

Chronic trauma. In case of a mild but permanent trauma (for example, in patients with an abnormal bite, artificial crown, bridge or overbite filling), the forces of traumatic action are concentrated in the area of the root apex. Such an injury causes gradual resorption of the alveolar bone, and the body also gradually compensates for it by producing granulation or fibrous tissue in the area of injury.

In addition, periodontitis can occur in pipe smokers, musicians playing wind instruments, people with bad habits of biting off the ends of threads with their teeth, chewing on pencils and in general in all cases when constant pressure is applied to the area of the apex of the tooth root. In this case, the crown of the tooth may remain intact, although a prolonged inflammatory process in the periodontium can lead to the death of the pulp tissue.

Traumatic periodontitis can be caused by the actions of the dentist who treated the inflamed pulp or by medical and instrumental treatment of the root canal. For example, during deep pulp extraction, periodontal tissue can be torn off, and over time, the periodontium can be damaged; in case of incorrect determination of the length of the canal, working with a file or a rimer can also lead to periodontal damage.

In the event of acute traumatic periodontitis, the patient experiences pain at the site of the knocked-down tooth, it becomes mobile, and may have bleeding gums, acute or aching pain.

The course of traumatic periodontitis is accompanied by the following symptoms:

- *Severe pain*

The cause of pain is a short impact of high force. This includes a blow or a fall, but in addition, discomfort can be caused by improperly prescribed and performed dental treatment. The pain occurs and intensifies during eating - biting and chewing, as well as when applying strong pressure to the tooth. It causes discomfort to the patient, but disappears for a while, which makes them postpone going to the doctor.

- *Redness and swelling of the mucous membrane*

Redness and swelling of the mucous membrane occurs for several reasons, they are accompanied by pain in the throat, gums, lips and tongue. The most common causes of redness and swelling are injuries or trauma to the oral cavity, such as eating too hot or hard food, poor dental treatment, and infectious inflammation. Periodontitis is characterised by swelling in the

gum area, the patient notices a deterioration in well-being and a feeling that the affected tooth has become higher than the others.

- *Mobility of the causative tooth*

The following classification was developed to determine the degree of tooth mobility:

1st degree. The tooth is unstable in relation to the adjacent teeth. The amplitude of swinging is no more than 1 mm

2nd degree. The mobility of the teeth increases. It is slightly more than 1 mm. The teeth wobble in different directions.

3rd degree. The teeth are loose in any direction. The patient can even lift one of them up.

4th degree. The tooth rotates around its axis.

- *Bleeding gums*

Bleeding gums are observed in various diseases of the oral cavity. In periodontitis, the ligaments that surround and hold the tooth are destroyed. Now the dental ligament is involved in the inflammatory process, and it is filled with blood vessels. Over time, the ligament begins to break down, and the teeth become loose and fall out.

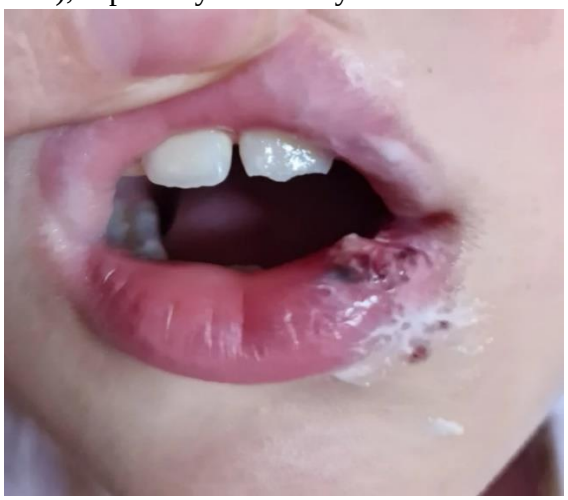
- *Tooth discolouration in pink colour*

The rupture of the neurovascular bundle is accompanied by haemorrhage into the pulp tissue, so the enamel turns pink. In some cases, the crown of the tooth darkens.

Toxicochemical (drug-induced) periodontitis occurs mainly after dental manipulations. In most cases, this is due to the use of arsenic paste, the effect of which extends to the periapical tissues in case of violation of its terms of use or dose.

Strong antiseptic agents (formalin, phenol, feresol, etc.) used to treat root canals can also cause irritation and inflammation of the periodontium.

Allergic periodontitis develops in patients with hypersensitivity to medications used for root canal treatment and filling. Quite often, allergic reactions occur after the use of iodine and iodide compounds (iodoform), especially when they are included in root canal filling pastes.



Pic.21. *Acute tooth trauma that leads to acute traumatic periodontitis.*

Acute serous periodontitis (periodontitis acuta serosa). In clinical practice, periodontitis is most commonly encountered under the influence of infection and develops mainly as a complication of pulp inflammation or as a result of errors made during endodontic therapy.

Symptoms. The patient's complaints are so characteristic that they are often enough to establish an almost unmistakable diagnosis. At first, the patient feels heaviness and tension in the tooth, which seems to be larger and longer than the others. Gradually, a rather severe pain of a spontaneous nature occurs. The pain is constant, localised, does not irradiate, worsens at night

and is almost not suppressed by common painkillers. As the process is constantly evolving, the intensity of the pain increases.

In addition, characteristic provoked pain may occur. Anything that can increase the blood flow in the tooth area and change its mobility provokes pain attacks. Thus, severe pain appears while eating. In the initial stage, however, deliberate, slow, long-term pressure relieves pain due to the outflow of exudate from the periodontium, reduced hyperaemia and compression of nerve endings. That is why, by pressing the tooth in the alveolus, patients temporarily improve their condition. Pain when pressing on the tooth can occur under the influence of heat if periodontitis is a complication of pulp gangrene with a closed tooth cavity. Temperature differences can cause pain if the change occurs suddenly. In the case of a gradual increase in temperature and prolonged exposure to heat, a calming effect is achieved due to sustained vasodilation, which promotes blood flow from the area of inflammation.

Objective. A diseased tooth may be intact, which does not exclude the presence of trauma (for example, when using an orthodontic appliance). Most often, however, it is carious, devitalised, with an open tooth cavity or filled with a large filling. The enamel loses its characteristic shine and becomes dark grey in colour. The gingiva in the apex area is often hyperaemic and swollen, sometimes hyperaemia is also present in the adjacent areas of the gingiva. Vertical percussion is painful. The reason for this reaction is an increase in the sensitivity of nerve receptors in the area of periapical inflammation. Palpation of the gums in the area of the tooth apex (especially the anterior ones) is painful, which is explained by the proximity of the root to the periosteum. Regional lymph nodes are enlarged and become painful during palpation. Depending on which lymph nodes are inflamed, in diagnostically difficult cases, a diseased tooth can be differentiated.

Thus, periodontitis of the lower front teeth is accompanied by inflammation of the submental lymph nodes, periodontitis of the upper incisors, as well as the upper and lower canines and premolars - by the anterior submandibular lymph node of the corresponding side, and periodontitis of the molars of both jaws - by the middle and posterior submandibular lymph nodes. Electrical excitability is above 100 microA, except in cases of traumatic periodontal injury, when the pulp is still alive and the response to direct current is related to its response.

Radiographic changes are usually undetectable, and only in the later stages of development may there be a slight widening of the periodontal gap. Depending on the etiology, the clinical picture of acute serous periodontitis may have its own specificity, which should be taken into account during the differential diagnosis. In patients with traumatic periodontitis, the clinical picture largely depends on the condition of the pulp that has been exposed to acute trauma. If the pulp is alive, the course of the process is milder and the prognosis is favourable. In the case of septic pulp necrosis, periodontal infection is always associated with a clinical picture of infectious periodontitis.

The differential diagnosis of acute serous periodontitis should be made with acute diffuse pulpitis. Pulpitis is characterised by pain radiation, acute onset, remissions and intermittent periodontitis. The pain in patients with periodontitis is duller, not as acute as in pulpitis. Lymph nodes are not affected in patients with pulpitis. The differential diagnosis between serous and purulent periodontitis is based on the severity of the patient's condition and the nature of the pain, as well as the overall clinical picture. In patients with serous periodontitis, the pain is less pronounced, not as intense, and strictly localised. Changes in the mucous membrane in the area of the root apex are minor, most often in the form of mild hyperaemia. The tooth is only slightly mobile in the transverse direction. The patient's general condition is not affected.

METHODOLOGICAL RESEARCH №19

TOPIC: ACUTE PURULENT APICAL PERIODONTITIS: ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSTICS

Determining the phase of purulent periodontitis is important for the choice of treatment methods, as each of these phases requires specific measures. With the equalisation of the forces of the damaging factor and the body's defences, proliferation processes begin to manifest themselves. Granulation tissue, which develops directly after the processes of alteration and exudation, restores the damaged periodontium and alveolar bone. In the future, it can act as a protective barrier that neutralises bacteria, toxins and other irritants.

Acute purulent periodontitis (periodontitis acuta purulenta) usually develops after serous periodontitis. However, it can often start spontaneously in case of massive penetration of virulent infection into the periodontium and a decrease in the reactivity of the patient's body. The clinical picture of such periodontitis is quite typical. Compared to the serous form, its course is more violent, and general manifestations are pronounced. Purulent exudate formed in the periodontal space, which is looking for a way out, most often breaks through, destroying periodontal tissue.

Patients complain of spontaneous acute continuous throbbing pain. At the beginning of the process, the pain is localised. However, it soon becomes diffuse, irradiating from the teeth of the lower jaw to the ear, and from the upper teeth to the temporal region. The patient always points to the diseased tooth, which they feel as "higher", very painful when pressing, contacting antagonists or even touching with the tongue during conversation. The pain is aggravated by heat, while cold, on the contrary, has a calming effect. Any physical effort leads to increased pain.

Objectively. A diseased tooth may be intact, although its colour may be discoloured, sometimes there is a significant carious defect or filling. The pulp cavity is closed in most cases, but may be open. Electrical excitability is 120-150 μA, which determines pulp necrosis. During probing, gangrenous decay is observed in the canals, and pus often comes out under pressure. Horizontal and vertical percussion of the tooth is very painful. The tooth is mobile in the mesiodistal direction and in the direction of the longitudinal axis. The mobility is especially significant if the pus reaches the circular ligament and seeks an exit in the gingival pocket. In this case, the tooth seems to be floating in the accumulation of pus. The tooth seems to have grown, which is not only subjectively felt by the patient, but is determined during the examination, as it is indeed somewhat displaced from the alveolus by the inflammatory exudate accumulated deep inside. The mucous membrane in the area of the apex is hyperemic and swollen. The transitional fold is smoothed due to the accumulation of inflammatory infiltrate, very painful during palpation. Depending on the stage of development of purulent periodontitis, palpation may reveal extremely painful hardening of the periosteum in the case of subperiosteal abscess formation.

In the case of a submucosal abscess, palpation reveals not only painfulness but also the phenomenon of fluctuation, collateral changes in the form of edema of the soft tissues of the face, the size of which does not always correspond to the severity of the lesion. Edema can lead to significant asymmetry and deformation of the face, especially in the case of pasty tissues. The presence of collateral edema should always be differentiated from phlegmon, but phlegmon is characterised by severe pain, as well as tension, thinness and shine of the skin. The progression of purulent exudate and the location of the abscess depend on the location of

the root that is the source of infection and the anatomical and histological features of the jaw area. In some cases, pus collected in the periodontium can be discharged through the tooth canal. This is the most favourable option for the evacuation of pus, but it is possible only if the canal is passable and open. Often, in the case of lower molar disease, pus flows marginally through the gingival pocket, which occurs after the circular periodontal ligament has melted. This pathway is unfavourable, as the cortical lamina is subsequently melted and a bone pocket is formed.

In addition to these pathways, purulent exudate can break through into the maxillary sinus or adjacent alveoli, penetrate the jawbone, and into the spongy substance. Under such conditions, limited osteomyelitis develops. This is a particularly unfavourable variant of pus spread, which leads to severe complications. In case of purulent periodontitis, the inflammatory reaction spreads to the regional and even cervical lymph nodes, which become painful and enlarged. Unlike serous periodontitis, purulent forms are most often accompanied by general symptoms. General disorders are observed in case of subperiosteal abscess formation, when unbearable pain, intoxication, general exhaustion, changes in complexion, and characteristic shadows under the eyes occur against the background of high body temperature (38-39 °C). Abuse of analgesics worsens the condition. Patients complain of headache, dizziness, and weakness.

Radiographic examination of purulent periodontitis in 24 to 48 hours reveals a darkening of the structure of the cancellous part of the bone due to bone marrow infiltration. The outlines of the compact lamina remain smooth and clear. In the case of severe collateral edema, the image of bone structures is superimposed by a light shadow of infiltrated, swollen soft tissue. The periodontal cleft is dilated. Radiography in patients with acute purulent periodontitis is advisable mainly for differential diagnosis with exacerbation of various forms of chronic periodontitis, when the image reveals changes characteristic of resorptive processes.

Differential diagnostics. The differential diagnosis between purulent and serous periodontitis is not difficult. Intense, unbearable pulsating pain with radiation is indicative of purulent periodontitis. The pain increases when pressing on the tooth or even touching it; tooth mobility is more pronounced, and mobility in the longitudinal axis is also characteristic; in case of circular ligament melting, the tooth seems to float in purulent exudate. The presence of abscess, pus discharge and general condition of the patient leave no doubt about the diagnosis. Acute purulent periodontitis, especially with severe general symptoms, must be differentiated from osteomyelitis. In the case of spontaneous osteomyelitis, the patient's general condition is quite severe. In patients with purulent periodontitis, the general intoxication is less pronounced, and local inflammatory changes affect only one or neighbouring teeth, and not a group of teeth or half of the jaw, as is the case with osteomyelitis. An X-ray examination will help to establish the final diagnosis.

Acute purulent periodontitis should also be differentiated from acute chronic periodontitis. Anamnestic data indicating the primary nature of the disease and relatively slow development of abscesses are in favour of purulent periodontitis. Whereas in patients with acute purulent periodontitis an abscess develops within 3-4 days, in the case of acute periodontitis, abscess formation occurs within one day or even several hours due to destructive changes in the bone tissue. During clinical examination of patients with inflammatory chronic periodontitis, especially granulating periodontitis, fistulas or scars are found. The absence of changes in the periapical area on X-rays confirms the diagnosis of acute purulent periodontitis. If the periodontitis is in the area of the upper 4, 5, 6 teeth, a differential diagnosis with maxillary sinusitis should be made. Patients with maxillary sinusitis complain of spontaneous pain in the

maxillary sinus area, which radiates to the posterior regions; percussion of several teeth, the roots of which are close to the floor of the maxillary sinus, is painful. In addition, in the case of a comparative examination of both sinuses, pain is detected when pressing over the affected sinus. Purulent exudate is also characteristic of the nostril, which is usually closed on the side of the affected maxillary sinus. Acute purulent periodontitis becomes chronic after drainage and drainage of purulent exudate.

METHODOLOGICAL RESEARCH №20

TOPIC: CHRONIC FIBROUS, GRANULATING, GRANULOMATOUS PERIODONTITIS: ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSTICS, DIFFERENTIAL DIAGNOSIS

Chronic fibrous periodontitis (periodontitis chronica fibrosa).

Symptoms. Chronic fibrous periodontitis is asymptomatic, only sometimes patients experience slight pain when chewing on rough food. The same can be determined in pulp gangrene if the carious cavity is filled with food debris. The disease is detected by radiography. From the anamnesis, it is established that earlier (1-2 years ago) the patient had spontaneous or causal pain and the tooth root was treated.

Objectively. A carious or filled devitalised tooth is detected. There is no pain from thermal stimuli and percussion. Palpation in the apex area is painless. If fibrous periodontitis has developed after treatment of acute purulent or chronic granulating periodontitis, an old scar may be detected. Sometimes fibrous periodontitis can occur in patients with intact teeth. In such cases, fibrous periodontitis is caused by chronic trauma or traumatic occlusion. Radiographically, the most commonly detected enlargement of the periodontal gap in the apical region is a sharp-edged cap. The compact alveolar lamina and root cementum are completely preserved.

In other cases, hypercementosis of the tooth root is detected, which causes its thickening in the apical part - the root looks like a drumstick. Hypercementosis is characterised by a positive immunological state of the body and a slow course of the chronic process. At the same time, hypercalcification of the alveolar bone can be observed, which looks like an osteosclerotic roller on the periphery of the periodontal fibrosis area on the radiograph.

Chronic granulomatous periodontitis occurs as a consequence of an acute inflammatory process, especially after acute purulent periodontitis, and can also develop in patients with chronic pulpitis, especially gangrenous pulpitis. The formation of granulation tissue rich in capillaries and fibroblasts indicates a higher level of the body's defences. Under the influence of stimuli from the root canal, the entire apical part of the periodontium is replaced by granulation tissue. As it grows, granulation tissue resorbs the adjacent layer of cement, on the one hand, and destroys the compact alveolar plate, on the other. Osteoblasts penetrate into the bone marrow cavities, resorbing the bone beams.

The granulation process can spread to soft tissues and create a gingival or skin fistula. The activation of the pathogenic factor causes an acute inflammatory process in the area of the root apex, and then the granulation tissue undergoes partial destruction. If at this stage the body's defences are superior, the granulation tissue restores the destroyed cells. It is this kind of periodontitis that is characterised in the clinic as unsealable or as periodontitis that is often exacerbated. This form of chronic periodontal inflammation is very difficult to treat.

Chronic granulating periodontitis (periodontitis chronica granulans) accounts for 65-70% of all cases of chronic periodontitis. Symptoms. Patients complain of tooth stiffness, some pain during eating and pressing. In the root area, the patient feels heaviness and some bulging. If there is a carious defect, filling it with food debris can cause exacerbation of the process and pain. The anamnesis reveals repeated exacerbations of the process with severe pain, swelling, abscess formation and the appearance of a fistula with pus discharge.

Objectively. A gangrenous or filled devitalised tooth with a discoloured tooth is detected. The tooth may also be externally intact or have a broken crown (in case of traumatic etiology of the

lesion). Vertical percussion of the tooth is quite tangible or gives a mild painful reaction. During horizontal percussion, if the bone wall is perforated or thinned, after inserting the index finger into the vestibule of the mouth in the apex area, a tapping sound is felt, which is transmitted directly from the crown of the tooth to its root. This transmission of percussion sound is called the phenomenon of the diverted blow and is most pronounced in the area of single-rooted teeth. Tooth mobility may vary depending on the degree of alveolar bone destruction. Examination of the mucous membrane in the area of the apex reveals hyperaemia with a bluish tint. However, the hyperaemia is not very pronounced, so to detect it, the oral cavity should be examined from the vestibule.

I.G. Lukomsky (1955) described a symptom of *vasoparesis* characteristic of granulating periodontitis, which is observed when pressing on the swollen gums - they seem like a swollen pillow. This is due to the infiltrative growth of the granulation focus, which spreads not only to the bone but also to the soft tissues surrounding the alveolus. After pressing on such gums with a small instrument (corkscrew head or blunt side of an excavator), depressions and pallor of the mucous membrane remain, which quickly changes to a bright red streak that lasts for a long time, sometimes several minutes (due to paresis of the gingival vessels).

In the case of frequent exacerbation, a permanent gingival or skin fistula (fistula) may occur on the mucous membrane, from which a drop of pus spills out when pressed. The fistulous passage connects the infectious focus to the oral cavity, where it opens with a fistulous opening, which is often tamponaded with granulation tissue protruding from it, giving the opening the appearance of a navel. Sometimes one or more scars can be seen in the fistula area. Palpation in the area of the apex, depending on the stage of the process, is accompanied by more or less severe pain. Palpation can reveal resorption of the cortical layer of the bone; the regional lymph nodes are usually enlarged and painful to pressure.

Chronic granulating periodontitis outside of exacerbation is not accompanied by general symptoms. Radiographs in the area of the apex reveal a focal point of lucency of varying size, the contours of which resemble flames. There is a consistent transition from the area of bone destruction to healthy bone in the form of a slight darkening. This indicates demineralisation of the inflamed bone. In the case of a prolonged course of the process, resorption of cement and dentin of the tooth root is noted in some areas, which may look like an obliquely cut cone of more than 1/3 of the root length.

In case of successful treatment of periodontitis, the defect begins to shrink in 4-8 months, and new bone tissue forms on its periphery. Bone trabeculae may also form, which can be seen as a characteristic grey shadow on the radiograph. After a year, the area of lucency is completely replaced by bone, sometimes denser than normal bone tissue. Differential diagnosis. Chronic granulomatous periodontitis can be easily differentiated from fibrous periodontitis by the nature of the signs detected by percussion and palpation, the presence of hyperaemia and swelling of the mucous membrane in the apex area, as well as a fistula or scar at the site of the latter. Diffuse, irregularly shaped lucency in certain areas of the apical alveolar region on X-rays allows for a more accurate diagnosis.

Chronic granulomatous periodontitis. This form of periodontal inflammation has 2 ways of formation.

1. The inflammatory process can occur spontaneously or after acute purulent periodontitis and is accompanied by the growth of granulation tissue, which takes the form of a local focus. This lesion does not tend to grow actively because the granulation of the outer layer is replaced by

fibrous connective tissue that separates it from the adjacent tissues. This formation is called a granuloma. It has a spherical shape, an outer dense capsule and soft granulation tissue inside.
2. Granuloma can develop from granulating periodontitis. In the case of sufficiently high defences of the body, high resistance of periodontal tissues and a weak etiological factor (microbes and their toxins), the granulating focus may lose its aggressive character. Under such conditions, the infiltrative growth of granulations stops, and a fibrous connective tissue capsule surrounding the granulation is formed along its periphery. A layer of bone tissue resembling a compact plate is deposited at the border of the capsule. The fibres of the capsule are woven into the periodontium, binding the granuloma to the tooth root. Such a granuloma is called simple or fibrous.
The granulation tissue inside the granuloma has the same cellular composition as in granulating periodontitis.
Granuloma is a fairly persistent, stable form of chronic periodontitis that can exist for a long time - several years. In case of a decrease in the body's defences or under the influence of local factors, an exacerbation of the inflammatory process may occur. Purulent exudate breaks through the fibrous capsule and causes diffuse inflammation of the periodontium and adjacent tissues. After treatment, this form of periodontitis can turn into fibrous periodontitis. Depending on the nature of the capsule and cell composition, epithelial granulomas and cystic granulomas are also distinguished.
Epithelial granulomas are granulomas in which the granulation tissue is permeated with epithelial strands. In the early stages of development, the epithelial strands are single, and later they become more numerous - epithelial cells almost replace the granulation tissue, which can lead to the formation of cystic granulomas.
A cystogranuloma is a cavity lined with epithelium and filled with a cloudy fluid. In the case of frequent exacerbations of periodontitis, the blood supply to the periodontium is disrupted, cells degenerate, and as a result of their decay, cavities filled with fluid containing degenerative forms of leukocytes, erythrocytes, epithelial cells, etc. appear. Any form of chronic periodontitis can be exacerbated when the body's defences are reduced. Most often, chronic granulating periodontitis is exacerbated. Thus, each of the three forms of chronic apical periodontitis can occur spontaneously or can transition from one to another. This factor is very important in the treatment of periodontitis.
Chronic granulomatous periodontitis (periodontitis chronica granulomatosa) is manifested by limited inflammation of the periodontium around the apical foramen, usually asymptomatic. The dentist detects it, like fibrous disease, by chance during an X-ray examination. Anamnesis can establish that sometimes during colds or active chewing of solid food, patients feel heaviness, tension, even pain in the area of the apex of the tooth root.
Objectively. A discoloured tooth is detected, it may be intact or filled.Vertical percussion is often painless.However, the causative tooth may be more sensitive compared to the adjacent teeth.Horizontal percussion in cases of large granulomas is used to determine the symptom of a reflected blow.The mucous membrane in the area of the apex is not altered; only in the acute stage is it hyperemic and swollen. Palpation reveals mild pain, and a hard protrusion without fluctuation of 3 to 5 mm in size can be detected, which is a reactive thickening of the periosteum. The diagnosis can only be confirmed by X-ray. In chronic periodontitis, radiography is the main examination, in fact, the only source that provides objective information about the condition of the periodontium. The radiograph reveals a limited lumen in the apex area, which is oval or round in shape, up to 5 mm in diameter. The contours of the

granuloma clearly delimit it from the healthy bone and resemble a compact alveolar plate. Along with the granuloma, there is often an osteosclerotic roller.

Differential diagnosis is based on clinical symptoms (appearance of the tooth and surrounding mucosa, percussion findings, etc.), electrical excitability test, and radiography. Granulomatous periodontitis differs from fibrous periodontitis by milder pain during palpation and sometimes percussion. It differs from granulomatous periodontitis in the absence of gingival edema and periodically opening fistula in the apex area. The main thing in the differential diagnosis is a typical radiological picture with a clearly limited periapical lumen, which confirms the diagnosis of chronic granulomatous periodontitis and allows to exclude granulating periodontitis.

METHODOLOGICAL RESEARCH №21

TOPIC: EXACERBATION OF CHRONIC APICAL PERIODONTITIS: ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSTICS. COMPLICATION OF PERIODONTITIS BY PERIOSTIS

The clinical picture of acute chronic periodontitis resembles the symptoms of acute periodontitis. The peculiarities of the clinic of acute chronic periodontitis are due to the presence of destruction in both the periodontium and the alveolar bone. Therefore, the history is characterised not only by repeated exacerbations with pain, swelling, general malaise, but also by a very rapid development of inflammation with the formation of fistulas. All symptoms - pain, collateral edema, lymph node reaction, etc. - appear in the same sequence as in the case of acute periodontitis. Its acuteness and severity, however, are significantly reduced due to the presence of a fistulous course. Pain during pressure and eating is less intense than in patients with acute purulent periodontitis.

Objectively. A carious or filled devitalised tooth is detected, the horizontal and especially vertical percussion of which is painful. Depending on the phase and stage of the disease, tooth mobility of the I-II degree is detected. The mucosa in the apex area is hyperemic and swollen. The transitional fold is smoothed and painful to palpate. There may be an abscess in one or another phase of development and collateral soft tissue edema. Lymph nodes can also be inflamed. The general condition may deteriorate.

The radiological picture corresponds to that of previous chronic periodontitis. Additional changes in it depend on the severity and duration of the inflammatory process. Thus, in the case of acute fibrous periodontitis, the periodontal space is more deformed - there is a clear widening of the periodontal gap in the apex area, and there are foci of bone softening.

In the case of exacerbation of granulomatous periodontitis, the clear contours of the compacted bone around the granulomas disappear, and the bone marrow spaces on its periphery become lucent.

The radiological picture of granulating periodontitis is generally blurred, but the contours of lucency are more pronounced. In the case of acute periodontitis, differential diagnosis with acute purulent periodontitis should be performed. Such anamnestic data as recurrence of acute symptoms and rapid development of inflammation up to fistula formation in a short time (within a few hours) indicate an exacerbation of chronic periodontitis. The diagnosis is confirmed by X-rays, which reveal periapical changes.

Sometimes, if periodontitis is not treated in a timely manner, a complication in the form of periostitis may occur.

Periodontitis is an inflammation of the periosteum (periosteum), the tissue that covers the jaw bones and alveolar branches in which the teeth are located. It usually occurs as a result of an infection that can occur due to tooth decay, trauma, gum disease or other causes. Localisation of the disease in the lower jaw occurs about 1.5-2 times more often than in the upper jaw. The course of jaw periostitis has a specific local and general clinical picture, and in case of timely treatment, the inflammatory process can be reversible. However, with the progression of the disease, the risk of severe purulent complications increases.

Tooth pain can be of a different nature: aching, dull, pulsating, constant, and can also radiate to the part of the head. This pain may be accompanied by complaints such as the inability to fully open the mouth, pain when chewing, swallowing, or moving the tongue. The localisation of swelling in periostitis on the gums is located in the area of the diseased tooth.

If you conduct an examination of the oral cavity, periostitis can be noticed due to its characteristic red colour, as well as elevation above the gum, swelling of the mucous membrane. Depending on the process of inflammation, a submucosal or subperiosteal abscess may form. In some cases, spontaneous release of the pathological process may occur - a breakthrough of pus from under the gingival areas. This can usually happen on the sixth or seventh day. However, it is also possible that the disease may need to be treated invasively.

Most patients also experience swollen, painful lymph nodes or some symptoms of intoxication in the form of weakness, poor appetite, malaise, and sleep disturbance. This can often be accompanied by elderly or senile people in the presence of characteristic diseases (diabetes mellitus, diseases of the cardiovascular system, gastrointestinal tract).

Periodontitis of the jaws can occur in different ways, but much depends on the location of the causative tooth. Thus, the pathology is characterised by an inflammatory process and swelling on the lip or wings of the nose. However, if the causative tooth is located on the upper jaw, the swelling can also spread to the area around the eyes, corners of the mouth, and buccal area. Sometimes even partial involvement of the facial nerve is possible, and it can reach the auricle. In case of development of periostitis on the lower jaw, swelling forms in the area of the lower lip and chin.

Diagnosis

The diagnosis can be determined from laboratory tests of blood samples taken from a person. In particular, a slight increase in leukocytes (10-11 * 109 / l), an increase in neutrophils (75-78%) is noted for a characteristic inflammation. Radiographic analysis of the jaws can also be performed.

METHODOLOGICAL RESEARCH №22

TOPIC: X-RAY DIAGNOSTICS OF PERIODONTITIS. CONCEPTS OF OSTEOPOROSIS, OSTEOSCLEROSIS, DESTRUCTION, DEFORMATION, HYPERCEMENTOSIS, ETC.

In the acute process, pathological changes in the periodontium may not be detected on the radiograph or the periodontal gap may be widened. In the case of exacerbation of the chronic process, changes occur that are characteristic of granulating, granulomatous, and rarely fibrous periodontitis.

In apical granulating periodontitis, changes are noted in the form of an expansion of the periodontal line near the apex of the tooth as a result of resorption of the socket walls. There is also bone thinning with uneven contours. The outline of the root apex may be uneven due to resorption of the tooth cement and dentin. In the presence of odontogenic granulomas in the soft tissues, the destructive focus at the root apex is always small. In patients with marginal granulating periodontitis, similar changes are found in the marginal periodontium, where bone resorption occurs both horizontally and vertically.

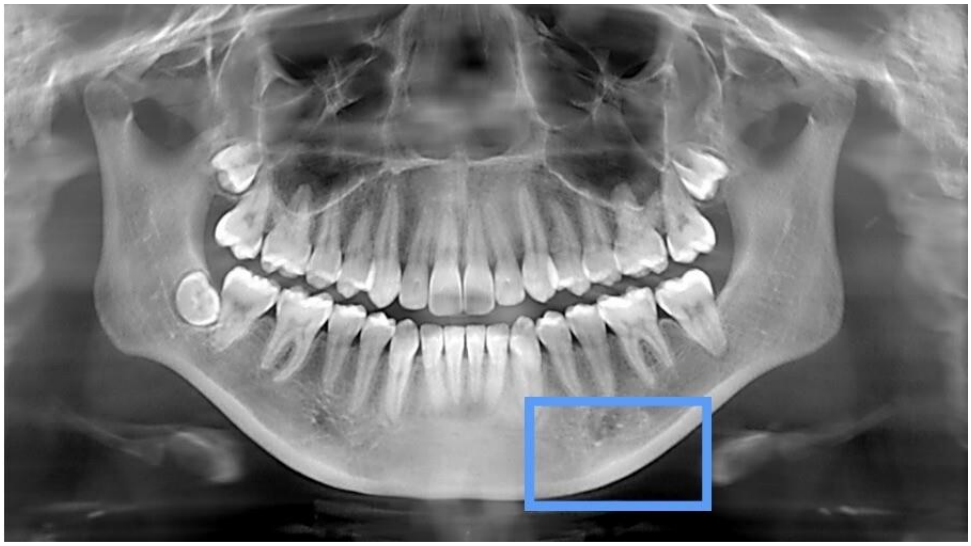
In case of granulomatous periodontitis, a rounded focus of bone thinning with clear, even borders is determined in the periapical area on the radiograph. In the inactive course of granulomatous periodontitis, the focus of liquefaction at the root apex is surrounded by a zone of sclerosed bone. If the treatment is carried out correctly, the granulomatous lesion should be destroyed and changes characteristic of fibrous periodontitis or the formation of a sclerosed bone area should be detected in its place.

On the radiograph, fibrous periodontitis is characterised by widening of the periodontal line, mainly at the apex of the tooth root. Sometimes, as a result of hypercementosis, a significant thickening of the apical part of the root is detected. The bone plate bordering the enlarged periodontal line is often thickened and sclerosed.

Radiological signs of exacerbation of chronic periodontitis. The diagnosis of chronic periodontitis in the exacerbation stage is based on the clinical manifestations of acute periodontitis and the radiological picture of chronic periodontitis. Granulating and granulomatous periodontitis are more likely to exacerbate, and fibrous periodontitis is less likely to exacerbate. Subjective symptoms indicating exacerbation of chronic periodontitis include recurrence of acute periodontitis symptoms and formation of fistulas on the gums or facial skin.

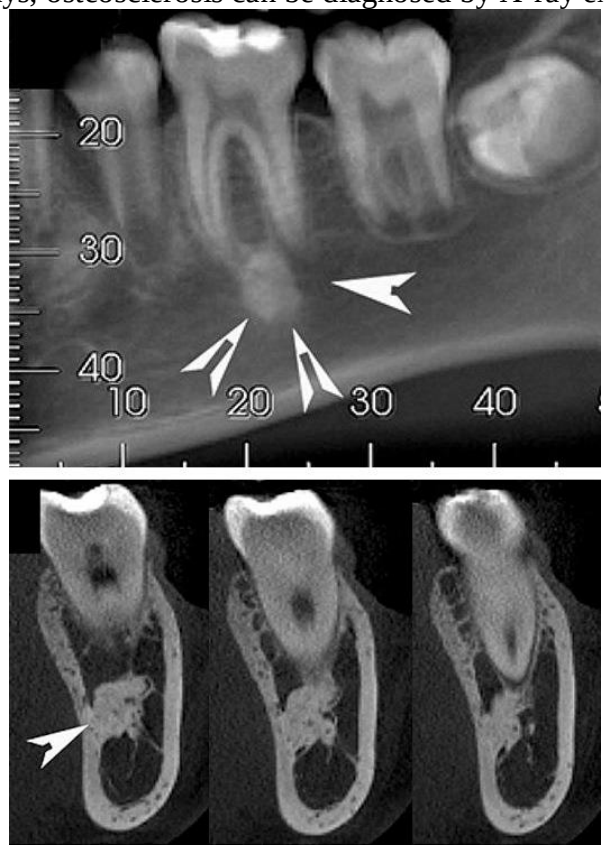
Exacerbation of chronic fibrous periodontitis on radiographs is represented by foci of destruction and osteoporosis in the area of a new inflammatory focus against the background of phenomena characteristic of fibrous periodontitis on the part of the periodontal crevice and the compact lamina of the tooth socket. The radiological picture of granulomatous periodontitis in the acute stage is characterised by a loss of clarity of the bone destruction boundaries in the apical part of the tooth, indistinct periodontal line in the lateral parts of the periodontium, as well as the appearance of destruction and osteoporosis in the periphery of the granuloma. Exacerbated chronic granulomatous periodontitis is radiographically characterised by more pronounced blurring of the destruction focus contours against the background of an enlarged osteoporosis zone.

Osteoporosis is a condition that causes bone loss. As the tissue breaks down at the microscopic level, bones become more fragile and can easily fracture.



Pic.22. Osteoporosis found by an AI

Osteosclerosis is a pathological condition that increases bone density, manifested in the form of thickening of bone trabeculae and compact bone substance. Spongy bone with osteosclerosis acquires a narrow looped structure. Due to the fact that the compacted bone tissue becomes less transparent to X-rays, osteosclerosis can be diagnosed by X-ray examination methods.



Pic. 23. X-ray of osteosclerosis

Destruction is the gradual destruction of bone with its replacement by other pathological tissue (pus, granulation, tumour mass, etc.). Destruction can be focal, diffuse, osteolytic, and marginal. The degree of destruction is assessed by localisation, number of foci or areas, shape, contours, structure and size.

Bone deformity is a group of congenital and acquired pathological conditions that are accompanied by changes in the length, shape and axis of bones, including the jawbone. External signs of deformity are accompanied by dysfunction of a particular bone.

Hypercementosis is a process of excessive deposition of secondary cement, during which the tooth root undergoes deformation: it thickens and characteristic protrusions are formed on it.

Рис.24. Tooth hypercementosis

METHODOLOGICAL RESEARCH №23

TOPIC: TREATMENT OF ACUTE SEROUS AND PURULENT APICAL PERIODONTITIS OF INFECTIOUS AND NON-INFECTIOUS (MEDICATION AND TRAUMA) ORIGIN. EMERGENCY CARE. SEQUENCE AND FEATURES OF THE TREATMENT STAGES. ANTIDOTE THERAPY. MODERN TECHNOLOGIES AND INSTRUMENTS FOR ROOT CANAL TREATMENT. FEATURES OF FILLING

The available methods of periodontitis treatment can be divided into 4 groups:

1) conservative - aimed at preserving the anatomical and functional value of a diseased tooth;
2) conservative-surgical - aimed at preserving the basic functions of the tooth. It involves the removal of a part of the root or peri-root tissue destroyed by a pathological process that cannot be treated;
3) surgical - removal of the diseased tooth and pathologically altered alveolar bone;
4) physical.

Conservative treatment of periodontitis is carried out to eliminate the focus of periodontal infection (pathologically altered pulp tissues, dentin, root canal microflora and microtubules) by thorough instrumental and medicinal treatment of root canals and their obturation, which creates conditions for the regeneration of periodontal tissues and the periapical area.

Indications for surgical and conservative surgical methods are the ineffectiveness or impossibility of conservative treatment in full or the presence of contraindications to its implementation, namely: - a diseased tooth is the cause of an acute septic condition, chronic infection and intoxication of the body; - complete destruction of the tooth crown, if its restoration is impossible; - large perforations of the root wall or the bottom of the tooth cavity.

Conservative method of periodontitis treatment. Treatment of acute and exacerbated chronic periodontitis. One of the main tasks of treatment of acute and acute chronic periodontitis is the fastest possible elimination of the inflammatory process in periodontal tissues, pain relief and prevention of the spread of the inflammatory process. Treatment of periodontitis involves the treatment of the root canal, microtubules and periapical inflammation. The doctor's tactics depend on the etiology of periodontitis, the stage of the inflammatory process and the general condition of the patient.

Treatment of acute infectious periodontitis. Acute infectious periodontitis has a very short phase of intoxication, but a strongly pronounced exudation that develops quite quickly. The exudate formed in the periapical space can move into the adjacent tissues in different ways: through the root canal, through the alveolar bone of the jaw under the periosteum on the vestibular or lingual (palatal) side and further under the mucous membrane, along the periodontal cleft to the circular ligament. The main tasks in the treatment of acute infectious periodontitis (both serous and purulent) are: pain relief, creation of conditions for exudate outflow, antimicrobial and anti-inflammatory treatment, cessation of the prevalence of inflammation of periodontal tissues, restoration of the anatomical shape and function of the tooth. The treatment of acute periodontitis consists of a number of stages and is carried out in several visits.

The first visit includes the following stages of treatment:

1. Anaesthesia. Taking into account inflammatory changes in the soft tissues around the diseased tooth, the presence of an abscess, and sometimes difficulty opening the mouth, it is advisable to perform a conduction anaesthesia.

2. Antiseptic treatment of the oral cavity. Patients with acute periodontitis, especially purulent periodontitis, due to their severe condition, cannot maintain oral hygiene, take only softened food, so the teeth are covered with plaque infected with various microorganisms. Before starting treatment, it is necessary to remove soft plaque with hydrogen peroxide and irrigation with furatsilin solution and herbal decoctions.
3. Preparation of the carious cavity taking into account the topographic and anatomical features of the diseased tooth. The tooth cavity should be opened so that there is free access to the root canal. In 2-3-rooted teeth, after opening and expanding the tooth cavity, the mouth of the root canal is opened using intra-root burs of the Gates Glidden type.
4. Removal of putrid masses from the root canal. The quality of further treatment depends on the thorough removal of infected pulp tissue decay, remnants of filling material, infected softened dentin and other irritants. Putrid masses are removed from the canal with the help of a pulp extractor gradually, layer by layer, very carefully, so as not to push infected tissues into the periapical opening, constantly treating the canal with antiseptic solutions. After removing all the putrid masses, a serous or purulent exudate with or without blood may be released. The exudate is sucked out with cotton pads or paper pins.
5. Medicinal treatment of the root canal. It is performed with one of the non-irritating and fast-acting medicinal solutions (1% chlorhexidine solution, 1% iodinol solution, 3% hydrogen peroxide solution, etc.) The treatment can be carried out using a syringe or cotton swabs until a clean swab is obtained.
6. Opening the apical foramen. If the exudate is not discharged into the canal, i.e. the apical foramen is not opened, then after thorough drug treatment and drying of the root canal, it is opened. For the outflow of exudate through the root canal, drainage of the apical foramen is mandatory. The apical foramen is opened with a root needle, file or rimer carefully, with rotational movements around the axis, pushing the instrument to the apical area. This manipulation should be carried out with great care to avoid injury and secondary infection of periodontal tissues.
Difficult to pass canals, as well as in case of obliteration of the apical foramen, its opening and dilation of the canals are performed using manual and machine reamers (reamers and files). After the exudate is drained, the canal is rinsed and a turunda abundantly moistened with enzymes (trypsin, chymotrypsin) and antibiotics (streptomycin, lincomycin) is left in it for 1-2 days under a loose or hermetic dressing.
In the presence of a subgingival or subperiosteal abscess, it is opened and drained. The type of dressing and the nature of the injected substance depend on the general condition of the patient, the severity of the inflammatory process, the amount and nature of exudate discharged through the root canal.
Acute purulent periodontitis, which is usually accompanied by a violation of the patient's general condition, severe collateral edema, and a large amount of purulent exudate, is treated by applying a loose dressing. Enzyme solutions with antibiotics in isotonic sodium chloride solution or 0.25% novocaine solution; sorbents, antiseptics are left in the root canal. The patient should be prescribed general treatment: detoxification therapy, plenty of warm vitamin drinks, analgesics, antipyretics, desensitising drugs. In addition, rinsing with 0.02% furatsilin solution, 1% sodium bicarbonate solution, and decoctions of medicinal plants is prescribed 6-8 times a day.
In the case of acute serous periodontitis, which is characterised by mild local pain, a small amount of exudate, and an unexpressed reaction of the oral mucosa, the root canal is treated

with aqueous solutions of enzymes, antiseptics, etc., an emulsion of enzymes with nitrofurans or combined sorbent preparations is left in the root canal, and the tooth is covered with a sealed dressing. During the second visit, the choice of further treatment depends on the patient's complaints and the results of an objective examination: percussion, palpation, the condition of the root canal turunda, and the presence of exudate. It is also taken into account whether the dressing was applied hermetically or loosely. If there are no complaints after closing the tooth with an airtight dressing, the tooth has withstood the sealing well, the reaction to percussion is negative or slightly positive, the turunda is clean and dry, and there is no exudate in the canal, a thorough preparation of the carious cavity, medical and instrumental treatment of the canal, and filling of the canal and carious cavity are performed. These stages are carefully outlined in the treatment of chronic periodontitis.

Sometimes the patient has no complaints, but the examination reveals a positive reaction to percussion, and a small amount of purulent or serous exudate is found in the root canal. In such cases, after preparation of the carious cavity, tooth cavity and instrumental treatment of the root canal, the latter is thoroughly treated with an enzyme solution with an antibiotic or sorbent, a turunda with an emulsion of enzyme and antibiotic is left, a sealed dressing is reapplied for 2 days and the treatment is completed at the next visit.

The treatment of acute toxic (drug-induced) periodontitis does not fundamentally differ from the treatment of acute infectious periodontitis. The success of treatment depends on the prompt elimination of the cause of inflammation, removal of necrotic masses or toxic substances from the canal and the introduction of anti-inflammatory and antidote drugs into the periodontal tissues. **To treat periodontal inflammation caused by the use of devitalising pastes (arsenic), the following steps are performed:**

1. Removal of the devitalising paste.
2. Devitalisation of the pulp.
3. Treatment of the root canal with specific antidotes containing sulfhydryl groups - 5% solution of unithiol, sodium thiosulfate. These solutions are used to rinse the root canals abundantly and leave them on the socket for 1 to 2 days under a sealed dressing. In multi-rooted teeth and difficult root canals, iodine electrophoresis should be performed.
4. On the second visit after the disappearance of symptoms of exudation and exacerbation, further medical treatment of the canals with 5% iodine solution, 1% iodinol solution and instrumental treatment of the canals in full is carried out.
5. Filling of the canals up to the apical foramen with silers with prolonged antiseptic and anti-inflammatory effect.

Treatment of traumatic periodontitis. Acute periodontitis, which occurs as a result of errors and complications in the treatment of pulpitis, haematoma formation in case of pulp extirpation, removal of formalin-containing silers beyond the root apex, root perforation, etc., is treated mainly with physical methods: electrophoresis of 1% potassium iodide solution, 10% calcium chloride solution, ultraviolet light, laser therapy. If after 5-6 treatment sessions the pain does not decrease and even increases, it is necessary to unseal the root canal and treat the tooth using the lithium-ion method. If, after 5-6 treatment sessions, the pain does not decrease and even increases, it is necessary to unseal the root canal and treat the tooth according to the method of treating acute infectious periodontitis.

METHODOLOGICAL RESEARCH №24

TOPIC: TREATMENT OF APICAL PERIODONTITIS OF SINGLE-ROOTED AND MULTI-ROOTED TEETH. PRINCIPLES OF TREATMENT. METHODS OF INFLUENCING THE APICAL FOCUS OF INFECTION. SEQUENCE AND FEATURES OF TREATMENT METHODS. EFFECTIVENESS OF TREATMENT. INDICATIONS AND TREATMENT OF APICAL PERIODONTITIS IN A SHORT TIME. MISTAKES IN THE DIAGNOSTICS AND TREATMENT OF PERIODONTITIS. CAUSES, METHODS OF ELIMINATION AND PREVENTION

The main objectives of chronic periodontitis treatment are to eliminate the focus of periodontal infection and further influence the microflora of the root canals and their branches, eliminate the effects of toxins and biogenic amines - tissue protein breakdown products; eliminate or reduce inflammation in the periodontium; provide conditions for the regeneration of all periodontal components; and desensitise the patient's body.

Chronic periodontitis can be treated in one or several visits, but regardless of the number of visits, treatment consists of a number of stages. Only in case of conscientious implementation of each stage can success be achieved in the treatment of this complex dental pathology.

Stage 1. Preparation of the carious cavity and tooth cavity. The purpose of the first stage is to create free access to the root canal openings. For a successful preparation, it is necessary to clearly define the topographic and anatomical features of the diseased tooth, for which it is advisable to have a targeted radiograph. It is known that in a depulpated tooth, replacement dentin cannot be formed or remineralised, so necrotomy of the carious cavity should be carried out especially carefully, otherwise the areas of softened dentin will become a focus of infection, which can lead to the destruction of the rest of the tooth crown.

Stage 2. Expansion of the root canal mouths for free access to them and their further processing. The mouth is expanded with special intracanal burs or small spherical burs.

Stage 3. Removal of putrid (infected) masses from the canal. Under a layer of antiseptic solution, the remnants of necrotic pulp tissue are removed with a pulp extractor to prepare the canal for instrumental treatment. For this purpose, a 0.5-1 % chloramine solution, a 0.02 % chlorhexidine bigluconate solution, which release atomic chlorine that denatures microbial cytoplasmic proteins, and a 1 % iodinol solution are used. The root canal mouths, especially their apical third, are the most infected, so it is necessary to remove the putrid masses in fractions, changing the antiseptic baths during the removal of each new portion of decay. Work very carefully in the apical third of the root canal so as not to push the contents into the periapical tissues.

Stage 4. Instrumental root canal treatment is the most important stage of endodontic treatment. The purpose of this stage is to remove the infected dentin from the canal walls, provide access to the apical opening and, if necessary, open it and create conditions for further filling of the canal. Root canal treatment begins with determining its working length. The length of the root can be determined in one of three ways: radiographically, using calculation tables, and electronometrically using an apex locator. Root canal expansion and formation is carried out using special sets of endodontic instruments using different methods. The most common is the standard method, according to which the canal is expanded with K-reamers or K-files with a sequential increase in their diameter by 3-4 sizes, not reaching the radiological length by 1 mm. This technique is advisable for instrumental treatment of straight, well-passable canals, especially in anterior teeth.

Nowadays, the most popular technique for instrumental treatment of narrow canals is the "step-back" canal preparation technique (a "step back" is the expansion of the canal from the apical opening to the mouth). According to this technique, endodontic instruments are used from

smaller to larger sizes. This technique is described in detail in the section "Pulpit". Start working in the canal with the smallest K-file (010), which freely passes to the marked depth of the canal, gradually use larger K-files; before the physiological narrowing, the root canal should be expanded to the file size 025. The instrument should be rotated only clockwise and constantly return to smaller instruments. After each instrument change, the canal is rinsed with an antiseptic to prevent blockage of the root canal tip with dental debris.
The next stage of this technique is to expand the canal with larger instruments (up to 040 - 045), but of shorter length to ensure the creation of a uniform conical shape of the canal in the direction of the mouth. Then the walls of the canal are levelled with a file (Hedström), rinsed after each instrument insertion, and the canal is ready for filling.
Recently, flexible endodontic instruments with a rounded tip made of nickel-titanium alloy have been developed and a technique for working with such instruments called "Crown down" ("step-down") has been proposed - the expansion of the canal from the mouth to the apex. This technique is based on the sequential replacement of endodontic instruments from large to smaller sizes. The canal is expanded from the oral part, gradually moving to the middle and then the apical part.
Root canal treatment using the *Balanced force method* has also become widespread. Methodology: the endodontic instrument is inserted 1/3 of the way into the canal and manually rotated counterclockwise. During its rotation, a slight pressure should be applied in the apical direction at the same time. The balance of these two forces will remove dentin without clamping the file. It is advisable to use flexible instruments made of nickel-titanium alloy with a rounded tip to perform this technique.
Instrumental treatment of the root canal is considered complete if the root canal meets the following requirements:
- is completely free of infected dentin;
- has a conical shape along the entire length from the apex to the mouth;
- is sufficiently dilated;
- has a formed apical stop;
- dry, clean, sterile.

Drug treatment of root canals during the treatment of periodontitis. The main objectives of the drug treatment are:
1. Influence on the etiological factor - infection, toxins, chemical toxic substances, etc. contained in the root canal and its branches, microtubules and periapical area.
2. Anti-inflammatory effect on damaged periodontal tissues.
3. Stimulation of regeneration processes of periodontal tissues and damaged alveolar bone.

Depending on the duration of action on microorganisms and damaged periodontal tissues, all medicines can be divided into 2 groups:
1. Drugs of instant or short-term action. their effect begins in 5-10 s and lasts 1-3-5 min. These are mainly drugs whose active ingredient is gases and gaseous substances (chlorine, iodine, oxygen, etc.).
2. Long-acting drugs (1-3 - 5 - 7 days). These drugs or their mixtures have not only antiseptic or antimicrobial effects, but also have both anti-inflammatory and regenerative effects - they affect the inflammatory process in periapical tissues.

The success of the treatment depends on the correct choice of the drug for the root canal treatment. Since both aerobes and anaerobes, non-proliferative microorganisms, play a significant role in the etiology and pathogenesis of inflammatory processes in the periodontium (acute and chronic), a positive treatment effect can be obtained by using medications that act on all types of microflora. To do this, you need to know the main clinical signs of a particular type of microorganism.

For example, clinical signs of anaerobic infection include a pungent putrid odour from the canal, thick yellowish-grey pus, moist gangrenous grey-black decay, and no or very low therapeutic effect from previous use of antibacterial drugs. Coccal microflora is characterised by a large amount of thin purulent light exudate without a noticeable odour. In order to influence anaerobes, it is advisable to use nitrofurans, 1-0.5 % dioxidin solution, bactrim suspension, as well as metronidazole, fusidine sodium, which in this case should be prescribed to patients according to the regimen.

Since patients with chronic periodontitis or its exacerbation have a significant contamination of the root canal with pathogenic staphylococcus aureus resistant to other antiseptics, the use of an ectericide or chlorophylliptic is justified.

When choosing a drug for root canal treatment, one should take into account not only the duration of its bactericidal and bacteriostatic action, solubility in water and biological fluids, but also the nature and stage of periodontal tissue inflammation and the patient's general condition.

Root canal filling in patients with periodontitis is an important stage of endodontic therapy. In case of its high-quality performance, two main tasks are solved. First of all, root canal filling aims to create a barrier between the external and internal environments of the body. The peculiarities of the tooth cavity morphology do not allow to completely remove the decayed organic substances and to achieve a state of sterility of the canal. Therefore, hermetic obturation of the canals is the only way to block and neutralise toxic decay products and microorganisms in the canal, apical delta and dentinal tubules. Secondly, the root canal filling material inevitably comes into contact with the periodontium, so it must be a biologically neutral substance that does not have an irritating effect on the tissues.

The root canal filling should protect the periodontium from external irritants. In addition, since there is an inflammatory process in the periapical tissues, the filling should play the role of a kind of therapeutic dressing that has a medicinal effect on inflammation and thus stimulates regeneration processes. This role of a root canal filling can be realised, first of all, if it is inserted into the root canal and filled tightly to the periodontium. Only in this case, conditions are created for a biological effect on the pathological focus in the periapical tissues.

Regardless of which filling material is chosen for root canal obturation, the filling will be considered to be correctly performed only if the canal is densely obturated along its entire length, especially in the apical part. The optimal level of filling is the level of the apical foramen. It is quite difficult to achieve a root canal filling exactly at the level of the apical foramen. A slight extension of the filling material beyond the apex is not a very serious complication, but it is not allowed to deliberately extend the filling material beyond the apex or fill the focal point of destruction of the collar bone with it. Particular care should be taken when working with materials that have high irritant properties and do not resorb from the periapical area.

Thus, the procedure for the treatment of periodontitis, regardless of clinical features, consists of the following sequential stages:

1. Preparation of the carious cavity and tooth cavity.
2. Antiseptic treatment and removal of putrid masses from the tooth cavity and canals.
3. Determination of the length of the root canal.
4. Instrumental treatment of the canal.
5. Medicinal treatment of the canal.
6. Zaapical therapy.
7. Root canal filling.
8. Filling or restoration of the crown part of the tooth.

The number of patient visits depends on the time required to complete all these stages. Some forms of periodontitis can be treated in one session, while other forms require 2-3-5 visits. It all depends on the success of each stage and the dynamics of the clinical condition.

Complications arising after periodontitis treatment

1. At different times after the treatment of periodontitis, asymptomatic progression of the pathological process in the periodontium is possible, which can lead to chronic osteomyelitis, odontogenic cysts, etc. Most often, this complication occurs in patients with reduced immunity, an individual reaction to the filling material or in case of resorption of the endocanal filling. This pathology is detected by X-ray. It requires immediate tooth retreatment. If it is not possible to treat the diseased tooth qualitatively, it should be extracted as a focus of chronioseptic condition.

2. Odontogenic maxillary sinusitis is a fairly common complication of chronic periodontitis. Its development can be caused by destruction of the spongy substance of the alveolar ridge by a pathological process, infection of the maxillary sinus, its trauma during instrumental root canal treatment of the non-root 5, 6, 7 teeth, removal of filling material into the sinus cavity. Exacerbation may occur in 1-2 months or in 1 - 2 years. In such cases, the causative tooth is removed and sinusitis is treated.

METHODOLOGICAL RESEARCH №25
TOPIC: USE OF PHYSICAL FACTORS IN THE COMPLEX THERAPY OF PULPITIS AND PERIODONTITIS. METHODS. EQUIPMENT

Physical methods are used when it is necessary to influence the infection in the root canal and microtubules, as well as the inflammatory process in the periapical tissues. These methods affect the bacterial flora, which is difficult to access and eliminate due to the complex topographic and anatomical properties of the endodontic tissue. In addition to the effect on the bacterial flora, physical methods activate biochemical processes in the periapical tissues, which contributes to the reversal of the pathological process. As a result of their action, fibrosis of granulation tissue increases and the processes of alveolar bone regeneration are accelerated.

For the treatment of periodontitis, physical methods can be used both independently (e.g. diathermocoagulation, electrophoresis) and in complex therapy aimed at stimulating reparative processes in periapical tissues, as well as preventing complications. In some cases, when drug therapy is unsuccessful or cannot be used (for example, in patients with allergies to certain drugs), these methods become the means of choice.

Indications for the use of physical methods:

1. Treatment of periodontitis in teeth with obstructed root canals, with fragments of instruments in them.
2. Treatment of periodontitis that does not withstand hermetic closure or is very slow to respond to drug treatment.
3. Treatment of chronic granulating periodontitis with persistent fistulas.
4. In patients with allergies to drugs that are most often used in dental practice, as well as in patients with reduced body resistance.
5. For the prevention and treatment of complications after endodontic treatment.

The most common physical method of treating periodontitis is electrophoresis of medicinal substances. Depending on the electric charge carried by the ion of the drug substance injected into the canal and periapex, the active electrode can be an anode or a cathode. Since the anode made of noble metal (platinum) is inserted into the canal as an active electrode, chlorine, chloride and hypochlorous acids, and oxygen are produced in the canal as a result of electrolytic dissociation of tissue fluids. Chloride and hypochlorous acids are also dissociated, additionally releasing chlorine. In essence, the method is a kind of chlorotherapy that leads to sterilisation of obstructed canals and their branches. Anodic sterilisation with ions of various metals is also used. The most commonly used electrolytes are zinc chloride, zinc iodide, zinc sulphate and copper sulphate. Negatively charged microorganisms moving with metal ions can partially escape from the dentinal tubules and periapex into the macrochannel, creating additional conditions for the inhibition and elimination of bacterial flora. Zinc ions, in addition to having a bactericidal effect, are able to coagulate proteins in the endodontium. For the treatment of teeth with obstructed root canals, silver nitrate electrophoresis is used, except for the anterior teeth (they may be stained).

For the treatment of periodontitis, iodine electrophoresis is very common. The active electrode is a cathode that is placed in the canal or fixed in the tooth cavity. The electrolyte can be of three types:

1) an alkaline solution prepared ex tempore: Iodi puri - 7.5, Kalii iodati - 5.0, Aq. destil. - 10 ml (diluted 10 times);
2) saturated solution of potassium iodide (dissolve potassium iodide powder in 2-3 drops of distilled water until saturated). Potassium iodide solution is used to treat periodontitis of the anterior teeth;
3) in the treatment of molars, if the darkening of the crown is not significant, a 5-10 % tincture of iodine is used (Fig. 125). The electrophoresis procedure lasts 20 minutes, the current strength

is about Z mA. The procedures are performed daily or every other day, depending on the form of periodontitis. The course of treatment is 3 to 5 procedures.
The therapeutic effect is associated with the action of chlorine and iodine ions, which move to the apex and penetrate dentinal tubes and apical tissues. In addition, the reaction in the channel near the cathode releases potassium, which forms potassium alkali with water, which lyses all organic matter. Hydrogen is also released in the form of gas bubbles. It carries the substances lysed in the canal into the tooth cavity with foam. Iodine ions have a bactericidal effect. In addition, oxygen is released in the root canal, which enhances oxidative processes, and cellular decay products have a local stimulating effect.
In the treatment of apical periodontitis, electrophoresis of calcium, copper, and zinc is also used.
Electrophoresis of a 10% calcium chloride solution is indicated in the treatment of acute and exacerbated chronic periodontitis, as calcium ions have analgesic and dehydrating effects in inflamed tissues. Copper ions have a strong antiseptic effect on anaerobes, so copper hydroxide electrophoresis is most effective in chronic granulomatous periodontitis.
Zinc chloride electrophoresis provides anti-inflammatory and stimulating effects in patients with chronic granulomatous periodontitis, because zinc ions coagulate proteins and inhibit the growth of granulation tissue. The course of treatment is 3 to 5 procedures.
To accelerate the treatment of acute purulent and exacerbated chronic periodontitis, especially in teeth that do not withstand tightness, the use of electrophoresis of proteolytic enzymes and their compositions with antibiotics is effective. Antibiotic and enzyme solutions are prepared ex tempore: 2 mg of chymotrypsin (or trypsin) is dissolved in 2 ml of isotonic sodium chloride solution and ZO 000 U of penicillin (streptomycin) is added, or 3 mg of trypsin is dissolved in 3 ml of microcide. One of these solutions is injected into the root canal. The active electrode is the cathode, the current is 1 - 2 mA, the procedure lasts 15 minutes. The course of treatment is 3 - 4 procedures. In addition to endocanal electrophoresis, for the treatment of chronic periodontitis, 10% calcium chloride solution is used to influence the inflammatory process and accelerate bone regeneration.
Ultraphonophoresis of medicinal substances is also used in the complex therapy of periodontitis. Ultrasound intensifies metabolism, enzyme activity, increases cell membrane permeability, resulting in the release of biologically active substances. In addition, ultrasound causes the dissociation of drugs and the accumulation of active ions in cells. This leads to the formation of a drug depot, which has a therapeutic effect at the subcellular level. Ultraphonophoresis is prescribed in the following mode: intensity from 0.005 to 0.4 W/cm2 , oscillation frequency 800 kHz - 2 mHz in continuous mode. The duration of the procedure is 10 minutes, and the course includes 5-7 procedures. Depophoresis of copper-calcium hydroxide.
In order to increase the effectiveness of endodontic treatment, especially in the treatment of infected hard-to-reach canals, Professor A. Knappvost (1998) proposed the method of copper-calcium hydroxide depophoresis. The essence of the technique is that the root canals are passed and extended to about 2/3 of their length. After that, an aqueous suspension of copper-calcium hydroxide is injected into one of the canals, a needle electrode (-) is inserted, the electrical circuit is closed, and the procedure is performed. Then other channels are treated in the same way. At the end of the depophoresis course, the canals are sealed with a special alkaline cement containing copper.
Mechanism of depophoresis action. Under the influence of a constant electric current, hydroxyl ions (OH) and hydroxycuprate ions [Cu(OH)4]2" penetrate the apical part of both the main canal and the deltaic branches. In the lumen of the canal, copper hydroxide accumulates, partially precipitates and corrodes the walls. In the area of the apical opening, in a neutral environment, hydroxycuprate ions decompose and turn into poorly soluble copper hydroxide

[Cu(OH)2], which also precipitates. As a result, "copper plugs" are formed, which reliably seal all the exits of the apical delta to the root surface, soft tissue decomposition occurs in the root canal lumen and adjacent tissues, while decomposition products are eliminated into the periapical tissues and resorbed by the body. At the same time, the lumen of the main canal and the apical delta are sterilised due to the bactericidal effect of the drugs used. In the unsealed part of the main canal, as well as in the deltaic branches, copper-calcium hydroxide lines the walls and creates a depot. The resulting "copper plugs", which encircle all the exits of the apical delta to the root surface, ensure the tightness, disinfection and long-term sterility of this part of the root canal. Due to the alkalisation of the environment and the therapeutic effect of copper-calcium hydroxide, the function of odontoblasts and bone regeneration in the periapical area are stimulated.

Indications. Depophoresis with copper-calcium hydroxide is indicated primarily in endodontic treatment of teeth with obstructed root canals, in addition, this method is recommended in cases of significant infection of the canal contents, breakage of the instrument in the lumen of the canal (without going beyond the apex), in case of unsuccessful treatment of the tooth with traditional methods in the presence of a wide apical opening.

Contraindications to depophoresis: malignant tumours, severe forms of autoimmune diseases, pregnancy, intolerance to electric current, allergic reaction to copper.

Depophoresis is a medical procedure performed by a dentist directly in the dental chair. Method of copper-calcium hydroxide depophoresis. During the first visit, the root canals are passed and expanded by about 2/3 of the length. The canals should be processed until it is necessary to use ISO instrument No. 35-50 (International Standard Organisation). The mouths of the canals are widened slightly more to create a sufficient depot of copper hydroxide. After machining, the canals are rinsed with distilled water, a 10% calcium hydroxide suspension or a dilute copper-calcium hydroxide suspension. After the root canal treatment, the tooth is isolated from saliva and dried. The patient should be positioned in such a way that the preparation does not leak out of the canal: when treating the teeth of the lower jaw - sitting, when treating the teeth of the upper jaw - lying in a chair with the head tilted back. The suspension of copper-calcium hydroxide is diluted with distilled water to a creamy consistency and injected with a root canal filling material into the treated part of the canal. When treating anterior teeth, to avoid staining the tooth crown, it is recommended to dilute the paste with water in a ratio of 1:10. Then a negative needle electrode (cathode) is inserted into the canal to a depth of 4-8 mm, leaving the tooth cavity open. It is necessary to ensure that during the procedure this electrode does not touch soft tissues, metal crowns and fillings of other teeth. In addition, saliva, blood or gum fluid should not enter the tooth cavity. All mistakes can lead to a decrease in the effectiveness of treatment and electrochemical burns of the oral tissues. The positive passive electrode (anode) is placed behind the cheek on the opposite side and make sure that it does not touch the teeth. To improve the electrical contact between the electrode and the cheek, a cotton roller moistened with tap water or isotonic sodium chloride solution is placed.

Electrophoresis is performed using the Original II, Comfort (Germany) or Endo EST (Russia) apparatus. During the procedure, the current strength is slowly increased until a slight sensation of warmth or tingling appears in the tooth area, then the current strength is reduced and slowly increased, reaching 1 - 2 mA. The duration of the procedure is calculated based on the fact that during one session, 5 mA/min should be received per channel. For example, if the current strength is 1 mA, the procedure duration is 5 minutes, 1.2 mA - 4 minutes, 2 mA - 2.5 minutes, and if only 0.5 mA was achieved, the procedure duration is 10 minutes. In multi-rooted teeth, each canal is treated separately. After the procedure, the canals and the tooth cavity are rinsed again with distilled water, 10% calcium hydroxide suspension or diluted copper-calcium hydroxide suspension, and the tooth cavity is sealed with an artificial dentin dressing. In case

of inflammation in the periodontium, the tooth can be left open after depophoresis to allow the exudate to drain through the canal. Additional infection of the periodontium with oral microflora is excluded due to the high bactericidal activity of copper-calcium hydroxide. The patient is scheduled for a follow-up visit in 8 to 14 days.

On the second visit, copper-calcium hydroxide depophoresis is again performed at the rate of 5 mA/min per channel. The patient should receive an "amount of electricity" equal to 15 mA/min during the course of treatment. After the last procedure, the treated part of the canal (2/3 of the length) is sealed with a special alkaline cement "Atacamit" containing copper, which is included in the depophoresis kit, and a permanent filling is placed. Positive aspects of depophoresis: possibility of successful endodontic treatment of teeth with obstructed root canals; high (up to 96%) clinical effectiveness; reduction of the risk of complications arising during instrumental treatment of the canal (perforation, breakage of instruments, etc.). there is no need to determine the working length - reducing the number of X-ray examinations and, as a result, the radiation exposure of the patient; minimal risk of removal of the filling material beyond the root apex; disinfection of the entire apical area.

Disadvantages: absence of objective diagnostic tests that would allow reliable assessment of the quality of obturation of the entire root canal, because its apical part looks unsealed on the radiograph; technical complexity of the procedure; after the course of depophoresis, the tooth crown acquires a yellowish tint; in addition, it is impossible to completely exclude further discolouration due to chemical transformations of copper compounds in the canals and tooth cavity; long treatment time - 2-4 weeks - creates some discomfort for the patient; the need for significant material costs for the purchase of a "starter" kit, replenishment of domestic materials, and provision of the doctor with appropriate endodontic instruments. Despite these disadvantages, this method undoubtedly opens up new opportunities in endodontics.

Diathermocoagulation. For diathermocoagulation, a current of low voltage and high strength is used, which has a thermal effect. As a result, the structures of soft tissues and microorganisms are denatured.

Methodology. To sterilise root canals, the instrument (root needle) at the tip of the diathermocoagulator is inserted into the canal first 1/3, then 2/3 and then the entire length of the canal. The high temperature destroys microorganisms and denatures putrid masses. For zaapical therapy, the instrument is withdrawn beyond the root apex and the granulation tissue is coagulated. The coagulant gradually dissolves, stimulating regeneration. After the procedure, the root canal is treated with instruments and medications and filled.

Laser therapy. Recently, low-frequency helium-neon lasers with irradiation in the infrared part of the spectrum have been used. For the treatment of periodontitis in dental practice, the following parameters are prescribed: dose 50-100 mW/cm2 , exposure 1 - 2 min per field, total 10-12 min. The therapeutic effect of the laser is associated with photosensitisation of the affected periodontal cells, which results in analgesic, anti-inflammatory and regeneration stimulation. In addition, the laser stimulates the body's general reactions (desensitisation) and improves the functioning of adaptive mechanisms.

Ultrashort waves (USW) are used to treat chronic periodontitis to sterilise the root canal, as well as to stimulate anti-inflammatory and general immunobiological functions. They are applied both intracanally and to the periapical tissue in the area of the diseased tooth.

Dyadic current. The method is based on the effect of diadynamic current on tissue metabolism, so its use helps to stimulate regeneration processes in periapical tissues. Diadynamic current is prescribed both during treatment and after root canal filling to prevent and treat complications.

Physical methods have a fairly quick and effective effect on the periapical inflammatory focus. They activate metabolic processes in the periapical tissues, stimulate trophism and regeneration of connective and bone tissue. The use of physical methods significantly speeds up the treatment of pulpitis and periodontitis and prevents complications.

CONCLUSIONS

By complementing the study at the Faculty of Dentistry with the analysis and study of the information array of this textbook, each student will be able to achieve the goal of studying the discipline of "Therapeutic Dentistry", namely to be ready to work in a dental clinic, to study the anatomical and physiological features of the dental pulp, the causes, mechanism of development of the inflammatory process in it, the main differential diagnostic features of various forms of pulpitis, to know the histology and physiology of the periodontium, the causes and pathogenetic mechanisms of development of the inflammatory process in it, clinical and radiological differential diagnostic signs of various forms of periodontitis; to implement a set of practical skills for examination of a dental patient, justification and formulation of a preliminary diagnosis, pulpitis and periodontitis; justification of the choice of treatment of pulpitis depending on the form, course and general condition of the body; identification of the main errors and complications of treatment and diagnosis of caries, pulpitis and periodontitis, ways to eliminate them

CONTROL TASKS AND QUESTIONS

1.Physical methods in the complex therapy of pulpitis and periodontitis, indications for use.
2.The difference between depophoresis, electrophoresis, ultraphonophoresis.
3.The use of laser, ultrashort waves, diadynamic current in the complex treatment of pulpitis and periodontitis.
4.Methods of diathermocoagulation.
5.Modern physical methods of treatment of pulpitis and periodontitis.

1.The patient visited the dentist for rehabilitation. Objectively: a deep carious cavity was found in 37, connected to the tooth chamber. The tooth does not respond to stimuli. X-ray: enlargement and deformation of the periradicular gap in the apical region. What is the most possible diagnosis?
a.Chronic fibrous pulpitis
b.Chronic granulomatous periodontitis
c.Chronic gangrenous pulpitis
d.Acute chronic pulpitis
e.Chronic fibrous periodontitis
2.A 14-year-old child has a maxillary sinus perforation with penetration of the distal buccal root into the maxillary sinus during extraction of 16 for periodontitis. What are the doctor's further actions?
a.Form a clot, do not inform the patient
b.Try to remove the root on your own
c.Refer to the hospital for surgical intervention
d.Perform outpatient maxillary sinus surgery
e.Close the perforation with a muco-periosteal flap.
3.A 13-year-old child complained of discolouration of the upper front tooth. 4 years ago, there was an injury to the anterior region of the upper jaw. Objectively: 11 is intact, percussion is painless. The X-ray shows bone thinning at the apex of the root of 11 measuring 1.6x2.7 cm with clear rounded edges. What is the most likely diagnosis?
a.Exacerbation of granulomatous periodontitis
b.Chronic granulating periodontitis
c.Chronic fibrous periodontitis
d.Odontogenic cyst of the upper jaw
e.Chronic granulomatous periodontitis.
4.The patient complains of discomfort in the tooth 3.4. while eating. From the anamnesis: swelling periodically appears near the tooth. In tooth 3.4, the entrance to the carious cavity is wide open. Probing and percussion are painless. X-ray: bone destruction without clear contours in the area of the root apex, associated with periodontal disease. Make a diagnosis:
a.Chronic fibrous pulpitis
b.Chronic fibrous periodontitis
c.Chronic gangrenous pulpitis
d.Chronic granulomatous periodontitis
e.Chronic granulating periodontitis
5.Indicate in which form of complicated caries the Lukomsky triad occurs:
a.Chronic granulating periodontitis
b.Chronic gangrenous pulpitis
c.Chronic fibrous pulpitis
d.Chronic fibrous periodontitis
e.Chronic granulomatous periodontitis

LIST OF USED LITERATURE

6.1. Main literature

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2. Periodontal and Oral Mucosa Diseases: textbook. Vol. 2 / A.V. Borysenko, L.V. Lynovytska, O.F. Nesyn et al.; edited by A.V. Borysenko. - Kyiv: AUS Medicine Publishing, 2018. – 624 p.; color
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6.2. Additional literature

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2. Esthetic and Restorative Dentistry: Material Selection and Technique 3rd Edition by Douglas A Terry, Willi Geller. Publisher: Quintessence Publishing Co Inc.; 3rd edition, 2017. – 776 p.
3. Dental Composite Materials for Direct Restorations Softcover reprint of the original 1st ed. by Vesna Miletic. Publisher: Springer; 1st edition, 2018. – 327 p.
4. Posterior Direct Restorations 1st Edition by Salvatore Scolavino, Gaetano Paolone. Publisher: Quintessence Pub Co; 1st edition, 2021. – 264 p.
5. Smile Design Integrating Esthetics and Function Essentials of Esthetic Dentistry Volume Two by Jonathan B. Levine. Elsevier Health Sciences, 2015. – 240 p.