

**M.N. VOLKOVA, N.A.SAKHARUK,
N.A. KORENEVSKAYA**

**GUIDELINE
ON THERAPEUTIC DENTISTRY**
for the 5-th term

VITEBSK, 2016

**MINISTRY OF HEALTH
OF THE REPUBLIC OF BELARUS**

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DEPARTMENT OF THERAPEUTIC DENTISTRY

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**Educational and methodical edition for
the third-year students of the stomatological faculty**

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LESSON 1.DEONTOLOGY IN THERAPEUTIC DENTISTRY. DENTAL OFFICE. STERILIZATION OF DENTAL INSTRUMENTS. THE BASIC RULES OF ERGONOMICS IN THE WORK OF THE DENTIST.

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

1. Therapeutic dentistry as a science, subject matter, objectives, goals.
2. Structure and equipment of dental office. The basic requirements are presented to him.
3. Aseptic and antiseptic for dental seeing, definitely, main types.
4. Sterilization dental instruments, concepts. Main types of sterilization used in clinic therapeutic dentistry.
5. Key terms in the ergonomics dentists. Workplace of dentist.

Question 1. Therapeutic dentistry as a science, subject matter, objectives, goals.

Therapeutic dentistry is the science that studies the characteristics of diagnosis, clinics, treatment, prevention and rehabilitation of patients with diseases of hard tissues of teeth, periodontal, oral mucosa.

The goal of therapeutic dentistry: implementation of effective prevention, diagnosis and conservative treatment of diseases of the teeth, periodontal and oral mucosa.

Objectives of therapeutic dentistry:

- 1.Prevention and treatment of caries and its complications.
- 2.Prevention and treatment and non-carious lesions.
- 3.Treatment and prevention of periodontal disease.
- 4.Prevention and treatment of oral mucosa.
- 5.Clinical examination of dental patients.

The term "deontology" comes from the Greek words: «deontos» is due, proper and «logos» - knowledge, means the totality of ethical standards of professional conduct for health workers.

Deontology involves questions:

- The confidentiality;
- Medical Malpractice for the life and health of the patient;
- The relationship between the health workers themselves and others.

The term "deontology" proposed by the British sociologist and jurist Jeremy Bentham (J. Bentham) in the XIX century to refer to the theory of morality. However, the foundations were laid of ethics in medicine of the ancient world.

Medical deontology is a set of ethical standards and principles of conduct of health professionals in carrying out their professional duties.

Dentist is a professional possessing special knowledge and skills in the practice of diagnosis, treatment and prevention of diseases of the teeth and

oral cavity inhumans. On reception at the doctor may have ethical and professional ethical issues.

Professional and ethical problems may be related to:

- diagnosis of diseases of the teeth;
- anesthesia;
- preparation of cavities;
- endodontic treatment;
- occupational health;
- local drug treatment.

Diagnosis of diseases. One of the most important functions of your dentist correct diagnosis. However, the diagnosis may be correct inaccurate or erroneous. The professional duty of the physician to correct diagnosis of dental disease. At the heart of the ethical problems associated with incorrect diagnosis, the doctor is the professional level. The higher it is, the smaller the error, and vice versa.

Anesthesia. This feature is extremely important the dentist, as the painful surgery inhumane. This function is not always performed satisfactorily and therefore the majority of the population is afraid of dental procedures. In all civilized countries, all treatments in dentistry, which may be accompanied by tenderness, conducted under anesthesia.

Dissection of the cavity. The basis of possible ethical problems in dissection may be incorrect following steps: preparation painful, overheating of the tooth, injury to tissues of the mouth, accidental opening of the pulp cavity, the destruction of tooth structure, insufficient preparation.

Filling. Key ethical issues associated with this function dentist may arise in connection with the loss of seals. Often this happens in 2-3 days after the visit to the dentist. Correct to seal loss is due to substandard work dentist. The optimal solution is the recognition of their professional errors and improving the professional level.

Endodontic treatment. Professional and ethical issues arising from complications after endodontic procedures.

To prevent this kind of professional and ethical conflicts should:

- ✓ to master endodontic procedures;
- ✓ do not expose the patient at risk in cases of uncertainty about the success of treatment.

Professional hygiene.

- Roth dentist should be in perfect condition;
- You can not talk to the patient about the poor hygiene, without confirming it is an objective test;
- Discussion of oral hygiene should take place in a positive value.

Local drug treatment. Problems arise due to:

- Lack of information about new drugs dentist;

- Illiterate use of drugs;
- Complications in their application;
- Side effects of drugs.

Prevention of professional and ethical issues must be to maintain high professional competence of a doctor.

Question 2. Structure and equipment dental office.

The basic requirements are presented to him.

Sanitation requirements for the offices of therapeutic dentistry. The area of the dental office should be 14 square meters per dental chair (height of 3.3 meters and a depth of 6 meters) for each additional - 7 square meters, in the event of a universal dental unit - 10 square meters.

Finishing cabinet. The walls in the dental office should be smooth, without cracks, ornaments, cornices, painted with oil paint or tiled to 2/3 height. Wall color should be pale shades. The ceiling of the cabinet is painted in white. Windows and doors are painted in bright colors. Paul's office should be covered with linoleum and have no gaps. Lighting in the dental office should be natural and artificial. Artificial lighting can be general and local. The air temperature in the cabinet should be 22 degrees. Ventilation supply and exhaust.

Equipping the dental office. To equip the dental office is necessary equipment that diversion can be divided into groups:

1) The equipment and the equipment required to perform medical manipulations:

- ✓ Dental unit, dental chair, the chair of the doctor, the assistant chair, dental doctor's desk.
- ✓ Accessories: vitalometer, apex locator, diathermocoagulator and others.

2) Sterilizing equipment - drying ovens, glassperlen sterilizer, quartz lamp, and so on.

3) Equipment for nurses: a table for documentation, chair, computer and so on.

4) Facilities for processing and pre-treatment arms: a sink for hand washing and separately for tools, containers with disinfectant solutions.

5) Medical furniture: the cupboard of medicines, filling materials, couch, chairs for patients, kitchen sanitary equipment.

Main blocks of the dental unit tools for manipulations in the mouth:

- ✓ micromotors (speed 10,000-30,000 rpm)
- ✓ turbine handpiece (the rate of 300,000 - 500,000 rpm)
- ✓ other tools (skeler, curing light),
- ✓ spittoon,
- ✓ glass sink,

- ✓ saliva ejector,
- ✓ vacuum cleaner,
- ✓ water-air gun,
- ✓ dental chair,
- ✓ compressor,
- ✓ table dentist,
- ✓ chair dentist,
- ✓ chair assistant (15-20 cm above the chair of the doctor).

Question 3. Aseptic and antiseptic for dental seeing, definitely, main types.

Asepsis is a system of preventive measures aimed at preventing the entry of microorganisms into the wound.

Asepsis involves performing disinfection, cleaning and sterilization of dental instruments, the use of personal protective equipment health workers special treatment doctor's hands.

Disinfection (decontamination) - a complex of measures aimed at the complete destruction of vegetative and dormant forms of certain kinds of microorganisms (spores and viruses remain) at the facilities of the environment in order to prevent transmission of the pathogen from infected to non-infected organism.

Disinfection physical methods.

- ✓ boiling in distilled water for 30 minutes after boiling;
- ✓ boiling in distilled water with the addition of 2% sodium bicarbonate for 15 minutes after boiling;
- ✓ effect of water vapor at a pressure in a steam sterilizer at $t=110^{\circ}\text{C}$ for 20 minutes;
- ✓ effect dry hot air in an air sterilizer at $t=120^{\circ}\text{C}$ for 45 minutes.

The chemical method of disinfection is the most common (e.g., immersion tool 6% hydrogen peroxide for 60 minutes).

Presterilizing purification consists of several stages. Upon completion of disinfecting instruments washed over a sink with running water for 30 seconds and are soaked in detergent solution (Inkrasept at a temperature of $20-45^{\circ}\text{C}$) for 1 hour. Then, the washed medical instruments are rinsed under running water 3-10 min. Then medical instruments are rinsed under distilled water. The washed medical instruments are dried in a hot air oven at a temperature of 85°C until complete disappearance of moisture.

The quality of cleaning products from the blood is checked by setting the test of azopiramide. The presence of residues of detergent products is determined by setting the phenolphthalein test.

Antiseptic - a set of chemical, mechanical, physical, biological methods to reduce the number of, suppression or total destruction of microorganisms in order to prevent the development of infection.

Chemical antiseptics carried out through the use of different antiseptics and antibiotics.

Biological antiseptic carried out by means of active or passive immunotherapy.

Mechanical antiseptic involves the use of mechanical methods to remove infected tissue or dental plaque.

Physical antiseptic is the use of physical methods of influence on the microorganisms with a view to their complete destruction.

Question 4. Sterilization dental instruments, concepts.

Main types of sterilization used in clinic therapeutic dentistry.

Sterilization is a complex of measures aimed at total destruction on the products or in products of all kinds of organisms, including their spore forms.

All medical products are used for manipulation in violation of the integrity of the skin and mucous membranes or in contact with the surface of the mucous membranes must be sterilized.

The sterilization of dental products is carried out physical and chemical methods.

Sterilization of physical methods:

- ✓ air - in air sterilizer at $t=180^{\circ}\text{C}$ for 60 minutes, with $t=160^{\circ}\text{C}$ for 150 minutes;
- ✓ steam - in a steam sterilizer at $t=132^{\circ}\text{C}$ for 20 minutes, at $t=120^{\circ}\text{C}$ for 45 minutes;
- ✓ in an environment heated balls - in glassperlen sterilizers intended for small dental instruments.

Sterilizers designed for small dental instruments using as sterilizing medium heated glass beads. Tools, sterilized in glassperlen sterilizer not be stored.

Shelf life of sterile instruments in a sealed package (to bikse, package kraft) is 3 days.

Question 5. Key terms in the ergonomics dentists. Workplace of dentist.

The design of the chair should allow the doctor to work sitting with the patient lying in the chair.

The materials of which made the chair should be required to withstand repeated handling antiseptics and disinfectants. The desired colors of gentle tones.

The chair of the doctor and the assistant should be light and mobile, height adjustable products and seat back angle.

Suspended table doctor - «UNIT» (block) must be mobile and be located at "arm's length" and the order of arrangement of instruments determined by the order and the frequency of their use.

1. Depending on the arrangement of the modules «UNIT» installation are:

- *T-type*, «Dentsan», «Practic» (Chirana), «Ergostart 92 C» (Chirana), «Performer», «ADEC» (ADEC), «Cavo Systematica 1060 TK» (Cavo), «FimerFl Continental A»(Finland), «PM 2002 CC» (Planmeca OY), «BelDent (Belarus)», «Biotec CM 6-120»(Belarus), «Sirona MI», «Spirit SI»(Siemens), when modules «Unit» arranged vertically;
- *S-type*, «Quint 7000», «FD-5000 Comfort» (Finndent), «Fimet Fl Continental E» (Finland), «ADEC international, Model 2080» (ADEC), «Cavo Systematica 1060 SK» (Cavo), «Promatic 1075 S» (Cavo), «Unident 1001» (JUGODENT), «Castellini», when modules are arranged horizontally;
- *G-type*, «Promatic 1075 G» (Cavo) modules mounted in the table, it is a fixed installation;
- *C-type*, «Cavo Junior» (Cavo), «Fimet Fl Cart» (Finland), «Unident 1002» (JUGODENT), «Ergostar 90 SU» (Chirana), the mobile unit with «Unit» operator.

2. The optimum operating position of the doctor

Operating position of the doctor, with the patient lying on his back, will be the following:

- browsing,
- back straight.
- without bending.
- foot flat on the floor.
- body slightly tilted forward.
- upper arms are close to the body.
- a universal position for 12 hours.
- the distance from the eye doctor to the patient's mouth is 40 cm.

3. Position assistant dentist

Area of work is the assistant for 2-5 hours at 10 cm above the operator.
Area transportation tools 5-8 hours.

4. The position of the patient

Most physiological, comfortable and is optimal posture of the patient when he is in the chair, and the tip of the nose and toes form a single line. Exceptions are patients with the following contraindications:

- pregnant women;
- aged people;
- patients who have problems with the spine;
- patients who have respiratory diseases;
- people absolutely do not want to be treated in this position.

Test tasks

1. On the main chair in the dentist's office is needed area:

- a. 20 square meters.
- b. **14 square meters.**
- c. 10 square meters.
- d. 59 square meters.
- e. 13 square meters.

2. If you have universal systems in the dental office need more space:

- a. 14 square meters.
- b. **10 square meters.**
- c. 7 square meters.
- d. 13 square meters.
- e. 59 square meters.

3. Methods of disinfection:

- a. **physics, chemistry.**
- b. the physical, the air.
- c. chemical, steam.
- d. steam, the physical.
- e. steam, the air.

4. The processing steps dental instruments:

- a. **disinfection, cleaning, sterilization.**
- b. disinfection, cleaning, boiling, sterilization.
- c. autoclaving, pre cleaning, sterilization.
- d. disinfection, autoclaving, boiling.
- e. autoclaving, boiling, sterilization.

5. Disinfection of dental instruments chemical method involves the use of:

- a. 1% chloramine solution, 4% hydrogen peroxide solution.
- b. **3% chloramine solution, 6% hydrogen peroxide solution.**
- c. 1% chloramine solution, 4% hydrogen peroxide solution.
- d. 4% hydrogen peroxide solution, 3% chloramine solution.
- e. 4% hydrogen peroxide solution, 6% hydrogen peroxide solution.

6. Methods of sterilization of dental instruments:

- a. steam, chemical, boiling.
- b. **steam, chemical, air.**
- c. air, steam, boiling.
- d. steam, boiling.
- e. steam, boiling.

7. Phenolphthalein sample is carried out to detect:

- a. residual blood.
- b. residual chlorine compounds.
- c. *residues of detergent (alkaline components).*
- d. residual blood and residues of detergent.
- e. residual blood and residual chlorine compounds.

8. Treat tips twofold:

- a. 4% solution of hydrogen peroxide.
- b. *70⁰ alcohol.*
- c. 3% chlorhexidine.
- d. 1% chloramine solution.
- e. 4% hydrogen peroxide solution.

9. Ways of HIV transmission:

- a. fecal-oral.
- b. sex.
- c. contact-household.
- d. *transplacental.*
- e. *parenteral.*

10. Factors of transmission in the dental practice:

- a. hand medical officer.
- b. Tools, instruments, equipment.
- c. towels, door handles, faucets.
- d. dosage medium.
- e. *all answers are correct.*

11. The composition of kits for emergency medical services (anti-AIDS) does not include:

- a. fingertips (or gloves).
- b. plaster.
- c. *wadding.*
- d. ethyl alcohol 70%.
- e. albucidum 20-30%.

LESSON 2. DIAGNOSTIC METHODS IN THERAPEUTIC DENTISTRY.

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

1. Basic methods of examination of dental patient
2. Schematic survey of dental patients.
3. Dental status at WHO.
4. Clinical diagnostic tests
5. Special research methods
6. X-ray examination
7. Laboratory diagnosis
8. An index score of dental diseases
9. Preparation of a dental treatment plan of the patient.

Question1. Basic methods of examination of dental patient.

All methods are divided into basic and advanced. The main methods are: clinical (survey, inspection) and instrumental (sounding, percussion). Additional methods include: physical (EDI X-ray diagnostics, laser diagnostics), laboratory (bacteriological method, biochemical methods, cytology, histology, virology).

Question 2. Schematic survey of dental patients.

Complaints: on the therapeutic reception most common complaints:

- ✓ pain (nature, duration, irradiation, which provokes that takes),
- ✓ bleeding of the gums,
- ✓ halitosis,
- ✓ functions like disorders (speech, chewing),
- ✓ the presence of dental plaque,
- ✓ prevention inspection.

History of life. It is the patient's memories about the lifestyle, health and social factors that could affect it:

- Social and living conditions,
- Conditions of work (occupational hazard).
- Common diseases (cardiovascular, gastrointestinal, endocrine).

It is essential to HIV, infectious disease, tuberculosis, sexually transmitted diseases.

- whether the patient took drugs
- aggravating factors (alcohol, smoking)
- hereditary pathology,
- the frequency of visits to the dentist,
- features hygienic measures,
- -allergie.

History of the disease - the patient's memories about the causes, manifestations and course of the disease:

- How long it onset,
- What the patient relates the emergence of the disease,
- What held diagnostic and therapeutic measures and their effectiveness,
- About the exacerbation of the disease and their duration.

Question3.Dental status at WHO.

Dental status at the WHO include: extraoral (external inspection), intraoral (oral examination).

Intraoral examination examine: the lips, the eve of the mouth, mucous membrane of the cheeks. Hard and soft palate, tongue, floor of the mouth, the teeth.

Question4. Clinical diagnostic tests.

1. Probing
2. Palpation
3. Percussion
4. Determination of mobility.
5. Mechanical test (biting hard on the subject).
6. Follow the fistulous.
7. Drying.
8. Staining.
9. Temperature test.
10. Local anesthesia.

Probing. It is performed to determine the integrity of hard dental tissue using a probe. It allows you to determine:

- presence of caries and its complications,
- presence and depth of the periodontal pocket.

Percussion: tapping a pen tool or tweezers for cutting edge or the chewing surface of the tooth(horizontal and vertical). Normally it is painless.

Palpation. Held by pressing your index finger on the region of interest, or the capture of the entire stratum, or fold two fingers. You can define:

- pain
- any sharp bony prominences
- edema
- tumor
- tooth mobility
- consistency
- tissue turgor
- nodal status
- state of the musculoskeletal system

- separation of pathological periodontal pockets
- bleeding gums palpation

Definition of mobility. Mechanical test.

Held by biting hard on the subject, to diagnose diseases of the apical and marginal periodontium.

Colouration. A 2% solution of methylene blue is applied onto the dried portion of the tooth for 2-3 minutes. Then the mouth rinsed with water. Then determine the degree of staining. It is used for diagnosis of caries and non-carious lesions, determination of hygienic habits.

They used special cavities or caries detectors - markers to determine the pathological changed tooth tissues during dissection. They were offered by Professor Fusayama (Japan).

Temperature test. The test tool is heated with heating or gutta-percha and the flame is applied to the tooth in the middle third of the facial surface.

Test cooling jet Use cold water or a cotton chloroethyl on the shelf or the Ad Hoc refrigerants (eg, spray Coolan)

There are several types of reactions:

- there is no response (complete necrosis of the pulp, apical periodontitis, tooth cavity obliteration)
- just passing reaction (healthy tooth, carious lesions, dental caries)
- quickly passing reaction (hyperemia of the pulp)
- painful reaction from the cold, the long-term passes (acute serous pulpitis)
- pain from hot, not long passes (acute purulent pulpitis)
- long growing slowly passing pain (chronic pulpitis).

Drying. Most often used for the diagnosis of caries and non-carious lesions. When drying hard tissues of the tooth enamel healthy air jet has a shiny, smooth surface, and the expression in caries - matte and textured. Also, using this method can be determined, pour the fillings.

Question 5. Special research methods.

- electric pulp test
- transillumination
- X-ray examination
- laser immunofluorescence techniques
- definition of bioelectric potential

Electric pulp test.

Devices: PDE-1-PDE 2 (Russia), PULPOTESTER (Latvia), Digitest (USA).

The methodology of: a study carried out with the assistant. The doctor puts one passive electrode on the patient's forearm, active - on the tooth. For

canines and incisors at the cutting edge for premolars - to the top of hill. It is also possible to the bottom cavity or filling. This is due to the fact that the largest number of data points in the dentinal tubules containing water and reduce the electrical resistance of hard tissues. Assistant to turn the knob - the potentiometer until the patient senses and captures this value. One should note-tit that unformed teeth have a higher value of EDI, so the differential diagnosis should be investigated eponymous tooth on the opposite side.

1. Intact tooth 2-6 mA
2. Caries 2-10
3. Flushing pulp 12-18
4. Acute pulpitis 20-30
5. Purulent pulpitis 30-60
6. Chronic pulpitis 40-60
7. Chronic gangrenous 60-90.
8. Chronic hyperplastic 50-70
9. Necrosis of the pulp (dry) 60-80
10. Pulp necrosis (wet) 100
11. Chronic apical periodontitis more than 100

Transillumination. For the diagnosis of approximal caries. In the darkroom fiber optic nakonech nickname blue light is placed behind the investigated tooth perpendicular to its axis. Healthy tissue you-look transparent, cavities - as brown shadow in the shape of a hemisphere.

Laser diagnostics (fluorescence method). The device DIAGNOdent (KaVo, Germany). The device comprises a laser diode (wavelength 650 nm) and a photodiode. Activating light is transmitted by an optical fiber to the tooth, length-nofokusny filter collects and transmits the excitement back to the long-wavelength fluorescence. The digital display shows the maximum intensity of luminescence at the time the EC-repetition. The device has a nozzle for the diagnosis of fissure caries and caries on smooth-surfaces.

Question 6. X-ray examination.

Carried out for:

- Determination of cavities,
- With injuries of teeth, jaws,
- To determine the depth of the caries process,
- For differential diagnosis forms of caries and apical periodontitis,
- To identify the type of the pathological process in the marginal and apical periodontitis,
- To evaluate the quality endodontic treatment,
- To evaluate the efficacy of treatment of periodontal diseases marginal.

The main types of X-ray in restorative dentistry:

1. Pritselnye dental pictures.
2. Orthopantomography.

3. BITEWING-radiography (Bite pictures).

Sights dental images allows to obtain a detailed view of the interdental septum, furcations roots, periodontal ligament.

Orthopantomogram provides a cross-sectional image of the entire dentition used as a single functional complex, to plan and evaluate the treatment of marginal periodontal.

BITEWING-radiography (Bite shots) is used for the diagnosis of approximal caries.

The order of the X-ray:

1. The quality of radiographs (shadows, overlays, and so on)
2. Determine the type of snapshot (intraoral, extraoral and panoramic)
3. Determine the anatomy of teeth and group membership
4. Position, size, shape of the tooth crown, a contour violation, the presence of pathological ones it.
5. The contours of the tooth cavity and root canal, the severity of the coronal tooth cavity, length, and width in the root canal, the presence dentikley etc.
6. Periodontal gap - normal uniform illumination strip between cement and cortical plate, at a pathology it narrows or expands, or its integrity is violated.
7. Determination of bone structure. Evaluate the structure of the cortical plate, interdental partitions, state spongy substance. In the pathology can be observed atrophy, osteopenia, roses, destruction, osteonecrosis, sequestration.

Description pathological shadows:

1. Localization
2. The ratio of the various anatomical structures (bones, maxillary sinus, mandibular canal)
3. The number of (single or multiple)
4. The shape (round, oval, right, wrong, etc.)
5. The sizes (up to 5mm - granuloma, 5-8 mm - kistogranulema, 8 mm - a cyst)
6. The nature of contours (smooth or rough, clear or fuzzy, blurred and sharp, smooth and polycyclic).
7. Intensity (compared to the intensity of tooth tissues or cortical bone)
8. The nature of the structure of the shade (homogeneous or heterogeneous).

Question7. Laboratory diagnosis.

It held primarily for diseases of the periodontal and GPRS.

1. Bacteriological research: analysis of the flora obtained from the lesion.
2. Microscopic examination is carried out by light microscopy.
3. Molecular biological research methods (PCR and hybridization method).

4. Cytological methods (smear-imprint smear reprints, sediment washing liquid).
5. Histological examination (biopsy).
6. Virological studies (IFA, ELISA, infecting chicken embryos).
7. Allergic studies (intraoral tests in vivo, blood cell count, tests with a standard set of allergens).

Question8. An index score of dental diseases.

1. To evaluate the oral hygiene (OHI-S, 1964).
2. For evaluating gingival (GI).
3. To assess the state of periodontal tissue (CPI, Leus, 1988).
4. To assess the state of the hard tissue of teeth (DFM).

Question9. Preparation of a dental treatment plan of the patient.

Dental patient's treatment plan includes:

1. Emergency aid.
2. Motivation and training of oral hygiene.
3. Professional oral hygiene.
4. Selection of individual drugs and hygiene practices.
5. Therapeutic treatment.
6. Surgery treatment.
7. Orthopaedic treatment.
8. Medication treatment.
 - Local treatment.
 - General treatment.
9. Control visits 2 times a year

Test tasks

1. Which of the following diseases must be postponed sure to ask a dental patient:

- a. *Viral hepatitis*,
- b. Colds,
- c. *Tuberculosis*,
- d. *Venereal* diseases,
- e. Hereditary diseases.

2. Which of the following applies to intraoral examination:

- a. Evaluation of the symmetry of the face,
- b. Evaluation of the skin,
- c. *Inspection of dentition*,
- d. *Inspection vestibule of mouth*,
- e. Inspection of the oral mucosa.

3. EDI data 12-18 mA with the diagnosis:

- a. *Hyperemia of the pulp,*
- b. Acute pulpitis
- c. Caries of dentine,
- d. Acute apical periodontitis,
- e. Chronic apical periodontitis.

4. The methods of laboratory diagnostics include:

- a. *Microscopic examination,*
- b. *Virological examination,*
- c. Probing,
- d. *Biopsy,*
- e. Percussion.

5. The patient's dental treatment plan includes:

- a. *Motivation and training of oral hygiene,*
- b. *Professional oral hygiene,*
- c. *Drug therapy,*
- d. *Surgical treatment,*
- e. Orthodontic treatment.

LESSON 3. DENTAL PATIENT CARD. RECORDING AND REPORTING DENTAL RECORDS. HOW TO FILL OUT.

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

- 1. Outpatients dental health card, a concept components. Other recording and reporting on the dental admission.
- 2. How to fill the dental patient card. Examination of the patient during the initial treatment. Transferred and concomitant disease.
- 3. Dental status, rules of filling
- 4. Preparation of a dental treatment plan of the patient.
- 5. Making diary visits.

Question1. Outpatients dental health card, a concept components.

Other recording and reporting on the dental admission.

Recording and reporting on the dental admission form number 037/u-10 "Leaf daily accounting work of a dentist (dental paramedic)"; form number 039/10-in "Diary of accounting work of a dentist (dental paramedic); form number 039-W/Y-10, "Summary of dental health of the patients with primary-increment; form number 043/10-in "Dental patient card" and others.

The form number 037/u-10 "Leaf daily accounting work of a dentist (dental paramedic)" has the following columns: date, time of reception of

patients, the number of full years, the name, address, diagnosis (ICD code and description), treatment (view and description), type of visit, the key code of the group intact dentition indices.

The form number 039/10-in "Diary of accounting work of a dentist (tooth paramedic-foot) includes the following sections: the total number of visits, number of visits to primary, preventive work, the diagnosis of the completed treatment, therapeutic treatment.

The form number 043/10-in "Dental patient card" co-featured bout is the main document of primary medical documentation, legally binding and wearing auxiliary character, which is reflected and documented in the dynamics state of dental health of the patient, the process of diagnosis, treatment and prevention dental diseases.

Outpatient map consists of several sections:

- Data;
- "Taking into account the list of appointments and stress radiographic studies";
- "Examination of the patient in the primary treatment";
- "Prior consent to medical intervention";
- Journal of visits.

In the section "examination of the patient in the primary treatment" includes subsections:

- Complaints;
- State of general health with the words of the patient;
- External inspection;
- Stomatological status;
- Total treatment plan based on the results of the patient examination in primary increments.

Question 2. How to fill the dental patient card. Examination of the patient during the initial treatment. Transferred and concomitant disease.

Form 043/u - 10 conducted for each patient at each visit dentists.

Form 043/u - 10 is filled by a dentist in Russian or in legible handwriting on the computer. Filling in all the fields and subject lines, only in the case of the provision of emergency care to the patient is allowed to fill only those partitions which needed to reflect the process of emergency care. Filling the passport data held in the registry based on document of the patient.

Outpatient map is stored in the registry for 10 years after the last patient visit. Control over the conduct of the head of the patient card is carried out structural units.

Examination of the patient during the initial treatment. Transferred and comorbidities.

The primary visit is to visit a patient, first applied in the current year for dental care in this organization, regardless of the type of treatment. Any other visits to the dentist in the organization present year is repeated.

The string "Reason complaints" made the complaint of the patient and medical history data.

In the table, "the general state of health of the patient with the words" made data and related diseases, allergoanamneze, heredity, bad habits, occupational hazards, conditions of life.

In the column "delete as appropriate" designation "YES" crossed out on the diseases, whose presence at the patient denies; designation of "NO" crossed out on the diseases that the patient is at confirming or denying.

Under "If YES, specify" refined diagnoses identified diseases.

In the "other" information marked with a significant relation to withstanding the dental health of the patient.

In the "external examination" include data external examination of the patient: configuration persons, the state of the skin and red border, regional nodes, tic, temporomandibular joint.

Question 3. Dental status, rules of filling.

In the "dental status" include data on the state of the teeth, gums, periodontal, oral mucosa, oral hygiene.

Dental health noted in the appropriate boxes dentition conditional-governmental designations in the table.

- Status Designation tooth
- Healthy teeth 0
- Cavities 1
- Seal 3
- Removed 4
- Artificial crowns 7
- Impacted tooth 8
- Bridges 7 April 7

Status defined oral hygiene in tables «OHI-S» for children and ADULT-bite-integer constant.

Method for determining «OHI-S». Method for determining the CPI (A comprehensive periodontal index), GI(adult, child).To complex medical interventions in the provision of outpatient dental care include:

- invasive methods of diagnosis and treatment;
- ortopedic treatment;
- orthodontictreatment.

Juvenile persons found incompetent, the consent of their legal representatives provide, in the case of patients who are unable for health

reasons to make an informed decision, the husband, and in its absence - a close relative. In the case of oral consent to medical intervention and refusal to issue it in writing in the line "give voluntary consent to medical intervention" word "intervention conducted by written consent of the patient or his legal representative, of" put the date, the signature of the attending physician and the head structural unit.

Consent to medical intervention may be withdrawn, except when the doctor began to intervene.

Question 4. Preparation of a dental treatment plan of the patient.

Planning for dental treatment carried out on the basis of the first survey was conducted, and the diagnosis is individual, is an integrated, providing the unity of purpose of the medical activities. It presupposes a certain amount and the scheme of dental procedures.

The treatment plan includes:

1. Emergency aid.
2. Motivation and training of oral hygiene.
3. Professional oral hygiene.
4. Selection of individual drugs and hygiene practices.
5. Non-surgical treatment.
6. Surgery.
7. Orthopedical treatment.
8. Orthodontic treatment.
9. Medication:
 - Local.
 - General.
10. Additional diagnostic measures.
11. Consultation other professionals.
12. Visits 2 times a year.

The physician should inform the patient or his legal representative with the treatment plan.

Question 5. Making diary visits.

This section shall be entered information reflecting the dynamics of the process of providing assistance to dentists, indicating the date of visits, procedures carried out and the names of the physician with his signature.

The attending dentist says complaints clinical picture, results of studies, makes a diagnosis. It makes a treatment plan and records of the treatment process. When you visit records are maintained until the end of treatment.

On the department of therapeutic dentistry is available electronic version of the Order of the Ministry of Health of Belarus from 14.01.2011 № 24 "On approval of forms of primary medical documentation in dentistry."

Test tasks

1. Which of the following applies to intra-oral examination:

- a. Evaluation of the symmetry of the face
- b. Evaluation of the skin
- c. *Inspection dentition*
- d. *Inspection vestibule of mouth*
- e. Assessment of the nodes.

2. Clinical diagnostic tests include:

- a. Sounding
- b. Percussion
- c. Colouration
- d. Palpation
- e. Thermodiagnosics
- f. *All answers are correct*

3. EOD is 12-18 mA with the diagnosis:

- a. *Hyperemia of the pulp*
- b. Acute pulpitis
- c. Caries
- d. Apical periodontitis
- e. Chronic pulpitis

4. The methods of laboratory diagnostics include:

- a. *Microscopic examination*
- b. *Virological*
- c. Sounding
- d. *Biopsy*
- e. Microbiologically study

5. Types of dental indices:

- a. *index of oral hygiene*
- b. *periodontal indices*
- c. dental codes
- d. gingival index
- e. indexes condition of the oral mucosa

6. The patient's dental treatment plan may include:

- a. Motivation and training of oral hygiene
- b. Professional oral hygiene
- c. Drug therapy
- d. Surgical treatment
- e. Orthodontic treatment

f. *All answers are correct.*

LESSON 4. INDIVIDUAL AND PROFESSIONAL ORAL HYGIENE.THE DISPLAY METHOD OF DENTAL PLAQUE. HYGIENIC INDEXES.

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

1. Dental deposits. Classification of dental deposits.
2. Non-mineralized dental plaque. The cuticle, pellicle, plaque. Definition, composition, mechanism of formation.
3. Characteristics, mechanism of formation, properties of dental plaque.
4. The mineralized dental plaque. Types of tartar. Theories formation of tartar. Role in the pathology of the oral cavity.
5. Methods for detection of dental plaque and hygiene codes.
6. Motivational conversation with patients instructed in oral hygiene.
7. Objects and personal of oral hygiene.
8. Basic methods of tooth brushing hand brush.
9. Professional oral hygiene.

Question 1. Dental deposits. Classification of dental deposits.

They are cuticle, pellicle by origin natural and microbial pellicle, dental plaque, tartar.

Localization all dental deposits are divided into: supragingival (pellicle, plaque, dental plaque, supragingival tartar) and subgingival (subgingival calculus).

According to the degree of infection all dental deposits are divided into: not infected (cuticle pellicle) and infected (dental plaque, dental plaque, tartar).

According to the degree of mineralization of all dental deposits are divided into: saline (tartar), non-mineralized (pellicle, plaque, dental plaque).

International Classification of Diseases (ICD-DA WHO, 1995)

- K03.6 Deposits [accretions] on teeth.
- K03.60 Pigmented dental deposits (black, green, orange).
- K03.61 Due to tobacco habit.
- K03.62 Due to betel-chewing habit.
- K03.63 Other gross soft deposits (Materia alba).
- K03.64 Supragingival calculus.
- K03.65 Subgingival calculus .
- K03.66 Dental plaque.
- K03.68 Other specified deposits on teeth.

- K03.69 Deposit on teeth, unspecified.

Question 2. Non-mineralized dental plaque. The cuticle, pellicle, plaque.

Definition, composition, mechanism of formation.

Cuticle is the reduced epithelium of the enamel organ. Tooth loses cuticle shortly after the eruption of the tooth, so the clinical significance it has.

Pellicle is structureless uninfected acellular glycoprotein on the membrane surface in the tooth. It is a product of protein-carbohydrate complexes oral liquid, such as mucin glycoproteins, sialoprotein. The thickness is from 1 to 10 microns. The size of it is thinner on the crests perikemata thicker - in the furrows, on the contact surfaces of the tooth and gingival margin. Under the layer of plaque-eat it thickens over carious spot becomes thinner.

The role of the pellicle. It controls the diffusion processes in the surface layer of enamel, teacher-exists acids on the tooth and the diffusion of calcium and phosphate from the tooth. It gives the enamel electingtonal permeability.

Distributed also felt that the pellicle gives rise to the formation of dental plaque. However Y. Erricson and B. Forsman reported that pellicle not only accelerates microbial colonization, and may even impede it. Possibly, pellicle plays a role in the development of caries (Leont'ev V.K., 1976), because regulates the diffusion and permeability in the surface layer of the enamel-prefecture, its solubility in an acidic environment. Changes in the composition and properties of the pellicle may favor the development of caries.

The mechanism of formation of pellicle. A spontaneous deposition of protein oral liquid on the enamel surface. This process is not depend on the bacterial activity.

Precipitated protein oral liquid increases significantly upon acidification its hole, and in the presence of calcium ions and phosphate. Calcium ions are involved in the attachment of bacteria to the pellicle. While maintaining these conditions in the oral cavity as part of pellicle amino sugars begin to predominate - derived bacterial membranes as well as high-molecular glycoproteins having the ability to agglutinate bacteria. Bacterial-cells are found in the pellicle. The organic composition of the pellicle should be considered as a mixture of salivary proteins of bacterial origin.

As part of the pellicle layer 3 release: sub-surface (it is chemically bonded to the enamel), in surfactant (formed by the precipitation of further components of the oral fluid) surface (has a rough surface and facilitates the connection of the following types of dental plaque).

Pellicle is subject to mechanical failure, but recovers quickly (the speed of its formation is 20-30 minutes).

Plaque is a sticky, loose adjacent to the tooth surface, a conglomerate consisting of microorganisms exfoliated epithelial cells, leukocytes, the mixture versus-new (human and bacterial), lipid particles with food (including chromogenic substances) or without them.

It accumulates on the surface of teeth, fillings, gums mainly during speech and chewing of rest and in the absence of efficient oral hygiene. The spread on the surface of plaque tooth comes from between the teeth and gum grooves. When the precient oral hygiene, use of solid and solid food of the soft plaque from surfaces in the teeth and gums are constantly removed, but he quickly re-formed. Thus, after brushing plaque begins to form after 2 hours.

The composition of the plaque. Soft plaque than water (80%), containing microorganisms and a relatively small amount of inorganic substances in the form of calcium (5 nr/mg dry weight), total phosphorous (16 Hr/mg dry weight), of which 4 nr/mg dry weight inorganic phosphorus represented. The structure of the tooth-foot plaque may include chromogenic substance. Depending on the availability of chromogenic substances, plaque may have different colors. If the stain of plaque is white or yellow, this is ordinary plaque. Green plaque is more common in children. It is formed by the activity of chromogenic micro-organisms containing chlorophyll. The brownplaque is more common in the smokers. The plaque is colored due to nicotine.

The dental plaque contains a large number of microorganisms. Of these, the most aggressive is Str.mutans.For this organism is characterized by:

1. Development of adhesive polysaccharides (levan, dextran).
2. Tolerance to acid (survive at pH <5.5).
3. The presence of adhesion molecules to the mucosa and tooth.
4. Development of organic acids (water demineralization action).

Contact bacteria pellicle of the tooth can occur via surface structures micro-organisms is called lectins. Lectins are typically form fibrils or fimbriae. The non-specific attachment of the bacteria can occur with the participation of teichoic acid cell wall. Teichoic acid binds to calcium ions of hard tissues of the tooth, or Pell molecule. Important factors are pathogenic plaque and microbial enzymes.

There are 3 stages of plaque formation:

1. Colonization.
2. Rapid bacterial growth.
3. Update plaque.

Question 3. Characteristics, mechanism of formation, properties of dental plaque.

Dental plaque is a dense structured education, located above the pellicle. Plaque is a matrix formed by proteins, polysaccharides, lipids, inorganic substance such as: calcium, phosphate, magnesium, potassium, sodium. The plaque has a high-pathogenic properties.

The mechanism of formation of dental plaque. Changing the microbial composition of plaque, the appearance of the matrix indicates the transition plaque dental plaque. Some authors do not tend to make a sharp distinction between the plaque and dental plaque. Dental plaque is seen as the final stage of plaque formation.

Initially, plaque approximately 50% consists of a predominance of streptococci and *S.mutans* *S.salivarius*. As the plaque thickens and creates anaerobic conditions within it, changes and microflora. On 2 - 3 day in the dental plaque appear Gram-WIDE cocci and rods. Subsequently, an increase in their number up to 30%, of which approximately 15% are anaerobic bacillus. On 4 - 5 day there *fuzobakterii*, *acti-nomyces* and *veillonella*. Dramatically increasing the number of strict anaerobes, and *veillonella* co-constitutes 16% of the total microflora. After 7 days in plaque and appear *spirella* *spirochetes*, gram-positive rods represent 50% of total microflora. The plaque becomes mature in three weeks.

Question 4. The mineralized dental plaque. Types of tartar. Theories formation of tartar. Role in the pathology of the oral cavity.

Plaque is a dense, amorphous, mineralized dental plaque. In the structure comprises a crystalline structure of calcium phosphate.

The chemical composition of supragingival tartar is slightly different from the subgingival plaque

Composition of tartar. The organic substance is 10-30%. These include: protein, polysaccharide complexes, desquamated epithelial cells, leukocytes, microbial body.

Inorganic substances constitute 70-90%. These include: brushite, phosphate, hydroxyapatite, calcite, coins, apatite.

By origin, as well as localization, tartar divided into supragingival and subgingival.

The mechanism of the formation of tartar. At the heart of tartar is the process of mineralization. The essence of the process concludes in binding of calcium ions to the protein-polysaccharide complex organic matrixes plaque and deposition of calcium phosphates. Initially formed crystals in intercellular matrix and on the surfaces of bacteria and then the bacteria inside. . The original stone is formed along the inner surface of the plaque adjacent to the tooth, and then deposits forms a solid mass of monolithic stone. The crystals of calcium phosphate, which are deposited in the inner

layers of dental plaque, may be closely related to the surface of EMA-Do, it is sometimes difficult to determine where it ends and begins a stone enamel. Home and speed mineralization varies in different individuals and different teeth of the individual. This allows to select people with fast, moderate, minor stone formation, as well as those who have stone is formed. The reasons for these differences are not fully understood.

The process of stone formation is divided into three phases, depending on the mineralization centers. Sufficient time for the formation of tartar is the 2nd of the month, followed by internal adjustment and replacement of the organic matter.

The theories of tartar.

1. Salivary theory suggests that phosphate and calcium carbonate are dissolved in saliva due to an excess of carbonic acid. Drop salt from solution is a result of evaporation of a portion of carbonic acid saliva in contact with air.

2. Theory of qualitative and quantitative changes in the saliva claims that colloidal protein saliva bind calcium and phosphate ions and keep a supersaturated solution with respect to the calcium phosphate. With increasing congestion saliva colloids break supersaturated state is not saved and calcium phosphate precipitate.

3. Phosphatase theory involves the release phosphatase from plaque exfoliating epithelial cells, bacteria. Phosphatase precipitates the calcium phosphate-hydrolyzing organical phosphates and saliva, thereby increasing the concentration of free phosphate ions. Another enzyme is contained in coccileptotrihiyah, actinomycetes, leukocytes plaque may also start mineralization by hydrolysis of fatty esters to free fatty acids, which form with calcium and magnesium soaps, subsequently turning into a less soluble calcium phosphate. In the formation of tartar play a role of microorganisms. Plaque mineralization begins intracellularly in some gram positive-negative bacteria, and proceeds as long as the matrix solidifies and the bacteria.

Pathogenic significance of tartar. Tartar mechanically irritating the epithelium of the gingival sulcus and its promotion in apical direction leads to the destruction of periodontal connection. Along with this exists the opinion that calculus is inert, and its formation is a natural attempt to reduce the pathogenic effects of plaque. Most scholars recognize that the main negative feature of tartar is its ability to accumulate on the surface plaque, pathogenetic role is convincingly proved.

Question 5. Methods for detection of dental plaque and hygiene codes.

1. Visual.
2. Staining.
3. Drying.
4. Probing.

One of the most informative methods for the detection of dental plaque is a painting. With this method you can determine the size, thickness, location of dental plaque.

To paint using special dyes, which can be divided into 2 groups: persistent (erythrosine) and unstable (iodine).

The method of application of the dye: dye is applied to the dried teeth with a cotton swab for 2 - 3 minutes, then allow the patient to rinse the mouth with water and evaluate the results. When the dental plaque is thicker, the intensity of the color greater.

On drying the tooth surface coated plaque has a roughened surface.

Probing for the detection of dental plaque was performed as follows: the probe is moved from the cutting edge or a knoll to the gum. In the case of dental plaque, it remains at the tip of the probe. With the sensing area can be determined plaque and the quantity.

Determining the level of oral hygiene with sanitary codes.

There are several groups of indices:

1. To evaluate the oral hygiene (OHI-S; Green, Vermillion, 1964), the efficiency index oral hygiene (PHP) Podshadley, Haley (1968).
2. To assess the gingival (GI; Loe, Silness, 1963).
3. To assess the state of periodontal tissue: periodontal index (CPITN; WHO, 1982; CPI; Leus PA, 1988).

Periodontal Index CPITN (WHO, 1982).

Performance Index ORAL HYGIENE (PHP) Podshadley, Haley (1968).

To quantify the plaque stained teeth 6:

- 16, 26, 11, 31 - vestibular surface;
- 36, 46 - lingual surfaces.

In the absence of the index can be inspected adjacent tooth, but within the homonymous group of teeth. Artificial crowns and fixed prosthesis part examined as well as the teeth.

Observed surface of each tooth is divided into 5 sections:

- 1 - Medial.
- 2 - Distal.
- 3 - Mid-occlusive.
- 4 - Central.
- 5 - Mid-Cervical.

CODES AND EVALUATION CRITERIA plaque:

0 - no staining

1 - staining revealed

Index calculation is performed by determining a code for each tooth by adding codes for each segment. Then summarize the codes for all examined teeth and divide the total by the number of teeth.

The index is calculated by the following formula:

$$PHP = \frac{\text{SUM CODES all teeth}}{\text{number of examined teeth}}$$

If interpretation of the result is:

0 - it is an excellent level of hygiene

0.1-0.6 - it is a good level of hygiene

0.7-1.6 - it is a satisfactory level of hygiene

1.7 and more - it is an unsatisfactory level of hygiene.

Question 6. Motivational conversation with patients instructed in oral hygiene.

Training oral hygiene is a major component of health education a dentist, his assistant and hygienist. Dental hygiene training staff will not only patients but also doctors of other specialties, teachers, educators, parents who, in turn, engaged in teaching hygiene charges. Training can be conducted by various methods and techniques (both individual and group, both office utilities).

The most common form of learning the rules of oral care is a lesson in hygiene. Basic structure of a lesson Hygiene built in accordance with the objectives of training: Stage 1 - motivation, Stage 2 - the choice of means and methods of hygiene, Stage 3 - practical training method chosen hygiene.

1. Motivation. In general, during the conversation the patient should make sure that:

- patients with dental problem is directly related to it in the present (the patient to demonstrate the problem in his mouth);
- healthy teeth better than patients (say about the absence of discomfort and pain, of beauty, of the possibility not to give up any food, the preservation of the health of the stomach);
- dental health can be saved by means of dental care using modern knowledge and relatively inexpensive means of effective prevention;
- dental disease associated with many different factors (list system and general risk factors);
- plaque at the moment to attack the patient's teeth (demonstrate his plaque on the probe on the Floss, after staining on the teeth, in the native preparation of plaque in a microscope).

The result of this step should be the appearance of the patient's desire to immediately get rid of the dental plaque.

2. The choice of means and methods of hygiene. At this stage, the doctor informs the patient about which the brush, paste, dental floss and other hygiene required in the individual case it for effective cleaning of teeth.

3. Training in the dentifrice. For training use two basic techniques:

- demonstration training model;
- practical training in the oral cavity (supervised tooth brushing).

Question 7. Objects and personal of oral hygiene.

The main subjects of individual oral hygiene are:

1. Toothbrushes.

2. Aids:

- The toothpicks;
- Dental floss (dental floss);
- Brushes;
- Irrigators;
- Brush for language.

Toothbrushes

Bristle. The ability to transfer the bristles pressure force from its base on the working surface known as hardness (softness). Stiffness depends on the bristles:

- on the material of construction of the bristle;
- the diameter of the bristles (the thicker, the stiffer);
- the length of the bristles (the shorter, the harder).

There are two basic types of materials for making the bristles: natural and synthetic. Currently used synthetic materials for the bristles.

For children only recommended soft bristles. Brushes are often labeled stiffness index in accordance with standard of ISO 8627 (1987 year): soft, medium-hard, hard.

There are some representatives of toothbrushes for special purposes.

Toothbrush NUR-1-TROPFEN (for language)

Indications:

1. If there is a large amount of plaque on the tongue.
2. With deep grooves and fissures on the tongue.
3. "Hairy" tongue.
4. Heavy smokers. Mode of application:

Cleaning the tongue is the final step of oral hygiene. The toothbrush should be placed closer to the root of the tongue and move forward, gently pressing on the tongue.

Toothbrushes type DENTURE. Bioactive head with a rigid and very stiff bristles. They are used to clean the inner and outer surfaces of dentures.

Toothbrushes type ORTHO. Have a V-shaped arrangement of the bristle type that allows a well to clear locks and arc brekkt system.

Toothbrushes type INTERDENTAL. The cone-shaped working part of their brush. Used to clean between the teeth, locks brekkt complete cleaning system.

Toothbrushes type MONOTAFTS (single-beam). Used for cleaning the wash space under bridges, adhesive bridges, as well as to clean the locks brekkt system.

Toothbrushes type SULCUS. Having a narrow design brush head enables it to penetrate into inaccessible places for cleaning the oral cavity (retromolar area, lateral teeth when equipped with tires, etc.).

Kids Toothbrushes. Toothbrushes for children, as well as toothbrushes for adults, divided by the hygienic and prevention. Keep in mind that children's toothbrush to make special demands:

1. Small toothbrush head.
2. Soft and very soft bristles.
3. Large and easy to grip handle.
4. It is desirable to control the presence of indicator brushing.
5. Presence in the design of the brush - toys, cartoon characters, sounds, color sensor.

The use of children's toothbrushes and have age criteria.

Besides the usual, there are toothbrushes toothbrushes for electrophoresis of fluoride ions, electric toothbrushes.

Toothbrush electrophoresis fluoride ion has two electrodes made of different materials. One electrode is pressed against the teeth, the other is hidden beneath the brush bristles. In the treatment of teeth occurs between the electrodes, and an electric potential difference in electrolyte (it is dissolved in saliva toothpaste) begins to flow imperceptible to the user of the toothbrush weak current, which facilitates the penetration of fluoride ions into the enamel.

Electro. In addition to a cleaning brush massage gums can be made, however, for the last procedure must be supplemented by a set of special rubber nozzle. An example of such a brush is an electric toothbrush «Braun Oral II Control Ultra». Significantly greater cleaning effect is achieved due to the high frequency reciprocating rotary motion of the brush head - 3800 strokes per minute - in the range of 70 degrees. The unique design of the brush head reflects the most recent advances in the field of dentistry. Two power projection and the cup-shaped cleaning head allows brush thoroughly remove plaque from all tooth surfaces, penetrating deeper between teeth and below the gum line.

The main advantage of electric toothbrushes is a three-dimensional cleaning effect, the combination of back - rotational and pulsating movements.

The presence in the construction of the brush pressure sensor eliminates the use of increased pressure on the tissues of the tooth during brushing, the brush is stopped.

Built in timer allows you to accurately record the working time duration of brushing.

Having multiple speeds the active brush head allows to achieve a more thorough cleaning.

A special type of Flexi Soft bristles has increased flexibility, which allows it to penetrate deeply into the interdental space and effectively clean the cervical area of the teeth.

Terms of use of electric toothbrushes:

1. The brush head set strictly perpendicular to the tooth surface.
2. The head moves slowly through the adjacent gingiva from the tooth to the tooth.
3. Each tooth was purified cheek, chewing, lingual surfaces.
4. The applied pressure should not be large, it may be less than that of a conventional toothbrush.

Electric toothbrushes are shown to use for daily oral hygiene. It must be remembered that the electric toothbrush can be a daily oral hygiene only if the person has learned to brush your teeth simple toothbrush.

Contraindications to the use of electric toothbrushes:

1. Severe periodontal disease, and gingivitis hypertrophic.
2. Increased sensitivity of tooth enamel.
3. Presence of a pacemaker.
4. Cancer of the oral cavity.
5. Persons with severe mental development.

Ultrasonic toothbrushes. The only representative of today is a toothbrush ULTRASONEX. This brush generates two frequencies of 150 Hz and 15 MHz. Ultrasonic vibration frequency of 15 MHz have an antibacterial effect, break the chains of microorganisms, and sound waves have a frequency of 150 Hz, foaming cleansing effect.

Ultrasonic toothbrush and prevent the development of gingivitis, thanks to preventive action of ultrasound. This toothbrush is recommended for patients with orthodontic appliances, fixed prosthetic designs, implants, periodontal tissue in diseases of mild severity.

Dental floss (dental floss). Dental floss or dental floss are auxiliary mechanical hygiene and are designed to clean between the teeth intervals. Their use is recommended for all, since the structure of the toothbrush does not allow it to adequately penetrate the interdental spaces.

Dental floss subdivided into groups:

1. The shape of the cross-sectional shape:
 - Flat (interdental tapes);
 - Round.
2. Count fibers
 - Monofilament;
 - Multifilament.
3. Surface treatment:
 - Waxed;
 - Unwaxed.
4. In the presence of impregnation:
 - Without special impregnation;
 - Subsistence curative substances.
5. According to the method of application.

- For personal use;
- For office.

Interdental thread consists of at least one fiber subjected to a special on-processing in the production in order to increase the tensile strength, and is covered with liquid paraffin mixture to reduce the coefficient of friction.

Superfloss presented a broad thread (3-4 times wider than floss), designed to cleanse the interdental spaces, mainly shows individuals with diastema.

There are also disposable devices for tensioning dental floss - flossety.

We recommend the following method of using dental floss. Thread a length of 35-40 cm, is wound around one phalanx of middle finger of each hand. Slowly and carefully introduced into teeth intervals, and then pull at the bottom of the gingival sulcus. With a few movements of the thread (6-7 times) back - forward, up and down remove all soft dental plaque the distal surface of the tooth. Then purified medial surface of the tooth. To do this, gently move the thread tightly pressed to the surface of the tooth, moving back - through the contact point remove plaque. You can use the 2% solution of sodium fluoride to prevent tooth decay. Just thread may be impregnated with menthol or different antiseptics.

Additional objects, means and methods of personal oral hygiene for persons with altered periodontitis.

To cleanse the wide interdental spaces free gum for the treatment of proximal surfaces of detached teeth and use a variety of special materials at hand, for example:

Onebeam brush their design differ from the conventional brushes only those that have very little rounded head (1 cm in diameter) 1-6 tufts of bristles, wherein sheared working field has a conical shape. The sharp tip of the brush is introduced into the interdental space, pushing him out of food waste; when the side surfaces of the vibrating movements clean the proximal portions of the teeth.

Interdental brushes is a small brush spiral fibers are fixed between the two metallic wires intertwined, employees and the head and the handle. Brushes can be cylindrical in shape and working surface of different diameters. The workpart of their brush is introduced into the recess or three, and clean the surface of the teeth reciprocating oral-vestibular movements. Brushes can be used to treat concave surfaces of the root.

Toothpicks are the most simple means to remove food residue of their mouth. Currently used as toothpicks wooden or plastic rods of length 5-8 cm, round or triangular in cross section with a diameter of 2-3 mm.

Toothpicks can not be used by people with untreated periodontitis, as in this case, the gums can respond to constant mechanical effect recession.

Hygiene products used in teeth cleaning hand brush. Tooth powders are medications, consisting mainly of abrasive. The formula toothpaste contains a number of components:

- 1) abrasive (5-50%);
- 2) humectants (20-30%);
- 3) water (20-30%);
- 4) binders (1-2%);
- 5) detergents (1-3%);
- 6) aromatizing and flavoring agents (1-2%);
- 7) preservatives (0.05-0.5%);
- 8) the therapeutic agent (0.4-1.0%).

The main active principle of pastes is abrasive. In recent years, giving way to paste hygienic medical preventive paste having in its composition special farmakological supplements. If the treatment and prevention paste contains some components against risk factors and/or symptoms of the disease, it refers to a combination, if the paste ingredients are combined, active in the risk factors and/or symptoms of diseases some - it belongs to complex paste. The anticaries paste includes:

- influencing the activity and growth of plaque: with antiseptics, enzymes, fluorine compounds;
- promoting remineralization and mineralization of hard tissue of the tooth with fluorides, soluble compounds of calcium and phosphorus (including hydroxyapatite) complexes with macro- and microelements (Remodent, crushed eggshells and t. D.).

Question 8. Basic methods of tooth brushing hand brush.

The standart methods is recommended for adults. The method of Martaller is recommended for primary school pupils. and the method KAI is recommended for pre-school children.

The standard method for cleaning teeth

This method is considered the most complete and effective way to clean teeth with a manual toothbrush. Dental arch is conventionally divided into a relatively straight segments - segments where possible planar contact surfaces of several adjacent teeth with a working field brushes.

From right to left the upper and lower jaw segments isolated 6:

- right molars (C1);
- right premolars (C2);
- right canines and incisors (C3);
- the left canines and incisors (C4);
- the left premolars (Cs);
- the left molars (Ce).

When performing the standard method of consistently clean the vestibular surfaces of the teeth of each segment, followed by oral and

chewing surfaces. Use several kinds of movement: vestibular and oral surfaces are cleaned to sweep the vertical, horizontal and circular movements, chewing surfaces - horizontal and circular movements of the brush.

A. Cleaning the vestibular surfaces begins with the first segment

1. The vertical movement. Brush put in the cervical area of the teeth of the upper jaw at an angle of 45° to the vertical axis of the tooth. Perform sweeping movements from the gum to the chewing surface. Perform 10 such movements, as well clean the vestibular surface of the 1st segment of the mandible.

2. Horizontal movement. Brush put perpendicular to the vestibular surface of the teeth of the 1st segment of the upper jaw and doing the horizontal reciprocating motion. Perform 10 such movements as clean vestibular surface of the lower jaw.

3. The circular movements. The bristles are perpendicular to the vestibular surface of the teeth of the upper jaw. Brush describes circles on the vestibular surface with) the upper and lower jaws. To prevent damage to the gingival margin, force is applied when moving from chewing gum to the surface, and the arc of the counter - brush passes without pressure on the teeth and gums.

After completing the vertical, horizontal and circular movements on the vestibular surfaces of the teeth of both jaws C1, rearrange the brush on the upper teeth and the C2 sequence is repeated the entire complex, and so on - up to and including C6.

B. Cleaning the oral surfaces. Just as in the vestibular surface cleaning are performed vertical, horizontal and circular movements on the palatal and lingual surfaces of C1-C6. To provide access to the brush head surface to be cleaned, the handle of the brush often takes a position close to the vertical.

C. Clean chewing surfaces

1. Horizontal movement. Brush put on the chewing surface C1 of the upper jaw, perform reciprocating motion in a horizontal plane; After the brush 10 movement is transferred to the surface of chewing C1 mandible.

2. The circular movement is made from the same initial position on the chewing surfaces of the upper C1, and then - the lower jaw.

By alternating horizontal and circular motion, clean the chewing surfaces of the teeth in the C2-C6.

Question 9. Professional oral hygiene

For the first time a professional cleaning, as a component of programme prevention of dental caries and periodontal inflammatory disease was proposed Axelsson and Lindhe in 1970 in the so-called " The model of Karlstad " (Sweden). In this comprehensive program for the prevention of dental caries were the following components:

- advice on nutrition

- topical application of fluoride
- regular training for cleaning teeth
- professional cleaning

Features removal of dental plaque. Scaler there are several ways:

1. Mechanical.
2. Chemical.
3. Physical (ultrasonic and pneumatic).
4. Combination.

1. *The mechanical method* - removal of tartar with hand tools. Scaler can be used the following basic tools: shovel, sickle hook curette, files, rasps.

2. *The chemical method* generally used for pre-soften tartar before mechanical removal, especially on movable teeth. The active principle in the formulations are acid, mostly hydrochloric acid. After the pre-insulation to the surface gingival tartar is applied to the drug 20 to 30 seconds, then rinse with water, after which the stone is removed by other means, such as a manual.

Methods of removing dental plaque (mechanical).

1. Begin removing deposits from the distal surface of the lower tooth 8th left move medially, removing deposits from all surfaces of the tooth.

2. Remove dental plaque begins with the cervical area of the tooth, gradually moving to the apex of the root.

For this sum under a rock the appropriate tool and sliding movements separate it from the hard tissues of the tooth up or sideways.

3. Then remove deposits from the distal surface 8th tooth right in the same sequence.

4. Move to the anterior teeth, starting with the lingual surface, removing deposits from all tooth surfaces.

5. The teeth of the upper jaw begin to clean the surface with distal eighth tooth from the right, moving in the direction medial removing deposits from all teeth surfaces. Turning to remove deposits left and complete cleaning of the front teeth.

6. When removing the tooth layering tool at "writing pen" and the jaw and teeth from which dental accretions removed, is fixed by left hand.

7. Minor dental plaque is removed in one session. Abundant dental plaque is removed in two stages: first supragingival dental calculus on the lower and upper jaw, then subgingival dental plaque.

Physical method involves the use of special sound or ultrasonic devices (magnetostrictive and piezoelectric). Sound tips are placed on the panel of dental units. The top of the nozzle is vibrated at a frequency of 2300- 6300 cps. Necessarily the presence of water cooling.

The benefits of physical methods:

- Reduces the time needed for the procedure;
- maximal cleaning of periodontal pockets;

- -antimikrobical action;
- -comfort for the patient.

Combined method of removing dental plaque. This method is used in the presence of abundant dental deposits. Usually begin removing dental plaque mechanically, ultrasonic method is the final stage.

Test tasks

1. Ninfected dental plaque includes:

- a. *pellicle*,
- b. dental plaque,
- c. tartar,
- d. *cuticle*,
- e. plaque.

2. According to the International Classification of Diseases to dental plaque include:

- a. *supragingival tartar*
- b. subgingival plaque,
- c. food residues,
- d. *pigment deposits*,
- e. subgingival calculus.

3. Dental plaque begins to form after his complete removal later:

- a. *2 hours*
- b. 8hours
- c. 20-30 minutes
- d. 12hours
- e. 15 minutes.

4. The plaque consists of the following microorganisms:

- a. Str. Mutans,
- b. Str. Salivarius,
- c. Lactobacilli,
- d. Actinomycetes
- e. *All the answers are correct.*

5. Dental plaque can be removed by:

- a. Individual oral hygiene,
- b. *Professional oral hygiene*,
- c. Can not be deleted,
- d. There is no need to remove it,
- e. It is possible to remove only the chemical method.

6. Methods of detection of dental plaque:

- a. *Visual*,
- b. *Drying*,
- c. *Proubing*,
- d. Percussion,
- e. Palpation.

7. Methods for the removal of tartar:

- a. *Chemical*,
- b. *Mechanical*,
- c. *Physical*,
- d. All answers are wrong,
- e. Combined.

LESSON 5. CLINICAL MANIFESTATIONS OF GINGIVITIS

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

1. Anatomical and histological structure of the gums. Signs and stage of inflammation.
2. Risk factors in the occurrence of gingivitis.
3. Relationship between oral hygiene and inflammation in periodontal tissue.
4. Clinical manifestations of gingivitis.
5. The definition of GI index and PMA.
6. Determination of the periodontal CPI INDEX.

Question 1. Anatomical and histological structure of the gums. Signs and stage of inflammation.

Gum is the part of the oral mucosa surrounding the teeth. Outside gums verge on the oral mucosa covering the alveolar bone of the jaw. This boundary has the form of a wavy line and is well-traced because of different colors. Gum covering the alveolar bone, has a bright red color, because it is lined by non-keratinizing epithelium through which blood is good shine suck-hole lamina propria. Gums covered stratum epithelium, has a pale shade. Inside the gum goes into the gum edge zone of the hard palate, or floor of the mouth. Gums are divided into three parts: the attached free gingival and interdental papillae.

Sticky part of the gums tightly adherent to the periosteum of the alveolar processes of the jaws. The free part of the gums - the edge - free adjacent to the tooth surface and is separated from it only by a narrow slit (sulcus). It has a strong attachment to periosteum and has some mobility. The dividing line between the free-fastening and gum is gum groove running parallel to the gingival edge of a distance 0.5-1.5 mm and level roughly corresponds to the bottom of the gingival sulcus. Gingival interdental

papillae are areas gums triangular filling gap between adjacent teeth. Gumin the sulcus has the highest permeability, is used for administration of drugs.

Gum is subjected to constant mechanical stress during chewing, and this explains the features of the structure of its epithelium and connective tissue.

Gum is lined with stratified squamous epithelium of the stratum thickness approximate, but 255 microns.

Lamina propria of the gingiva is presented papillary and reticular layers. The papillary layer is formed by loose connective tissue containing a large amount of blood from the vessels and nerve-fibers with numerous nerve endings. Reticulated layerpre-sented a layer of dense fibrous connective tissue rich in collagen fibers, thick bundles which are firmly attached to the periosteum of the gingiva (attached gingiva). In the lamina propria also gums woven bundles of collagen fibers are firmly bonded with cement gum tooth. Glands and submucosaare absence of gum.

The border between the attached gingiva and the free pass at the enamel-cement border and 40% of adults with vestibular surface appears in the form of gum sulcus. Free and attached gingiva is a certain size.

Free gingiva covers the cervical surface and has a smooth surface. The width of the free gingival is 0.8-2.5 mm. The width of the attached gingiva is 1-9 mm, and with age, it may increase. The surface of the attached gingiva bumpy, uneven.

Gum between adjacent teeth called interdental. On the labial and gingival sides of the top of the gingival papillae are located, and between them are saddle recess referred to as "saddle". There are three areas of gingival epithelium.

Sulcusis a groove between the edge of the gums and the tooth surface. The depth of the gingival sulcusis usually 0,1-0,5 mm, but in some patients it reaches 2-3 mm The bottom of the sulcus is located on the neck of the tooth level. The transition of tooth enamel in cement. Gingival fluid is a physiological environment that normally fills the gingival sulcus, jutting out into the gum to a depth of 1-3 mm. Depending on its depth varies the amount of gingival fluid. During the day in the mouth comes cavity 0.5-2 ml of gingival fluid. The maxillary its quantity more than in the lower.

The composition of gingival fluid are water, white blood cells, microorganisms, enzymes, proteins, exfoliated epithelium, immunoglobulins, however it has antimicrobial action. When clinically healthy gingiva, gingival fluid can be detected or not it appears in small quantities. With increasing intensity of gingivitis amount of fluid in the gingival sulcus is significantly increased.

Gingival fluid takes part in a mechanical furrow-washing and is nutritious environment for subgingival plaque microorganisms.

Inflammation is a pathological process that occurs when tissue damage and manifested by blood circulation, changes in the blood and connective tissue in the form of alteration, exudation and proliferation. Inflammation always starts with tissue damage (primary alteration that changes the tissue cells and affects the blood). Following the primary alteration occurs secondary alteration at which formed mediatory inflammation (histamine, serotonin and other kinins), causing changes in the lumen of the capillaries and permeability. Increased pro-permeability resulting in exudation (exit liquid part of blood - exudate - in inflamed tissue). Exudation is manifested by swelling of tissues. Change the lumen of the capillaries, reducible to a change of color fabrics (narrowing of the capillaries - pale tissue expansion - erythema - redness (arterial or venous)).

Inflammation always starts with cell damage and death, and it ends with recovery-damaged tissue by proliferation. Depending on the nature of the dominant local process (alteration, exudation, proliferation) are three types of inflammation:

- 1) alterative - dominated necrosis, degeneration and damage.
- 2) exudative - characterized by impaired blood flow to the phenomena of exudation and leukocyte migration. By the nature of fluid inflammation may be serous, purulent, hemorrhagic, catarrhal (if a lot of mucus exudate).
- 3) proliferative - dominated by cell proliferation or hematogenous histogens pro-origin in the inflammation and cell infiltrates occur.

Question 2. Risk factors in the occurrence of gingivitis.

Inflammation of the gums can be caused by a number of general and local factors.

1. Local factors:

1.1- microorganisms in the plaque is the main risk factor for sore-of gums. Studies show that 80 - 90% of periodontal disease caused by activity of microorganisms of dental plaque;

1.2- local exposure to chemicals (acids, alkalis, metal salts);

1.3- exposure to high and low temperatures, radiation energy;

1.4- infringement of functional load on periodontal (with malocclusion, edentulous, dysfunction of the temporomandibular joint, oral parafunctions and bad habits, carious tooth decay, excessive occlusion prosthesis or seal, and so on);

1.5- Pathology architectonic vestibule of mouth: improper attachment of the ultrasonic bridle, buccal bands, small vestibule of the oral cavity.

1.6- retention factors (orodonticheskie and orthopedic structures, overhanging edges of fillings and so on);

1.7- chronic autotravma (smoking, mechanical injury due to improper wire-my technique oral hygiene).

1.8- Violation amount and composition of the oral fluid, which is normally a natural protective factor in the oral cavity.

2. Common factors:

2.1- chronic somatic diseases (for example, diabetes, cardiovascular disease, and so on);

2.2- stress;

2.3- malnutrition;

2.4- taking certain medications;

2.5-hereditary factor (the individual characteristics of the protective potential of the human organism);

2.6- environmental factors (socio-economic, environmental conditions). Considered, for example, that insufficient cultural level of the person causes his dismissive attitude toward oral hygiene and thus creates an enabling environment for the development of periodontal disease.

2.7- occupational hazards.

Question 3. Relationship between oral hygiene and inflammation in periodontal tissue.

Most modern scientists believe that in 80% of cases of inflammatory processes occurring in the gums, leading role of bacterial plaque, which occurs gradually accumulates at unsustainable oral health. Plaque is a chemical, mechanical and biological irritant of periodontal tissues.

Ripened plaque causes tissue irritation by, microorganisms and their newly-methoxy, resulting in damage to the junctional epithelium, and inflammation of the gums. On-consisting of periodontal tissues affect microbial products - toxins. Exotoxins are derivated of Gram-positive bacteria. They are common in the oral cavity and not you-binding inflammation. Endotoxins arederivated of Gram-negative bacteria resistant to temperature, showing aggressive action at the site of plaque and stimulate the formation of antibodies that cause an increase in the permeability of the capillary-ditch violate cellular metabolism, leading to hemorrhagic necrosis. Gingivitis is a typical reaction of inflammation of connective tissue in response to the activities of microorganismsplaque. One of the possible mechanisms of inflammation of periodontal tissue can be schematically represented as follows:

1. Bacterial endotoxins + protease plaque and gingival fluid -> Zoom-set the number of gingival fluid.

2. The mast cell degranulation (the accumulation of serotonin, histamine, heparin) + change of enzyme activity of the kallikrein-kinin system and their inhibitors -> increase in the lumen of the capillaries, discoloration of the gums.

3. The disintegration of glycosaminoglycans - a change in vascular permeability -> swelling of the gums, bleeding.

4. Violation of the re-synthesis of collagen (collagenase activity change), changes in barrier function of periodontal tissues (local immunity) and total body resistance -> destruction of periodontal tissue.

Question 4. Clinical manifestations of gingivitis.

When the doctor make clinical examination for gums, he pay attention to the color of the gums. Normally, it is pale pink or coral pink, and the attached gingiva has a bright color, as non-keratinizing lined epithelium, which is well blood shines-bearing vessels. The color change indicates the presence of gum disease. Pale, anemic gums observed at keratoses and anemia. In acute inflammation of the gums the color becomes of bright red (hyperemia), and chronic inflammation of the gums becomes bluish color (congestive hyperemia). The systematic ingestion of brown-century salts of lead, bismuth and mercury along the gingival margin there is a dark gray or black border, due to the accumulation of metals in the subepithelial layer. Discoloration des us can be generalized and local, occur only in gingival papillae, or cover all the gums and depends on the increase in the lumen of the capillaries.

The surface of the gums is normally evenly spaced the top that give it the appearance of orange peel. If you have inflammation in the gum surface becomes smooth and shiny due to the development of edema.

The consistency of the gum is elastic. Palpation of the inflamed gums is looseness or pasty.

The contours of healthy gums presented pointed interdental papillae in frontal section, trapezoidal is in the posterior region. In chronic inflammatory processes, gums valicorethickens, which leads to a change in the shape of papillae. Sometimes gum marked erosion, ulceration.

Bleeding gums is the main feature of inflammation. It is defined by a light-sensing gingival sulcus using a special probe bellied. Signs of bleeding may be appear immediately after sensing or 30-40 seconds. The emergence of bleed-east due to the increased permeability of the walls of blood capillaries, cause inflammation of the gums.

In determining the location of the gingival margin in relation to the necks of the teeth we can meet with the phenomena of recession and hyperplasia.

Hyperplasia - an increase in the gums due to the quantitative growth of tissue cells. There are three degrees of hyperplasia:

1 degree - the increase in the gums to 1/3 the length of the crown;

2 degree - the increase in gingival 1/2 of the length of the crown;

Grade 3 - gums increase by more than half the length of the crown.

Gingival recession is a progressive shift of the gingival margin in apical direction.

Question 5. The definition of GI index and PMA.

GI index .We study the gum near teeth 16, 21, 24, 36, 41, 44. Gum is examined on 4 sides. They are distal-vestibular, mesial, buccal, vestibular and oral. Taking into account the following parameters: 0 - normal gum, 1 - slight inflammation of the gum, bleeding on probing is absent; 2 - moderate inflammation of the gum, bleeding on probing and palpation are present; 3 - pronounced inflammation of the gum, spontaneous bleeding. Then the performances are summarized and divided by the product of the number of surveyed surfaces (4) and the number of the examined teeth (6). If interpretation of the results is 0.1 - 1 it is gingivitis mild severity; 1.1 - 2 it is moderate gingivitis; 2.1 - 3 it is heavy gingivitis.

PMA index (Schour, Massler, 1948) is designed to determine the intensity of the inflammation of the gums - gingivitis. Inflammation visually determined for each of the three parts of the gingiva: medial interdental papilla (P), the free marginal gingival (M) and attached gingiva (A). The absence of inflammation registered number 0, the presence of inflammation - figure 1. Examine the gums in the area of incisors, canines, and premolars. At the request of the doctor can examine the teeth. The individual index is defined by the formula: = PMA score investigated teeth.

Normally PMA index is 0. The larger the numerical value PMA, the greater the intensity of gingivitis. A number of modifications of the PMA index, so be sure to indicate the author's modifications, or can not be the correct interpretation of the index. In practice, the most often used to modify the index PMA Parma (1960). Calculations are made on the index formula $PMA = S / 3Z \times 100\%$, where S - the sum of all the ratings, Z - number of teeth with complete tooth. With 15 years and older are examined teeth available in the oral cavity.

Interpretation:

- ✓ up to 25% - mild gingivitis;
- ✓ 25-50% - moderate gingivitis;
- ✓ 50% - severe gingivitis.

Question 6. Determination of the periodontal CPI INDEX.

The assessment of the state of periodontal tissues is made with the help of index CPI (comprehensive periodontal index). We study the teeth 16, 11, 26, 31, 36, 46.

Each tooth following symptoms are recorded: 0 – all well, 1 – is determined by any number of plaque, 2 – bleeding, 3 – tartar, 4 – periodontal pocket, 5 – tooth mobility. If interpretation of the result is: 0.1 – 1 it is risk of disease; 1.1 – 2 it is easy disease severity; 2.1 – 3,5 it is average degree of severity of the disease; 3.6 - 5 it is severe degree of severity of the disease.

Test items

1. Specify the key to determine the teeth gingival index GI:

- a. 16,17,11,26,36,31,46,47;
- b. **16,21,24,36,41,44;**
- c. Consider all the teeth;
- d. 22, 23, 24, 13,16;
- e. 11, 45, 33, 44, 16, 27.

2. What shape is the top of the interdental papillae in the anterior teeth is normal?

- a. round;
- b. spherical;
- c. crater;
- d. **peaked;**
- e. truncated.

3. End of the clinical signs of inflammation of the gums:

- a. a color change, the presence of periodontal pockets, tooth mobility;
- b. **a change of color, texture, contour the gingival margin, surface bleed-ness;**
- c. changing the consistency, bleeding, increased number of gingival crevicular fluid;
- d. availability of gums, discoloration of the gums;
- e. changing the consistency of the gums, teeth tenderness to percussion.

4. Select a color gum in chronic inflammation:

- a. coral-pink;
- b. a bright red;
- c. **dark red with cyanosis;**
- d. pink;
- e. brown.

5. Assess presence of bleeding gums is necessary to:

- a. In the time of sensing;
- b. **Within 30-40 seconds after probing;**
- c. 1-2 seconds after sensing;
- d. 20 seconds after the probe;
- e. 10 seconds after the probe.

6. Histologically, the gum comprises:

- a. stratified epithelium, lamina propria, the submucosa;
- b. stratified epithelium, basement membrane, submucosa, minor salivary glands;

- c. *stratified epithelium, lamina propria*;
- d. the lamina propria of the mucosa, submucosa;
- e. minor salivary glands; submucosa.

LESSON 6. METHODS AND MEANS OF ANESTHESIA

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

1. Local anesthetics. Classification.
2. Mechanism of local anesthetics.
3. Composition of local anesthetics
4. Contraindications and limitations for use local anesthetics

Question 1. Local anesthetics. Classification.

Adequate anesthesia creates psychophysiological comfort and greatly reducing the emotional burden on both patient and physician. Anesthesia promotes better cooperation between doctor and patient, will largely determine the success and reducing the time of the forthcoming treatment. Painless treatment testifies to the high professionalism of doctors, increase its credibility in the eyes of the patient. Local anesthesia in dentistry becomes mainly away anesthesia.

Local anesthetics, acting on the sensitive nerve endings or conductors, reduce or completely eliminate the flow of impulses from the place of painful manipulations in prices, central nervous system, causing a temporary loss of sensitivity reversible anesthesia.

Development of local anesthesia is currently in two directions: on the one hand, the search and introduction into medical practice of new high-level tools for local anesthesia, on the other hand, development of new application technologies and improved methods of administration used tools. When interventions on the tissues of the maxillofacial area is preferred injection anesthesia. A great achievement for dental anesthesiology was the development of carpool technology based on the release of local anesthetics in the cartridge sealed. This ensures the purity and sterility of the drug, the exact dosage of anesthetic and vasoconstrictor, but also relieves doctors and medical sisters responsible for the quality and accuracy of drug dosages and shifts it to the vendors. Creation of a special carpool syringe device to swipe-aspiration of the sample made it possible to minimize the risk of intravascular injection of anesthetic solution. Widespread use of local anesthesia in dentistry may be for its relative safety, the relative ease and speed of implementation.

History of local anesthetics injection

In 1879 Russian scientist pathologist V.K. Anrep were first proved local anesthetics properties of cocaine. It proved that the subcutaneous

solution cocaine-crystalline temporary loss of sensitivity. But the big distribution, this method was not until the XX century, when A. Einhorn, in the laboratories of the company Hoechst AG in 1904-1905 received a procaine, who is known to us under the name of Novocain. Novocaine remained virtually the only anesthetic injection before the end of the Second World War, there was no alternative to it. In 1943, Nils Lofgren in Sweden synthesized lidocaine. Mepivacaine was synthesized by a group of scientists, natives of Sweden under the direction of A.F. Ekenstam in the US in 1956, the same A.F. Ekenstam a half year later has been synthesized bupivacaine. In 1969, in the laboratories of the company Hoechst AG was established H. Ruschingartine authorized for use as a local anesthetic in 1974 and introduced to the dental practice under the name of articaine in 1976.

Chemical structure, physicochemical properties and pharmacokinetics of a local anesthetic.

Most local anesthetics are weak bases. The molecules of these drugs has three structural components:

- ✓ lipophilic center to coordinate the passive diffusion of compounds through the membrane-nerve fiber;
- ✓ hydrophilic center which interacts with protein receptors on the integrated on the inner surface of the membrane of the nerve fibers;
- ✓ intermediate group - aliphatic chain connecting the hydrophilic and lipophilic moieties (Pryanishnikova N.T., 1970; Lee A.G., 1976; Butterworth J.F., Strichartz G.R., 1990; Glinert R.J., Zachary C.B., 1991 et al.).

According to the structure of the intermediate part of the local anesthetics are divided into two groups, esters and amides.

Type of connection determines the metabolism of the compound in the body and the duration of its validity.

The esters (procaine). Essential communications fragile, anesthetics that group hydrolyzable and form a blood esterases, have a small half-life and short-acting.

The amides (lidocaine, mepivacaine, articaine). Amides are metabolized by hepatic microsomal enzymes, are more durable, the best diffuse into the tissues and is currently the most widely used in dental anesthesia. When you select a local anesthetic should be aware that their chemical structure and physico-chemical properties. Directly related to the therapeutic activity and toxicity of drugs and their metabolites.

Question 2. Mechanism of local anesthetics.

Local anesthetics are weak bases. Hydrolysis of the salt of the local anesthetic should occur in the tissues at the injection site. Ceteris paribus anesthetic faster and stronger effect, the higher the concentration of

anesthetics base on the outer side of the membrane of the nerve fibers and the concentration depends on the pK of the drug and tissue pH.

The dissociation constant (pKa) of the basic local anesthetic is in the range from 7.5 to 9.0

Most local anesthetics has a dissociation constant (pK) 7.6-7.8, so the guide is well-hydrolysis in a weakly alkaline environment of intact tissues (pH 7.4). The lower the pKa of the local anesthetics, the closer it is to the pH, the more anesthetic-base on the outer side of the membranes and faster is its diffusion. Therefore, local anesthetics with a relatively low-constants of the dissociation (lidocaine, mepivacaine, articaine) are fast (2-5 minutes), and the drag with a higher pKa (bupivacaine, and procaine especially having pKa 8.9) are slower and have a latency period of 8 to 18 minutes. The rate of onset of effect is also influenced by the dose and concentration of local anesthetic in the tissues. Thus, articaine and lidocaine have identical pKaarticaine but used as a 4% solution, and lidocaine - 2% solution, so articain acts faster.

In inflammatory conditions, when tissue acidosis develops pH drops to 5-6, the hydrolysis of the salt of the local anesthetic difficult and local anesthetic activity decreases.

For penetration through the membrane of the nerve fiber, consisting essentially of lipids essential solubility in fats local anesthetics, or lipophilic, non-ionized form which is provided by a base-anesthetic. With the receptor interacts cationic form of anesthetic. So, for the manifestation of the effect of local anesthetic are important both forms of local anesthetic: uncharged base, soluble in lipids of the membrane of the nerve fiber, which delivers the anesthetic to a receptor, to the place of reaction and cationic form, which carries out direct interaction with local anesthetic receptor.

The anesthetic-base enters the cell by simple diffusion. This passive trans-port is the concentration gradient at a rate directly proportional to dissolve the drug bridge in fats. Other things being equal, the higher the solubility of the local Anestick in fat, the easier it gets through the fabric membrane. The highest rate of fat-solubility of bupivacaine from having both the most active and greatest to toxicness.

For the manifestation of local anesthetic action and systemic toxicity pain-large value it has the ability to bind to the protein-membrane receptors on the nerve dies, and blood plasma.

The high rate of plasma protein binding correspondence high-linking of the protein receptor that increases the activity and duration of drug action. Relations of local anesthetic plasma protein prevents entry of the drug into the tissue, reducing its systemic toxicity. The highest rate of binding to plasma proteins is articaine, somewhat lower - bupivacaine, surpassing other local anesthetics of activity. Articaine has high anesthetic activity by fixing the membrane protein receptors of nerve fibers and blood plasma, thus, poorly

soluble in fats has less toxicity than other amide local anesthetics. Fat solubility of bupivacaine is significantly higher, which causes it to be more pronounced activity and a higher toxicity than articaine. High toxicity of bupivacaine is caused that drugs based on it though, and are approved for use in Russia, but are not used.

Of great importance in the study of the pharmacokinetics of drugs attached to the period of half-life of drugs ($t_{1/2}$). It represents the time during which the content of the drug in blood plasma is reduced by 50%. This process is important not only local anesthetic excretion from the body, but also its biotransformation and deposit in tissues. The largest plasmatic clearance characteristic of articaine, which, combined with the lowest period half-life, low lipid solubility and a high percentage of protein binding determines the little toxicity (Table. 2). Lidocaine and mepivacaine, despite the longer than that articaine, the half-life and reduced clearance of plasmatic worse bind to proteins that causes their shorter action and greater systemic toxicity.

Question 3. Composition of local anesthetics

Vasoconstrictor. These substances are required to narrow the blood vessels. Anesthesia will be longer and more severe by the action.

Adrenaline is the strongest catecholamine vasoconstrictor. There are the following relative contraindications to the use of adrenaline as a local anesthetic:

- ✓ cardiovascular diseases (hypertension (EH), ischemic heart disease (IHD, heart failure)
- ✓ pregnancy
- ✓ concomitant medication corticosteroids, tricyclic antidepressants, MAO inhibitors, chlorpromazine (and other drugs with α -adrenoceptor blocking activity)

Absolute contraindications to the use of adrenaline:

- ✓ diabetes
- ✓ glaucoma (narrow-angle shape)
- ✓ thyrotoxicosis
- ✓ decompensated form of cardiovascular disease (stage III GB, paroxysmal tachycardia, tahiaritmin).

Norepinephrine is similar to adrenaline, but the effect is weaker.

Mezaton is a catecholamine, with the same adrenaline, and noradrenaline properties, but will recompense, exists only on α -adrenergic receptors (vasoconstriction). Vasoconstrictor effect of 5-10 times weaker than adrenaline. Contraindicated in hypertension and hyperthyroidism. Used at a dilution of 1: 2500 (0.3 - 0.5 mL of 1% solution of 10 ml of anesthetic solution).

Felypressin (oktapressin) not catecholamine, it has no effect on adrenergic receptors, so devoid of all of the above disadvantages. It is an analogue of the posterior pituitary hormone - vasopressin. Causes only vasoconstriction, so hemostatic effect is not pronounced, as a result of its little used. Contraindicated during pregnancy because it can cause a reduction of myo-meters, and it is characterized by antidiuretic effect, so patients with ischemic heart disease and heart failure should be given no more than one drug the cartridge containing felypressin. Please note that the use of all of the above-vazokonstriktorov contraindicated in children under 5 years.

Preservatives or stabilizers

As a preservative commonly used esters of parahydroxybenzoic acid (parabens), they possess antibacterial and antifungal "effect. These substances can be an allergen.

Stabilizers (disulfite sodium or potassium) used in conjunction with catecholamines-vasoconstrictors and protects them from oxidation. They can cause allergic reactions if you are sensitive to sulfites. An allergy to sulfites is most often found in patients with bronchial asthma (frequency - about 5%), so the treatment of these patients should be particularly careful.

Question 4. Contraindications and limitations for use local anesthetics

All contraindications and limitations to the use of local anesthetic are reduced to three bases positions (Special itesSeptodont, 1995; Petrikas A., 1997).

1. Allergic reactions to local anesthetics.

2. Lack of systems metabolism and excretion. Local anesthetics can be an eye-binding toxic effects when they overdose, and the failure of their systems, metabolism and excretion. Essential local anesthetics are inactivated directly in the bloodstream by means of enzyme-pseudocholinesterase. Metabolism amide local anesthetics in the liver. In small amounts (less than 10%) as the amide and ester local anesthetics excreted unchanged by the kidneys. Thus, a relative contraindication to the use-of amide local anesthetics are liver disease, essential - de deficit of plasma pseudocholinesterase and (for all local anesthetics) kidney disease. In these cases, you should use the local anesthetics in small doses, with the dishes-all necessary precautions.

3. The age limit. Note that for children minimum toxic dose of local anesthetic is significantly less than for adults.

Lidocaine. The maximum dose is 1.33 mg per 1 kg of body weight of the child, which corresponds to 1.3 ml of 2% lidocaine.

Mepivacaine. The maximum dose is 1.33 mg per 1 kg of body weight of the child.

Articaine. The maximum dose is 7 mg per 1 kg of body weight of the child. Contraindicated utilization of the articaine children up to 4 years!

Test tasks

1. What are the structural components are molecules of local anesthetics:

- a. *hydrophilic center*
- b. chain amino acids
- c. *lipophilic center*
- d. *the intermediate group*
- e. the hydrogen bond

2. What anesthetics chemical structure belong to esters:

- a. articaine
- b. bupivacaine
- c. *cocaine*
- d. lidocaine
- e. *novocaine*

3. What are the chemical structure anesthetics are amides:

- a. benzocaine
- b. dicain
- c. *trimecaine*
- d. *articaine*
- e. *benzocaine*

4. What is a local anesthetic has the lowest toxicity:

- a. articaine
- b. mepivacaine
- c. lidocaine
- d. bupivacaine
- e. *novocaine*

5. What is local anesthetic has the greatest toxicity:

- a. articaine
- b. mepivacaine
- c. lidocaine
- d. *bupivacaine*
- e. novocaine

6. The index of lipid solubility articaine:

- a. 3
- b. 110
- c. 42
- d. **40**
- e. 560

7. The indicator lipid solubility of bupivacaine is:

- a. 3
- b. 110
- c. 42
- d. 40
- e. **560**

8. Absolute contraindications to the use of adrenaline as a vasoconstrictor:

- a. *thyrotoxicosis*
- b. *diabetes*
- c. *glaucoma (narrow-angle shape)*
- d. *decompensated form of cardiovascular disease*
- e. Pregnancy

9. What anesthetics exert foetotoxic effects on the fetus:

- a. lidocaine
- b. *novocaine*
- c. ultracaine.
- d. *trimecaine*
- e. mepivacaine

LESSON 7.DENTAL CARIES.DEVELOPMENT. MECHANISM.EPIDEMIOLOGY. CLASSIFICATIONS.

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

1. Dental caries. Definition. Theories of caries appearance.
2. Etiology of dental caries. Microbiology of caries. The concept of dental plaque, role of biofilm.
3. Pathogenesis of dental caries.
4. Histopathology of dental caries.
5. Epidemiology of dental caries.
6. Caries classifications. Black's classification. International diseases classification. Anatomical classification.

Question 1.Dental caries.Definition.Theories of caries appearance.

Dental caries is an irreversible microbial disease of the calcified tissues of the teeth, characterized by demineralization of the inorganic portion and destruction of the organic substance of the tooth, which often leads to cavitations. The word caries is derived from the Latin word meaning 'rot' or 'decay'. It is a complex and dynamic process where a multitude of factors initiate and influence the progression of disease.

Caries is still a major oral health problem in most industrialized countries, affecting 40-90% of schoolchildren and about 90% of adults.

Caries is the most prevalent oral disease in several Asian, European and Latin American countries.

The etiology of dental caries is generally agreed to be a complex problem complicated by many indirect factors that obscure the direct cause or causes. There is no universally accepted opinion for the etiology of dental caries. Numerous references on dental caries, including early theories attempting to explain its etiology, have been found in recorded history of ancient people. However, many theories have evolved through years of investigation and observation; the acidogenic theory of Miller (Miller's chemico-parasitic theory), the proteolytic theory and the proteolysis chelation theory, are among those which have stood the test of time.

The early theories

The Legend of Worms. The earliest reference to tooth decay is probably from the ancient Sumerian text known as the «Legend of Worms» from about 5,000 BC. The idea that caries is caused by worms was possibly prevalent for a long time as evident from the writings of Homer who made a reference to worms as the cause of toothache.

Endogenous theories. Keeping with the humoral theory of Greek physicians, dental caries was thought to be produced by internal action of acids and corroding humors. Along with this, the early Greek physicians such as Hippocrates, Celsus, and Galen, proposed the vital theory of tooth decay, which postulated that tooth decay originated, like a bone gangrene, from within the tooth itself.

Chemical theory. Parry in 1820s observed that dental decay affected externally, not internally, as had been thought previously. It was proposed that an unidentified 'chymalagent' was responsible for caries. This was further supported by Robertson in 1835 who proposed that dental decay was caused by acid formed by fermentation of food particles around the teeth.

Parasitic theory. The first to relate microorganisms to caries on a causative basis as early as 1843 was Erdl who described filamentous organisms in the membrane removed from teeth. Shortly thereafter, Ficus in 1847, a German physician in Dresden, attributed dental caries to 'denticolae' the generic term he proposed for decay related microorganisms. Leber and Rottenstein, two German physicians, disseminated the idea that dental caries commenced as a chemical process but that living microorganisms continued the disintegration in both enamel and dentin. In addition to these observations, Clark (1871, 1879), Tomes (1873) and Magitot (1878) concurred that bacteria were essential to caries, although they suggested an exogenous source of the acids. In 1880, Underwood and Miller presented a septic theory with the hypothesis that acid capable of causing decalcification was produced by bacteria feeding on the organic fibrils of dentin. They

reported sections of decayed dentin having micrococci as well as oval and rod shaped forms.

Miller'schemico-parasitic theory or the acidogenic theory.The chemico-parasitic theory is a blend of the above mentioned two theories. Willoughby D Miller, an American who was working at the University of Berlin, is probably the best known of the early investigators on dental caries. He published extensively on the results of his studies, beginning in 1882, which culminated in the hypothesis, "Dental decay is a chemico-parasitic process consisting of two stages, the decalcification of enamel, which results in its total destruction and the decalcification of dentin as a preliminary stage, followed by dissolution of the softened residue. In case of enamel; however, the second stage is practically wanting, the decalcification of enamel signifying its total destruction". The acid, which affects this primary decalcification, is derived from the fermentation of starches and sugar lodged in the retaining centers of the teeth. Miller found that bread, meat and sugar incubated in vitro with saliva at body temperature, produced enough acid within 48 hours to decalcify sound dentin. Subsequently, he isolated numerous microorganisms from the oral cavity, many of which were acidogenic and some were proteolytic. Since a number of these bacterial forms were capable of forming lactic acid, Miller believed that caries was not caused by any single organism, but rather by a variety of microorganisms. He assigned an essential role to three factors in the caries process: the oral microorganisms in acid production and proteolysis; the carbohydrate substrate; and the acid which causes dissolution of tooth minerals. Miller's chemico-parasitic theory is the backbone of current knowledge and understanding of the etiology of dental caries. However, Miller's chemico-parasitic theory could not explain the predilection of specific sites on a tooth to dental caries and the initiation of smooth surfaces. Also, why some populations are caries-free and the phenomenon of arrested caries.

So, Miller's chemico-parasitic theory or acidogenic theory:

1. Caries is caused by acids produced by microorganisms of the mouth.
2. Dental decay is a chemico-parasitic process consisting of two stages:
 - Decalcification of enamel and dentin (preliminary stage)
 - Dissolution of the softened residue (subsequent stage). Acids resulting in primary decalcification are produced by the fermentation of starches and sugar from the retaining centers of teeth.

The concept of dental plaque adhering to teeth and serving to localize bacterial enzymatic activity was proposed later in 1897 by Williams. This theory has been accepted by majority of investigators in a form essentially unchanged since its inception. The bulk of scientific evidence does implicate carbohydrates, oral microorganisms and acids, and for this reason, these deserve further consideration.

Question 2. Etiology of dental caries. Microbiology of caries. The concept of dental plaque, role of biofilm.

In 1920s Clarke described a spherical bacterium that formed chains of cells, isolated from dental caries lesions. He named this organism *Streptococcus mutans* (different morphological forms which he believed were mutants). *S. mutans* produced lactic acid as a main by-product from glucose fermentation (homolactic). But there was a strong cohort of *Lactobacillus* microbiologists at the time, and Clarke's attempts to have *Streptococcus* named as a new genus were foiled.

By the late 1950s, Keyes and Fitzgerald began working on the nature of this transmissible factor. A *Streptococcus* was purified from carious lesions of hamsters (also from rats) that was strongly acidogenic (producing acid) and non-proteolytic.

It was not until 1968 that it was accepted that the *Streptococcus* isolated from hamsters was the same *S. mutans* as that described by Clarke in 1924.

Streptococcus mutans. Gram-positive cocci in chains. More accurately, a collection of closely related species known as mutans streptococci and comprising seven species and eight serotypes, a–h. *S. mutans* serotypes c, e and f and *S. sobrinus* serotypes d and g are most closely associated with human disease. *S. cricetus*, *S. ferus*, *S. rattus*, *S. macacae* and *S. downei* are more usually found in animals. Mutans streptococci possess adhesins for salivary receptors allowing attachment to saliva-coated smooth surfaces. In addition, these organisms produce extracellular polysaccharides from sucrose that facilitate retention on surfaces. Mutans streptococci are associated with all forms of caries.

Virulence factors of *S. mutans*. *S. mutans* possess several attributes that contribute to its success as a cariogenic organism: (1) ability to adhere to the tooth surface and develop plaque communities; (2) production of glucans and other polysaccharides from excess carbohydrate (often sucrose) in the diet, leading to plaque accumulation; (3) production of acids (principally lactic acid), that generate a low pH environment and enrich for aciduric organisms.

S. mutans is adapted to the biofilm lifestyle and there is coordinated production of bacteriocins along with an increase in competence in high density situations. *S. mutans* may thus acquire DNA from other organisms in close proximity either for nutrition or increasing genetic diversity or both.

S. mutans can metabolize a variety of sugars, resulting in the production of a number of weak acids, including lactic, formic and acetic acids. Lactic acid is the strongest of these acids, with an ionization constant (pKa) of 3.5. When the plaque pH drops below about 5.5 the balance between enamel demineralization and remineralization shifts toward solubility and the caries process is initiated.

S. mutans produces polymers of glucan and fructan from dietary sucrose through glucosyl- and fructosyl-transferases.

S. mutans participates in Initial attachment to tooth surfaces producing a major surface protein. A major surface protein is the AgI/II family protein called SpaP (or P1). This protein contributes to the fibrillar layer that is on the outside of *S. mutans* cells, made up of proteins, polysaccharides and teichoic acids.

S. mutans is highly aciduric, and resistance to the adverse effects of low pH.

Organisms such as Lactobacilli that produce and tolerate large amounts of lactic acid are not thought to be the initiators of smooth surface caries as they lack specialized colonization mechanisms. Actinomyces and non-mutans streptococci produce less acid and so may be more important in root caries as cementum (and dentin) is less mineralized and thus more easily dissolved, as compared to enamel.

Lactobacilli. Gram-positive rods lactobacilli are efficient producers of lactic acid and are tolerant to low pH values (two important caries associated traits). However, lactobacilli are poor colonizers of smooth surfaces and probably do not initiate caries at these sites. Most likely lactobacilli are secondary colonizers of established caries lesions, where their aciduric properties allow them to out compete other organisms.

Acid production will then exacerbate the lesion and facilitate extension into the dentin. If lactobacilli become embedded in pits and fissures they may be able to initiate caries at these sites. Different species and strains of lactobacilli exhibit differing cariogenic potential.

Actinomyces species. Gram-positive rods Actinomyces, especially *A. naeslundii*, are frequently isolated from root caries lesions and can cause root caries in experimental animals. However, the organisms are also commonly found on healthy root surfaces so the role of actinomyces in the disease process has been unclear. More recent molecular detection techniques are re-establishing the importance of Actinomyces species in both root and coronal caries.

Question 3. Pathogenesis of dental caries.

Demineralization of enamel. The role of critical pH.

Critical pH is the term given to the highest pH at which there is a net loss of minerals from tooth enamel. This is the pH at which saliva and plaque fluid cease to be saturated with calcium and phosphate, thereby permitting hydroxyapatite to dissolve. Critical pH is generally accepted to be 5.5, but it can be a little higher or lower depending on individual factors. During the demineralization process, acid diffuses between the rods and reaches deeper areas of the enamel and into dentin, where carbonated hydroxyapatite crystals are more susceptible to dissolution. The calcium and phosphate ions that are

lost from the tooth diffuse out into dental plaque fluid and saliva. If the acid attack is chronic and prolonged, progressively greater amounts of calcium and phosphate minerals diffuse out of the tooth, causing the crystalline structure of the tooth to shrink in size, while pores enlarge. Eventually, a carious lesion develops; its rate of development is a function of the degree of undersaturation of fluid in its environment and rates of diffusion of ions into and out of enamel.

Host and environmental factors in caries.

Dental caries is multifactorial disease in which there is an interaction among three principle factors

1. Susceptible host tissue
2. Cariogenic microflora
3. Suitable local substrate - fermentable carbohydrates

The initiation and progression of caries requires that host, diet and bacterial factors are all conducive to disease.

Host factors

1. Teeth

1) Composition and structure of enamel

- Composition of surface enamel due to dense mineralization and high F-content, surface enamel more resistant to caries than subsurface layers of enamel
- Type of crystalline lattice of enamel.

Teeth become less susceptible to caries over time. Such post-eruptive resistance is due in part to an increase in the concentration of fluoride in the surface layer of enamel. Fluoride ions substitute for hydroxyl ions in hydroxyapatite, forming fluorapatite which is less soluble in acid than hydroxyapatite.

2) Tooth morphology contributes to caries susceptibility on the basis of ease of bacteria colonization. Deep, narrow occlusal, buccal and lingual pits promotes to trap food, bacteria or debris.

3) Tooth position rotated, misaligned teeth, deformed dentitions are difficult for cleanse and tends to accumulation of food and debris

2. Saliva

1) Flow rate. The flow of saliva physically washes away weakly attached bacteria and acids, and delivers salivary buffers. Xerostomia (low salivary flow < 0.1 ml/min) leads to rampant caries. Impaired salivary flow is deleterious to oral health. For example, an inadequate salivary flow rate increases the incidence of dental caries, for at least three reasons. First, there is greater bacterial retention in the mouth and more dental plaque forms; second, the acids produced by bacteria such as mutans group streptococci are inefficiently neutralized; and third, the enamel surface does not efficiently remineralize.

Around 0.5 to 1.5 liters of saliva are secreted into the mouth each day.

Saliva is responsible for flushing the epithelial surfaces and for lubrication and protection of tissues and an adequate flow of saliva is essential for the maintenance of both hard and soft tissue integrity. Saliva is hypotonic, with an average pH of around 6.7. Saliva contains both organic compounds (2–3 g/l protein, notably the enzyme amylase) and inorganic compounds including the electrolytes bicarbonate, chloride, potassium and sodium.

Salivary flow rate and composition can be affected by a range of infectious diseases, clinical conditions, e.g. wearing of dentures, clinical treatments, e.g. radiation therapies for oral cancer, or pharmaceutical drugs.

Saliva also contains a number of anti-microbial compounds that can restrict plaque accumulation and kill bacteria and other microorganisms.

2) Buffering capacity. Saliva has two major buffering systems: bicarbonate-carbonic acid $\text{HCO}_3^-/\text{H}_2\text{CO}_3$ (is the most important) and phosphate. Buffering by saliva helps prevent bacterial acids, from reducing the pH to levels that dissolve apatite.

3) Protective role of saliva. There are between 1 million and 100 million bacteria present in 1 ml of saliva, depending upon oral hygiene, frequency of food consumption, and salivary flow rate. Saliva in the fluid phase acts principally to flush out bacteria from the mouth. Saliva contains agglutinins that aggregate bacteria, thus preventing adherence to surfaces, and the bacterial clumps are removed by swallowing or expectoration.

Anti-microbial components in saliva

(1) Lysozyme. Lysozyme is a basic protein found in most secretions, including saliva, where it is present in high concentrations. Salivary lysozyme originates from both the salivary gland secretions and from gingival crevicular fluid (GCF). Lysozyme digests the cell walls of Gram-positive bacteria by breaking the bond between N-acetylmuramic acid and N-acetylglucosamine in peptidoglycan. Lysozyme can also activate autolysins in bacterial cell walls. Not surprisingly, many successful oral colonizers are relatively resistant to killing by lysozyme. Lysozyme can also bind and aggregate bacteria and facilitate clearance by swallowing or expectoration. In addition, lysozyme contains small amphipathic sequences in the C-terminal region that are capable of killing bacteria.

(2) Salivary peroxidase. Peroxidase in saliva is derived from the salivary glands and PMNs, and catalyzes the oxidation of thiocyanate (SCN^-) to hypothiocyanite (OSCN^-) by hydrogen peroxide, which is produced by the aerobic metabolism of oral bacteria. At acid pH, OSCN^- becomes protonated (and uncharged) and readily passes through bacterial membranes. Hypothiocyanite oxidizes SH groups in bacterial enzymes and inhibits bacterial metabolism. Reduction of hydrogen peroxide to water by peroxidase also prevents oxidative damage to the host soft tissues.

(3) **Lactoferrin.** Lactoferrin is an iron binding glycoprotein produced from glandular acinar cells, epithelial cells and phagocytic cells. Lactoferrin inhibits bacterial growth by binding and sequestering Fe²⁺ ions, and in the apo (iron free) state can be toxic to bacteria and interfere with bacterial adhesion. A 25-residue N-terminal proteolytically derived peptide fragment termed lactoferricin also kills bacteria through depolarization of cytoplasmic membranes.

(4) **Histatins.** Histatins are cationic histidine rich proteins that kill *Candida albicans* and some bacteria.

(5) **Cystatins.** Cystatins are cysteine rich peptides that inhibit bacterial cysteine proteases.

(6) **Chromogranin A.** Chromogranin A is produced by the submandibular and sub-lingual glands, and is processed to release an N-terminal peptide, vasostatin-1, which is antibacterial and antifungal protein from epithelial cells.

(7) **Immunoglobulins** (IgA from saliva, IgG and IgM from serum via GCF).

4) **Supersaturation** of saliva with calcium, phosphate and fluoride allows remineralization of enamel. At physiological pH saliva is supersaturated with respect to calcium and phosphate, that helps prevent loss of calcium and phosphate from enamel mineral.

3. Systemic factors

- Heredity
- Pregnancy and lactation.

4. Dietary factors

In order to produce acid, cariogenic bacteria require a fermentable carbohydrate substrate, in particular sucrose. Studies have shown that in addition to total consumption, the frequency of intake and physical form of the sucrose containing food are important. The potential cariogenicity of food can be assessed by measuring the pH changes in plaque over time following ingestion. In order to produce acid, cariogenic bacteria require a fermentable carbohydrate substrate, in particular sucrose.

Sucrose is the most cariogenic sugar because:

- it can be processed into glucan and fructan
- it is efficiently fermented into lactic acid
- sucrose and other sugars are transported into SM cells by the high affinity and high capacity phosphoenolpyruvate (PEP) sugar: phosphotransferase (PTS) uptake system
- has the additional cariogenic property of providing a substrate for bacterial glucosyltransferases and fructosyltransferases

Role of carbohydrates. Reference has been made previously to the finding that members of isolated primitive societies who had a relatively low caries index manifested a remarkable increase in caries incidence after

exposure to refined diets. The presence of readily fermentable carbohydrates has been thought to be responsible for their loss of caries resistance. The early studies of Miller showed that when teeth were incubated in mixtures of saliva and bread or sugar, decalcification occurred. There was no effect on the teeth when meat or fat was used in place of the carbohydrate. Both cane sugar and cooked starches produced acid, but little acid was formed when raw starches were substituted. Volker and Pinkerton reported the production of similar quantities of acid from mixtures of either sucrose or starch incubated with saliva with no difference in acid production between raw and refined sugar cane. The etiology of dental caries involves interplay between oral bacteria, local carbohydrates and the tooth surface that may be shown as follows: Bacteria + sugars + teeth → organic acids → caries.

The cariogenic carbohydrates are dietary in origin, since uncontaminated human saliva contains only negligible amounts regardless of the blood sugar level. Salivary carbohydrates are bound to proteins and other compounds, and are not readily available for microbial degradation. The cariogenicity of a dietary carbohydrate varies with the frequency of ingestion, physical form, chemical composition, route of administration and presence of other food constituents. Sticky, solid carbohydrates, soft retentive foods those that are cleared slowly, monosaccharides and disaccharides are more caries-producing. Plaque organisms produce little acid from the sugar alcohols, sorbitol, and mannitol. Glucose or sucrose fed entirely by stomach tube or intravenously, does not contribute to decay as they are unavailable for microbial breakdown. Meals high in fat, protein or salt reduce the oral retentiveness of carbohydrates.

Question 4. Histopathology of dental caries.

Zones of enamel caries:

- The early (submicroscopic) lesion
- Phase of nonbacterial enamel crystal destruction
- Cavity formation
- Bacterial invasion of enamel.

Early submicroscopic lesion. In early lesion, earliest visibility changes appear as white opaque spot on the surface of tooth and adjacent to contact point other than this chalking white appearance the enamel is hard and smooth on appearance. This caries is known as incipient caries.

1st observable changes occur in this **translucent zone (1)**. It appears as by formation of submicroscopic spaces or by pores located at the prism boundaries and other junctional sites such as on the striae of Retzius. Here 1% demineralization occurs.

2. Dark zone. It lies adjacent to translucent zone. It is defined as positive zone because it is always present. This zone is formed by demineralization and here 2-4% demineralization occurs.

3. Body of the lesion. It lies between surface zone and body of the lesion. Here greatest amount of demineralization occur. Here 5% demineralization occurs near the periphery and 25 % demineralization occur on the center of the lesion.

4. Surface zone. When it is examined by polarized microscope, micro radiography shows relatively unaffected. The greater resistance of this layer may be due to greater amount of remineralization or may be due to greater concentration of surface enamel.

Once bacteria have penetrated the enamel they reach the amelodentinal junction and spread laterally to undermine the enamel. This has three major effects. First, the enamel loses the support of the dentine and is therefore greatly weakened. Second, it is attacked from beneath. Third, spread of bacteria along the amelodentinal junction allows them to attack the dentine over a wide area. Thus the primary lesion provides the bridgehead for the attack on enamel, but undermining of the enamel determines the area of a cavity. Clinically this is frequently evident when there is no more than a pinhole lesion in an occlusal pit, but cutting away the surrounding enamel shows it to be widely undermined. As undermining of the enamel continues, it starts to collapse under the stress of mastication and to fragment around the edge of the (clinically obvious) cavity. By this stage, bacteria damage to the dentine is extensive.

Question 5.Epidemiologyof dental caries.

Caries in Prehistoric Man. Dental caries is probably a disease of modern civilization. Anthropologic studies of von Lenhossek revealed that the dolichocephalic skulls of men from preneolithic periods (12,000 BC) did not exhibit dental caries, but brachycephalic skulls of the neolithic period (12,000–3000 BC) contained carious teeth. Apparently the carious lesions were found at or just below the contact areas and an increased frequency of caries at the cemento-enamel junction was noted.

Caries incidence in modern societies. By about the 17th century, there was a significant increase in the total caries experience and a smaller increase in the number of carious lesions involving the interproximal contact areas of teeth, characteristic of the pattern and occurrence of caries in modern population. Extensive studies on the incidence of dental caries from various geographic areas have illustrated the apparent influence of civilization on dental disease. Mellanby in 1934 reviewed the literature on caries in existing primitive races and noted that the incidence was invariably less than that in modern man suggesting isolated populations that have not acquired the dietary habits of modern, industrialized man retain a relative freedom from dental caries. Native population living in the North West territories of Canada, Alaska and Greenland who consumed native food, had a lower evidence of carious lesion (0.1%) compared to those living at trading posts

(13%). A comparable effect of diet upon caries was demonstrated by Mellanby in studies on natives of Southern Rhodesia. The determinants of the carious process are essentially local and limited to the oral cavity. Although there may be a certain degree of racial resistance to dental caries, dietary factor appears to be more significant, especially since caries incidence is increased by contact with 'civilized' food.

DMF AND def INDEX. The most commonly employed method to measure the extent of previous damage to permanent dentition is by a measure known as the DMF index. The designation DMF (T) is used to denote decayed, missing, and filled teeth; DMF(S) denotes decayed, missing and filled surfaces in permanent teeth and therefore takes into account the number of surfaces attacked on each tooth. A similar index def (t) or def (s) index is used for primary dentition. The DMF/def index can be used to quantify both caries prevalence and caries incidence in a given population. It is an arithmetic index of the cumulative caries attack in a population. A commonly used modified form of this test is the caries increment, which refers to the number of new carious lesions occurring in a specified time interval, either for an individual or averaged over a population. The assessment of the caries increment involves at least two examinations—one at the beginning and one at the end of the period in question. In children, primary teeth may be lost due to natural exfoliation and, for the purpose of the def index, it is essential that the examiner designates as missing only those teeth that are lost due to caries.

The oral health of children 12 years old is the object of several epidemiological studies conducted around the world. According to the World Health Organization (WHO, 1997), the importance given to this age group is due to the fact that it is this age that children leave primary school. Thus, in many countries, is the last age at which data can be easily obtained through a reliable sample of the school system. Moreover, it is possible that at this age all the permanent teeth except third molars, have already erupted. Thus, the age of 12 was determined as the age of global monitoring of caries for international comparisons and monitoring of disease trends. Even considering the large number of scientific evidence from several epidemiological studies in schoolchildren worldwide, the majority are regional studies. In addition, the information is too outdated for some countries, which does not make easy international comparison. The index that measures the number of permanent teeth decayed; missing and filled teeth (DMFT) is the common outcome for such studies.

The WHO Oral Health Program (Petersen, 2003) presented in its report on the global oral health conditions, a four-level scale for the classification of the DMFT index at 12 years-old. They are: very low (less than 1.2), low (1.2 to 2.6), moderate (2.7 to 4.4) and high (over 4.4).

Factors Affecting Caries Prevalence

Race. Some studies show remarkable differences in the caries experience between races. American blacks and whites, living in the same geographic areas under similar conditions, offer an excellent opportunity for comparison. Investigations indicate that the blacks have fewer carious lesions than the whites. Most studies concerning other races have been relatively unsatisfactory because of complicating factors such as differences in diet or exposure to fluoride, which tend to mask any differences due to racial background. Nevertheless, there is some evidence to indicate that blacks, Chinese, and East Indians have considerably less caries than American Whites. The English have a higher caries incidence than Italians, Russians, and Chinese.

Age. Carious lesions that result in cavitation are irreversible and therefore, cumulative with age. There is a strong correlation between age and DMF indices. Several studies have shown that by the age of 6 years, about 20% of children have experienced dental caries in their dentition and a DMFT of 0.5 can be expected. By the age of 12 years, 90% of children would have experienced a DMFT of approximately 5.5. The decayed, missing and filled surface (DMFS) accelerates at a greater rate than the DMFT beyond the age of eight years. By the age 12, an average DMFS of 7.5 is seen in most populations. In general, other reports of caries prevalence among children in various parts of the world show rates that seem to be comparable to those cited here. Another common element is that children from families in lower socioeconomic groups consistently have greater caries prevalence than their peers from families at a higher socioeconomic level.

Gender. Studies indicate that the total caries experience in permanent teeth is greater in females than in males of the same age. This is attributable largely to the fact that the teeth of girls erupt at an earlier age. This time difference is particularly significant during the formative years because teeth have been shown to be maximally susceptible to dental caries immediately after eruption since, the chemical structure of teeth in the immediate post eruptive stage is suboptimal in terms of caries resistance. As teeth are exposed to saliva and constituents in the diet, the outer layers of the tooth take up additional minerals from the oral environment in a process known as post eruptive maturation. This maturation process confers a greater resistance to dental caries on the tooth.

Familial. Siblings of individuals with high caries susceptibility are also generally caries active, whereas siblings of caries immune individuals generally exhibit low caries rates. Children of parents with a low caries experience also tend to have low caries; the converse is true for children whose parents have a high caries rate. Studies of the dental caries experience in monozygotic and dizygotic twins indicate that concordance for carious sites in monozygotic twins is much higher than in dizygotic twin pairs.

Question 6. Caries classifications. Black's classification.

Anatomical classification. IDC.

The different features of dental caries are differentiated.

1. Anatomical caries classification is according to morphological cite:

- ✓ Pit and fissure caries
- ✓ Smooth surface caries.
- ✓ Cemental or root caries

Pit and fissure caries develops in:

- ✓ Occlusal surface of molar and premolar.
- ✓ Buccal surface of molars.
- ✓ Lingual surface of maxillary incisors.

Deep pits and fissures are naturally more prone to caries due to their poor self cleansing properties and tendency to entrap food debris and bacteria. Also, enamel at the base of such pits and fissures is thin. Early caries appears brown or black discoloration which is rough in probe. Staining is due to pigmentations from tobacco and bacteria. Enamel bordering the pit appears bluish-white as it is undermined. Upon reaching the DEJ, there is lateral spread of caries and penetration of dentinal tubules. All this occurs without apparent fracturing away of overlying enamel. Thus there may be a large carious lesion inside with only a pinpoint opening seen on the tooth surface. Sometimes caries begins as open cavity, this type is slower.

Smooth surface caries develops in:

1) *Proximal surface.* Here, the caries is preceded by formation of dental plaque, unlike the pit and fissure caries. Presence of plaque ensures retention of carbohydrate and bacteria on tooth surfaces, leading to subsequent acid production and demineralization of enamel.

Smooth surface caries usually begins just below the contact point and appears in initial stages as a faint white opacity of enamel without loss of continuity of enamel surface. The early white spot becomes roughened due to superficial decalcification of enamel.

As caries reaches the DEJ, there is rapid lateral spread.

2) *Gingival third of buccal and lingual surface (cervical caries).* It always occurs as an open cavity unlike the smooth surface or pit and fissure caries. It occurs on all the teeth without predilection as it is directly related to lack of oral hygiene.

Root caries. Root caries is concerning for the elderly population who often have gingival recession exposing the root surfaces and they have also reduced salivary flow.

Carious lesions form more quickly on root surfaces than coronal caries because the cementum on the root surface is softer than enamel and dentin. Microorganisms invade cementum along Sharpey's fibers.

2. Classification according to rapidity of caries:

Caries is divided into:

1) *Acute dental caries*. It runs rapidly so it leads to early pulp exposure. It occurs in young adults because the dentinal tubules are large and so, there is little time for secondary dentin formation. The entrance of carious cavity is small. The small point of opening doesn't allow the buffering ions of saliva to neutralize acids formed within the cavity.

Rampant caries: It is the sudden, rapid destruction of teeth affecting even relatively caries free surfaces like proximal and cervical surfaces of mandibular teeth. 10 or more carious lesions over a one year period is characteristic of rampant caries.

Nursing bottle caries. It is a rampant caries affecting deciduous teeth in babies due to prolonged use of milk after eruption of deciduous teeth.

Radiation caries: It is a type of rampant caries occurring in patients receiving radiotherapy in head & neck region. Xerostomia is a major complication of radiotherapy of head and neck.

2) *Chronic dental caries*. This type of caries is slowly progressive. It occurs in adult. The entrance for caries is large. So, there is less food retention and greater saliva access. This allows time for sclerosis of dentin and deposition of secondary dentin.

3) *Arrested caries*. Caries which becomes static. This occurs in case of:

- Occlusal surface with large cavity, so there is lack of food retention which leads to hard polished surface (eburnation of dentin). Secondary dentin is formed.
- In proximal surface if adjacent tooth is extracted.
- Following topical application of fluoride.

3. Caries classification according to onset of occurrence:

Caries is divided into:

a) *Primary caries*. Caries that occurs for the first time.

b) *Secondary (recurrent) caries*. Secondary, or recurrent, caries starts to form in the small spaces or gaps between the tooth and the margins of a restoration.

4. Caries classification Based on tissue involved:

- ✓ Enamel caries
- ✓ Dentin caries
- ✓ Cemental caries

5. Classification of caries lesions. Black's classification:

Lesions of class I. Locations include:

- Occlusal surface of molars and premolars
- Lingual surface of anterior teeth
- Occlusal two thirds of buccal and lingual surfaces of molars and premolars, i.e. blind pits of teeth

Lesions of class II. Lesions occur on the proximal surfaces of the posterior teeth - molars and premolars.

Lesions of class III. Lesions occur on the proximal surfaces of anterior teeth – incisors and canines. Class III cavities do not involve an incisal angle.

Lesions of class IV. Lesions occur on the proximal surfaces of anterior teeth when the incisal angle is involved and requires restoration.

Lesions of class V. Lesions occur on smooth facial and lingual surfaces in gingival third of teeth. Lesions begin close to gingival may involve a cementum or dentinal surface as well as enamel.

Lesions of class VI. Involve the incisal edges of anterior teeth and the occlusal surfaces posterior teeth that have been worn away due to abrasion.

International classification ICD-10

K02 Dental caries

K02.0 Caries limited to enamel. White spot lesion [initial caries]

K02.1 Caries extending into dentine

K02.2 Caries of cementum

K02.3 Arrested caries

K02.4 Odontoclasia

K02.8 Other specified dental caries

K02.9 Dental caries, unspecified

Test tasks

1. What acid is *Streptococcus mutans* capable of metabolizing, and in the process, further promoting demineralization?

- a. *lactic acid*
- b. *acetic acid*
- c. *pyruvate acid*
- d. *formic acid*
- e. hydrochloric acid

2. Which factors affect the rate at which acid is produced in plaque?

- a. The microbial composition of the dental plaque.
- b. The density of plaque.
- c. The speed at which bacteria are able to metabolize the dietary carbohydrate.
- d. *All of the above.*

3. At what pH does tooth enamel begin to demineralize?

- a. 8.3
- b. 7.5
- c. **5.5**
- d. 3.2

4. What is the clinical appearance of the initial stage of a carious lesion?

- a. A large cavitation that extends into the dentin.

- b. *A chalky white and softened spot on the tooth surface.*
- c. Evidence of tooth erosion caused by acid attack.
- d. Completely demineralized tissue.

5. Which of the following is a remineralization-promoting characteristic of saliva?

- a. Saliva promotes acidity that promotes hydroxyapatite crystal growth.
- b. Saliva does not promote remineralization.
- c. *Saliva is supersaturated with calcium and phosphate ions.*
- d. None of the above.

6. How is the biological hydroxyapatite of tooth enamel different than pure hydroxyapatite?

- a. It readily incorporates trace minerals, such as fluoride and carbonate into its crystal lattice.
- b. It is stronger.
- c. It has the following formula: $\text{Ca}_{12}(\text{PO}_4)_8(\text{OH})_4$
- d. *All of the above.*

7. Which of the following is true about fluorapatite?

- a. It has crystal structure resulting from the replacement of hydroxyl ions (OH^-) in the hydroxyapatite structure with fluoride ions.
- b. Crystal structure of fluorapatite is a more compact structure than hydroxyapatite.
- c. It is stronger and more acid resistant than hydroxyapatite.
- d. *All of the above.*

8. A cavity which located on the proximal surface of molars and premolars is classified as:

- a. Black's Class I
- b. *Black's Class II*
- c. Black's Class III
- d. Black's Class IV
- e. Black's Class V

9. Which of the following is true about acute caries?

- a. It runs rapidly so it leads to early pulp exposure
- b. *The entrance of carious cavity is small*
- c. Lesions are dark or brown
- d. *Lesions are soft and are light colored*

10. What index can be used to quantify both caries prevalence and caries incidence in a given population?

- a. *DMFT*
- b. GI
- c. PI
- d. PMA.

LESSON 8.ENAMEL CARIES. CLINICAL APPEARANCE. DIAGNOSTICS.TREATMENT.

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

1. Composition of enamel caries.
2. Histopathology of enamel caries.
3. Clinical appearance of enamel caries.
4. Main and additional methods of diagnosis of enamel caries.
5. Treatment methods of enamel caries.

Question 1.Composition of enamel caries.

Enamel is the most mineralized tissue of the body, forming a very hard, thin, translucent layer of calcified tissue that covers the entire anatomic crown of the tooth. Enamel is so hard because it is composed primarily of inorganic materials: 95% of enamel is calcium and phosphate ions combined to make up strong hydroxyapatite crystals. Hydroxyapatite crystals contain calcium and phosphate ions in the following proportions: $\text{Ca}_{10}(\text{PO}_4)_6\text{OH}_2$. Hydroxyapatite readily incorporates trace minerals into its crystal lattice. These ions can be negatively charged, such as fluoride or carbonate, or positively charged, such as sodium, zinc, strontium, or potassium. The concentrations of these trace minerals change the solubility of enamel. For example, the presence of fluoride in the crystal structure strengthens the structure and decreases solubility, while carbonate incorporation increases solubility. It has been found that hydroxyapatite crystals have more fluoride and less carbonate than crystals in the interior, making the outer surface less soluble than deeper layers of enamel.

Approximately 1% to 2% of enamel is made up of organic materials, particularly enamel-specific proteins called enamelin, which have a high affinity for binding hydroxyapatite crystals. Water makes up the remainder of enamel, accounting for about 4% of its composition.

The inorganic, organic, and water components of enamel are highly organized: Millions of carbonated hydroxyapatite crystals are arranged in long, thin structures called rods that are 4 μm to 8 μm in diameter.^{2,4} Viewed in cross section, these rods appear as keyhole-shaped structures. It is estimated that the number of rods in a tooth ranges from 5 million in the lower lateral incisor to 12 million in the upper first molar. In general, rods extend at right angles from the dento-enamel junction (the junction between

enamel and the layer below it called dentin) to the tooth surface. Surrounding each rod is a rod sheath made up of a protein matrix of enamelines. The area in between these rods is referred to as interrod enamel, or interrod cement. While it has the same crystal composition, crystal orientation is different, distinguishing rods from interrod enamel.

Some spaces exist where crystals do not form between rods. Typically called pores, they contribute to enamel's permeability, which allows fluid movement and diffusion to occur, but they also cause variations in density and hardness in the tooth, which can create spots that are more prone to demineralization - the loss of calcium and phosphate ions - when the oral pH becomes too acidic.

So, Enamel is most highly mineralized biological hard tissue. Enamel consists of apatite crystallites which are oriented in structural layers known as enamel prisms. The enamel crystals are surrounded by water. Enamel comprises 98% mineral present in the form of hydroxyapatite. The hydroxyapatite in human enamel is not pure. The hydroxyapatite contains carbonate ions. The presence of carbonate ions makes the enamel structure much more soluble and less resistant to acid dissolution. The hydroxyapatite is often described as a calcium-deficient carbonate hydroxyapatite $\text{Ca}_{10-x}(\text{PO}_4)_6-x(\text{CO}_3)x(\text{OH})_2$. A crystal structure resulting from the replacement of hydroxyl ions (OH^-) in the hydroxyapatite structure with fluoride ions (F^-) - Fluorapatite $\text{Ca}_{10}(\text{PO}_4)_6\text{F}_2$. Fluorhydroxyapatite is stronger and more acid resistant than hydroxyapatite.

Question 2.Histopathology of enamel caries.

Caries process in enamel progresses through following stages.

A. Early submicroscopic lesion. There is prominence of Transverse striation of enamel and incremental striae of Retzius.

B. Nonbacterial enamel destruction. As caries advances, triangle or cone-shaped lesion appears the apex at A.D.J.

C. Cavity formation and Bacterial invasion of enamel.

Before complete disintegration of enamel (stage of white spot) several zones can be identified:

1. Stage 1. Translucent zone, characterized by presence of more pores, 1% of enamel surface (normal porosity is 0.1%) initial demineralization at the microscopical level. Formation of the translucent zone. No clinical or radiographical signs.

2. Stage 2. Dark zone, as a result of demineralization, presence of 2-4% pores. Further demineralization. Spread of the translucent zone. Initial redeposition of minerals within the defect. No clinical or radiographical signs.

3. Stage 3. Body lesion, Area of great demineralization and large pores, presence of 5-25% pores. Formation of the classical structure of the lesion

Increased porosity in the body of the lesion clinically on the dry surface layer remains intact.

4. *Stage 4.* The surface zone, relatively unaffected as it has greater resistance due to a greater mineralization and presence of fluoride. Progression of the lesion towards the enamel dentin junction

Question 3. Clinical appearance of enamel caries

ENAMEL CARIES (ICD-10)

K02.0 Caries limited to enamel:

- Enamel caries.
- Initial caries.

A non cavitated caries is the first clinically notable sign of the disease. Demineralization of hard dental tissues has reached the level when it can be seen with the naked eye but without a visible breakdown of dental enamel.

Under the microscope a variety of different irregularities can be observed:

- Destroyed perikymata pattern.
- Minor cracks and fractures.
- Microcavities.
- Scratches.

Histologically it appears as a wedge shaped defect with the base at enamel surface. The shape of the non cavitated lesion is determined by the distribution of the microbial deposits.

White spot lesion. The white spot lesion is the first visible evidence of a caries in the enamel, characterized by demineralized lesion underneath an intact surface. The lesion is caused by the accumulation of plaque and bacteria, developing among young adolescents with insufficient oral hygiene. The increased pore volume inside the demineralized lesion body leads to a different refraction index from the sound enamel. An inactive white spot lesion might act as arrested dental caries and impair only the esthetic appearance by displaying a milky white color from its interior opacity.

INITIAL CARIES

Typical location. Approximal surfaces involve an interdental facet area toward the gingival margin, possible extensions buccally and lingually.

Occlusal surfaces natural pits and fissures are the most vulnerable sites. The process starts in the deepest parts of fissures, depending on tooth specific anatomy. Smooth surfaces along the gingival margin

Patient complains. No pain. Aesthetical discomfort especially in anterior teeth

Clinical appearance

Active lesion:

- Whitish, Opaque, Chalky.
- On smooth surfaces, close to gingival margin.

- Covered by plaque.

Inactive lesion:

- Whitish, Yellowish, brownish.
- Glossy and shiny.
- On smooth surfaces with small distance from gingival margin.
- Clean from plaque.

Diagnosis:

1. Gentle probing:
 - Active lesion feels rough.
 - Inactive feels smooth.
2. Dye method.
3. Radiograph detection conical shape in enamel, sometimes involve dentin.

Differential diagnosis: enamel hypoplasia, dental fluorosis.

SUPERFICIAL CARIES

Patient complains

- Short pain as reaction to sweets or thermal agents.
- Aesthetical discomfort especially in anterior teeth.

Clinical appearance

Active lesion:

- Whitish, Opaque, Chalky.
- On smooth surfaces shallow defect close to gingival margin.
- Covered by plaque.
- In fissures local enamel defect.

Inactive lesion:

- Whitish, Yellowish, brownish.
- Glossy and shiny.
- On smooth surfaces with small distance from gingival margin.
- Clean from plaque.
- On occlusal surfaces localized enamel defect.

Diagnosis:

1. Gentle probing:
 - Active lesion feels rough.
 - Inactive feels smooth and hard.
2. Radiograph detection conical shape in enamel, EDJ and outer dentin involve too.
3. FOTI for approximal surfaces a shadow in the area of a carious demineralization.
4. Electrometrical test show normal tooth vitality.

Differential diagnosis: enamel hypoplasia, dental fluorosis, usura cervicalis, dental erosion.

Question 4. Main and additional methods of diagnostics of enamel caries

The main methods of diagnosis:

1. Questioning:

- Complaints.
- Patient history.
- Medical history.

2. Examination:

- Probing.
- Drying.

3. Visual assessment:

- Reveals enamel caries (spot, cavity).
- Determines lesion activity.

Caries Lesion Diagnosis

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.

Tools - dental mirror, dental probe. 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.

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 first visual indication of caries in enamel is generally a small white lesion on smooth surfaces or light to dark brown lesion in pits or fissures where demineralization has occurred under the dental plaque.

Careful visual inspection of well-dried tooth surfaces is useful in detecting discolored and demineralized areas of enamel and cementum. Incipient enamel caries lesions look whiter than the surrounding sound enamel because of the strong scattering of light within the lesion. Use of magnification can be especially helpful in assessing the integrity of the tooth surface.

The basis of visual inspection of caries is based upon the phenomenon of light scattering. Sound enamel is comprised of modified hydroxyapatite crystals that are densely packed, producing an almost transparent structure. The colour of teeth, for example, is strongly influenced by the underlying dentin shade. When enamel is disrupted, for example in the presence of demineralisation, the penetrating photons of light are scattered (i.e. they change direction, although do not lose energy) which results in an optical disruption. In normal, visible light, this appears as a ‘whiter’ area—the so called white spot. This appearance is enhanced if the lesion is dried; the water is removed from the porous lesion. Water has a similar refractive index to

enamel, but when it is removed, and replaced by air, which has amuch lower refractive index than enamel, the lesion is shown more clearly. This demonstrates the importance of ensuring the clinical caries examinations are undertaken on clean, dry teeth.

Table 1

Drying	
<i>Visual assessment</i>	<i>Histopathologic feature</i>
Enamel optical properties do not change after drying over 5 second	Lack of enamel demineralization
Enamel opacity or discoloration are not visible on the wet surface, but clearly visible after drying	Demineralization of the upper third of enamel
Enamel opacity or discoloration are clearly visible without drying	Entire demineralization of enamel and upper third of dentine

Lesion depth assessment.

Ekstrand et al (1997) presented a visual ranked scoring system for lesion depth assessment that is still commonly used. Using no probe, they examined tooth surfaces to devise the following diagnostic levels:

- no or slight change in enamel translucency after 5 seconds of air-drying
- opacity or discoloration that is hardly visible on wet surfaces, but visible after 5 seconds of air drying
- opacity or discoloration that is visible without air-drying
- localized enamel breakdown with opaque or discolored enamel and/or grayish discoloration from underlying dentin
- cavitation in opaque or discolored enamel exposing dentin.

Recording both cavitated and non-cavitated lesions.

Pitts and Fyffe (1988) devised the following diagnostic levels that are still used today, and devised this method with the help of a mouth mirror and probe:

- D1 (enamel lesion, no cavity).
- D2 (enamel lesions, cavity).
- D3 (dentin lesions, cavity).
- D4 (dentin lesions, cavity to the pulp).

Lesion activity assessment

This is a newer diagnostic method developed by Nyvad et al in (1999). Diagnostic method focuses on the surface characteristics of lesions, namely activity as reflected in the surface texture of the lesion, and surface integrity, as indicated by the presence or absence of a cavity or microcavity in the surface. The rationale behind the method is that the surface characteristics of enamel change in response to changes in the biofilm covering the tooth surface. The diagnostic categories are as follows: active, non-cavitated;

active, cavitated; inactive, non-cavitated; inactive, cavitated; filling; filling with active caries; filling with inactive caries.

- Active, non-cavitated enamel caries lesions have a whitish/yellowish opaque surface, with a chalky or neon-white appearance, and the surface feels rough when a probe is moved across it.
- Inactive, non-cavitated lesions, on the other hand, are shiny and can vary in color from white, brown, or black, and will feel smooth with gentle probing.
- Active, cavitated lesions feel soft or leathery, while inactive, cavitated lesions are shiny and feel hard with probing.
- In general, active, non-cavitated lesions have a higher risk of progressing to a cavity than inactive, non-cavitated lesions, which have a higher risk of becoming a cavity than healthy surfaces.

**Diagnostic Criteria for Assessing Coronal Caries Lesion Activity
according to International Caries Detection and Assessment System
(ICDAS) (it was developed in 2002)**

The ICDAS lesion evaluation criteria serve as the basis for determining the stages of the caries process and lesion activity for the purpose of caries management. This is valid and reliable caries reporting system.

Table 2

ICDAS II Criteria

Score	Criteria
0	No or slight change in enamel translucency after prolonged air drying (five seconds)
1	First visual change in enamel (seen only after prolonged air drying or restricted to within the confines of a pit or fissure)
2	Distinct visual changes in enamel
3	Localized enamel breakdown in opaque or discoloured enamel (without visual signs of dentinal involvement)
4	Underlying dark shadow from dentin
5	Distinct cavity with visible dentin
6	Extensive distinct cavity with visible dentin (involving more than half of the surface)

For the purpose of caries management, individual tooth surfaces are categorized and described, based on an evaluation of each surface affected. For pits and fissures, the evaluation criteria are as follows:

Additional methods of diagnosis

Fluorescence. Laser fluorescence device (e.g. DIAGNOdent; Kavo GmbH, Bibberach, Germany) has been reported to be invalid in detecting carious lesions in occlusal surfaces

- Quantitative light-induced fluorescence (QLF, Inspektor, Amsterdam, The Netherlands).
- Fiber-optic transillumination (FOTI).
- Digitally imaging fiber-optic transillumination (DIFOTI).
- Electrical conductivity.
- Electrical impedance (CarieScan PRO™, CarieScan Ltd, Dundee, Scotland).
- Photothermal radiometry (Canary System™, Quantum Dental Technologies, Toronto).
- Radiologic Clinical Examination.

Fibre optic transillumination takes advantage of these optical properties of enamel and enhances them by using a high intensity white light that is presented through a small aperture in the form of a dental handpiece. Light is shone through the tooth and the scattering effect can be seen as shadows in enamel and dentine, with the device's strength the ability to help discriminate between early enamel and early dentine lesions. A further benefit of FOTI is that it can be used for the detection of caries on all surfaces; and is particularly useful at proximal lesions.

Bitewing Radiography. The most commonly used radiographic method for detecting caries lesions is the bitewing technique. This technique is used for identifying carious lesions on the proximal surfaces that may be inaccessible to visual and tactile examination.

It is meant to find lesions that are hidden from a clinical visual examination, such as when a lesion is hidden by an adjacent tooth, as well to estimate depth of lesions. Another way in which bitewing radiography complements the visual–tactile examination is in the diagnosis of recurrent caries lesions.

To get the radiographic images, a central beam of X-rays is positioned to pass at right angles to the long axis of the tooth. If film is used, a beam-aiming device on the film holder guides the position, directing the beam at right angles to the film.

Bitewing Radiography Diagnosis is that it is not invasive, and does not damage tooth structure like an incorrectly used dental probe might. Radiographs can also be filed and reexamined at a later date to compare with a more recent image to detect whether a lesion is progressing or not.

Limitations of Bitewing Radiography Diagnosis

Besides concerns about low-dose radiation and variations in how images are interpreted by dentists, the main limitation is that the validity in diagnosing early lesions is rather low. Also, the bitewing radiograph cannot always distinguish between sound surfaces, those with initial caries activity and cavitated lesions, or non-carious demineralizations, so clinical inspection is still needed to determine what is happening to the tooth. Bitewing radiographs also tend to underestimate the depths of lesions, so a lesion that

appears confined to the inner enamel on an image is often actually in the dentin, and this can lead to insufficient or improper treatment.

However, digital radiography is replacing radiography based on film. It has been proven as accurate as traditional radiography for detecting caries, and it comes with additional advantages of using a lower radiation dose, being less time-consuming, and does not require wet chemicals in the processing of the image.

Digital radiography – which is increasingly replacing bitewing radiography and that is as accurate as film for the detection of caries lesions.

Digital image enhancement – which studies show can provide superior results to radiographs when enhanced correctly but takes a significant amount of technical skill.

Determination of the patient's caries risk based on past caries experience and current lifestyle factors is the logical first step in the diagnostic process. Properly exposed radiographs can detect proximal lesions that are only one third of the way through the enamel. Use of rectangular collimation and a film holding device can be beneficial by reducing the penumbra effect and providing a consistent exposure angle. Digital radiographic techniques may also improve the detection of early caries since the images acquired are digital and can be processed or analyzed to enhance diagnostic performance.

A key outcome of these efforts is the International Caries Classification and Management System (ICCMS™) a standardized method based on the best evidence currently available. This system, which is focused on improving long-term caries outcomes, combines history taking, clinical examination, risk assessment and personalized care planning at the individual patient level. A goal of the system is to develop a comprehensive care plan that incorporates:

- •Preventing caries initiation (primary prevention).
- •Preventive management of early caries (secondary prevention).
- •Tooth preserving operative plan (minimally invasive).
- •Review, monitoring and recall. This comprehensive care plan takes into account key risk factors for the individual patient, recommends inclusion of caries detection aids and lesion activity assessments and then lays out clear caries management strategies to obtain optimal results.

Early caries detection and caries risk assessment

The caries disease process is dynamic and multi-factorial in nature. Caries risk is defined as 'the probability of future caries disease development'. Disease development includes both primary disease (new carious lesions) and secondary disease (lesion progression or reactivated carious lesions). Risk assessment for such a dynamic disease is complex as it is only able to provide a snapshot at that particular moment and risk factors may change over time. Most importantly, for assessing lesion activity

accurately in one session, using a combination of indicators (visual appearance, location, tactile sensation and gingival health) might still provide the best way to determine lesion activity.

The risk should be documented in a patient's chart and be used to influence a treatment plan.' One of the tools that assist clinicians worldwide in motivating patients is the 'Cariogram', an interactive validated program for patient motivation. This informative program illustrates caries risk in an instructive but simple graphical way, including the interaction between the various caries related factors. The Cariogram demonstrates the 'chance to avoid new carious lesion development' in the near future and to what extent the various factors will affect this chance. The software is available in 13 languages and can be downloaded as 'shareware'.

Caries-risk assessment is usually described at the level of the individual patient. It provides information needed to determine the most appropriate management decision for an individual patient. Risk prediction in a group is also pursued to enhance healthcare efficacy by focusing on those individuals with the largest risk, thus aiming to prevent or reduce a disease burden in the near future. This provides the oral healthcare professional with both individualized and population-based strategies for improving oral health.

The caries risk prediction may guide the best use of available funds to support preventive caries management, While the dental profession needs to embrace a more primary preventive approach to caries management based on common risk factors, secondary prevention and management will continue to focus on patient-centered caries management, including both preventive and minimally invasive tissue-preserving operative interventions

Question5.Treatment methods of enamel caries.

Therapeutics to promote the remineralization process.Remineralisation of enamel and dentine carious lesions.

Fluoride.Mechanisms of action of fluoride in enamel.The presence of fluoride during the remineralisation/demineralisation cycle leads to its incorporation into the crystalline structure of the carbonated hydroxyapatite, which not only decreases crystal solubility, but also increases the precipitation rate of enamel mineral in the presence of calcium and phosphate due to the lower solubility of fluorapatite. The fluoride decreases enamel solubility in two ways: (1) the fluoride ion is more stable in the crystal lattice than the hydrogen ion and (2) it interacts with the calcium ions on the crystal surface, interacting closely and binding strongly.

The effect and penetration of fluoride into the biofilm on the tooth surface is dependent on the type of fluoridated product and the time of exposure. When a clinical biofilm was exposed to 1,000 ppm (0.22%) sodium fluoride solution, exposure of up to 120 seconds increased plaque surface

fluoride concentrations only, while 30-minute exposure allowed penetration of more than 1,000 ppm (0.22%) fluoride up to 900 μm into the plaque.

Fluoride therapy has been the main caries preventive strategy since the introduction of water fluoridation schemes several decades ago. Fluoride is the most available and commonly used remineralization agent. Fluoride salts can be commonly found in drinking water, toothpastes and mouth rinses which are available to majority of the population in the world. However, it has been pointed out that, even with such a high availability, an increase in caries-free population reached a plateau in 1990s, and there were still at least 60% of teenagers around that time, and most likely still today, had observable dental decay. But the action of fluoride is limited by the amount of calcium derived from saliva, without extrinsic bioavailable sources of calcium and phosphate. Increased concentrations of calcium would also increase the retention of fluoride in the plaque biofilm by increasing calcium-bridging. Therefore, for remineralisation to occur during increased caries risk, an increase in bioavailable calcium and phosphate is fundamental to improving the effectiveness of the agent. Increased calcium and phosphate can be stabilised by macromolecules inherent in the saliva and plaque.

Thus the anti-caries effect of traditional fluoride therapy is still limited.

Many clinical studies have point out that fluoride therapy alone is not enough to overcome high caries challenges. Its retention in oral cavity is also another challenge.

Remineralizing products based on calcium phosphate remineralization systems. The technology which involves casein phosphopeptide stabilized amorphous calcium phosphate or casein phosphopeptide-amorphous calcium phosphate complexes (CPP-ACP).

The casein phosphopeptides (CPP) are derived from casein by tryptic digestion. In 1987, Reynolds found that CPPs were incorporated into the intra-oral appliance plaque and were associated with a substantial increase in the plaque's content of calcium and phosphate. Casein phosphopeptides (CPP) have the ability to stabilize high concentrations of calcium and phosphate in metastable solution, CPPs have a marked ability to stabilize calcium phosphate ions in solution and to form a amorphous calcium phosphate (ACP) complex, referred to as CPP-ACP. CPP-ACP binds strongly to hydroxyapatite and can diffuse and retained in dental plaque, therefore is able to buffer acid and substantially raise the level of calcium phosphate in plaque or close proximity to the tooth surface, and thus inhibits enamel demineralization and enhances remineralization. Stable and highly soluble CPP-ACP has been trademarked as RecaldentTM and has now been commercialized in sugar-free gum and mints and in dental professional products (Tooth MousseTM).

Several randomized clinical trials (RCT) have shown that CPP-ACP added to sugar-free chewing gums, tooth paste or dental cream increased

enamel subsurface remineralization. These RCT results suggested both a short-term remineralization effect of CPP-ACP and a caries-preventing effect for long-term clinical CPP-ACP use. Besides ACP, CPP also stabilize calcium fluoride phosphate (ACFP) and forming CPPACFP. In this case, calcium and phosphate ions co-localize at the tooth surface with fluoride ion, therefore increases the degree of saturation with respect to fluorapatite and promoting remineralization of enamel with fluorapatite.

A dicalcium phosphate anhydrous (DCPA) nanocomposite was developed by Xu, et al. as a restoration material that can slowly release high levels of CaPO_4 requisite for remineralization.

The technology is an unstabilized amorphous calcium phosphate (ACP, Enamelon™).

The technology is a bioactive glass containing calcium sodium phosphosilicate (NovaMin™). Since all systems rely on calcium and phosphate compounds, their effect is mainly based on an enhancement of the natural capacity of saliva to remineralize mineral loss.

Techniques for Minimally Invasive Treatment

The minimally invasive treatment options for dental demineralization or early caries include the following:

- 1) Treatment with topical fluoride and/or other remineralizing agents with repeated applications;
- 2) Surgical removal of demineralized enamel and placement of resin bonded restorative material;
- 3) Use of microinvasive infiltration resin;

Caries infiltration has been proposed as an alternative for management of non-cavitated enamel carious lesions on approximal and buccal surfaces.

The resin infiltration technique prevents further progression of the lesion using a low-viscosity resin with a high penetration coefficient, filling the enamel intercrystalline spaces. This technique has been reported to remove the whitish opaque color thereby changing the color and translucency of the white lesion. As such, the infiltrant creates a diffusion barrier for hydrogen ions preventing lesion progression.

Manufacturer's directions for the use of the infiltrant Icon® (DMG) are for treatment of proximal carious lesions extending no deeper than the outer third of the dentin layer radiographically, with demineralized "white spots" on the facial surfaces of teeth without visible cavitation.

On the enamel surface, careful application of 15% hydrochloric acid gel (ICONetch; DMG, Hamburg, Germany) is performed for 120 seconds to remove the surface layer less than 30 to 40 μm . The acid gel was suctioned and washed away thoroughly. The lesions is desiccated using ethanol (ICON-Dry; DMG) for 30 seconds. After thorough dessication with ethanol, an infiltrant resin (ICONInfiltrant; DMG) is placed on the tooth surface for 3 minutes for inside penetration. Excessive resin is wiped away from the

surface and the proximal spaces. Light polymerizing is performed for 40 seconds. Applying the infiltrant resin was repeated to compensate for the shrinkage after polymerization. The tooth surface was polished with polishing discs.

Pits and fissure sealants. Pits and fissures of permanent molars are particularly prone to carious lesion development during and after tooth eruption. The morphology of pits and fissures has been reported to be one of the main caries risk factors, with molars being more frequently affected than premolars. Sealing aims to modify patent pits and fissures into smooth surfaces that are protected from bacterial colonization and exposure to fermentable substrate and can be cleaned easily. The strategy is effective not only as a preventive measure, but also in arresting non-cavitated enamel carious lesions in pits and fissures. The superiority of pit and fissure sealants over fluoride varnish application in the prevention of occlusal carious lesions has been reported.

Resin composites and glass-ionomer cements are the dental materials generally used to seal pits and fissures.

Chemicomechanical Removal of Caries. Method of removing caries is the chemicomechanical method as by using this procedure there are very less chances of having pain. The use of local anaesthesia while preparing the cavity has been reduced a lot by this procedure and the chemicals that are used are mainly available in two forms i.e. liquid form (caridex) and in gel form (carisolv).

Procedure of using Carisolv gel. In this procedure of chemicomechanical method of removing caries there is requirement to maintain the proper moisture control and there should be no chances of contamination. Rubber dam can be used as this can help a lot while working with chemicomechanical method of removing caries. The main steps that are to be followed by the specialist during the procedure are.

- First the tooth is properly diagnosed that where the carious lesion is present and this can be done with taking the x-ray of the tooth.
- Now apply the rubber dam on the tooth and then take small amount of gel and apply on the carious lesion with the help of any hand instrument.
- After 30 seconds take a sharp instrument with scraping end and remove the carious part.
- Now apply more gel and then again scrap out the carious part.
- Repeat the procedure until there left no carious part.

Treatment of white spot lesions. For esthetic improvement of non-cavitated white spot lesions with remineralized surface, treatment may consist of tooth bleaching, microabrasion, composite resin bonding, prosthetic restoration or some combination depending on the severity of the lesion and its etiology.

Some of the recent advances in dentistry regarding the cavity preparation method is air abrasion.



Picture 1. Air abrasion instrument

In air abrasion there is make the use of narrow and very powerful beam and it contains the aluminium oxide particles. The particles in this have the capability of being abrading the surface against which they got hit during the procedure. It produces no noise, no vibrations and no heat like the other cutting instruments. Also the procedure of cutting tooth material with the help of this is less painful and there occurs no shattering of enamel and other structure of the tooth. Air abrasion is most important to use in cavity preparation as it saves most of the tooth structure and prevent the excess cutting of it.

Treatment of Superficial caries. Surgical removal of demineralized enamel and placement of resin bonded restorative material;

Test tasks

1. Are the first visual indication of caries in enamel?

- a. Grey lesions
- b. Green lesions
- c. ***White lesions***
- d. Black lesions

2. What are the basic tools for detecting caries lesions of enamel by visual inspection?

- a. ***Bright light and a dental mirror***
- b. An x-ray
- c. Fiber-optic transillumination
- d. Electrical current

3. What is the primary purpose of caries diagnosis?

- a. To only detect cavitations.
- b. To help identify early signs of tooth demineralization in order to halt its progression.
- c. To only detect non-cavitated tooth lesions.
- d. To only prevent teeth from falling out.
- e. *All of the above.*

4. Which of the following is true about using a dental probe?

- a. Excessive probing can cause irreversible damage to the surface of a developing lesion.
- b. Gentle probing does not disrupt the surface integrity of non-cavitated lesions.
- c. The blunt side can be used to remove biofilm.
- d. *All of the above.*

5. Which visual–tactile set of diagnostic criteria is considered the best choice for performing caries lesion diagnosis?

- a. Recording cavitated lesions only.
- b. Recording cavitated and non-cavitated lesions only.
- c. Activity assessment.
- d. Assessing lesion depth.
- e. *All of the above.*

6. How do moisture levels on the tooth surface affect the visibility of a lesion?

- a. *White spot lesions become more opaque (visible) in dried dental tissue.*
- b. While spot lesions become less visible in dried dental tissue.
- c. White spot lesions look greenish in dried tissue.
- d. Moisture levels do not affect the visibility of a lesion.

7. Which of the following is a benefit of bitewing radiography?

- a. It is not invasive and does not damage tooth structure like an incorrectly used probe might.
- b. It allows accessibility to surfaces that may not be seen in the clinical visual–tactile examination.
- c. Radiographs can be filed and reexamined at a later date to detect whether a lesion is progressing or not.
- d. *All of the above.*

8. What is the main limitation of bitewing radiography?

- a. *The validity in diagnosing early lesions is rather low.*
- b. It does a bad job of detecting caries in children's teeth.

- c. It requires too much equipment.
- d. This technique is too expensive.

9. List remineralization agent:

- a. *fluoride*
- b. *calcium*
- c. phosphate
- d. resin composite

10. How do increased concentrations of calcium influence on retention of fluoride in the plaque biofilm?

- a. *increase*
- b. decrease
- c. do not influence.

**LESSON 9.CARIES OF DENTIN.CLINICAL APPEARANCE.
METHODS OF DIAGNOSTICS.**

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

1. Structure and composition of dentin.
2. Histopathology of dentin caries.
3. Clinical appearance of dentin caries.
4. Main and additional methods of diagnosis of dentin caries.
5. Differential diagnosis of dentin caries.

Question1.Structure and composition of dentin.

Dentin formation begins immediately before enamel formation. Odontoblasts generate an extracellular collagen matrix as they begin to move away from the adjacent ameloblasts. Mineralization of the collagen matrix, facilitated by modification of the collagen matrix by various noncollagenous proteins, gradually follows its secretion. The most recently formed layer of dentin is always on the pulpal surface. This unmineralized zone of dentin is immediately next to the cell bodies of odontoblasts and is called predentin. Dentin formation begins at areas subjacent to the cusp tip or incisal ridge and gradually spreads to the apex of the root. In contrast to enamel formation, dentin formation continues after tooth eruption and throughout the life of the pulp. The dentin forming the initial shape of the tooth is called primary dentin and is usually completed 3 years after tooth eruption (in the case of permanent teeth).

The dentinal tubules are small canals that extend through the entire width of dentin, from the pulp to the DEJ. Each tubule contains the cytoplasmic cell process (Tomes fiber) of an odontoblast and in is layer of

peritubular dentin, which is much mineralized than the surrounding intertubular dentin.

The surface area of dentin is much larger at the DEJ or dentinocemental junction than it is on the pulp cavity side. Because odontoblasts form dentin while progressing inward toward pulp, the tubules are forced closer together. The number of tubules increases from 15,000 to 20,000/mm² at the DEJ to 45,000 to 65,000/mm² at the pulp. The lumen of the tubules also varies from the DEJ to the pulp surface. In coronal dentin the average diameter of tubules at the DEJ is 0,5 to 0.9 μ m, but this increases to 2 to 3 μ m near the pulp.

The course of the dentinal tubules is a slight S-curve in the tooth crown, but the tubules are straighter in the incisal ridges, cusps, and root areas. The ends of the tubules perpendicular to the DEJ. Along the tubules walls are small lateral openings called canaliculi. As the odontoblastic process proceeds from the cell in the pulp to the DEJ, lateral secondary branches extend into the canaliculi and can communicate with the lateral extensions of adjacent odontoblastic processes. Near the DEJ, the tubules divide into several terminal branches, forming an intercommunicating and anastomosing network.

Dentin is significantly softer than enamel but harder than bone or cementum. The hardness of dentin averages one-fifth that of enamel, and its hardness near the DEJ is about three times greater than near the pulp. Dentin becomes harder with age, primarily as a result of increases in mineral content. Although dentin is a hard, mineralized tissue, it is flexible, with a modulus of elasticity of approximately 18 GPa. This flexibility helps support the more brittle, nonresilient enamel. Dentin is not as prone to fracture as is the enamel rod structure. The ultimate tensile strength of dentin is approximately 98 MPa, whereas the ultimate tensile strength of enamel is approximately 10 MPa. The compressive strength of dentin and enamel are approximately 297 and 384 MPa, respectively.

During tooth preparation, dentin usually is distinguished from enamel by color and opacity, reflectance, hardness, and sound. Dentin is normally yellow-white and slightly darker than enamel. In older patients, dentin is darker, and it can become brown or black when it has been exposed to oral fluids, old restorative materials, or slowly advancing caries. Dentin surfaces are more opaque and dull, being less reflective to light than similar enamel surfaces, which appear shiny. Dentin is softer than enamel and provides greater yield to the pressure of a sharp explorer tine, which tends to catch and hold in dentin.

Sensitivity is encountered whenever odontoblasts and their processes are stimulated during operative procedures, even though the pain receptor mechanism appears to be within the dentinal tubules near the pulp. Physical, thermal, chemical, bacterial, and traumatic stimuli are transmitted through the dentinal tubules, although the precise mechanism of the transmissive

elements of sensation has not been conclusively established. The most accepted theory of pain transmission is the hydrodynamic theory, which accounts for pain transmission through rapid movements of fluid within the dentinal tubules. Because many tubules contain mechanoreceptor nerve endings near the pulp, small fluid movements in the tubules arising from cutting, drying, pressure changes, osmotic shifts, or changes in temperature account for most pain transmission.

Dentinal tubules are filled with dentinal fluid, a transudate of plasma. When enamel or cementum is removed during tooth preparation, the external seal of dentin is lost, allowing tubular fluid to move toward the cut surface. Pulpal fluid has a slight positive pressure that forces fluid outward toward any breach in the external seal. Permeability studies of dentin indicate that tubules are functionally much smaller than would be indicated by their measured microscopic dimensions as result of numerous constrictions along their paths. Dentin permeability is not uniform throughout the tooth. Coronal dentin is much more permeable than root dentin. There also are differences within coronal dentin. Dentin permeability primarily depends on the remaining dentin thickness (i.e., length of the tubules) and the diameter of the tubules. Because the tubules are shorter, more numerous, and larger in diameter closer to the pulp, deep dentin is a less effective pulpal barrier compared with superficial dentin.

Question2.Histopathology of dentin caries.

Caries in enamel is clearly a dynamic process, and this tissue does not contain cells and therefore is incapable of reacting in a vital manner. As soon as the process has reached dentin there is an immediate vital response by the odontoblasts and their processes within the dentinal tubules which is assumed as defense reaction. After reaching enamel dentin junction (EDJ), caries spreads laterally along the junction of least resistance and therefore undermines sound enamel. The established occlusal dentinal lesion is conical in shape with its basis on the EDJ and its apex directed towards the pulp. Continuous irritation of odontoblasts processes by acids diffusing through the porous enamel or even by bacteria themselves at the stage of enamel destruction activates the pulp/dentin organ in such a way that a region of reactionary or reparative irregular dentin begins to form from the pulpal side.

Dentin contains much less mineral and possesses microscopic tubules that provide a pathway for the ingress of bacteria and egress of minerals. The DEJ has the least resistance to caries attack and allows rapid lateral spreading when caries has penetrated the enamel. Because of these characteristics, dentinal caries is V-shaped in cross-section with a wide base at the DEJ and the apex directed pulpally. Caries advances more rapidly in dentin than in enamel because dentin provides much less resistance to acid attack owing to

less mineralized content. Caries produces a variety of responses in dentin, including pain, sensitivity, demineralization, and remineralization.

Often, pain is not reported even when caries invades dentin except when deep lesions bring the bacterial infection close to the pulp. Episodes of short-duration pain may be felt occasionally during earlier stages of dentin caries. The pain is caused by stimulation of pulp tissue by the movement of fluid through the dentinal tubules that have been opened to the oral environment by cavitation. When bacterial invasion of the dentin is close to the pulp, toxins and possibly a few bacteria enter the pulp, resulting in inflammation of the pulpal tissues and, thus, pulpal pain.

The pulp-dentin complex reacts to caries attacks by attempting to initiate remineralization and blocking off the open tubules. These reactions result from odontoblastic activity and the physical process of demineralization and remineralization. Three levels of dentinal reaction to caries can be recognized: (1) reaction to a long-term, low-level acid demineralization associated with a slowly advancing lesion; (2) reaction to a moderate-intensity attack; and (3) reaction to severe, rapidly advancing caries characterized by very high acid levels.

Dentin can react defensively (by repair) to low-intensity and moderate-intensity caries attacks as long as the pulp remains vital and has an adequate blood circulation.

In slowly advancing caries, a vital pulp can repair demineralized dentin by remineralization of the intertubular dentin and by apposition of peritubular dentin. Early stages of caries mild caries attacks produce long-term, low-level acid demineralization of dentin. Direct exposure of the pulp tissue to microorganisms is not a prerequisite for an inflammatory response. Toxins and other metabolic products, especially hydrogen ion, can penetrate via the dentinal tubules to the pulp. Even when the lesion is limited to enamel, the pulp can be shown to respond with inflammatory cells. Dentin responds to the stimulus of its first caries demineralization episode by deposition of crystalline material in the lumen of the tubules and the intertubular dentin of affected dentin in front of the advancing infected dentin portion of the lesion. Hypermineralized areas may be seen on radiographs as zones of increased radiopacity (often S-shaped following the course of the tubules) ahead of the advancing, infected portion of the lesion. This repair occurs only if the tooth pulp is vital.

Dentin that has more mineral content than normal dentin is termed sclerotic dentin. Sclerotic dentin formation occurs ahead of the demineralization front of a slowly advancing lesion and may be seen under an old restoration. Sclerotic dentin is usually shiny and darker in color but feels hard to the explorer tip. By contrast, normal, freshly cut dentin lacks a shiny, reflective surface and allows some penetration from a sharp explorer tip. The apparent function of sclerotic dentin is to wall off a lesion by

blocking (sealing) the tubules. The permeability of sclerotic dentin is greatly reduced compared with normal dentin because of the decrease in the tubule lumen diameter.

Crystalline precipitates form in the lumen of the dentinal tubules in the advancing front of a demineralization zone (affected dentin). When these affected tubules become completely occluded by the mineral precipitate, they appear clear when a section of the tooth is evaluated. This portion of dentin has been termed affected zone of dentin and is the result of mineral loss in the intertubular dentin and precipitation of this mineral in the tubule lumen. Consequently, affected dentin is softer than normal dentin.

The second level of dentinal response is to moderate-intensity irritants. More intense caries activity results in bacterial invasion of dentin. Infected dentin contains a wide variety of pathogenic materials or irritants, including high acid levels, hydrolytic enzymes, bacteria, and bacterial cellular debris. These materials can cause the degeneration and death of odontoblasts and their tubular extensions below the lesion and a mild inflammation of the pulp. The pulp may be irritated sufficiently from high acid levels or bacterial enzyme production to cause the formation (from undifferentiated mesenchymal cells) of replacement odontoblasts (secondary odontoblasts). These cells produce reparative dentin (reactionary dentin) on the affected portion of the pulp chamber wall. This dentin is different from the normal dentinal apposition that occurs throughout the life of the tooth by primary (original) odontoblasts. The structure of reparative dentin varies from well-organized tubular dentin (less often) to very irregular atubular dentin (more often), depending on the severity of the stimulus. Reparative dentin is an effective barrier to diffusion of material through the tubules and is an important step in the repair of dentin. Severe stimuli also can result in the formation within the pulp chamber of unattached dentin, termed pulp stones, in addition to reparative dentin.

The success of dentinal reparative responses, either by remineralization of intertubular dentin and apposition of peritubular dentin or by reparative dentin, depends on the severity of the caries attack and the ability of the pulp to respond. The pulpal blood supply may be the most important limiting factor to the pulpal responses.

The third level of dentinal response is to severe irritation. Acute, rapidly advancing caries with high levels of acid production overpowers dentinal defenses and results in infection, abscess, and death of the pulp. Compared with other oral tissues, the pulp is poorly tolerant of inflammation. Small, localized infections in the pulp produce an inflammatory response involving capillary dilation, local edema, and stagnation of blood flow. Because the pulp is contained in a sealed chamber, and its blood is supplied through narrow root canals, any stagnation of blood flow can result in local anoxia and necrosis. The local necrosis leads to more inflammation, edema,

and stagnation of blood flow in the immediately adjacent pulp tissue, which becomes necrotic in a cascading process that rapidly spreads to involve the entire pulp.

Maintenance of pulp vitality depends on the adequacy of pulpal blood supply. Recently erupted teeth with large pulp chambers and short, wide canals with large apical foramina have a much more favorable prognosis for surviving pulpal inflammation than fully formed teeth with small pulp chambers and small apical foramina.

Zone 1: normal dentin. The deepest area is normal dentin, which has tubules with odontoblastic processes that are smooth, and no crystals are present in the lumens. The intertubular dentin has normal cross-banded collagen and normal dense apatite crystals. No bacteria are present in the tubules. Stimulation of 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: affected dentin. Also called inner carious dentin, affected dentin is a zone of demineralization of intertubular dentin and of initial formation of fine crystals in the tubule lumen at the advancing front. Damage to the odontoblastic process is evident. Affected dentin is softer than normal dentin and shows loss of mineral from intertubular dentin and many large crystals in the lumen of the dentinal tubules. Stimulation of affected dentin produces pain. Although organic acids attack the mineral and organic contents of dentin, the collagen cross-linking remains intact in this zone. The intact collagen can serve as a template for remineralization of intertubular dentin, and this region remains capable of self-repair, provided that the pulp remains vital. The affected dentin zone can also be subclassified in three sub-zones: (1) subtransparent dentin (2) transparent dentin (3) and turbid dentin.

Zone 3: infected dentin. Also called outer carious dentin, this is the outermost carious layer, the layer, that the clinician would encounter first when opening a lesion. The infected dentin is the zone of bacterial invasion and is marked by widening and distortion of the dentinal tubules, which are filled with bacteria. Little mineral is present, and the collagen in this zone is irreversibly denatured. The dentin in this zone does not self-repair. This zone cannot be remineralized, and its removal is essential to sound, successful restorative procedures and the prevention of spreading the infection.

In slowly advancing lesions, it is expedient to remove softened dentin until the readily identifiable zone of sclerotic dentin is reached. In rapidly advancing lesions little clinical evidence (as determined by texture or color change) exists to indicate the extent of infected dentin.

Question 3. Clinical appearance of dentin caries.

Typical location:

- Approximal surfaces a defect above the gingival margin, the ridge may be broken and the cavity extends to the occlusal surface.
- Occlusal surfaces a defect located in the pits and fissures and involving surrounding enamel.
- Smooth surfaces above the gingival margin.

Caries media

Patient complains:

Active lesion. Short pain as reaction to sweets, sometimes there is the pain on thermal agents.

Inactive lesion. There are not complains.

Clinical appearance

Active lesion. A cavity full of soft demineralized dentin. Light brown and dull.

Inactive lesion. Dark brown and shiny. On smooth surfaces with small distance from gingival margin.

Diagnosis. Gentle probing:

- Active lesion feels soft and sticking, sensitivity on probing.
- Inactive feels smooth and hard.

Caries profunda

- Short pain as reaction to sweets, thermal agents or mechanical pressure.
- High sensitivity on probing.
- Aesthetical discomfort especially in anterior teeth.

Clinical appearance

Active lesion. A gross cavity full of soft demineralized dentin. Light brown and dull. On smooth surfaces shallow defect close to gingival margin.

Inactive lesion. Dark brown and shiny. On smooth surfaces with small distance from gingival margin.

Diagnosis. Gentle probing:

- Active lesion feels soft and sticking.
- Inactive feels smooth and hard.

Radiograph detection conical shape in enamel, EDJ and dentin at varying depth is usually involved too.

FOTI for approximal surfaces – shadow in the area of a carious demineralization

Electrometrical test show normal tooth vitality.

Term «**secondary caries**» defines the process of caries development which occurs after treatment of the primary caries lesion. Clinically it can be presented with all mentioned clinical manifestations, starting from a non cavitated lesion and extending further in enamel or dentin.

The reasons for formation of secondary caries are:

- Marginal gap formation between the restoration and the tooth surface.
- Broken filling.
- Loss of a part of the filling.
- Initial caries development due to plaque accumulation around margins of the filling.

Diagnosis. Can be performed with the same methods as for other forms of caries but in this case the visual examination of dental surfaces should be supported with additional diagnostic tools (bite wing). The 100% seal of the restored surface cannot be guaranteed forever and as soon as the dentinal microbiota will obtain contact with oral environment, the destructive process in dentin towards the pulp will continue.

The marginal gap around the restorations cannot be detected with the naked eye at the initial stage but the products of microbial metabolism (acids) on the surface can penetrate the minor microporosities that appear in the restored tooth surface.

Hidden caries lesion in some areas which are relatively well protected from mechanical pressure, the layer of clinically intact enamel can be maintained for a long time, so hiding an ongoing demineralization in dentin.

In rare cases, if soft dentin is exposed to attrition the affected area gradually turned in to a smooth and polished surface, and the caries process may become arrested.

Typical location

- Approximal surfaces — a defect above the gingival margin, the ridge may be broken and the cavity extends to the occlusal surface.
- Occlusal surfaces — a defect located in the pits and fissures and involving surrounding enamel.
- Smooth surfaces above the gingival margin

Patient complaints

- Short pain as reaction to sweets, thermal agents or mechanical pressure.
- High sensitivity on probing.
- Aesthetical discomfort especially in anterior teeth.

Clinical appearance- a gross cavity full of soft demineralized dentin.

1. *Active lesion.* Light brown and dull. On smooth surfaces — shallow defect close to gingival margin.

2. *Inactive Lesion.* Dark brown and shiny. On smooth surfaces with small distance from gingival margin.

Question 4. Main and additional methods of diagnosis of dentin caries

Caries detection methods. Oral assessment and assignment of risk of developing dental caries. Radiologic and clinical examination. Emerging diagnostic techniques:

- Fluorescence
- Fiber-optic transillumination (FOTI)
- Digitally imaging fiber-optic transillumination (DIFOTI)
- Electrical conductivity.

Table 3

Methods of caries detection based on their underlying physical principles

<i>Physical principle</i>	<i>Application in caries detection</i>
X-rays	Digital subtraction radiography Digital image enhancement
Visible light	Fibre optic transillumination (FOTI) Quantitative light-induced fluorescence (QLF) Digital image fibre optic transillumination (DiFOTI)
Laser light	Laser fluorescence measurement (DiagnoDent)
Electrical current	Electrical conductance measurement (ECM) Electrical impedance measurement
Ultrasound	Ultrasonic caries detector

Clinical Examination for Dental Caries

Visual-tactile examination. Visual changes of the dental structure resulting from the demineralization process can be visually observed during caries development, 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 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 color.

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. Moreover, use of an explorer can cause irreversible damages to the iatrogenic and demineralized tooth structure.

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 characteristics of active coronal lesions (not all characteristics need to be present to decide activity status) are as follows:

Table 4

Characteristics of Active Coronal Lesions

<i>ICDAS code</i>	<i>Characteristics of Lesion</i>	
	<i>Signs of Active Lesion</i>	<i>Signs of Inactive Lesion</i>
Initial to Moderate Stage Caries (1-4)	Surface of enamel is whitish/yellowish; opaque with loss of luster; feels rough when the tip of the probe is moved gently across the surface. Lesion is in a plaque stagnation area, i.e. in the entrance of pits and fissures, or near the gingiva, and in approximal surfaces below the contact point. Lesion was covered by thick plaque prior to cleaning.	Surface of enamel is whitish, brownish or black. Enamel may be shiny and feels hard and smooth when the tip of the probe is moved gently across the surface. For smooth surfaces, the caries lesion is typically located at some distance from the gingival margin. Lesion was not covered by thick plaque prior to cleaning.
Extensive Stage Caries (S-6)	Dentine feels soft or leathery on probing.	Dentine is shiny and hard on probing.

For the purpose of caries management, individual tooth surfaces are categorized and described, based on an evaluation of each surface affected. For pits and fissures, the evaluation criteria are as follows:

Table 5

Pits and Fissures Evaluation Criteria

<i>Tooth Surface Description</i>	<i>Evaluation Criteria</i>	<i>ICDAS Code</i>
Sound surfaces	No visible caries when viewed clean and dry. Non-carious white or brown marks on tooth surfaces must be differentiated from early caries lesions.	0
Initial stage caries	Characterized by the first visual change in enamel (seen only after prolonged air drying or restricted to the confines of a pit or fissure). OR A distinct visual change in enamel (seen on a wet or dry surface).	1
		2

Moderate stage caries	<p>Characterized visually by either localized enamel breakdown (without visual signs of dentinal exposure).</p> <ul style="list-style-type: none"> - Enamel breakdown is often viewed best when the tooth is air dried. <p>OR</p> <p>An underlying dark shadow from dentin.</p> <ul style="list-style-type: none"> - Shadowing from dentinal caries is often best seen with the tooth surface wet. 	3
		4
Extensive stage caries	<p>Characterized by distinct cavitation exposing visible dentine.</p> <ul style="list-style-type: none"> - Lesions exhibiting cavitation Involving less than half the tooth surface - Lesions involving half of the tooth surface or more 	5
		6

For mesial and distal surfaces, the evaluation criteria are as follows:

Table 6

Mesial and Distal Surfaces Evaluation Criteria

<i>Tooth Surface Description</i>	<i>Evaluation Criteria</i>	<i>ICDAS Code</i>
Sound surfaces	No visible caries when viewed clean and dry. Non-carious white or brown marks on tooth surfaces must be differentiated from early caries lesions.	0
Initial stage caries	<p>Characterized by the first visual change in enamel (seen only after prolonged air drying).</p> <p>OR</p> <p>A distinct visual change in enamel (seen on a wet or dry surface).</p> <ul style="list-style-type: none"> - These lesions are usually seen directly from the lingual or buccal directions but may be viewed from the occlusal direction as a shadow confined to enamel. 	1
		2
Moderate stage caries	<p>Characterized visually by either localized enamel breakdown (without visual signs of dentinal exposure).</p> <ul style="list-style-type: none"> - Enamel breakdown is often viewed best when the tooth is air dried. <p>OR</p> <p>An underlying dark shadow from dentin.</p> <ul style="list-style-type: none"> - Shadowing from dentinal caries is often best seen with the tooth surface wet. 	3
		4
Extensive stage caries	<p>Characterized by distinct cavitation exposing visible dentine.</p> <ul style="list-style-type: none"> - Lesions exhibiting cavitation Involving less than half the tooth surface - Lesions involving half of the tooth surface or more 	5
		6

Table 7

Buccal-lingual Smooth Surfaces Evaluation Criteria

<i>Tooth Surface</i>	<i>Evaluation Criteria</i>	<i>ICDAS Code</i>
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<i>Description</i>		
Sound surfaces	No visible caries when viewed clean and dry. Developmental defects like enamel hypoplasias, fluorosis, tooth wear (attrition, abrasion and erosion), and extrinsic or intrinsic stains should be recorded as sound in the absence of other signs of caries lesions as described below.	0
Initial stage caries	Characterized by the first visual change in enamel (seen only after prolonged air drying). OR A distinct visual change in enamel (seen on a wet or dry surface). - Initial stage lesions on free smooth surfaces are located in close proximity (in touch or within 1 mm) to the gingival margin or adjacent to orthodontic or prosthetic attachments on a tooth surface	1
		2
Moderate stage caries	Characterized visually by either localized enamel breakdown (without visual signs of dentinal exposure). - Enamel breakdown is often viewed best when the tooth is air dried. OR An underlying dark shadow from dentin. - Shadowing from dentinal caries is often best seen with the tooth surface wet.	3
		4
Extensive stage caries	Characterized by distinct cavitation exposing visible dentine. - Lesions exhibiting cavitation involving less than half the tooth surface - Lesions involving half of the tooth surface or more	5
		6

Radiologic Clinical Examination for Dental Caries

The use of X-rays and radiographic films promoted a significant jump in the direction of dental therapy, since it provided substantial contribution in obtaining the diagnosis. In addition, radiographic techniques have been modified to acquire optimum X-ray quality and to increase diagnostic possibilities, as for detecting caries lesions.

Radiography is the most common caries lesion detection aid. It is fundamentally based on the fact that as the caries progress proceeds, the mineral content of enamel and dentin decreases, resulting in a decrease in the attenuation of the X-ray beam as it passes through the teeth. This feature is recorded on the image receptor as an increase in radiographic density. Clinically, the detection of carious lesions is based on a combination of visual-tactile and radiographic examination.

Bitewing radiography has been used for the detection and evaluation of caries lesions depth, which are invisible or poorly visible for inspection. Thus, radiography is mainly used for the detection of carious lesions in approximal surfaces, but is also recommended as a supplement for occlusal

caries detection. However, experiments have shown that, once an occlusal carious lesion is clearly visible on radiographs, histological examination shows that demineralization has extended to or beyond the middle third of the dentin.

Regarding the performance of bitewing radiography, studies have found that the X-rays show a high sensitivity (50-70%) to detect caries lesions in dentin of both approximal and occlusal surfaces, compared to clinical visual detection. However, the validity of detecting enamel lesions is limited on the approximal surfaces and low for the occlusal surfaces. This difference can be explained by the fact that radiography is a 2-dimensional image of a 3-dimensional anatomy of the tooth structure.

Several criteria are used to classify the extent of carious lesions on radiographs, such as:

- (0) absence of radiolucency
- (1) radiolucency in the outer half of the enamel
- (2) radiolucency on the inner half of the enamel, which can extend up to the dentin-enamel junction (DEJ)
- (3) radiolucency in the outer half of the dentin
- (4) radiolucency in the inner half of the dentin toward the pulp chamber.

Bite-wing (BW) films for proximal decay detection

Directions in Caries Diagnosis

Occlusal caries. Caries researchers recommend against using a sharp explorer when examining pits and fissures. Histological evidence shows the explorer can disrupt incipient caries and bacteria can be moved from groove to groove by a sharp explorer. Reliance on a sharp explorer has the potential to underutilize other detection methods and leads to a false positive diagnosis of occlusal caries.

Visualization of pit and groove areas for discoloration under the enamel is essential for pit and fissure diagnosis. Teeth must be clean, dry, and well illuminated to be properly evaluated. With the greater exposure to fluorides and more frequent placement of sealants deep caries can exist without a visible indication. Radiographs, in the past considered of limited use in the detection of occlusal caries, have been shown to be valuable in detecting deep dentin caries beneath pit and fissures and sealants.

Proximal caries. Proximal radiographs provide a good representation of caries in enamel and dentin. When exposed with consistent angulation radiographs can provide an indication of whether a lesion is progressing. Film holding devices are valuable in producing consistent radiographic exposures, and should be used whenever possible. Visualization of the tooth looking for clinical cavitation and marginal ridge discoloration is an important adjunct to radiographic examination. Transillumination of the tooth is a valuable adjunct when recent films are not available, but provides limited

additional diagnostic yield beyond that afforded by a radiograph. An evaluation of risk is a required part of the prescription of a radiographic recall interval.

The radiographic examination recommendations are presented based. The recommendations are meant only as a guide and may be customized by the dentist to satisfy the individual needs of the patient in a given situation. As an example, specific monitoring of an early carious lesion would be appropriate at other intervals.

As the patient's caries risk level increases, the need for timely clinical dental examination increases.

Using criteria based on the caries risk level helps to ensure that patients will not be exposed to unnecessary ionizing radiation and that the radiographs taken will have significant diagnostic value. Intervals for clinical examination are lengthened as the dentist has gathered information that indicates the caries risk is reduced. Monitoring of patient compliance with lifestyle changes and interventions

The detection of carious lesions has been primarily a visual process, based principally on clinical-tactile inspection and radiographic examination. Caries detection methods should be capable of detecting lesions at an early stage, when progression can be arrested or reserved, avoiding premature tooth treatment by restorations. However, none of the conventional methods fulfill this requirement and are highly subjective. The development of some alternative non-invasive detection methods, such as laser fluorescence devices (DIAGNOdent and DIAGNOdent pen), quantitative light-induced fluorescence (QLF), fluorescence camera (VistaProof), LED technology (Midwest Caries I.D.), fiber-optic transillumination (FOTI), digital imaging fiber-optic transillumination (DIFOTI) and electrical caries monitor (ECM), can offer objectives assessments, where traditional methods could be supplemented by quantitative measurements.

Question 5.Differential diagnosis of dentin caries

Differential diagnosis pulpitis symptomatica, pulpitis asymptomatica, periodontitis asymptomatica.

Table 8

	Caries profunda	Pulpitis symptomatic	Pulpitis asymptomatic	Periodontitis asymptomatic
State of the cavity	Deep, full of soft discolored dentin, no pulp exposure.	Deep, full of soft discolored dentin, may be pulp exposure.	Deep, full of soft discolored dentin, may be pulp exposure.	Deep, full of soft discolored dentin, may be pulp exposure.
Character of pain	No irritation. No pain.	Prolonged pain, Self starting pain. Irritation induce pain.	Prolonged pain after irritation.	No pain.
Reaction to thermal irritants (cold, heat)	Short pain, Pain disappear after removal of agent.	Prolonged pain to cold. Doesn't stop after removal of agent. Irradiates to nerve.	Prolonged throbbing (pain to cold or heat).	No reaction to thermal agents.
Electro test	Normal 10-20 μ a.	Lowered 20-40 μ a.	Lowered 20-40 μ a.	No reaction 100-200 μ a
X-ray examination	Focal radiolucency in dentin, Thin layer of dental bridge above pulp, No changes in periodontium.	Radiolucency in dentin extending to the pulp. No changes in periodontium.	Radiolucency in dentin extending to the pulp. Periodontal gap slightly widened.	Radiolucency involves the pulp. Pathological changes are present in periodontium.

Test tasks

1. What differentiates dentin from enamel?

- a. There are no significant differences.
- b. Enamel can repair and regenerate, while dentin cannot.
- c. *Unlike enamel, dentin is living tissue with the ability for constant growth and repair, thanks to cells called odontoblasts that create new dentin.*
- d. Dentin is harder than enamel.

2. What is the basic dentin element?

- a. *Dentinal tubule.*
- b. Odontoblast.
- c. Enamel rod.
- d. Lamella.

3. What is the average diameter of dentinal tubules at the DEJ?

- a. *0,5 to 0.9 μm .*
- b. 2 to 10 μm .
- c. 100 to 200 μm .
- d. 50 to 100 μm .

4. What is the average diameter of dentinal tubules near the pulp?

- a. *0,5 to 0.9 μm .*
- b. 2 to 10 μm .
- c. 100 to 200 μm .
- d. 2 to 3 μm .

5. Dentin includes the following parts:

- a. *Peritubular dentin.*
- b. *Intertubular dentin.*
- c. Dentinal tubules.
- d. Odontoblasts.

6. What does the dentinal tubule contain?

- a. *Cytoplasmic cell process of an odontoblast.*
- b. *Dentinal fluid.*
- c. Intertubular dentin.
- d. Pulp.

7. What is the most accepted theory of pain transmission in the hard tissues of teeth?

- a. **Hydrodynamic theory.**
- b. Neurological theory.

- c. Microbial theory.
- d. Receptor theory.

8. What are the patient complaints at caries profunda?

- a. *Short pain as reaction to sweets, thermal agents or mechanical pressure.*
- b. Extending pain.
- c. Pain when biting
- d. Lasting pain as reaction to thermal agents or mechanical pressure.

9. What are the patient complaints at caries media?

- a. *Short pain as reaction to sweets and others chemical agents.*
- b. Extending pain.
- c. Pain when biting.
- d. Lasting pain as reaction to thermal agents or mechanical pressure.

10. What are clinical appearances of acute caries profunda?

- a. A gross cavity full of soft demineralized dentin.
- b. High sensitivity on probing of cavity bottom.
- c. Walls of cavity are light brown and dull.
- d. Small enter of caries cavity.
- e. *All of the above.*

11. What are clinical appearances of chronic caries profunda?

- a. A gross cavity full of thick pigmented dentin.
- b. Weak sensitivity on probing of cavity bottom, feels smooth and hard.
- c. Walls of cavity are dark brown and shiny.
- d. Wide enter of caries cavity.
- e. *All of the above.*

12. What are patient complaints at hidden caries on proximal surface?

- a. Sticking of food.
- b. Inflammation of interdental gingival.
- c. Aesthetical discomfort.
- d. laceration of floss.
- e. *All of the above.*

13. Select additional methods of diagnosis of hidden caries:

- a. X-ray examination.
- b. Thermometry investigation.
- c. Electrical conductivity.
- d. Methods of transillumination.
- e. *All of the above.*

LESSONS 10.CARIES OF DENTIN.TREATMENT METHODS.HEALING LINERS.

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

1. Principles of tooth preparation.
2. Instrument and Equipment for Tooth Preparation.
3. Stages of carious cavity preparation.
4. Pulp protection.
5. Classification of healing liners.
6. Treatment of deep caries lesions.

Question 1. Principles of tooth preparation.

- To restore function.
- To prevent further spread of an active lesion which is not amenable to preventive measures.
- To preserve pulp vitality.
- To restore aesthetics.

Preparation design. With caries prevalence declining, emphasis has changed from extension for prevention, to minimizing removal of tooth tissue. Tooth preparation should be based on the morphology of the carious lesion and the requirements of the restorative material being used.

General principles of tooth preparation:

- 1) Gain access to caries.
- 2) Remove all caries at ADJ (to prevent spread laterally).
- 3) Cut away all significantly unsupported enamel.
- 4) Extend margins so that they are accessible for instrumentation and cleaning.
- 5) Shape preparation so that remaining tooth tissue and restorative material will be able to withstand functional forces.
- 6) Shape preparation so that restoration will be retained, i. e. undercut for amalgam, none required for resin composite or bonded amalgams.
- 7) Check preparation margins are appropriate for the restorative material. Small areas of unsupported enamel may be left if a resin composite restoration is being placed.
- 8) Remove remaining caries unless indirect pulp cap to be carried out.
- 9) Wash and dry preparation.
- 10) While care must be exercised not to overcut a preparation, do not skimp on access so that caries removal is compromised by poor visibility.
- 11) Mark centric stops with articulating paper prior to tooth preparation and try to preserve if possible, or place the preparation margins past the occlusal contact areas.
- 12) Avoid crossing marginal ridges.

- 13) In removing caries a tactile appreciation of the hardness of dentine is important, therefore use slow-speed instruments or excavators.
- 14) The base of the preparation should not be flattened as this runs the risk of pulp exposure.
- 15) Unless caries dictates, margins should be supragingival.
- 16) All internal line angles should be rounded to internal stresses. Removing caries with a large diameter round bur automatically produces the desired shape.
- 17) In a proximal box, the margin should extend below the contact point because this is where the caries is!

Approach to caries preparation. The old surgical approach to cavity design was adopted in the absence of adhesive techniques and on the basis of Black's principle of "extension for prevention," but this theory is no longer tenable.

The current availability of adhesive bioactive restorative materials makes it possible to maintain areas of tooth structure even though they appear to be undermined and weakened. Thus, the concept of geometric designs for cavities is no longer valid.

Question 2. Instrument and Equipment for Tooth Preparation.

Removal and shaping of tooth structure are essential aspects of restorative dentistry.

Rotary Power Cutting Equipment. Powered rotary cutting instruments, known as dental hand-pieces, are the most commonly used in contemporary dentistry. Two technologies are used today for dental handpieces. The air-driven handpiece was, for many years, the mainstay for cutting teeth in dentistry. The electric motor-driven handpiece is now becoming increasingly popular for use in all cutting applications in dentistry. The advantages of electric handpieces are that they are quieter than air-driven handpieces, they cut with high torque with very little stalling, they maintain high bur concentricity, and they offer high-precision cutting. Cutting with electric handpieces is smoother and more like milling, whereas cutting with the air-driven handpiece is more like chopping the tooth with the bur. Another advantage of electric handpieces is that they offer multiple attachment for the motor that can be used for different cutting applications such as denture adjustments and endodontic instrumentation. Some disadvantages of air-driven handpieces are that they create a loud, high-pitched noise. The torque and concentricity of the air turbines degrade in a relatively short period. More vibration and bur chatter are associated with air-driven handpieces.

Rotary speed Ranges for different cutting applications

The rotational speed of an instrument is measured in revolutions per minute (rpm). Three speed ranges are generally recognized: low or slow speeds (<12.000 rpm), medium or intermediate speeds (12.000-200.000 rpm),

and high or ultra-high speeds (>200.000 rpm). Most useful instruments are rotated at either low speed or high speed. Electric handpiece motors generate up to 200.000 rpm of rotation. This speed is significantly less than the 400.000 rpm generated by air-driven handpieces. However, the Electric handpiece motor has attachments with speed increase multipliers that can increase rotation in ratios of 5:1 or 4:1, which makes them effective in the same range as air-driven handpieces.

The crucial factor for some purposes is the surface speed of the instrument, that is, the velocity at which the edges of the cutting instrument pass across the surface being cut. This speed is proportional to the rotational speed and the diameter of the instrument, with large instruments having higher surface speeds at any given rate of rotation. Low-speed cutting is ineffective, is time-consuming, and requires a relatively heavy force application; this results in heat production at the operating site and produces vibrations of low frequency and amplitude.

Heat and vibration are the main sources of patient discomfort. At low speeds, burs have a tendency to roll out of the tooth preparation and mar the proximal margin or tooth surface. In addition, carbide burs do not last long because their brittle blades are easily broken at low speeds. Many of these disadvantages of low-speed operation do not apply when the objective is some procedure other than cutting tooth structure. The low-speed range is used for cleaning teeth, caries excavation, and finishing and polishing procedures. At low speeds, tactile sensation is better, and generally, overheating of cut surfaces is less likely. The availability of a low-speed option provides a valuable adjunct for many dental procedures.

At high speed, the surface speed needed for efficient cutting can be attained with smaller and more versatile cutting instruments. This speed is used for tooth preparation and removing old restorations. Other advantages are the following: (1) diamond and carbide cutting instruments remove tooth structure and with less pressure, vibration, and heat generation; (2) the number of rotary cutting instruments needed is reduced because smaller sizes are more universal in application; (3) the operator has better control and greater ease of operation; (4) instruments last longer; (5) patients are generally less apprehensive because annoying vibrations and operating are decreased; and (6) several teeth in the same arch can be treated at the same appointment (as they should be).

Rotary Cutting Instruments.Common Design. Characteristics

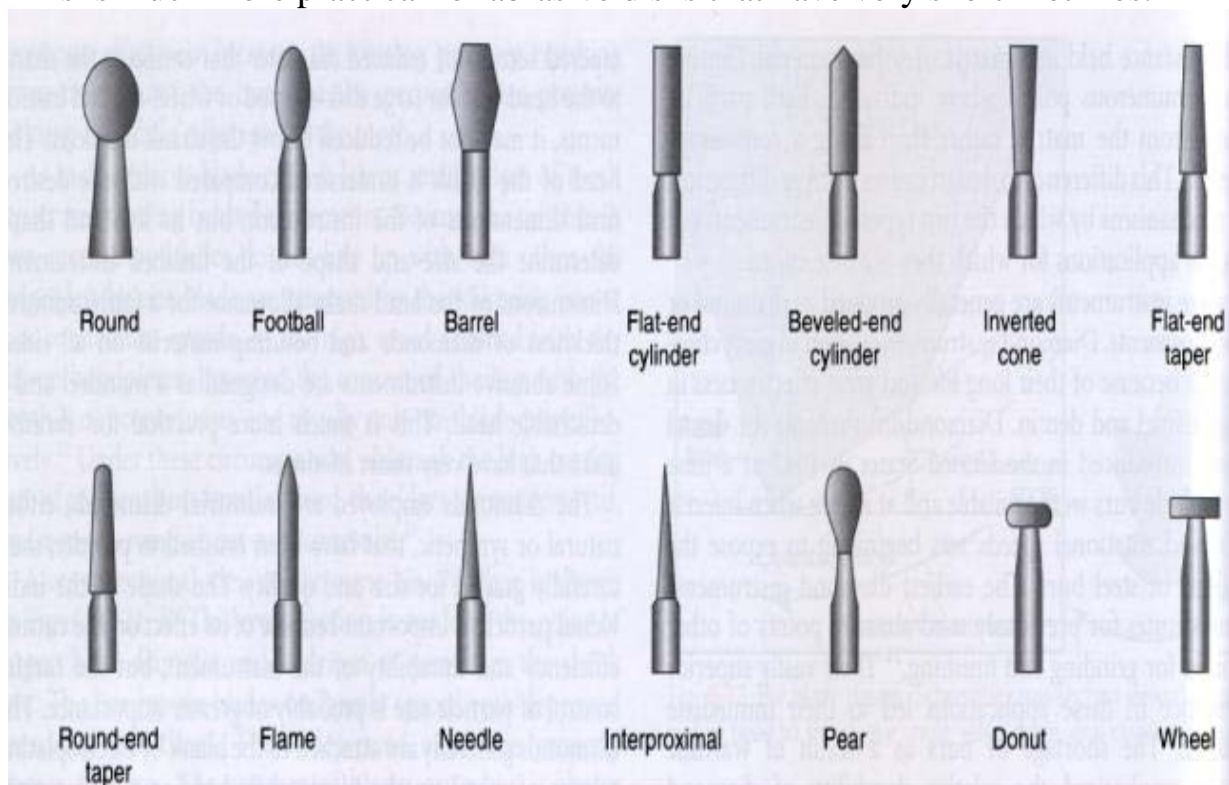
Despite the great variation among rotary cutting instrument they share certain design features. Each instrument consists of three parts: (1) shank, (2) neck, and (3) head. Each has its own function, influencing its design and the materials used for its construction. The term shank has different meanings as applied to rotary instruments and to instruments.



Picture 2. Normal designation of three parts of rotary cutting instruments.

Diamond instruments consist of three parts: a metal blank, the powdered diamond abrasive, and a metallic bonding material that holds the diamond powder onto the blank. The blank in many ways resembles a bur without blades. It has the same essential parts: head, neck, and shank.

The shank dimensions, similar to those for bur shanks, depend on the intended handpiece. The neck is normally a tapered section of reduced diameter that connects the shank to the head, but for large disk-shaped or wheel-shaped instruments, it may not be reduced below the shank diameter. The head of the blank is undersized compared with the desired final dimensions of the instrument, but its size and shape determine the size and shape of the finished instrument. Dimensions of the head make allowance for a fairly uniform thickness of diamonds and bonding material on all sides. Some abrasive instruments are designed as a mandrel and a detachable head. This is much more practical for abrasive disks that have very short lifetimes.

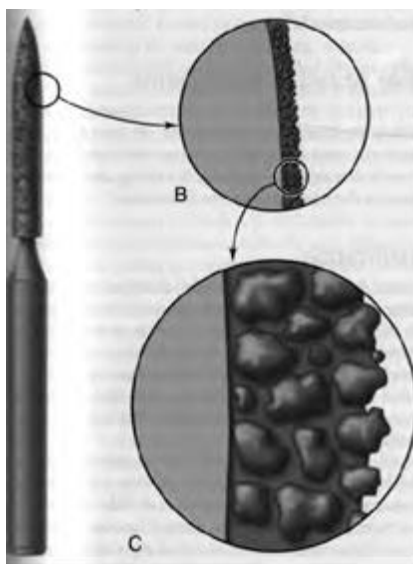


Picture 3. Characteristic shapes and designs for a range of diamond cutting instruments

Head Shapes and Sizes. Diamond instruments are available in a wide variety of shapes and in sizes that correspond to all except the smallest-diameter burs. The greatest difference lies in the diversity of other sizes and

shapes in which diamond instruments are produced. Even with many subdivisions, the size range within each group is large compared with that found among the burs. More than 200 shapes and sizes of diamonds are currently marketed. It is essential to indicate the manufacturer when attempting to describe diamond instruments by catalogue number.

Diamond Particle Factors. The clinical performance of diamond abrasive instruments depends on the size, spacing, uniformity, exposure, and bonding of the diamond particles. Increased pressure causes the particles to dig into the surface more deeply, leaving deeper scratches and removing more tooth structure. Diamond particle size is commonly categorized as coarse (125-150 μm), medium (88-125 μm), fine (60-74 μm), and very fine (38-44 μm) for diamond preparation instruments. These ranges correspond to standard sieve sizes for separating particle sizes.



Picture 4. Diamond particles.

When using large particle sizes, the number of abrasive particles that can be placed on a given area of the head is decreased. For any given force that the operator applies, the pressure on each particle tip is greater. The resulting pressure also is increased if diamond particles are more widely spaced so that fewer are in contact with the surface at any one time. The final clinical performance of diamond instruments is strongly affected by the technique used to take advantage of the design factors for each instrument.

Diamond finishing instruments use even finer diamonds (10-38 μm) to produce relatively smooth surfaces for final finishing with diamond polishing pastes. Surface finish less than 1 μm are considered clinically smooth and can be routinely attained by using a series of progressively finer polishing steps.

Question 3. Stages of carious cavity preparation.

1. Opening of cavity

2. Enlargement of cavity.
3. Necrectomy is a removal of the necrotic dental hard tissues from carious cavity.
4. Formation the cavity.
5. Smoothing the edges of enamel.

The use of modified cavity designs for the treatment of initial carious lesions can be justified on the grounds that, because no restorative material can adequately replace natural tooth structure for the long term, preservation of natural tooth structure is important. It is apparent that it is possible to remineralize and heal demineralized tooth structure to some degree. Therefore, neither enamel nor dentin should be removed simply because it has lost calcium and phosphate ions as a result of acid attack. The older surgical approach to cavity design was adopted in the absence of adhesive techniques and on the basis of Black's principle of "extension for prevention," but this theory is no longer tenable. The current availability of adhesive bioactive restorative materials makes it possible to maintain areas of tooth structure even though they appear to be undermined and weakened. Thus, the concept of geometric designs for prescribed cavities is no longer valid. The purpose of this article is to describe a series of simplified, modified cavity designs for small initial lesions; preservation of natural tooth structure is the principle objective of these designs.

Question 4. Pulp protection.

Pulpal Responses

<i>Types of stimulus</i>	<i>Examples of stimulus</i>
Physical	Thermal, electrical
Mechanical	Handpiece, traumatic occlusion
Chemical	Acid from dental materials
Biologic	Bacteria from saliva

- 1) Heat generated by rotary instruments,
- 2) Some ingredients of various materials
- 3) Thermal changes conducted through restorative materials
- 4) Forces transmitted through materials to the dentin
- 5) Galvanic shock
- 6) The ingress of different products and bacteria through microleakage.

It has been generally accepted that the materials that were used to restore teeth posed a danger to the tooth and allowed for the occurrence of postoperative sensitivity. If this were true, then a barrier or protective layer needed to be placed on the tooth before the final restoration. This buffer would, in part, act to reduce or even eliminate postoperative sensitivity.

Over time we have come to learn that it is not the restorative material that causes problems, but bacteria and the by-products of bacteria. These

bacteria, present in the oral cavity, enter the tooth at the margin of the restoration through capillary action of oral fluids. This is referred to as microleakage. Others have defined microleakage as ‘the marginal permeability of bacterial, chemical, and molecular invasion at the interface between the teeth and restorative material.

Liner and bases Intermediate supplementary restorative materials are materials that are placed between a restoration and the dentine with a primary function of protecting the pulp.

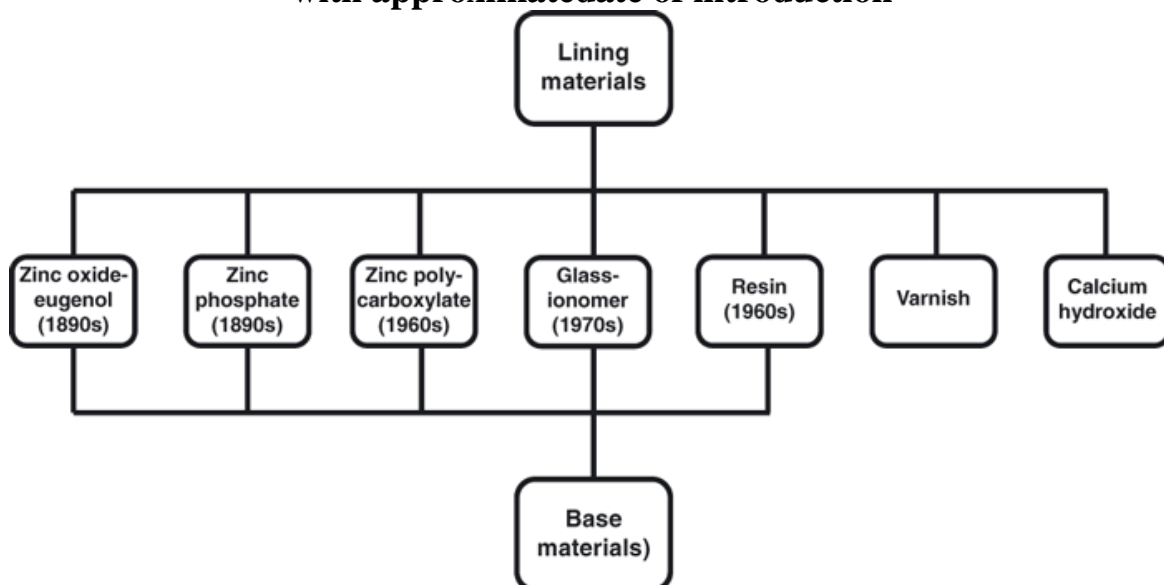
Liners include suspensions or dispersions of zinc oxide, calcium hydroxide, resin-modified glass ionomer. It can be applied to a tooth surface in a relatively thin film. Dental liners provide a thin barrier that protects the pulpal tissue from irritation caused by physical, mechanical, chemical and biologic element. Liners protects dentin from irritans agents from either the restorative materials or oral fluids. Bases are materials, most coments, that are used in thicker dimensions beneath restorations to provide for mechanical, chemical, thermal protection of the pulp.

Examples: phosphate, zinc oxide-eugenol, polycarboxylate cements and most common, some type of glass ionomer (usually an RMGI).

If the removal of infected dentin does not extend deeper than 1 to 2 mm from the initially prepared pulpal or axial wall, usually no liner is indicated.

If the excavation extends into or within 0.5 mm of the pulp, a calcium hydroxide liner usually is selected to stimulate reparative dentin (indirect pulp cap procedure).

Materials which can be used as liners and bases, with approximatedate of introduction



Many of these materials belong to the family of so-called water-based «cements».

Table 9

Liners and Bases application in order of use

<i>Type of restorative material</i>	<i>Shallow Preparation</i>	<i>Moderately Deep Restoration</i>	<i>Deep Restoration</i>
Amalgam	1.Dentin sealer 2.Bonding system	1.Base 2.Dentin sealer 3.Bonding system	1.Liner 2.Base 3.Dentin sealer 4.Bonding system
Composite resin	1.Bonding system	1. Bonding system	1.Liner 2.Bonding system
Gold inlays/onlays		1. Base	1.Liner 2.Base
Ceramic	1.Bonding system	1. Bonding system	1.Liner 2.Bonding system

Question 5. Classification of healing cavity liners.

Calcium hydroxide. Calcium hydroxide (CaOH) has two components: a base and a ‘catalyst’. The base is composed of calcium tungstate, tribasic calcium phosphate and zinc oxide. The catalyst is composed of calcium hydroxide, zinc oxide and zinc stearate. Radiopacity is provided by calcium tungstate, or in some cases by barium sulphate fillers. Calcium hydroxide should not be applied in a thickness greater than 0.5 mm, which would make it a liner.

Calcium hydroxide is considered to be bactericidal due to its high pH, approximately 12, which is provided by the catalyst. This alkaline property can cause cytotoxic effects to both the pulp and any bacteria in the preparation. Additionally, the acidic by-products of the bacteria are counteracted by the high pH. This high pH continues even after the material has set due, to hydroxyl ions that continue to leach out of the material when it comes in contact with the dentinal fluid. Calcium hydroxide can also irritate the pulp due to its high alkaline nature. This results in the formation of reparative dentine (a dentine bridge). This new dentine forms because CaOH can stimulate growth factors in the dentine matrix, and this process may occur more quickly when a resin-based calcium hydroxide formulation is used. Calcium as well as hydroxyl ions play an important role on the pulpal healing by modifying the environmental pH in the zone of inflammation to levels favourable for pulp matrix mineralization.

There are light-cured resin-based versions of calcium hydroxide and such formulations are not harmful to the pulp but do not show any antibacterial characteristics. They have a demand set and are less soluble than the self-cured products. The high solubility of conventional CaOH materials requires that clinicians ensure that restoration margins are sealed.

Properties of Calcium Hydroxide

- Low thermal conductivity.

- Stimulates the production of irregular secondary (tertiary) dentine pH of 11–12 (i.e. alkaline).
- Bactericidal properties.
- Highly soluble (not applicable to resin version).

Advantages

- Easily manipulated.
- Stimulates the formation of irregular secondary (tertiary) dentine.

Disadvantages

- Moisture sensitive.
- Low strength.

Indications for use of Calcium Hydroxide

- Protects the pulp from chemical irritation by its sealing ability.
- Stimulates the production of reparative or secondary dentin.
- Compatible with all types of restorative materials.
- Used in the deepest portion of cavity preparation.
- For use with direct or indirect pulp capping.
- Only used when within 1–2 mm of pulp or direct pulp capping.
- May be used underneath a base.

Contraindications. Cannot be applied thick enough to provide thermal protection for the pulp due to poor strength.

Operators will find that CaOH is easy to manipulate, hardens rapidly when applied in thin layers, provides a relatively good seal and has positive effects on both carious dentine and exposed pulp. Unfortunately, it is low in strength, undergoes plastic deformation and is highly soluble in water, and resins-based restorative materials will not bond to conventional CaOH.

Calcium hydroxide products are available in either a paste-paste version or a liquid formulation. Trade names: examples of calcium hydroxide paste-paste products are Dycal, VLC Dycal (Caulk Dentsply, Milford, DE, USA), Life (Kerr, Orange, CA, USA). Liquid versions of CaOH are the resin-based products Hydroxyliner (George Taub Products, Jersey City, NJ, USA) and Timeline (Caulk Dentsply, Milford, DE, USA).

Hydroxyliner and Hydroxyliner TC – Calcium hydroxide cavity liner (Taub Products, USA).



Picture 5. liners with Calcium hydroxide.

Zinc oxide eugenol (ZOE). The powder is composed of zinc oxide (70% by weight) with rosin added to reduce the brittleness of the set material.

The eugenol is in the liquid portion, derived from oil of cloves (one of the «essential oils»). The eugenol is bactericidal on its own, but is more potent when combined with zinc oxide.

ZOE has been available for over 100 years. Despite having a pH of about 7 and having a sedative effect on the pulp, the eugenol can be toxic to the pulp, especially when present in high concentrations. It is for this reason that ZOE should not be placed in direct contact with the pulp.

Eugenol is released from the mixture by hydrolysis. The wet dentine causes enough eugenol to be released to form a concentration gradient that kills bacteria, but does not damage the pulp. Hume showed that the dentine protects the pulp from chemical irritation and as the remaining dentine thickness increases, so does the protection.

Even though ZOE does not bond to the tooth, it does afford an excellent marginal seal, which is better when a lower powder-liquid ratio is used. The advantage of this seal is the prevention of diet-derived substrate from reaching the micro-organisms found below the restoration. This results in the reduction of both acid production and of the formation of secondary caries. Essentially, ZOE inhibits bacterial cell metabolism, the end result being a low incidence of postoperative sensitivity.

Hydrolysis of zinc oxide precedes a reaction between the resulting zinc hydroxide and eugenol, and this allows the ZOE mixture to set. The reaction occurs in the presence of water acting as a catalyst, which is why the reaction occurs faster when wet than when no moisture is present.

ZOE is not marketed as a cavity liner, but as a base (as well as other uses not relevant to this paper). Some products contain polymethylmethacrylate, which is incorporated in order to strengthen the material, making it more appropriate for use as a cavity base.

There are no ZOE products that are marketed for use as a liner. An example of ZOE as a base is IRM (Intermediate Restorative Material; Caulk Dentsply, York, PA, USA), available both in powder-liquid and encapsulated versions.

Mineral trioxide aggregate (MTA), a material currently being used in pulp therapy, has been demonstrated to provide an enhanced seal over the vital pulp and is non-resorbable. MTA has been used experimentally for a number of years and was approved for human usage by the FDA in 1998.

MTA is an ash-colored powder made primarily of fine hydrophilic particles of tricalcium aluminate, tricalcium silicate, silicate oxide, and tricalcium oxide. When the material is hydrated it becomes a colloidal gel. The main components of MTA are calcium phosphate and calcium oxide. The material sets in approximately 3-4 hours, and, for radiopacity, vismuth oxide powder has been added, achieving a radiopacity similar to gutta percha. The initial pH of MTA when hydrated is 10.2 and the set pH is 12.5, which is comparable to that of calcium hydroxide. MTA has been found to have a set

compressive strength of about 70 MPA. This is approximately equal to that of IRM but much less than amalgam (311 MPA).

MTA stimulated the release of cytokines and the production of interleukin, new bone formation.

The material has also been shown to have antimicrobial properties, have low cytotoxicity

The setting ability of MTA is uninhibited by blood or water. Place a 1-1.5 mm thick layer of freshly mixed MTA directly over the exposed pulp. Place a wet, thinned, flattened cotton pellet over the MTA.

Examples: ProRoot™ (Tulsa Dental), Grey MTA PlusR _Avalon Biomed.

Indications:

- Pulp capping.
- Cavity lining.
- Pulpotomies.

Question 6. Treatment of deep caries lesions.

1) Traditional complete caries removal (removal of all soft and leathery dentin). The traditional caries removal technique involves the removal of all soft and leathery dentin until hard dentin is reached before placing a final restoration. In shallow to moderate dentinal cavitated caries lesions (that radiographically appear to extend less than 75 percent into the dentin) this technique is often used without the risk of exposing the pulp.

Deep caries lesions are cavitated caries lesions that radiographically extend more than 70 to 75 percent into dentin. When the traditional caries removal technique is used to treat the deep caries lesions of vital asymptomatic teeth, the risk of pulp exposure is high. Complete removal of the soft and leathery dentin may cause a pulpal exposure, introducing bacteria into the pulp. Such outcomes require either root canal therapy or extraction. Evidence indicates that the traditional complete caries removal procedure may be detrimental to the pulpo-dentinal complex and does not take into consideration the biological natural response of the tooth to the caries stimulus.

Table 10

Indications, advantages and limitations for complete caries removal

<i>Indications</i>	<i>Advantages</i>	<i>Limitations</i>
Indicated for shallow to moderate caries lesions with no risk of pulp exposure	One visit (time for dentist and patient)	Invasive treatment for deep caries lesions
Only for deep caries lesions when:		Excessive removal of tooth structure
– Tooth is treatment plan for indirect restoration		Does not follow a biological approach to maintain pulp

		vitality
– Key Teeth abutment for FPD or RPD		
– Tooth will benefit from elective RCT		High risk of pulp exposure in deep caries lesions
– To clarify diagnosis when suspecting irreversible pulpitis		
– By patient informed decision		
FPD: Fixed Partial Denture RPD: Removable Partial Denture RCT: Root Canal Treatment		

For deep lesions, this lack of clinical evidence may result in an excavation that risks pulp exposure.

In a tooth with a deep caries lesion, no history of spontaneous pain, normal responses to thermal stimuli, and a vital pulp, a deliberate, incomplete caries excavation may be indicated. This procedure is termed indirect pulp capping (also referred to as stepwise caries excavation or partial caries excavation) and is supported by a large body of evidence. In brief, indirect pulp capping consists of complete caries excavation peripherally to a sound, caries-free DEJ; axially and pulpally, caries is excavated to within approximately 1 mm of the pulp; a glass ionomer (e.g., Fuji IX, GC, Alsip, IL) sedative restoration or a definitive restoration is then placed. The glass ionomer is used when the clinician anticipates a follow-up appointment will be needed to re-enter the preparation and complete the caries excavation.

Pulp exposures can be due to mechanical reasons, caries or trauma. When the pulp exposure of a vital asymptomatic tooth is mechanical or due to trauma, the Direct Pulp Capping procedure has been used in an attempt to preserve tooth vitality. After rinsing and disinfecting the exposure site, a liner usually from calcium hydroxide or MTA material is placed directly over the exposed pulp followed by a sealing liner of resin modified glass ionomer and the final restoration.

It is thought that pulp exposures due to trauma or mechanical reasons (iatrogenic) have a better success rate than a caries exposure since there is no bacterial contamination. When the pulp is exposed due to caries, the bacterial contamination will cause inflammation, decreasing the healing ability of the pulp and resulting in irreversible damage or necrosis. In deep caries lesions of asymptomatic, vital restorable teeth the pulp exposure should be avoided; it is preferable to use an incomplete caries removal approach rather than the direct pulp treatment.

The use of incomplete caries removal techniques have been proposed based on the deeper understanding of the biological response of the tooth to caries stimulus and the structural changes that occur as a protective response of the tooth to bacterial invasion.

The incomplete caries removal technique involves the partial removal of soft caries infected dentin starting peripherally (at the DEJ) and the sealing of the remaining caries lesion with an interim or final restoration (in one or two visits) with the goal to seal the partially demineralized affected dentin and arrest or reverse caries lesion progression.

2) The Conservative Approach to Partial Caries Removal. The most widely known and used techniques are the Indirect Pulp Treatment (formerly termed as "capping") and Stepwise Caries removal. They differ in the amount of soft dentinal tissue removed, number of appointments involved (one or two), and restorative materials. Each technique has indications, advantages and limitations. To properly utilize these techniques for different clinical situations, tooth and pulpal diagnosis is crucial, as well as the understanding of the caries lesion activity, and related changes in the dental structures.

Stepwise caries removal

Stepwise excavation is an alternative technique for removal of deep caries lesions that radiographically involve 75 percent or more of the total dentin thickness and do not already penetrate to the pulp. The purpose of the stepwise excavation approach is to change the cariogenic environment of deep caries lesions by removing only the soft wet infected dentin and then sealing the remaining demineralized dentin with an interim restoration. The goal is to arrest the active caries lesion and stimulate dentinal tubule sclerosis and the formation of reparative dentin while maintaining pulp vitality.

Table 11

Indications, advantages and limitation of stepwise treatment

<i>Indications</i>	<i>Advantages</i>	<i>Limitations</i>
Deep caries lesions in vital asymptomatic (reversible pulpitis & non periradicular pathosis), restorable teeth	Preserves tooth structure	Time (2 visits)
Active\soft\rapid progressive lesions	Promote remineralization of soft remanent dentin	Cost (patient may need to pay for provisional and final restorations)
Usually in compliant young patients	Thicker remaining dentin over pulpal and axial walls, less likely to cause pulp exposure	May not be covered by third party payers
Patient OK with 2 steps and 6.0 months re-entry	Biological approach promoting tubule sclerosis and formation of reparative, reactionary dentin	Patient compliance (patient may not come back for final restorations)
Primary lesions or RCA in shallow restorations with thick RDT	Provides time to clarify pulpal diagnosis	Risk of pulp exposure at re-entry
In caries control cases		When compromise future

(multiple teeth with rampant caries)		restorability. Key teeth, abutments and teeth in need of large indirect restorations
RCA: Recurrent Caries RDT: Remaining Dentinal Thickness		

Technique for stepwise excavation

After a detailed evaluation and correct case selection using the previous criteria, the stepwise approach is performed in two separate appointments with an interval of six to eight months.

First Appointment

1. Inform the patient about the treatment options including benefits and possible drawbacks. Allow the patient to be part of the decision.
2. Rubber dam isolation is highly recommended.
3. Access to the caries lesion, peripheral excavation should be completed by cleaning the DEJ, removing the very soft, necrotic and infected dentin and leaving the soft, discolored yellow or dark leathery dentin over the pulpal floor and axial walls. Avoid excavating close to the pulp during this first step to reduce the risk of pulp exposure.
4. Restore with a temporary glass ionomer material. For example, first use Fuji Triage GC® as a liner (color coded for reentry) and then place Fuji IX or Fuji II LC GC® as a restorative material.
5. Schedule appointment (six to eight months) for re-entry.

Second Appointment (Re- Entry)

1. Re-evaluate history of symptoms.
2. Clinical exam to evaluate for swelling or tenderness.
3. New periapical radiograph to verify lack of pathosis.
4. Pulp vitality tests.
5. If all of the above are normal isolate teeth, preferable with Rubber Dam.
6. Remove the sedative filling peripherally first and then be especially careful when approaching the Fuji Triage liner. The Fuji Triage does not need to be completely removed if arrested and well-sealed dentin is observed, it may be maintained over pulpal and axial wall to prevent pulp exposure.
7. Dentin assessment (peripheral) and careful removal of any remaining soft dentin.
8. Placement of glass ionomer liner over the exposed dentin (Vitrebond 3M®)
9. Restore with the material of choice for final restoration.
10. Six month recall for evaluation of vitality tests and periapical radiograph.
11. Continue with similar annual recalls.

The difference between Stepwise Caries Removal and Indirect Pulp Treatment is that the stepwise procedure is performed in two visits (usually months apart). In the first visit, the soft necrotic carious dentin is removed

partially and peripherally and the tooth is sealed with an interim restoration. The time interval between the two visits allows remineralization to occur and tertiary dentin to develop. At the second visit, the tooth is re-entered, the residual affected soft dentin is removed and the final restoration is placed. Two recent systematic reviews, Rickets et al. and Schewendicke et al. have compiled and analyzed the evidence suggesting that there are potential benefits to reducing the risk of pulp exposure in using either one or two steps techniques compared with complete caries removal. There is still a need of more evidence and good standardize clinical research to determine whether is necessary to re-enter.

Indirect pulp treatment (IPT)

ITP consists in the removal of all peripheral soft dentin of the deep caries lesion, leaving a thin residual layer (0.5mm–1.0mm) of leathery affected dentin over the pulpal floor or axial wall followed by a liner and placement of the final restoration with the goal of preventing pulp exposure.

Use of incomplete caries removal techniques significantly decreases the risk of pulp exposure in deep caries lesions compared with the traditional complete caries removal procedure, and these restorations have shown similar success.

If there is any remaining bacteria after the caries is partially removed, the placement of a restoration providing a good seal will arrest the lesion progression by isolating the bacteria from the substrate and decreasing acid production.

Table 12

Indications, advantages and limitation of indirect pulp treatment

<i>Indications</i>	<i>Advantages</i>	<i>Limitations</i>
Deep caries lesions in vital asymptomatic (reversible pulpitis & non periradicular pathosis), restorable teeth.	One visit (time for dentist and patient)	More structure removed than Stepwise technique but less than complete caries removal technique
Presence of sclerotic dentin in a chronic slow progressing lesion	Cost	Greater risk of pulp exposure by accident than SWE (1st appointment)
Desire for final restoration in same appointment	Insurance coverage	Less RDT is left (around 0.5 mm of residua affected dentin)
RCA under deep existing restoration with thin RDT		
Asymptomatic patient that deny RCT and have above indications		No time interval to clarify diagnosis or promote tubule sclerosis
By patient informed decision		
RCA: Recurrent Caries RDT: Remaining Dentinal Thickness RCT: Root Canal Treatment		

Technique steps for indirect pulp treatment

1. No history of spontaneous pain.
2. Proper Diagnosis: EPT (Electric Pulp Test).
3. Periapical radiograph with normal periapical structures
4. Good Isolation (Preferable Rubber Dam).
5. Peripheral caries at the DEJ removed while maintaining thin residual caries dentin over pulpal and axial walls.
6. Clean DEJ at cavosurface margin to achieve a good restoration seal.
7. Finish cavity preparation (clean and smooth walls) with design depending on material selection.
8. Liner placement (either calcium hydroxide, resin modified glass ionomer (ex: Vitrebond™ 3M) or a resin-modified calcium silicate filled liner® Theracal (Bisco).
9. Final restoration providing good seal.
10. Follow-up\Recall within three to six months.

Test tasks

1. Name the main principles of tooth preparation

- a. *To restore function.*
- b. *To prevent further spread of an active lesion which is not amenable to preventive measures.*
- c. *To preserve pulp vitality.*
- d. Provide access to pulpal chamber.

2. What is the approach of cavity formation, that has main principle «extension for prevention».

- a. *Black's technique*
- b. Technique of adhesive preparation

3. What are types of dental bors?

- a. Diamond
- b. Steel
- c. Carbide
- d. Hard-allow
- e. *All of the above*

4. What parts does any rotary cutting instrument include?

- a. Shank
- b. Neck
- c. Head
- d. *All of the above*

5. What are the stages of carious cavity preparation?

- a. Opening of cavity
- b. Enlargement of cavity
- c. Necrectomy
- d. Formation the cavity
- e. *All of the above*

6. Causes of pulp damage during tooth and treatment

- a. Heat generated by rotary instruments,
- b. Some ingredients of various materials
- c. Thermal changes conducted through restorative materials
- d. The ingress of diferrent products and bacteria through microleakage
- e. *All of the above*

7. What are functions of Liners?

- a. *Barrier that protects dentin from irritants agents from either the restorative materials or oral fluids*
- b. *Some thermal protection*
- c. To restore form and function of a tooth

8. Name properties of CaOH materials

- a. *Stimulates the production of irregular secondary (tertiary) dentine*
- b. *PH of 11–12 (i.e. alkaline)*
- c. *Bactericidal properties*
- d. Insoluble

9. What are disadvantages of CaOH materials?

- a. *Moisture sensitive*
- b. pH of 11–12 (i.e. alkaline)
- c. *Low strength*
- d. Biocompatibility

10. What are introductions to use of MTA materials?

- a. *Pulp capping*
- b. *Cavity lining*
- c. Restoration of tooth crown
- d. Treatment of hypersensitivity of dentin

CONTROL TEST:

«Etiology, pathogenesis, clinical features, diagnosis and treatment of dental caries and dental plaque».

1. Classification of tooth deposits.
2. The non-mineralized dental plaque. The cuticle, pellicle, definition, composition, mechanism of formation.
3. Dental plaque characteristics, mechanism of formation, properties.
4. The mineralized tooth deposits. Types of a dental tartar. Theories of the formation of a tartar. Influence of a tartar on the teeth and periodontal tissues.
5. Anatomical and histological structure of the gums.
6. Risk factors of gingivitis occurrence.
7. The relationship between oral hygiene and inflammation in the periodontal tissues.
8. Symptoms and stages of the gums inflammation.
9. Clinical manifestations of inflammation of the gums.
10. Indices GI, PMA, CPI.
11. Dental caries. Etiology.
12. Theories of dental caries.
13. The epidemiology of dental caries.
14. Pathogenesis of dental caries.
15. The classification of dental caries: Black's classification. International caries classification. Morphological classification. International Classification of caries.
16. The composition and structure of enamel.
17. The composition and structure of dentin.
18. Histopathology of enamel caries.
19. Clinical appearance of initial caries
20. Clinical appearance of superficial caries.
21. Histopathology of dentin caries.
22. Clinical appearance of dentin caries.
23. Diagnosis of dentin caries.
24. Differential diagnosis of dentin caries.
25. Clinical examination of enamel caries.
26. Clinical diagnosis tests of dentin caries.
27. Additional methods of diagnosis of enamel caries.
28. Differential diagnosis of caries enamel.
29. Treatment of caries enamel. Techniques for Minimally Invasive Treatment.
30. Treatment of dentin caries. Principles of tooth preparation.
31. Treatment of deep caries lesions.

LESSON 11.DIAGNOSIS AND TREATMENT OF OCCLUSION SURFACES CARIES.SELECTION OF FILLING MATERIAL.

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

1. Lesions of Black's Class I. Location. Clinical characteristics.
2. Diagnosis of carious lesions of Class I.
3. Peculiarities of Black's preparation of carious lesions of Class I.
4. Class I Amalgam Restoration.
5. Class I Composite Restoration.

Question 1.Lesions of Black's Class I. Location.Clinical characteristics.

Locations of lesions of Class I include:

- Occlusal surface of molars and premolars
- Lingual surface of anterior teeth
- Occlusal two thirds of buccal and lingual surfaces of molars and premolars, i.e. blind pits of teeth.

The occlusal surface is characterized by the pit and fissure systems, a favorable biofilm stagnation area where the bacterial accumulations receive the best protection against functional/mechanical wear (mastication, attrition, abrasion from brushing, flossing or toothpicks). Those aspects contribute to the high prevalence of caries on occlusal surfaces both in the primary and permanent dentition.

The complex anatomy of the occlusal surfaces requires professional special attention and deep understanding of how lesions develop on this surface. It is known that the deepest part of the fissure usually harbors non-vital bacteria or calculus. An enamel caries lesion begins along the pits and fissures through acids diffusion from bacterial metabolism in the biofilm. This diffusion occurs through the side walls of the pits and fissures, guided by prisms direction and striae of Retzius. Histologically, the lesion forms in three dimensions and assumes the shape of a cone, with its base toward the enamel-dentin junction. Acids lead to the demineralization underneath the enamel surface and there is an enlargement in intercrystalline spaces, increasing its permeability. Over time, the surface porosity has increased and leads to a considerable increase of the lesion body (a subsurface lesion starts to form). Occlusal enamel breakdown is the result of further demineralization, thus leading to cavity formation.

Question 2.Diagnosis of carious lesions of Class I Main and additional methods of diagnosis of carious lesions of Class I

The main methods of diagnosis

1. Questioning:

- ✓ Complaints.

- ✓ Patient history.
- ✓ Medical history.

2. Clinical Examination:

Probing.

- ✓ To determine of roughness of enamel surface.
- ✓ To determine of dentin density (soft or hard) at a bottom and a walls of caries cavity.
- ✓ To determine of sensitivity (a bottom and a walls of caries cavity).

Drying. Reaction on thermal agents (cold, heat).

For the purpose of caries management, individual tooth surfaces are categorized and described, based on an evaluation of each surface affected. For pits and fissures, the evaluation criteria are as follows.

Table 13

Pits and fissures evaluation criteria

Tooth surface	Evaluation criteria	ICDAS code
Sound surfaces	No visible caries when viewed clean and dry. Non-carious white or brown marks on tooth surfaces must be differentiated from early caries lesions.	0
Initial stage caries	Characterized by the first visual change in enamel (seen only after prolonged air drying or restricted to the confines of a pit or fissure). OR A distinct visual change in enamel (seen on a wet or dry surface).	1
		2
Moderate stage caries	Characterized visually by either localized enamel breakdown (without visual signs of dentinal exposure). - Enamel breakdown is often viewed best when the tooth is air dried. OR An underlying dark shadow from dentin. - Shadowing from dentinal caries is often best seen with the tooth surface wet.	3
		4
Extensive stage caries	Characterized by distinct cavitation exposing visible dentine. - Lesions exhibiting cavitation Involving less than half the tooth surface - Lesions involving half of the tooth surface or more	5
		6

The additional methods of diagnosis of carious lesions of Class I

- Radiologic and clinical examination
- Emerging diagnostic techniques
- Fluorescence
- Fiber-optic transillumination (FOTI)
- Digitally imaging fiber-optic transillumination (DIFOTI)
- Electrical conductivity.

Bite-wing (BW) films for proximal decay detection.

Directions in caries diagnosis for occlusal caries. Caries researchers recommend against using a sharp explorer when examining pits and fissures. Histological evidence shows the explorer can disrupt incipient caries and bacteria can be moved from groove to groove by a sharp explorer. Reliance on a sharp explorer has the potential to underutilize other detection methods and leads to a false positive diagnosis of occlusal caries.

Visualization of pit and groove areas for discoloration under the enamel is essential for pit and fissure diagnosis. Teeth must be clean, dry, and well illuminated to be properly evaluated. With the greater exposure to fluorides and more frequent placement of sealants deep caries can exist without a visible indication. Radiographs, in the past considered of limited use in the detection of occlusal caries, have been shown to be valuable in detecting deep dentin caries beneath pit and fissures and sealants.

Bitewing radiography diagnosis is that it is not invasive, and does not damage tooth structure like an incorrectly used dental probe might. Radiographs can also be filed and reexamined at a later date to compare with a more recent image to detect whether a lesion is progressing or not.

Limitations of bitewing radiography diagnosis. Besides concerns about low-dose radiation and variations in how images are interpreted by dentists, the main limitation is that the validity in diagnosing early lesions is rather low. Also, the bitewing radiograph cannot always distinguish between sound surfaces, those with initial caries activity and cavitated lesions, or non-carious demineralizations, so clinical inspection is still needed to determine what is happening to the tooth. Bitewing radiographs also tend to underestimate the depths of lesions, so a lesion that appears confined to the inner enamel on an image is often actually in the dentin, and this can lead to insufficient or improper treatment.

However, digital radiography is replacing radiography based on film. It has been proven as accurate as traditional radiography for detecting caries, and it comes with additional advantages of using a lower radiation dose, being less time-consuming, and does not require wet chemicals in the processing of the image.

Digital radiography – which is increasingly replacing bitewing radiography and that is as accurate as film for the detection of caries lesions.

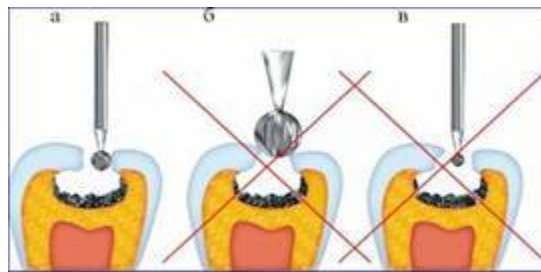
Digital image enhancement – which studies show can provide superior results to radiographs when enhanced correctly but takes a significant amount of technical skill.

Question3. Peculiarities of Black's preparation of carious lesions of Class I.

According to Black's principles of preparation, a cavity of the I class should be: with straight walls at right angle to the bottom, a form of the cavity should be cylindrical, box-shaped, rhombic, X-like.

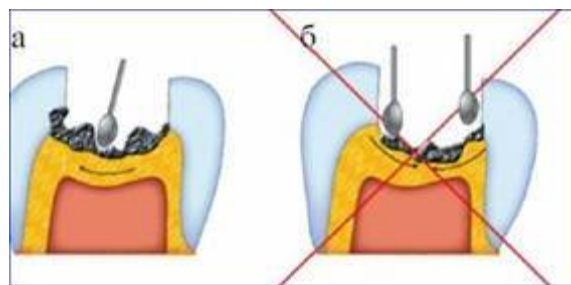
Regardless of cavity location, there are common stages of dental hard tissues preparation, which are come to:

Disclosure (opening and enlargement) of cavity (is conducted by using round-shaped, fissures burs, burs that is chosen, should have the size of the working end not bigger than the entrance aperture of this cavity).



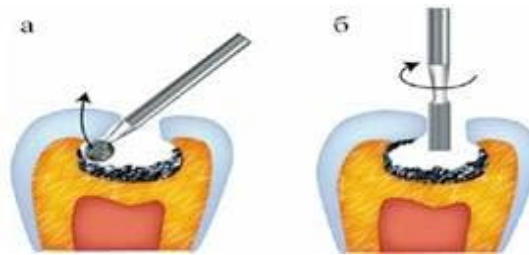
Picture 6. Correct and incorrect opening of cavity.

Necrectomy is a removal of the necrotic dental hard tissues from carious cavity. There are total and partial necrectomy. Total -is complete removal of necrotic dentin from the walls and bottom of the cavity. Partial - is complete removal of necrotic dentin from walls and partly from the bottom of the cavity. Partial necrectomy is allowed in the case of deep dental caries, when the bottom of the cavity is very thin and there is a danger of the pulp horn disclosure. In this case is permitted to leave on the bottom of the cavity a dense pigmented dentin, and in the course of acute deep caries – is allowed to leave a small layer of softened dentin with the next remineralization influence on it. Necrectomy is done by the round-shaped burs and the excavator.

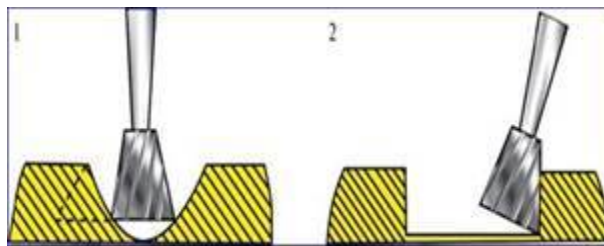


Picture 7. Correct and incorrect directions of bor movement during of necrotomy.

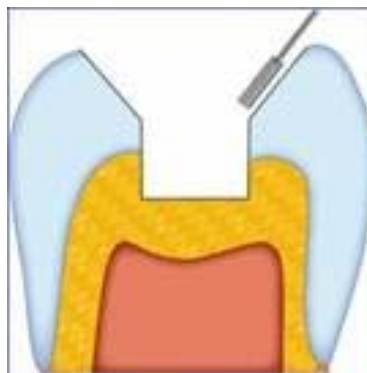
Formation the cavity for fillings (is done with fissures, inverted-cone and cone-shaped burs).



Picture 8. directions of movement of different burs during formation the cavity.



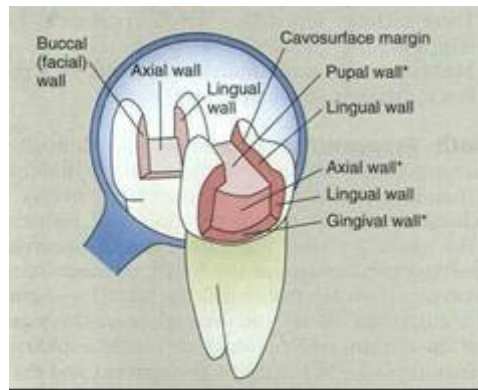
Picture 9.



Picture 10. Smoothing the edges of enamel.

Cavity wall: Side or surface of a tooth prepared for restoration.

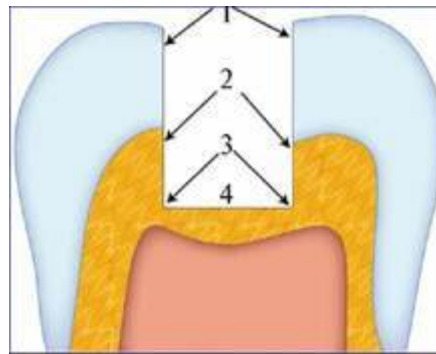
- Internal wall: Cavity wall that does not extend to the external tooth surface.
- External wall: Portion of the tooth preparation that extends to the external tooth surface, named according to the tooth surface involved: distal, mesial, facial, lingual, and gingival.
- Axial wall: Internal wall of prepared tooth that runs parallel to the long axis of the tooth.
- Pulpal wall: Internal wall of prepared tooth that is perpendicular to the long axis of the tooth; also known as the pulpal floor.
- Line angle: Angle formed by the junction of two walls in a cavity preparation.



Picture 11. Cavity wall.

There are general rules for the preparation of cavities according to Black's principles of preparation:

1. Transition the bottom of the cavity (the surface which is turned to a pulp) to the side wall should be at right angle.



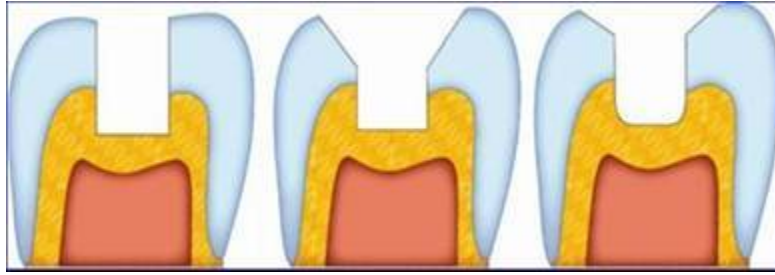
Picture 12.

2. Transition of one wall to another should be at an angle –the form of the cavity-box- shaped form (except the V class).

3. Enamel edges should be straight and smooth.

4. Bottom of the cavity should be flat or somewhat reminding the form of the occlusal surface of the tooth.

Dissection of tooth tissues for filling with composites materials is slightly different from the traditional preparation by Black. This is because the traditional preparation is used for mechanical fixing of fillings in the carious cavity. Composite materials have the ability to chemically bind to tissues, so there is no need to prepare a wall at right angles.



Picture 13. Variants of traditional preparation by Black and preparation for Composite materials.

Question 4. Class I Amalgam Restoration.

Types of Amalgam Restorative Materials

Low-copper amalgam. Low-copper amalgams were prominent before the early 1960s. When the setting reaction occurred, the material was subject to corrosion because a tin-mercury phase (gamma-two) formed. This corrosion led to the rapid breakdown of amalgam restorations. Subsequent research for improving amalgam led to the development of high-copper amalgam materials.

High-copper amalgam. High-copper amalgams are the materials predominantly used today in the World. The increase in copper content to 12% or greater designates an amalgam as a high-copper type. The advantage of the added copper is that it preferentially reacts with the tin and reduces the formation of the more corrosive phase (gamma-two) within the amalgam mass. This change in composition reduces possible deleterious corrosion effects on the restoration. However, enough corrosion occurs at the amalgam-tooth interface to result in the successful sealing of the restoration. These materials can provide satisfactory performance for more than 12 years. High-copper materials can be either spherical or admixed in the composition.

Spherical amalgam. A spherical amalgam contains small, round alloy particles that are mixed with mercury to form the mass that is placed into the tooth preparation. Because of the shape of the particles, the material is condensed into the tooth preparation with little condensation pressure. This advantage is combined with its high early strength to provide a material that is well suited for very large amalgam restorations such as complex amalgams or foundations.

Admixed amalgam. An admixed amalgam contains irregularly shaped and sized alloy particles, sometimes combined with spherical shapes, which are mixed to form the mass that is placed into the tooth preparation. The irregular shape of many of the particles makes a mass that requires more condensation pressure (which many dentists prefer) and permits this heavier condensation pressure to assist in displacing matrix bands to generate proximal contacts more easily.

New amalgam alloys. Because of the concern about mercury toxicity, many new compositions of amalgam are being promoted as mercury free or

low-mercury amalgam restorative materials. All with gallium or indium or alloys using cold-welding techniques are presented as alternatives to mercury-contain amalgams. None of these new alloys shows sufficient promise to become a universal replacement for current amalgams materials.

Important properties. The linear coefficient of the thermal expansion of amalgam is 2,5 times greater than that of tooth structure, but it closer than the linear coefficient of thermal expansion composite. Although the compressive strength of high-copper amalgam is similar to tooth structure, the tensile strength is lower, making amalgam restorations prone fracture.

Usually, high-copper amalgam fracture is a bulk fracture, not a marginal fracture. All amalgams are brittle and have low edge strength. The amalgam material must have sufficient bulk (usually 1 to 2 mm, depending on the position within the tooth) and a 90-degree or greater marginal configuration.

Creep and flow relate to the deformation of a material under load over time. High-copper amalgams exhibit no clinically relevant creep or flow. Because amalgam is metallic in structure, it also is a good thermal conductor. An amalgam restoration should not be placed close to the pulpal tissues of the tooth without the use of a liner or base (or both) between the pulp and the amalgam.

Amalgam restorations. Amalgam functions as a direct restorative material because of its easy insertion into a tooth preparation and, when hardened, its ability to restore the tooth to proper form and function. The tooth preparation form not only must remove the fault in the tooth and remove weakened tooth structure, but it must also be formed to allow the amalgam material to function properly.

There required tooth preparation form must allow the amalgam to:

- 1) possess a uniform specified minimum thickness for strength;
- 2) produce a 90-amalgam angle (butt-joint form) at the margin;
- 3) be mechanically retained in the tooth.

Amalgam is less technique sensitive or operator sensitive compared with composite. Amalgam is used for the restoration of many carious or fractured posterior teeth and in the replacement of failed restorations.

Material qualities and properties for Class I amalgam restorations include the following:

- Strength
- Longevity
- Ease of use
- Clinically proven success

Advantages. Primary advantages are the ease of use and the simplicity of the procedure. The placing and contouring of amalgam restorations are generally easier than those for composite restorations.

Disadvantages. The primary disadvantages of using amalgam for Class I defects are:

1) Amalgam use requires more complex and larger tooth preparations than composite resin,

2) Amalgams may be considered to have a non-esthetic appearance by some patients.

Clinical indications for direct amalgam restoration – moderate to large restoration, especially restoration that involve heavy occlusion, can not be isolated well, or extend onto the root surface.

Contraindications for amalgam restoration – amalgams contraindicated in patients who are allergic to the alloy components. The use of amalgam in more prominent esthetic areas of the mouth is usually avoided.

Clinical technique for class I amalgam restorations

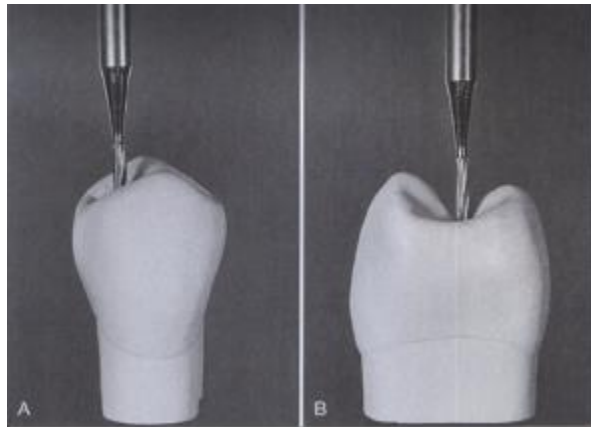
Conservative class I amalgam restorations. Conservative tooth preparation is recommended to protect the pulp, preserve the strength of the tooth, and reduce deterioration of the amalgam restoration. Such conservative preparation saves the tooth structure, minimizing pulpal irritation and leaving the remaining tooth crown as strong as possible. Conservative preparation also enhances marginal integrity and restoration longevity.

Initial clinical procedures. After the onset of profound anesthesia, isolation with the rubber dam is recommended to gain control over the operating field and for mercury hygiene. For a single maxillary tooth, where caries is not extensive, adequate control of the operating field may be achieved with cotton rolls and high-volume evacuation. A pre-operative assessment of the occlusal relationship of the involved and adjacent teeth also is necessary.

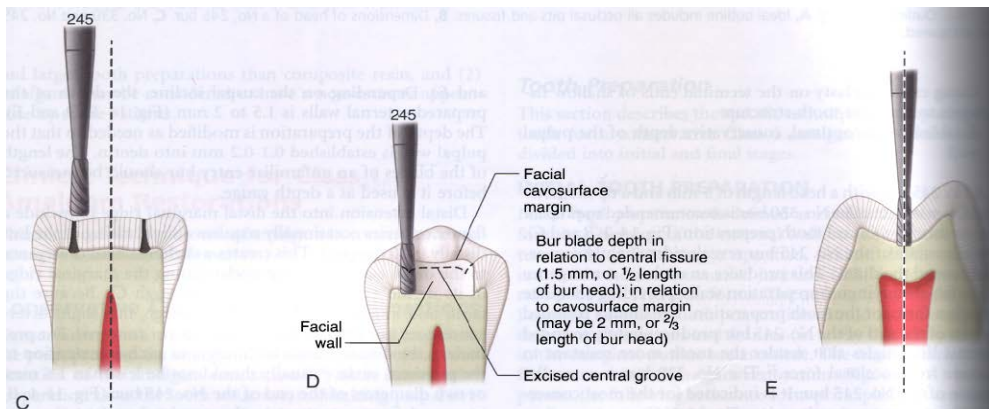
Initial tooth preparation is defined as establishing the outline form by extension of the external walls to sound tooth structure while maintaining a specified, limited depth (usually just inside the dentinoenamel junction [DEJ] and providing resistance and retention forms. The outline form for the Class I occlusal amalgam tooth preparation should include only the defective occlusal pits and fissures (in a way that sharp angles in the marginal outline are avoided). The ideal outline for a conservative amalgam restoration incorporates the following resistance form principles that are basic to all amalgam tooth preparations of occlusal surfaces. These principles allow margins to be positioned in areas that are sound and subject to minimal forces while conserving structure to maintain the strength and health of the tooth. The resistance principles are as follows:

- Extending around the cusps to conserve tooth structure and prevent the internal line angles from approaching the pulp horns too closely.
- Keeping the facial and lingual margin extensions as minimal as possible between the central groove and the cusp tips.

- Extending the outline to include fissures, placing margins on relatively smooth, sound tooth structure.
- Minimally extending into the marginal ridges (only enough to include the defect) without removing dentinal support.
- Eliminating a weak wall of enamel by joining two outlines that come close together (i.e., <0.5 mm apart).
- Extending the outline form to include enamel undermined by caries.
- Using enameloplasty on the terminal ends of shallow fissures to conserve tooth structure
- Establishing an optimal, conservative depth of the pulpal wall



Picture 14. Directions of instrument.



Picture 15. Stages of preparation:

A, No. 245 bur oriented parallel to long axis of tooth crown for entry as viewed from lingual aspect.

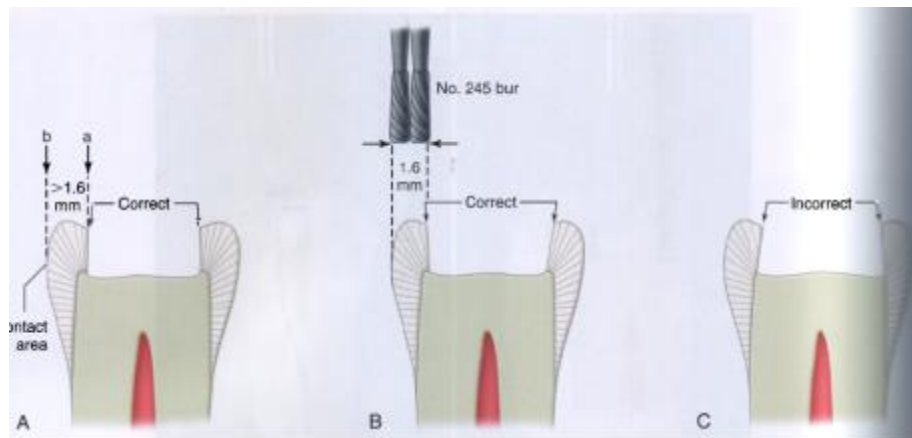
B, The bur positioned for entry as viewed from the distal aspect.

C, The bur is positioned over the most carious pit (distal) for entry. The distal aspect of the bur is positioned over the distal pit.

D, Mesiodistal longitudinal section. Relationship of head of No. 245 bur to excised central fissure and cavosurface margin at ideal pulpal floor depth, which is just inside the dentinoenamel junction (DEJ).

E, Faciolingual longitudinal section. Dotted line indicates the long axis of tooth crown and the direction of the bur.

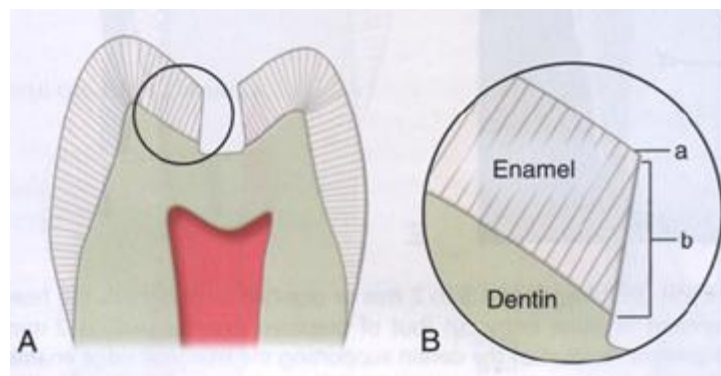
The direction of the mesial and distal walls is influenced by the remaining thickness of the marginal ridge as measured from mesial or distal margin to the proximal surface.



Picture 16: A. Mesial and distal walls should converge when the distance from a to b greater than 1,6 mm.

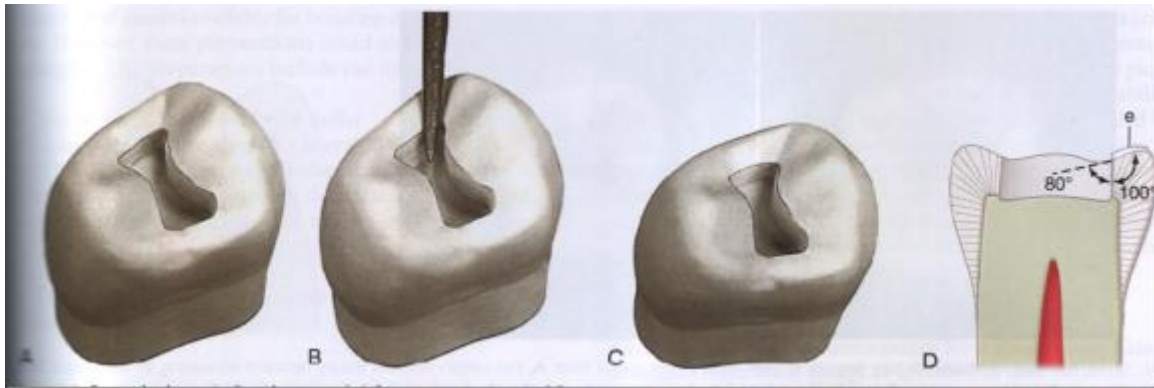
B. When the operator judges that the extension will leave only 1,6 mm thickness (two diameters of N 245 bur) of marginal ridge (i. e., premolars) as illustrated, the mesial and distal walls must diverge occlusally to conserve ridge-supporting dentin.

C. Extending the mesial or distal walls to a two-diameter limit diverging the wall occlusally undermines the marginal ridge enamel.

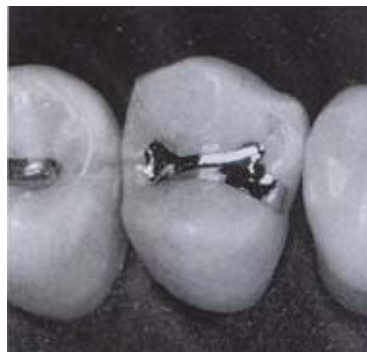


Picture 17.

The ideal and strongest enamel margin is formed by full-length enamel rods resting on sound dentin supported on the preparation side by shorter rods, also on sound dentin.



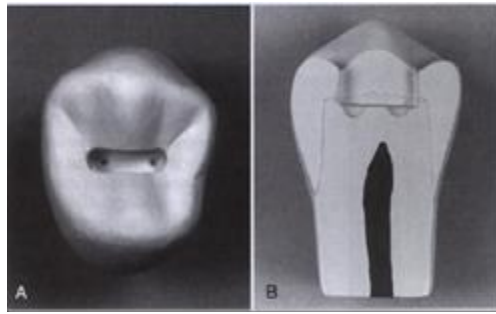
Picture 18. Enameloplasty. A. Development defect at terminal end of fissure.
 B. Fine – grit diamond bor in position to remove the defect.
 C. Smooth surface after enameloplasty.
 D. The cavosurface angle should not exceed 100 degrees, and the margin-amalgam angle should not be less than 80 degrees.



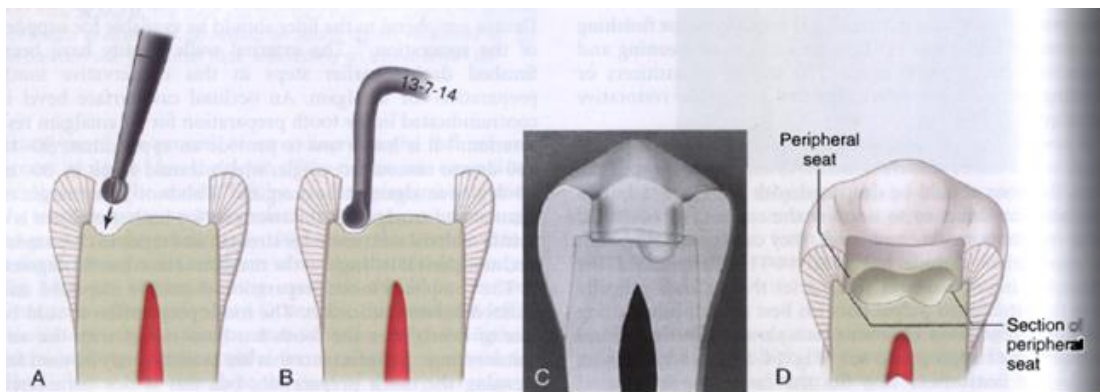
Picture 19. Mesial fissure that cannot be eliminated by enameloplasty may be included in the preparation if the margins can be lingual of contact.



Picture 20. Removal of enamel fissure extending over most of the pulpal floor.
 A, Full-length occlusal fissure remnant remaining on the pulp after the initial tooth preparation.
 B and C. The pulpal floor is deepened to a maximum depth of 2 mm to eliminate the fissure or uncover dentinal caries.



Picture 21. Removal of enamel pit and fissure and infected dentin that is limited to a few small pit-and-fissure remnants.
A, Two pit remnants on the pulpal floor after the initial tooth preparation.
B, Defective enamel and infected dentin have been removed.



Picture 22. A and B, Removal of dentinal caries is accomplished with round burs (A) or spoon excavators (B).
C and D, The resistance form improved with a flat floor peripheral to the excavated area or areas.

The final tooth preparation includes:

- 1) Removal of remaining defective enamel and infected dentin on the pulpal floor,
- 2) Pulp protection, where indicated,
- 3) Procedures for finishing the external walls,
- 4) Final procedures of cleaning and inspecting the prepared tooth.

The removal of carious dentin should not affect the resistance form further because the periphery would not need further extension. In addition, it should not affect the resistance form if the restoration is to rest on the pulpal wall peripheral to the excavated area or areas. The peripheral pulpal floor should be at the previously described initial pulpal floor depth just inside the DEJ.

If the tooth preparation is of ideal or shallow depth, no liner or base is indicated. In deeper caries excavations (where the remaining dentin thickness is judged to be 0.5 to 1 mm), a thin layer (i.e., 0.5-0.75 mm) of a light-activated, resin-modified glass ionomer (RMGI) material should be placed. The RMGI insulates the pulp from thermal changes, bonds to dentin, releases

fluoride, is strong enough to resist the forces of condensation, and reduces microleakage.

Dentin peripheral to the liner should be available for support of the restoration. The external walls already have been finished during earlier steps in this conservative tooth preparation for amalgam. An occlusal cavosurface bevel is contraindicated in the tooth preparation for an amalgam restoration. It is important to provide an approximate 90- to 100-degree cavosurface angle, which should result in 80- to 90-degree amalgam at the margins. This butt-joint margin of enamel and amalgam is the strongest for both. Amalgam is a brittle material with low edge strength and tends to chip under occlusal stress if its angle at the margins is less than 80 degrees. The completed tooth preparation should be inspected and cleaned before restoration. The tooth preparation should be free of debris after the tooth has been rinsed with the airwater syringe. Disinfectants that are available may be used for cleaning the tooth preparation, but this is not considered essential. A cotton pellet or a commercially available applicator tip moistened only with water is generally used.

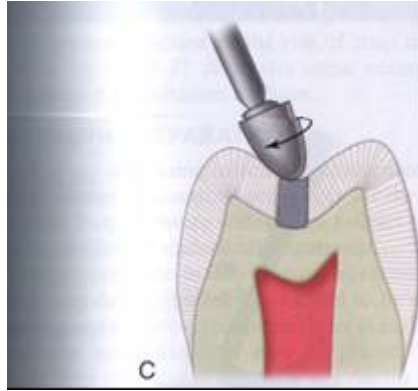
Restorative Technique for Class I Amalgam Preparations

The use of desensitizers or bonding systems is considered the first step of the restorative technique.

A dentin desensitizer is placed in the preparation before amalgam condensation. The dentin desensitizer is applied onto the prepared tooth surface according to manufacturer's recommendations; excess moisture is removed without desiccating the dentin; and then the amalgam is condensed into place. The dentin desensitizer precipitates protein and forms lamellar plugs in the dentinal tubules.

Insertion and carving of the amalgam

The principal objectives during the insertion of amalgam are to condense the amalgam to adapt it to the preparation walls and the matrix (when used) and produce a restoration free of voids. Thorough condensation helps to reduce marginal leakage. Optimal condensation also is necessary to minimize the mercury content in the restoration to decrease corrosion and to enhance strength and marginal integrity. Condensation of amalgam that contains spherical particles requires larger condensers than are commonly used for admixed amalgam. Smaller condensers tend to penetrate a mass of spherical amalgam, resulting in less effective force to compact or adapt the amalgam within the preparation. In contrast, smaller condensers are indicated for the initial increments of admixed amalgam because it is more resistant to condensation pressure. Because the area of a circular condenser face increases by the square of the diameter, doubling the diameter requires four times more force for the same pressure on a unit area.



Picture 23.

Carving should be accomplished so that opposing cusps contact on a surface that is perpendicular to the occlusal forces in maximum intercuspation. Occlusal contacts located on a cuspal incline or ridge slope are undesirable because they cause a deflective force on the tooth and should be adjusted until the resulting contact is stable (i.e., the force vector of the occlusal contacts should parallel the long axis of the tooth).

The patient has been instructed to close vertically in to maximum intercuspation. After placing the articulating paper over the tooth, the patient is asked to occlude lightly and to slide the teeth lightly from side to side. Any additional occlusal marks are evaluated, and undesirable contact areas are eliminated. Appropriate caution is indicated, as amalgam restorations carved out of occlusion may result in undesirable tooth movement. Finally, the patient should be cautioned to protect the restoration from any heavy biting pressure for 24 hours.

Most amalgams do not require further finishing and polishing. These procedures are occasionally necessary, however, to:

- 1) Complete the carving;
- 2) Refine the anatomy, contours and marginal integrity;
- 3) Enhance the surface texture of the restoration;

Additional finishing and polishing procedures for amalgam restorations are not attempted within 24 hours of insertion because crystallization is incomplete.

If used, these procedures are often delayed until all of the patient's amalgam restorations have been placed, rather than finishing and polishing periodically during the course of treatment. An amalgam restoration is less prone to tarnish and corrosion if a smooth, homogeneous surface is achieved.

Polishing of high-copper amalgams is less important than it is for low-copper amalgams because high-copper amalgams are less susceptible to tarnishing and marginal breakdown.

Question5. Class I Composite Restoration.

Advantages

The advantages of composite as a Class I direct restorative material relative to other restorative materials are:

1. Esthetics.
2. Conservative tooth structure removal.
3. Easier, less complex tooth preparation.
4. Insulation.
5. Decreased microleakage.
6. Increased short-term strength of remaining tooth structure.

Disadvantages

Disadvantages of Class I direct composite restoration as follows:

1. Polymerization shrinkage effects.
2. Lower fracture toughness than most indirect restorations.
3. More technique-sensitive than amalgam restorations and some indirect restorations.
4. Possible greater localized occlusal wear.

Pit-and-Fissure Sealants

Pits and fissures typically result from an incomplete coalescence of enamel and are particularly prone to caries. These areas can be sealed with a low-viscosity fluid resin after acid-etching. Long-term clinical studies indicate that pit and fissure sealants provide a safe and effective method of preventing caries. In children, sealants are most effective when they are applied to the pits and fissures of permanent posterior teeth immediately on eruption of the clinical crowns, provided proper isolation can be achieved. Adults also can benefit from the use of sealants if the individual experiences an increase in caries susceptibility because of a change in diet, lack of adequate saliva, or a particular medical condition. Most currently used sealant materials are light-activated urethane dimethacrylate or BIS-GMA (bisphenol A-glycidylmethacrylate) resins.

Clinical Technique for Class I Direct Composit Restorations

Tooth Preparation

As a general rule, the tooth preparation for direct posterior composites involves:

- 1) creating access to the faulty structure,
- 2) removal of faulty structures (caries, defective restoration),
- 3) creating convenience form for the restoration. Retention is obtained by bonding.

Small to Moderate Class I Direct Composite restorations

Small to moderate Class I composite restorations may use minimally invasive tooth preparations and do not require typical resistance and retention form features. Instead, these conservative preparations typically use more

flared cavosurface forms without uniform or flat pulpal or axial walls. These preparations are less specific in form, having a scooped-out appearance. They are prepared with a small round or elongated pear diamond or bur with round features.

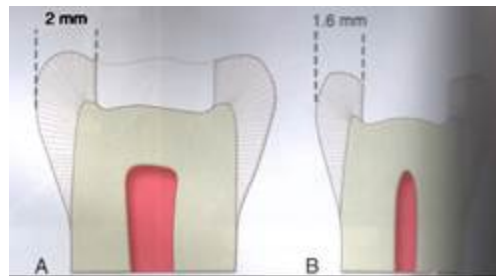
The initial pulpal depth is approximately 0.2 mm inside the DEJ but may not be uniform (i.e., the pulpal floor is not flat throughout its length). Usually, a more rounded, and perhaps smaller, cutting instrument is used for this preparation, in an attempt to be as conservative as possible in the removal of the tooth structure. If a round instrument is used, the resulting cavosurface margin angle may be more flared (obtuse) than if an elongated pearl instrument is used.

Various cutting instruments may be used for Class I preparations; the size and shape of the instrument generally dictated by the size of the lesion or other defect or type of defective restoration being replaced. Both diamond and pearl instruments can be used effectively. It be noted that diamond instruments create a thicker smear layer, however, which might make bonding more difficult self-etch bonding systems.

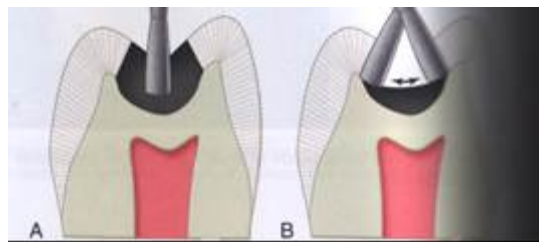
The objective of the tooth preparation is to remove all of the caries or fault as conservatively as possible. Because the composite is bonded to the tooth structure, other less involved, or at-risk, areas can be sealed as part of the conservative preparation techniques. Sealants may be combined with the Class I composite restoration.

In large composite restorations, the tooth is entered in the area most affected by caries, with the elongated pearl diamond or bur positioned parallel to the long axis of the crown. When it is anticipated that the entire mesiodistal length of a central groove will be prepared, it is easier to enter the distal portion first and then transverse mesially. This technique permits better vision to the operator during the preparation. The pulpal floor is prepared to an initial depth that is approximately 0.2 mm internal to the DEJ. The instrument is moved mesially, following the central groove, and any fall and rise of the DEJ. Mesial, distal, facial, and lingual extensions are dictated by the caries, old restorative material, or defect, always using the DEJ as a reference for both extensions and pulpal depth. The cuspal and marginal ridge areas should be preserved as much as possible. Although the final bonded composite restoration would help restore some of the strength of weakened, unprepared tooth structure, the outline form should be as conservative as possible. Extensions toward cusp tips should be as minimal as possible. Extensions into marginal ridges should result in at least 1.5 mm of remaining tooth structure (measured from the internal extension to the proximal height of contour) for premolars and approximately 2 mm for molars. These limited extensions help preserve the dentinal support of the marginal ridge enamel and cusp tips. As the instrument is moved along the central groove, the resulting pulpal floor is usually moderately flat (as a result

of the shape of the tip of the instrument) and follows the rise and fall of the DEJ. If extension is required toward the cusp tips, the same depth that is approximately 0.2 mm inside the DEJ is maintained, usually resulting in the pulpal floor rising occlusally. The same uniform depth concept also is appropriate when extending a facial or lingual groove radiating from the occlusal surface. When a groove extension is through the cusp ridge, the instrument prepares the facial (or lingual) portion of the faulty groove at an axial depth of 0.2 mm inside the DEJ and gingivally to include all caries and other defects.



Picture 24. Mesiodistal extension.
Preserve dentin support of marginal ridge enamel
A, Molar. B, Premolar.



Picture 25. A, After initial entry cut at correct initial depth (1,5 mm),
the caries remains facially and lingually.
B, Orientation of diamond must be tilted as the instrument
is extended facially or lingually to maintain a 1,5 mm depth.

After extending the outline form to sound tooth structure, if any caries or old restorative material remains on the pulpal floor, it should be removed with the appropriately-sized round bur or hand instrument. The occlusal margin is left as prepared. No attempt is made to place additional beveling on the occlusal margin because it may result in thin composite in areas of heavy occlusal contact. Because of the occlusal surface enamel rod direction, the ends of the enamel rods already are exposed by the preparation, which further reduces the need for occlusal bevels.



Picture 26. Directions of enamel rods

Restorative technique. Insertion and Light-Activation of the Composite

Composite insertion hand instruments or a compule may be used to insert the composite material. The dispenser, for example, a syringe or compule, must be kept covered when not in use to prevent premature hardening of the material. Small increments of composite material are added and successively light-activated. It is important to place (and light-activate) the composite incrementally to maximize the polymerization depth of cure and possibly to reduce the negative effects of polymerization shrinkage.

The term "configuration factor" or "C-factor" has been used to describe the ratio of bonded to unbonded surfaces in a tooth preparation and restoration. A typical Class I tooth preparation will have a high C-factor of 5 (five bonded - pulpal, facial, lingual, mesial, and distal-vs. one unbonded surface-occlusal). The higher the C-factor of a tooth preparation, the higher the potential for composite polymerization shrinkage stress, as the composite shrinkage deformation is restricted by the bonded surfaces. Incremental insertion and light-activation of the composite may reduce the negative C-factor effects for Class I composite restorations.

Test tasks

1. Point the main requirements for the formation of the classical class I cavities by Black:

- a. ***Bottom is flat.***
- b. Cavity walls are at an acute angle to the bottom.
- c. ***Walls are vertical.***
- d. ***The right angle between the bottom and the walls***
- e. Bottom is convex.

2. What are the peculiarities of class I cavity preparation by Black with deep caries:

- a. ***The formation of retention points***
- b. ***Softened dentin may be remained at the bottom***
- c. Necessarily steep wall
- d. ***Pigmented dentin may be remained at the bottom***
- e. ***The bottom can be a relief***
- f. Pins are used.

3. What are the peculiarities of class I cavities preparation using composite materials:

- a. *Mesial-distal walls are parallel to the axis of the tooth or have a slight expansion of the occlusion (<10 degrees)*
- b. Bottom is flat
- c. *Bucco-lingual walls are parallel to the axis of the tooth or slightly converge (<10 degrees)*
- d. *The angles between the bottom and walls of the cavity are rounded.*
- e. The angle between the bottom and walls of the cavity are right.

4. What are the advantages of amalgam?

- a. *High compressive strength*
- b. *Ease of use*
- c. Esthetic
- d. *Excellent wear resistance*
- e. *Favorable long-term clinical research results*
- f. *Lower cost than composite restoration*
- g. *Chemically resistant*

5. What are the disadvantages of amalgams:

- a. *Non-esthetic*
- b. *Require increased tooth structure removal during tooth preparation*
- c. *Require insulating layers.*
- d. *Initial marginal leakage*
- e. *High thermal conductivity*
- f. Easy to form a seal.

6. What are the indications for the use of amalgams:

- a. Class III cavities
- b. *Class I cavities*
- c. *Class V cavities*
- d. *Class II cavities*
- e. Class IV cavity

7. What are the advantages of composites?

- a. *Aesthetics*
- b. *Low thermal conductivity*
- c. Not toxic
- d. Strengthening tooth structure
- e. *Good polishing.*

8. What complications polymerization shrinkage causes?

- a. *Secondary caries*

- b. *Marginal gap formation between the restoration and the tooth surface*
- c. *Violation of marginal fit*
- d. *Microcracks*
- e. *Microleakage*
- f. *Hypersensitivity*
- g. Discoloration of seals

9. In what time is recommended to make a final finishing and polishing amalgam fillings?

- a. In 2 hours
- b. In 4 hours
- c. In 12 h
- d. *In 24 h.*

10. What are the advantages of glass ionomer cements?

- a. Chemical adhesion
- b. Coefficient of thermal expansion close to that of hard tissue
- c. The content of fluoride ions
- d. *All of the above*

11. The disadvantages of conventional glass ionomer cements:

- a. Moisture sensitivity in the early stages of curing
- b. Sensitivity to desiccation
- c. Mechanical instability
- d. *All of the above*

12. The way of improving the amalgam currently is the following:

- a. *Removing α -2 phase and a zinc*
- b. Reducing the copper content
- c. *Increasing copper content*

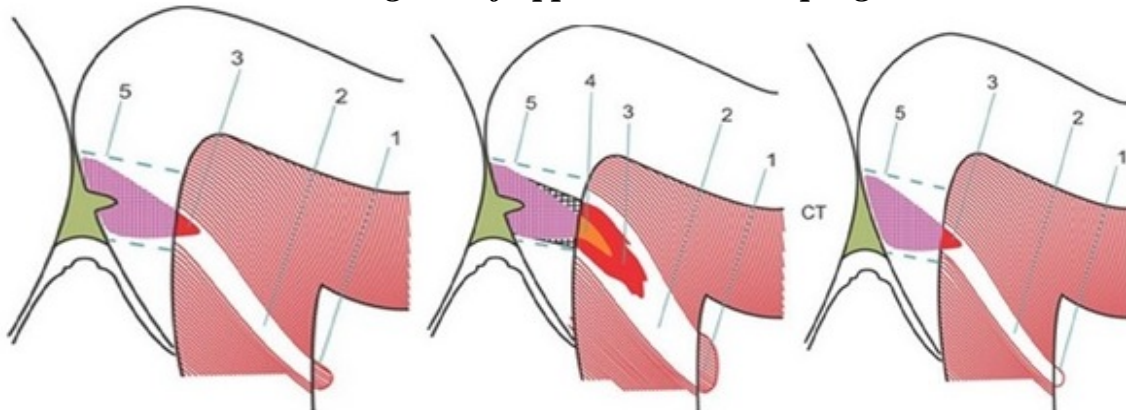
LESSON 12. DIAGNOSIS AND TREATMENT OF PROXIMAL SURFACES CARIES OF POSTERIOR TEETH. PROXIMAL CONTACT POINT. TECHNIQUES OF THE RE-ESTABLISHMENT CONTACT POINT. SELECTION OF FILLING MATERIAL.

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

1. Locations lesions of Class II. Physiological role of proximal contact point.
2. Main and additional methods of diagnosis of carious lesions of Class II.
3. Principles of preparation of Class II carious lesions.
4. Peculiarities of preparation of Class II carious lesions for composite filling materials.
5. The methods of re-establishment of proximal contact point.
6. Matrix system for re-establishment of proximal contact point.

Question 1. Locations lesions of Class II. Physiological role of proximal contact point.

Schematic diagram of approximal caries progression



- 1 - tertiary or reparative dentine, 2 - dentine tubules, 3- affected layer of carious dentine, 4-infected layer of carious dentine, 5- enamel lesion.

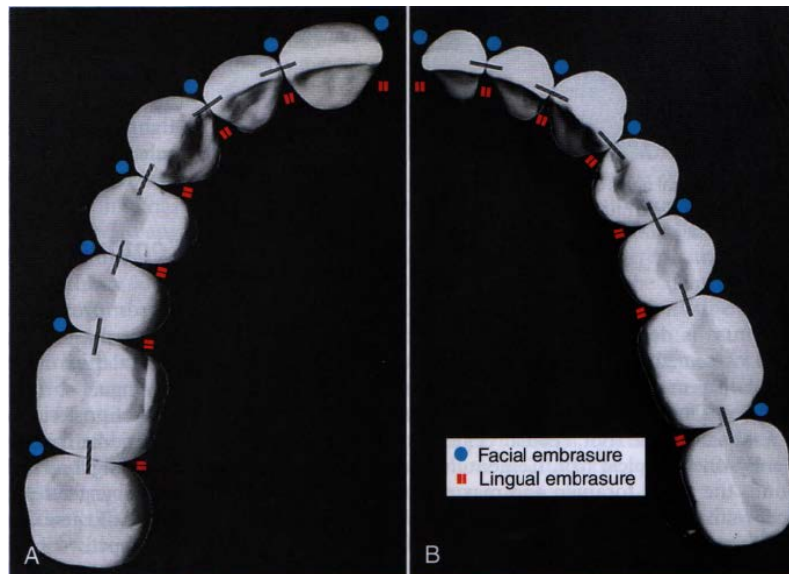
Lesions of Class II occur on the proximal surfaces of the posterior teeth - molars and premolars.

Proximal Contact Area. When teeth erupt to make proximal contact with previously erupted teeth, initially a contact point is present. The contact point increases in size to become a proximal contact area as the two adjacent tooth surfaces abrade each other during physiologic tooth movement.

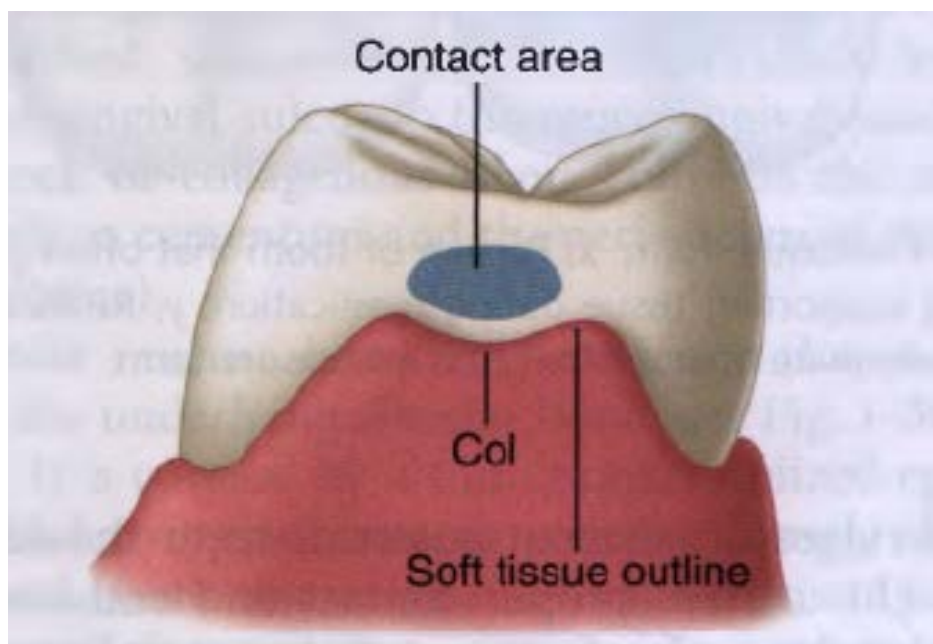
The proximal contact area is located in the incisal third of the approximating surfaces of maxillary and mandibular central incisors. It is positioned slightly facial to the center of the proximal surface faciolingually.

Proceeding posteriorly from the incisor region through all the remaining teeth, the contact area is located near the junction of the incisal (or occlusal) and middle thirds or in the middle third.

Proximal contact areas typically are larger in the molar region, which helps prevent food impaction during mastication.



Picture 27.



Picture 28. Relationship of ideal interdental papilla to molar contact area.

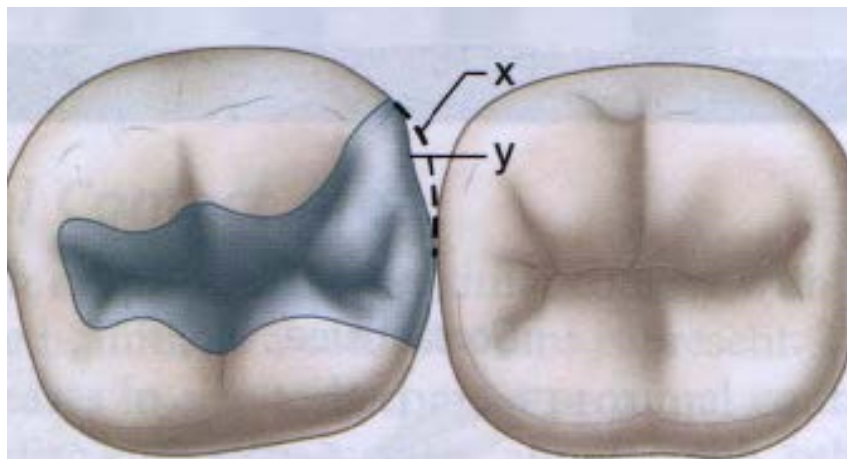
Adjacent surfaces near the proximal contacts (embrasures) usually have remarkable symmetry.

Initially, the interdental papilla fills the gingival embrasure. When the form and function of teeth are ideal and optimal oral health is maintained, the interdental papilla may continue in this position throughout life. When the gingival embrasure is filled by the papilla, trapping of food in this region is prevented.

In a faciolingual vertical section, the papilla has a triangular shape between anterior teeth, whereas in posterior teeth, the papilla may be shaped like a mountain range, with facial and lingual peaks and the col (“valley”) lying beneath the contact area. This col, a central faciolingual concave area beneath the contact, is more vulnerable to periodontal disease from incorrect contact and embrasure form because it is covered by nonkeratinized epithelium. The physiologic significance of properly formed and located proximal contacts and associated embrasures cannot be overemphasized; they promote normal healthy interdental papillae filling the interproximal space.

Improper contacts can result in food impaction between teeth, potentially increasing the risk of periodontal disease, caries, and tooth movement. In addition, retention of food is objectionable because of its physical presence and the halitosis that results from food decomposition. Proximal contacts and interdigitation of teeth through occlusal contacts stabilize and maintain the integrity of the dental arches.

The correct relationships of embrasures, cusps to sulci, marginal ridges, and grooves of adjacent and opposing teeth provide for the escape of food from the occlusal surfaces during mastication. When an embrasure is decreased in size or absent, additional stress is created on teeth and the supporting structures during mastication. Embrasures that are too large provide little protection to the supporting structures as food is forced into the interproximal space by an opposing cusp. A prime example is the failure to restore the distal cusp of a mandibular first molar when placing a restoration.

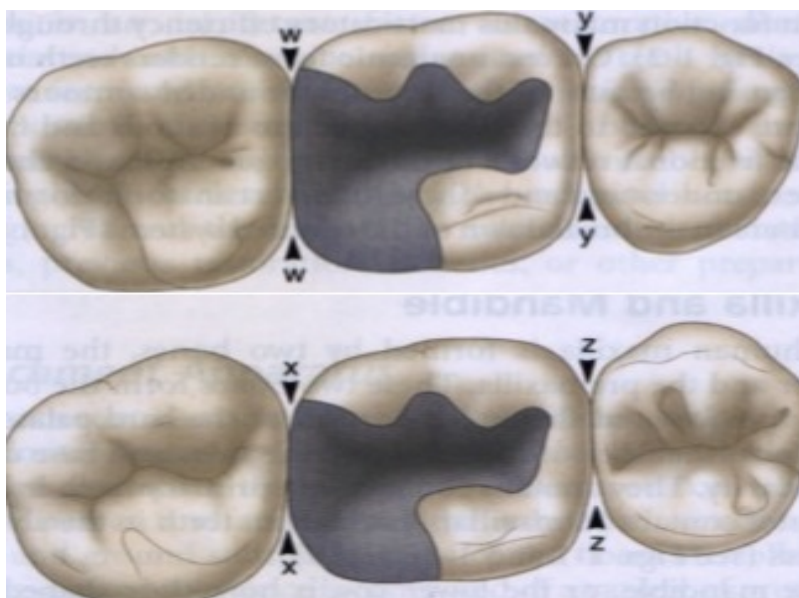


Picture 29. Embrasure form, x, Portion of tooth that offers protection to underlying supporting tissue during mastication, y. Restoration fails to establish adequate contour for good embrasure form

Lingual embrasures are usually larger than facial embrasures and this allows more food to be displaced lingually because the tongue can return the food to the occlusal surface more easily than if the food is displaced facially into the buccal vestibule. The marginal ridges of adjacent posterior teeth should be at the same height to have proper contact and embrasure forms.

When this relationship is absent, it causes an increase in the problems associated with weak proximal contacts and faulty embrasure forms.

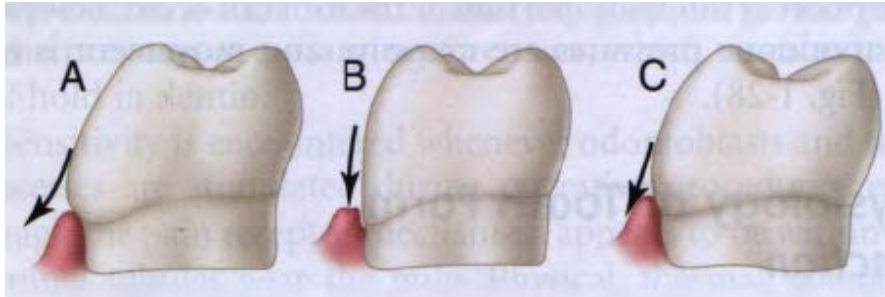
Preservation of the curvatures of opposing cusps and surfaces in function maintains masticatory efficiency throughout life. Correct anatomic form renders teeth more self-cleansing because of the smoothly rounded contours that are more exposed to the cleansing action of foods and fluids and the frictional movement of the tongue, lips, and cheeks. Failure to understand and adhere to correct anatomic form can contribute to the breakdown of the restored system.



Picture 30. Embrasure form, w, improper embrasure form caused by overcontouring of restoration resulting in unhealthy gingiva from lack of stimulation, x, Good embrasure form, y, Frictional wear of contact area has resulted in decrease of embrasure dimension, z, When the embrasure form is good, supporting tissues receive adequate stimulation from foods during mastication

Contours. Facial and lingual surfaces possess a degree of convexity that affords protection and stimulation of supporting tissues during mastication. The convexity generally is located at the cervical third of the crown on the facial surfaces of all teeth and the lingual surfaces of incisors and canines. The lingual surfaces of posterior teeth usually have their height of contour in the middle third of the crown. Normal tooth contours act in deflecting food only to the extent that the passing food stimulates (by gentle massage) and does not irritate supporting tissues. If these curvatures are too great, tissues usually receive inadequate stimulation by the passage of food. Too little contour may result in trauma to the attachment apparatus.

These tooth contours must be considered in the performance of operative dental procedures. Improper location and degree of facial or lingual convexities can result in serious complications, in which the proper facial contour is disregarded in the placement of a cervical restoration on a mandibular molar.



Picture 31. Contours. Arrows show pathways of food passing over facial surface of mandibular molar during mastication.

A, Over-contour deflects food from gingiva and results in under-stimulation of supporting tissues.

B, Under-contour of tooth may result in irritation of soft tissue.

C, Correct contour permits adequate stimulation for supporting tissue, resulting in healthy condition

Over-contouring is the worst offender, usually resulting in increased plaque retention that leads to a chronic inflammatory state of the gingiva. The proper form of the proximal surfaces of teeth is just as important to the maintenance of periodontal tissue as is the proper form of facial and lingual surfaces. The proximal height of contour serves to provide: (1) contacts with the proximal surfaces of adjacent teeth, thus preventing food impaction, (2) adequate embrasure space apical to the contacts for gingival tissue, supporting bone, blood vessels, and nerves that serve the supporting structures.

Question 2. Main and additional methods of diagnosis of carious lesions of Class II.

The main methods of diagnosis:

1. Questioning:

- Complaints.
- Patient history.
- Medical history.

2. Clinical Examination:

- Probing:
- To determine of roughness of enamel surface.
- To determine of dentin density (soft or hard) at a bottom and a walls of caries cavity.
- To determine of sensitivity (a bottom and a walls of caries cavity).
- Drying.

- Reaction on thermal agents (cold, heat).

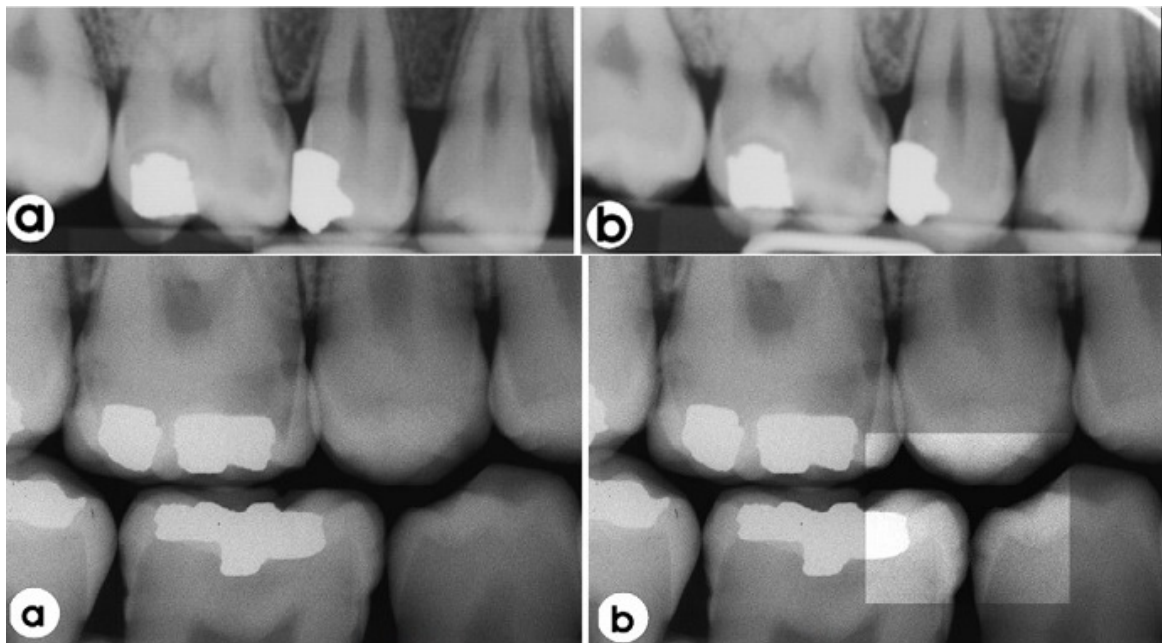
The additional methods of diagnosis of carious lesions of Class II.

- ✓ Radiologic and clinical examination.
- ✓ Emerging diagnostic techniques.
- ✓ Fluorescence.
- ✓ Fiber-optic transillumination (FOTI).
- ✓ Digitally imaging fiber-optic transillumination (DIFOTI).
- ✓ Electrical conductivity.

Radiographic examination is useful to confirm the extent of caries, to detect lesions where visual examination of the tooth surface is hampered and to serve as an aid in making appropriate clinical decisions.

Radiography. The most commonly used radiographic method for detecting caries lesions is the bitewing technique. It is meant to find lesions that are hidden from a clinical visual examination, such as when a lesion is hidden by an adjacent tooth, proximal caries.

Bitewing radiography allows accessibility to surfaces that may not be seen in the clinical visual–tactile examination, and allows the depth of lesions to be assessed. Other advantages are that it is not invasive, and does not damage tooth structure like an incorrectly used dental probe might. Radiographs can also be filed and reexamined at a later date to compare with a more recent image to detect whether a lesion is progressing or not.



Picture 32. Approximal caries on bitewing radiographs.

Digital radiography – which is increasingly replacing bitewing radiography and that is as accurate as film for the detection of caries lesions.

Digital image enhancement – which studies show can provide superior results to radiographs when enhanced correctly but takes a significant amount of technical skill.

Digital subtraction radiography – which is not typically used in a clinical setting, also because of the high level of technical skill needed to perform correctly.

Tuned aperture computed tomography – which shows improved diagnostic accuracy in caries lesion detection, but with equipment that is too expensive for most clinical practices.

Question 3. Principles of preparation of Class II carious lesions.

If the cavity is localized below the equator of the teeth, cavity should be prepared, as a Class 5 cavity (with the possibility of access). Additional box is formed on the vestibular surface.

If the cavity is located on the equator or higher, it is required to prepare the box on the occlusal surface. The bottom of the proximal box is vertically. Gingival wall can be formed not only by direct, but at an acute angle to the bottom of the cavity. This prevents injuries of the gingiva and improves the retention of the filling.

In a place of convergence of the gingival wall and of the cavity bottom the corner slightly rounded in order to avoid stress of hard dental tissues.

The occlusal box has the following parameters:

- ✓ The bottom is perpendicular to the proximal box.
- ✓ The length is $\frac{1}{2}$ - $\frac{1}{3}$ of the mesiodistal size of the teeth
- ✓ The width is equal to the width of the proximal box.
- ✓ The depth is a little deeper enamel-dentine connection.

If there are carious cavities of both interproximal surfaces, it is perhaps to combine of these cavities.

In order to improve the fixation of the seal it is necessary to perform retention points in the dentin in the gingival area.

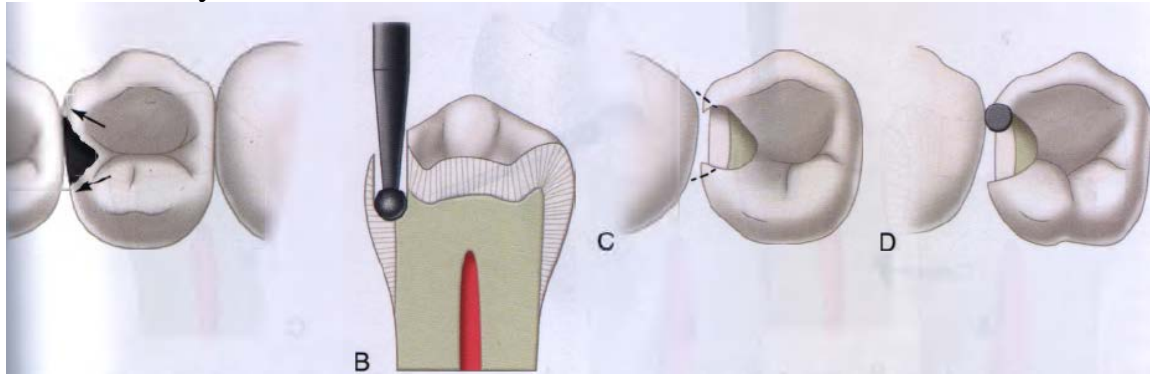
Question 4. Peculiarities of preparation of class II carious lesions for composite filling materials.

The tooth preparation for Class II direct composites involves:

- 1) creating access to the faulty structure,
- 2) removal of faulty structures (caries, defective restoration and base material, if present),
- 3) creating the convenience form for the restoration. Retention, use mechanical retention features in the tooth preparation Class II composite restorations.

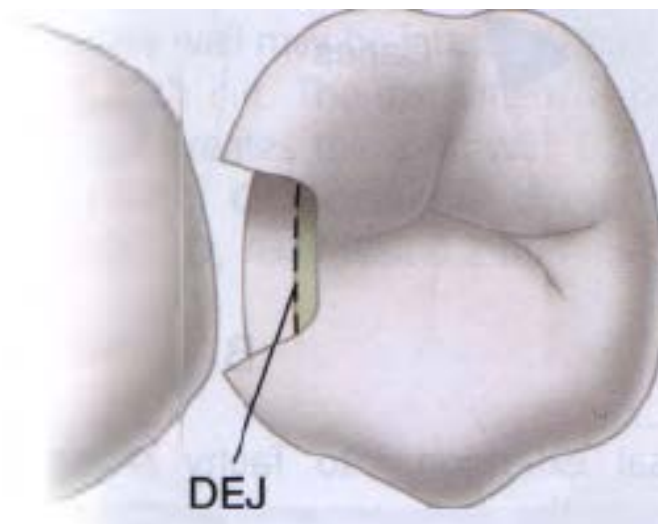
Small Class II direct composite restorations are often on primary caries lesions, that is, initial restorations. A round or elongated pearl diamond or bur with round features may be used for this preparation to scoop out the carious

or faulty material from the occlusal and proximal surfaces. The pulpal and axial depths are dictated only by the depth lesion and are not uniform. The proximal extensions likewise are dictated only by the extent of the lesion but may require the use another instrument with straight sides to prepare walls 90 degrees or greater. The objectives are to remove caries or the defect conservatively and remove friable tooth structure.



Picture 33. *A, Pre-operative visualization of faciolingual proximal box extensions. Arrows indicate desired extensions. B, Round or oval, small elongated pear instrument used. C and D, Facial, lingual, and gingival margins may need undermined cavosurface enamel (endicated by dotted lines) removed with straight-sided thin and flat-tipped rotary instrument or hand instrument*

Another conservative design for small Class II composites is the box only tooth preparation.

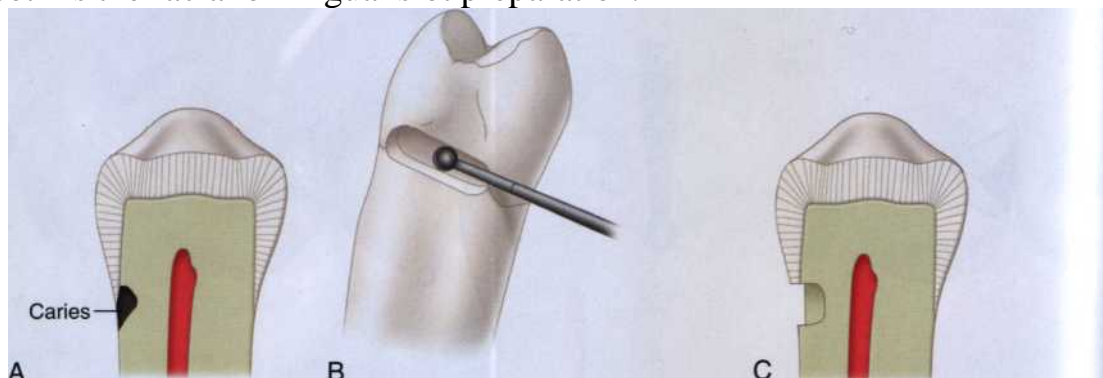


Picture 34. *Box-only Class II composite preparation.*

This design is indicated when only the proximal surface is defective, with no lesions on the occlusal surface. A proximal box is prepared elongated pear or round instrument, held parallel to the long axis of the tooth crown. The instrument is extended through marginal ridge in a gingival direction. The axial depth dictated by the extent of the caries lesion or fault. The form of the box depends on which instrument shape is used the more boxlike with

the elongated pear and the more scooped with the round. The facial, lingual, and gingival extensions are dictated by the defect or caries. No beveling or secondary retention is indicated.

A third conservative design for restoring proximal lesions on posterior teeth is the facial or lingual slot preparation.



Picture 35. Facial or lingual slot preparation.*A, Cervical caries on the proximal surface.*

B, The round diamond or bur enters the tooth from accessible embrasure, oriented to the occlusogingival middle of the lesion. C, Slot preparation

Here, a lesion is detected on the proximal surface, but the operator believes that access to the lesion can be obtained from either a facial direction or a lingual direction, rather than through marginal ridge in a gingival direction. Usually, a small diamond or bur is used to gain access to the lesion. The instrument is oriented at the correct occluso – gingival position, and the entry is made with the instrument adjacent tooth as possible, preserving as much of the facial or lingual surface as possible. The preparation is extended occlusally, facially, and gingivally enough to remove the lesion. The axial depth is determined by the extent of the lesion. The occlusal, facial, and gingival cavosurface margins are 90 degrees or greater. Care should be taken not to undermine the marginal ridge during the preparation.

Moderate to Large Class II Direct Composite Restorations.

The tooth preparation for moderate to large Class II direct composite restorations has features that resemble a more traditional Class II tooth preparation (amalgam) and include an occlusal step and a proximal box.

Occlusal step

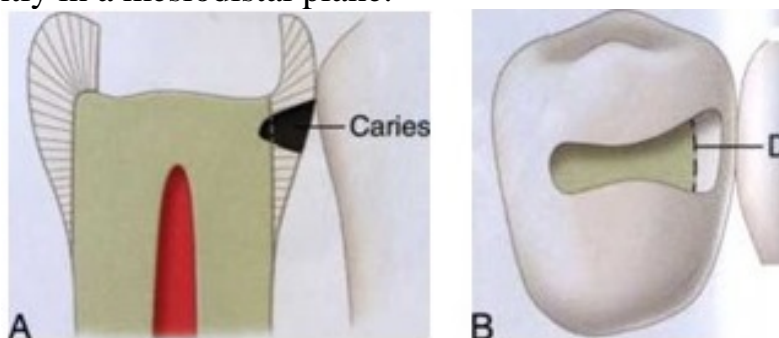
The occlusal portion of the Class II preparation is prepared similarly as for the Class I preparation. The primary differences are related to technique of incorporating the faulty proximal surface. Pre-operatively, the proposed facial and lingual proximal extensions should be visualized.

Initial occlusal extension toward the involved proximal surface should go through the marginal ridge area at initial pulpal floor depth, exposing the

DEJ. The DEJ serves as a guide for preparing the proximal box portion of the preparation.

When only one proximal surfaces is affected, the opposite marginal ridge should be maintained. The pulpal floor is prepared with the instrument to a depth that is approximately 0.2 mm inside the DEJ. The instrument is moved to include caries and all defects facially or lingually or both, as it transverses the central groove. Every effort should be made, however, to keep the faciolingual width of the preparation as narrow as possible. The initial depth is maintained during the mesiodistal movement, but follows the rise and fall of the underlying DEJ.

The pulpal floor is relatively flat in a faciolingual plane but may rise and fall slightly in a mesiodistal plane.



Picture 36. Occlusal extension into faulty proximal surface. A and B, Extension exposes the dentinoenamel junction (DEJ) but does not hit adjacent tooth. Facial and lingual extensions as preoperatively visualized

If caries remains in dentin, it is removed after preparation outline, including the proximal box extensions, has been established.

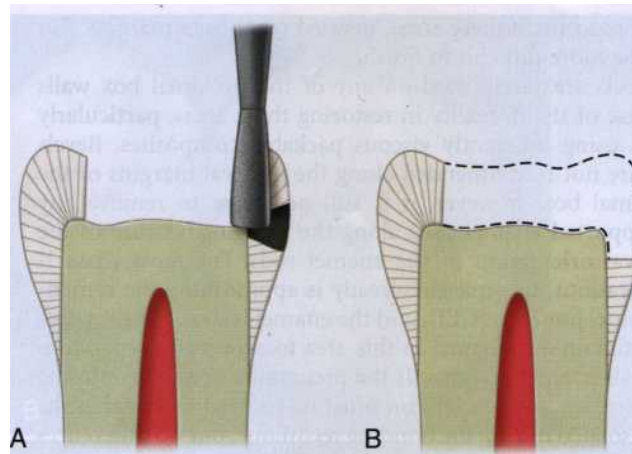
Because the facial and lingual proximal extensions of the faulty proximal surface were visualized preoperatively, the occlusal extension toward that proximal surface begins to widen facially and lingually to begin to outline those extensions as conservatively as possible. Care is taken to preserve cuspal areas as much as possible during these extensions. At the same time, the instrument extends through the marginal ridge to within 0.5 mm of the outer contour of the marginal ridge. This extension exposes the proximal DEJ and protects the adjacent tooth. At this time, the occlusal portion of the preparation is complete except for possible additional pulpal floor caries excavation. The occlusal walls generally converge occlusally because of the inverted s the instrument.

Proximal box

Typically, caries develops on a proximal surface immediately gingival to the proximal contact. The extent of the caries lesions and amount of old restorative material are two factors that dictate the facial, lingual, and gingival extensions of the proximal box of the preparation. Although it is not required to extend the proximal box beyond contact with the a tooth (i.e.,

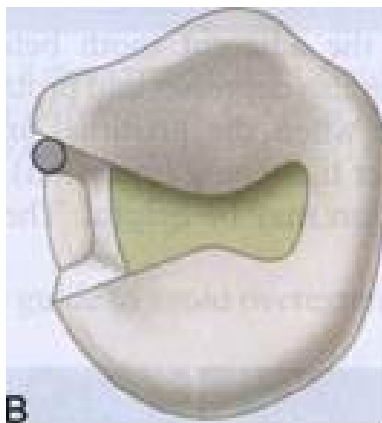
provide clearance with the adjacent tooth), it may simplify the preparation, matrix placement, and contouring procedures. If all of the defect can be removed without extending the proximal preparation beyond the contact, however, the restoration of the proximal contact with the composite is simplified.

Before the instrument is extended through the marginal ridge, the proximal ditch cut is initiated. The operator holds the instrument over the DEJ with the tip of the instrument positioned to create a gingivally directed cut that is 0,2 mm inside the DEJ. For a № 245 instrument with a tip diameter of 0.8 mm, this would require one-fourth of the instrument's tip positioned over the dentin the side of the DEJ (the other three fourths of the tip over the enamel side). The instrument is extended facially, lingually and gingivally to include all of the caries or old material, or both.



Picture 37.

The faciolingual cutting motion follows the DEJ and is usually slightly convex arc outward.

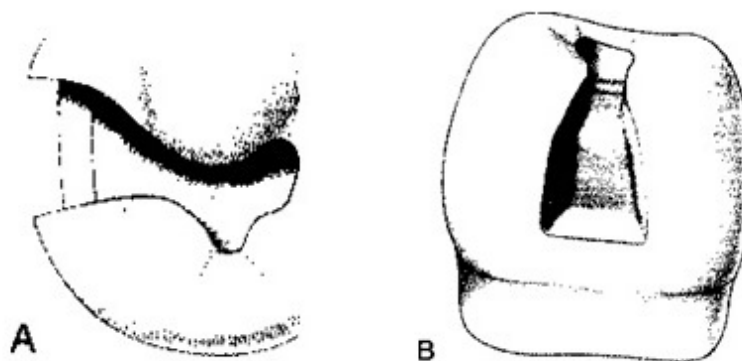


Picture 38. Using a smaller instrument to prepare the cavosurface margin and lingual proximal walls.

During this entire cutting, the instrument is held parallel to the long axis of the tooth crown. The facial and lingual margins are extended as necessary and should result in at least a 90 degree margin, more obtuse being acceptable as well. If the preparation is conservative, a smaller, thinner instrument is used to complete the faciolingual wall formation, avoiding the adjacent tooth. Alternatively, a sharp hand instrument such as a chisel, hatchet, or a gingival margin trimmer can be used to finish the enamel wall. At this point, the remaining proximal enamel that was initially maintained to prevent damage to the adjacent tooth has been removed. The gingival floor is prepared flat (because of the tip instrument) with an approximately 90-degree cavosurface margine. Gingival extension should be as minimal as possible, in an attempt to maintain an enamel margin. The axial wall shuld be 0.2 mm inside the DEJ and have a slight outward convexity. For large caries lesions, additional axial wall caries excavation may be necessary later, during final tooth preparation.

Proximal extension. The enamel margin on the gingival floor is critical for bonding, so it should be preserved, if not compromised. Any remaining infected dentin on the axial wall (or the pulpal floor) is excavated as part of the final tooth preparation.

If no carious dentin or other defect remains, the preparation is considered complete at this time. Because the composite is retained in the preparation by micromechanical retention, no secondary preparation retention features are necessary. No bevels are placed on the occlusal cavosurface margins because these walls already have exposed enamel rod ends because of the enamel rod direction in this area. A bevel placed on an occlusal margin may result in thin composite on the occlusal surface in areas of potentially heavy contact. This feature also could result in fracture or wear of the composite in these areas. Beveled composite margins also may be more difficult to finish.



Picture 39. Final Class II composite tooth preparation.A, Occlusal view.B, Proximal view.

Bevels are rarely used on any of the proximal box walls because of the difficulty in restoring these areas, particularly when using inherently viscous packable composites. Bevels also are not recommended along the gingival margins of the proximal box; however, it is still necessary to remove any unsupported enamel rods along the margins because of the gingival orientation of the enamel rods. For most Class II preparations, this margin already is approaching the cemento-enamel junction (CEJ), and the enamel is thin. Care is taken to maintain any enamel in this area to achieve a preparation with all-enamel margins. If the preparation extends onto the root surface, more attention must be focused on keeping the area isolated during the bonding technique, but no differences in tooth preparation are required. When the gingival floor is on the root surface (no enamel at the cavo- surface margin), the use of a glass ionomer material may decrease microleakage and recurrent caries. Usually, the only remaining final tooth preparation procedure that might be necessary is additional excavation of carious on either the pulpal floor or the axial wall. If necessary, a round bur or appropriate spoon excavator is used to remove any remaining caries.

Question5. The methods of re-establishment of proximal contact point.Restorative Technique. Matrix Application.

One of the most important steps in restoring Class II preparations with direct composites is the selection and proper placement of the matrix. In contrast to amalgam, which can be condensed to improve the proximal contact, Class II composites are almost totally dependent on the contour and position of the matrix for establishing appropriate proximal contacts. Early wedging and re-tightening of the wedge during tooth preparation aid in achieving sufficient separation of teeth to compensate for the thickness of the matrix band. Before placing the composite material, the matrix band must be in absolute contact with (touching) the adjacent contact area.

Generally, the matrix is applied before adhesive placement. An ultra-thin metal matrix band generally is preferred for the restoration of a Class II composite because it is thinner than a typical metal band and can be contoured better than a clear polyester matrix. No significant problems are experienced in placing and light-activating composite material when using a metal matrix as long as small incremental additions (2 mm each or less) are used.

Although a Tofflemire-type matrix can be used for restoring a two-surface tooth preparation, pre-contoured sectional metallic matrices are preferable, because only one thickness of metal matrix material is encountered instead of two, making contact generation easier.

These sectional matrices are relatively easy to use, very thin, and come in different sizes that can be used according to the clinical situation. There

are several systems available, and selection is based on operator preference. These systems may use a bitine ring:

- 1) aid in stabilizing the matrix band.
- 2) provide additional tooth separation while the composite is inserted.

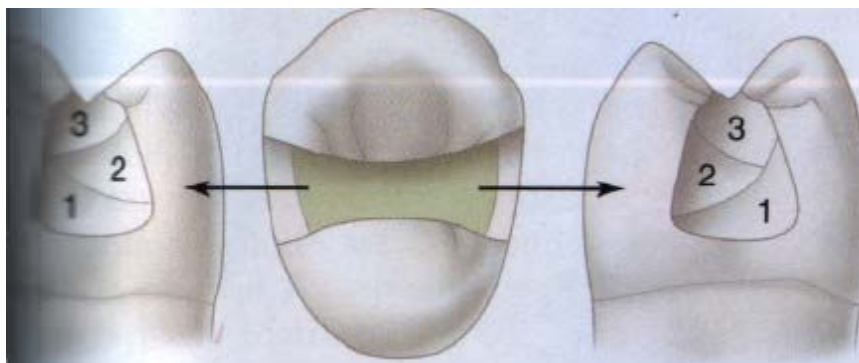
The primary benefit of these systems is a simpler method for establishing an appropriate composite proximal contour and contact. Use of these systems for restoring wide faciolingual proximal preparations requires careful application; otherwise, the bitine ring prongs may cause deformation of the band, resulting in a poor restoration contour.

When both proximal surfaces are involved, a Tofflemire retainer with an ultra-thin (0.001 inch), burnishable band is used. The band is contoured, positioned, wedged, shaped, as needed, for proper proximal contacts and embrasures. Before placement, the metal matrix band for po: composites should be burnished on a paper pad to proper proximal contour to the band (the same as a mfl for amalgam). Alternatively, an ultra-thin precontoured metal matrix band may be used in the Tofflemire retainer.

If the Tofflemire matrix band is open excessively along lingual margins of the preparation (usually because of the contour of the tooth), a “tinner’s joint” can be used to close the matrix band. This joint is made by grasping the lingual portion of the matrix band with No. 110 pliers and the band tightly together above the height of contour of the tooth.

Insertion and Light-Activation of the Composite. Hand instruments or a «composite gun» may be used to insert the composite material. It is important to place (and light-activate) the composite incrementally to maximize the curing potential and to reduce negative effects of polymerization shrinkage. There are many techniques for the restoration of the proximal box. Research comparing different insertion and light- activation techniques is not conclusive, and no single technique has been universally accepted. The number of increments depend on the size of the proximal box.

Oblique incremental technique: the first increment(s) should be placed along the gingival floor and should extend slightly up the facial (or lingual) wall.



Picture 40. Directions of insertions of composite material

This increment (or increments for large box) should be only approximately 1 to 2 mm thick because it is the farthest increment from the curing light and most critical in establishing a proper gingival seal. A second increment is then placed against the lingual (or facial) wall, to restore about two thirds of the box. The final increment is then placed to complete the proximal box and develop the marginal ridge. Subsequent additions, if needed, are made and light-activated (usually not exceeding 2 mm in thickness at a time) until the proximal box is fully restored.

Increments should be light-activated for as long as needed, depending on the shade and opacity of the composite used, the distance of the composite from the light tip, and the power of the unit. Regardless of the number of increments needed, when restoring the proximal box, an effort should be made to develop the anatomy of marginal ridge without excessive composite, to reduce the amount of cutting needed during contouring and polishing.

When the proximal box is completed, the occlusal step of the preparation is restored exactly as for the Class I direct composite restoration, that is, using an anatomic layering technique.

The incremental insertion and light-activation technique described provides enhanced control over the application and polymerization of individual increments of composite. The incremental technique also allows for (1) orientation of the polymerization light beam according to the position of each increment of composite, thus enhancing the curing potential; (2) intrinsic restoration characterization with darker or pigmented composites; and (3) sculpture of the restoration occlusal stratum with a more translucent material simulating the natural enamel. Tight proximal contacts can also be better achieved when composite is applied in increments. The matrix can be held in close contact with the adjacent proximal surface while the contact-related increment of composite is light-activated. A hand instrument with a large surface area (e.g., a small football-shaped or round-shaped burnisher) is well suited for that purpose. Once this increment is cured, the proximal contact is established, and remaining increments can be inserted and light-activated. The matrix is removed, and the restoration is cured from the facial and lingual directions. The restoration can be contoured and finished immediately after the last increment is cured.

When a stiffer or packable composite is used for the restoration of the proximal box, a very small increment of a flowable composite first in the proximal box can be used to improve marginal adaptation of the restoration.

Contouring and Polishing of the Composite. Contouring can be initiated immediately after the composite material has been fully polymerized. If the occlusal anatomy was developed as described in the previous sections, the need for additional contouring is greatly minimized. If contouring is needed, the occlusal surface is shaped with a round or oval, 12-bladed carbide finishing bur or finishing diamond. Excess composite is

removed at the proximal margins and embrasures with a flame-shaped, 12-bladed carbide finishing bur or finishing diamond and abrasive discs. Narrow fin- strips may be used to smooth the gingival proximal surface. Care must be exercised in maintaining the position of the finishing strips gingival to the proximal contact area to avoid inadvertent opening of the contact. The occlusion is evaluated for proper contact. Further adjustments are made if needed, and the restorations are finished with appropriate polishing points, cups, brushes, or discs.

Technique for Extensive II Direct Composite restorations

Direct composite is not usually indicated for extensive posterior restorations but may be indicated when economic and others factors prevent the patient from selecting a more expensive indirect restorations.

The primary differences for very large preparations includes the following:

- 1) some or all of the cusps may be capped;
- 2) extensions in most directions are greater,
- 3) secondary retention features are used;
- 4) more resistance form features are used.

A cusp must be capped if the operator believes it is likely to fracture if left in a weakened state. Cupping a cusp usually is indicated when the occlusal outline form extends more than two thirds the distance from a primary groove to a cusp tip. An operator sometimes may choose to ignore this general rule when using a bonded restoration if cusp will be capped as part of the preparation the subsequent indirect restoration.

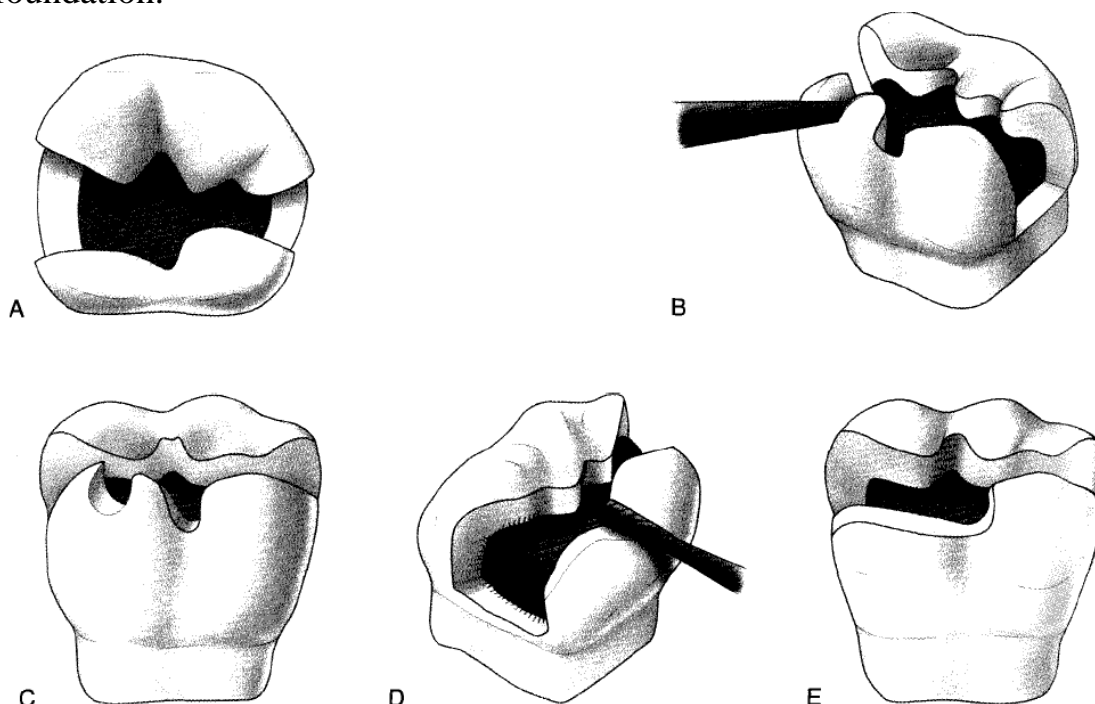
If the tooth has had endodontic treatment, the pulp chamber can be opened, and extensions can be made several millimeters into each treated canal. Because of the increased surface area for bonding and the mechanical retention from extensions into the canals, usually fewer secondary retention features are incorporated into the remaining tooth preparation.

Tooth Preparation. The elongated pear diamond or bur is used to prepare the occlusal step. As already indicated, the occlusal outline form is usually extensive. When moving the instrument from the central groove area toward a cuspal prominence, the pulpal depth that is approximately 0.2 mm inside the DEJ should be maintained, if possible. This creates a pulpal floor which rises occlusally as it is extended either facially or lingually. If a cusp must be capped, the side of the rotary instrument can be used first to make several depth cuts in the remaining cuspal form to serve as a guide for cusp reduction. Cusps should be capped as early in the tooth preparation procedure as possible, providing more access and visibility for the preparation. The depth cut is made with the instrument held parallel to the cuspal incline (from cusp tip to central groove) and approximately 1.5 to 2 mm deep. For a large cusp, multiple depth cuts can be made. Then, the instrument is used to join the depth cuts and extend to the remainder of the cuspal form. The reduced

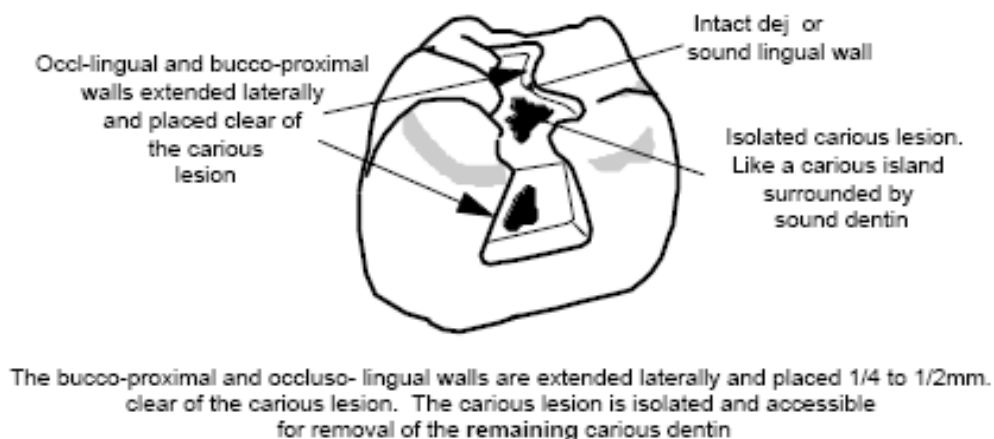
cusps have a relatively flat surface that may rise and fall with the normal mesial and distal inclines of the cusps. It also should provide enough clearance with the opposing tooth to result in approximately 1.5 to 2 mm of composite material to restore form and function. The cusp reduction should be blended in with the rest of the occlusal step portion of the preparation.

The proximal boxes are prepared as described previously. The primary difference is that they may be much larger, that is, more extension in every direction. The extent of the lesion may dictate that a proximal box extend around the line angle of the tooth to include caries or faulty facial or lingual tooth structure. When the outline form has been established (the margins extended to sound tooth structure), caries at the pulpal and axial walls is excavated and the preparation is assessed carefully for additional retention form needs.

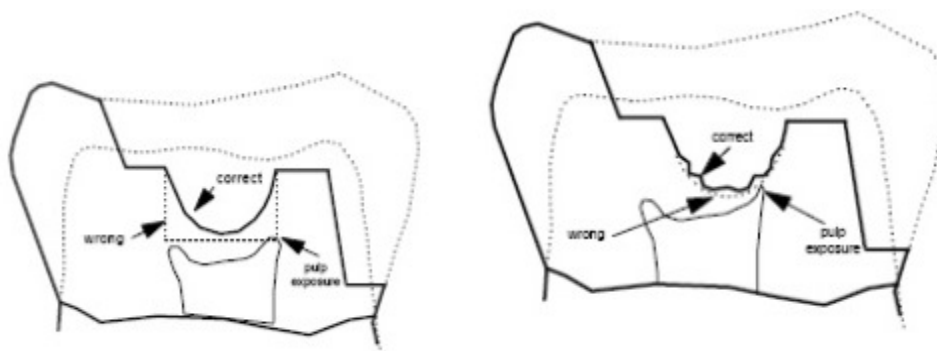
Retention form can be enhanced by the placement of grooves, locks, coves, or slots. All such retention form features must be placed entirely in dentin, not undermining and weakening any adjacent enamel. At times, bevels may be placed on available enamel margins to enhance retention form, even on occlusal areas. Retention form for foundations must be placed far enough inside the DEJ (at least 1 mm) to remain after the crown preparation is done subsequently. Otherwise, the potential retentiveness may be lost for the foundation.



Picture 41. Cusp reduction.*A, The initial outline form weakens the mesiolingual cusp enough to necessitate capping. B, Depth cuts made. C, cuts. D, Cusp reduction prepared. E, Vertical wall maintained between reduced and unreduced cusps.*



Picture 42.



Picture 43. Correct and incorrect variants of preparations of cavity bottom.

Restorative Technique. Matrix Application. Matrix placement is more demanding for these large restorations because more tooth structure is missing, and more margins may be subgingival. Proper burnishing of the matrix band to achieve appropriate axial contours is important, unless immediate full coverage of the tooth is planned. It also may be necessary to modify the matrix band to provide more subgingival extension in some areas and prevent extrusion of the composite from the matrix band-retainer tooth junction.

Insertion and Light-Activation of the Composite. When a light-activated composite is used, first it is placed in 1- to 2-mm increments into the most gingival areas of the proximal boxes. Each increment is cured, as directed. It may be helpful to use a hand instrument to hold the matrix against the adjacent tooth while light-activating the composite. This may assist in restoring the proximal contact.

Self-activated and dual-activated composite resin materials are frequently used for large composite foundations because these can be injected in the preparation in a single increment. However, it is recommended that even when dual-cured composites are used, they be carefully light-activated

during after the final placement, as needed. When this technique used, the operator should carefully select the adhesive system, as some simplified adhesives have been shown to be incompatible with some self-activated composite foundation materials. Acidic monomers in these adhesives scavenge the activators (tertiary amines) in the self-cure composite. If the activator does not function properly, the composite at the adhesive interface does not polymerize thoroughly and does not bond to the adhesive. Some manufacturers have introduced op chemical catalysts that can be mixed with the light adhesive to reduce or prevent this problem.

Question 6. Matrix system for re-establishment of proximal contact point.



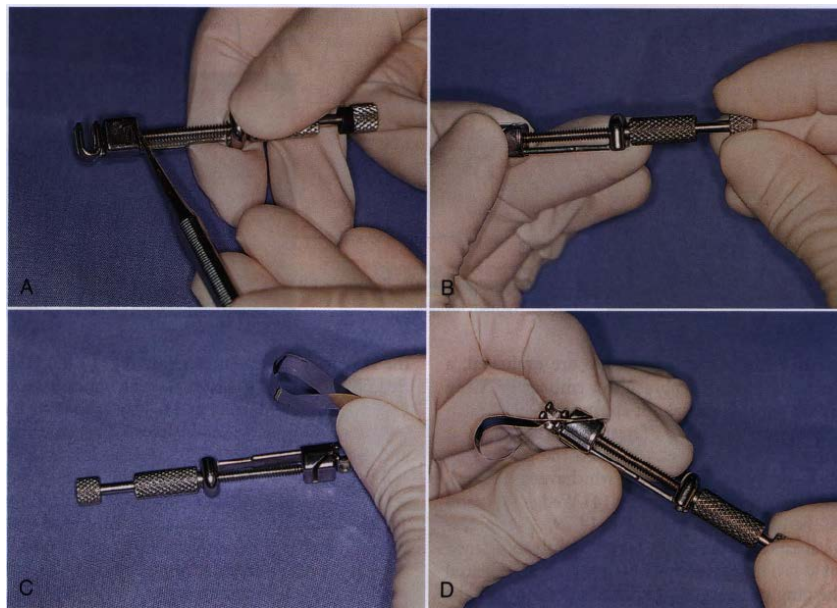
Picture 44. Straight and contra-angled Universal (Tofflemire) retainers. Bands with varying occlusogingival measurements are available.



Picture 45. Lingual positioning requires a contra-angled Universal retainer.



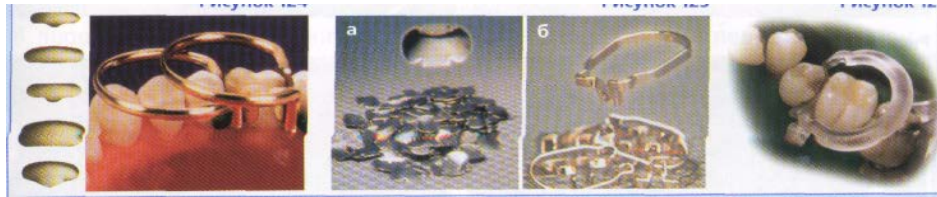
Picture 46. Precontoured bands for a Universal retainer



Picture 47.



Picture 48. Contoured sectional matrices are ideal for posterior composite.



Picture 49. Hawe Adapt Sectional Matrix System (KerrHawe), Palodent (Dentsply).



Picture 50. Composite placement instruments with blunt cone-shaped ends are useful for placing increments of composite resin.



Picture 51. A large spoon makes a good contact-forming instrument.

Test tasks

1. Locations of lesions of Class II include:

- a. Occlusal surfaces of molars and premolars;
- b. *Proximal surfaces of molars and premolars;*
- c. Cutting edge;
- d. Lingual surface of anterior teeth

2. The role of contact proximal point:

- a. Protect the papilla and periodontal tissues of the damage;

- b. Promotes the proper distribution of occlusal pressure;
 - c. Preserves the integrity of dentition;
 - d. *All of the above.*
- 3. The most informative for the diagnosis of proximal caries are:**
- a. *X-ray diagnosis*;
 - b. Electrotest;
 - c. *Transillumination*;
 - d. Dye method.
- 4. Name a general rule of tooth preparation for direct posterior composite restoration:**
- a. creating access to the faulty structure
 - b. removal of faulty structures (caries, defective restoration and base material, if present)
 - c. creating the convenience form for the restoration.
 - d. *All of the above.*
- 5. What are criteria of final class II composite tooth preparation?**
- a. The axial wall has a slight outward convexity;
 - b. The gingival floor is prepared flat with approximately 90-degree cavosurface margin;
 - c. The lingual and facial walls diverge to the proximal surface;
 - d. The lingual and facial walls converge slightly to the occlusal surface;
 - e. The lingual and facial margins are extended to 90-degree margin or more obtuse.
 - f. *All of the above.*
- 6. Is indicated beveling or secondary retention when preparation for small Class II Direct Composite Restoration?**
- a. *no*
 - b. yes.
- 7. Name the primary differences of preparation technique for Extensive class II direct composite restoration:**
- a. some or all of the cusps may be capped;
 - b. extensions in most directions are greater,
 - c. secondary retention features are used;
 - d. more resistance form features are used;
 - e. *All of the above.*

8. What matrixe system are preferable for re-establishment of proximal contact point.

- a. *System of precontoured sectional metallic matrize;*
- b. Tofflemire matrixe system;
- c. Universal retainer;

9. What technique of insertion of composite provides control over the polymerization of composite?

- a. *Incremental technique*
- b. Technique of tunnel tooth preparation
- c. Technique of insertion in single increment.

10. What are in the gingival embrasure?

- a. *Interdental papilla*
- b. gingival sulcus
- c. trapped food
- d. cement.

LESSON 13. DIAGNOSTICS AND TREATMENT OF CERVICAL CARIES.

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

1. Cervical caries, definition.
2. Etiology of cervical caries.
3. Symptoms of cervical caries.
4. Diagnosis of cervical caries.
5. Treatment of cervical caries.
6. Prevention of cervical caries.

Question1.Cervical caries, definition.

Cervical caries is a special type of tooth decay which is characterized by destruction of teeth tissue at the cervical margin of the tooth. This disease progresses very rapidly permeating into the dental tissues and affecting the nerve canals. It is very difficult to detect this dental caries type at early stages and hard to manage it at more advanced stages. Frequently cervical caries affects people over thirty years of age but it is often observed in children.

There are several stages of cervical caries development in children and adults. They are: a demineralized spot lesion, superficial caries, median and deep stages of dental caries. Cervical caries can be accompanied by different disorders of the thyroid, diabetes mellitus in particular. That is why it is necessary to undergo endocrinological examination along with cervical caries

management. This disease often provokes the onset of circular caries in children. It can be accompanied by baby bottle tooth decay.

Most commonly they are seen in mandibular molars, followed by premolars, canines and incisors. This order is reversed in the maxilla. The buccal and interproximal surfaces are more susceptible than the palatal or lingual surfaces.

Cervical caries can occur in the areas of abrasion, erosion, and abfraction, or as primary root caries and recurrent decay.

Question2. Etiology of cervical caries.

All etiological factors of cervical caries are divided into two parts: intraoral and extraoral.

Intraoral:

- -Xerostomia.
- -Low salivary buffer capacity.
- -Poor oral hygiene.
- -Periodontal disease and periodontal surgery.
- -Gingival recession.
- -Frequency of carbohydrate intake.
- -Unrestored and restored coronal and root caries.
- -Overdenture abutments and removable partial dentures.
- -Malocclusion.
- -Abfraction lesions.
- -Tipped teeth which make areas of teeth inaccessible for cleaning.

Extraoral:

- -Advanced age.
- -Medications that decrease the salivary flow.
- -Antipsychotics, sedatives, barbiturates, and antihistamines.
- -Diabetes, autoimmune disorders (e.g. Sjogren's syndrome).
- -Radiation therapy.
- -Gender-males are affected more than females.
- -Lower educational and socioeconomic levels.
- -Physical disability where patients have limited manual dexterity for cleaning of teeth.
- -Limited exposure to fluoridated water.
- -Consumption of alcohol or narcotics.

Next, consider the most significant of these factors.

Continuous multiplication of microorganisms which are present in dental plaque and dental calculus is the principal cause of cervical caries development. The layer of tooth enamel is thinner in cervical regions and it is more difficult to brush the bacteria away from these zones. Hence, dental plaque accumulates very quickly. Some patients are predisposed to gingival layering which can result in cervical caries. Food gets into periodontal

pockets and a great amount of pathogenic bacteria accumulate in these regions. These bacteria produce lactic acid which demineralizes both the enamel and the crown. Thus, the main causes of cervical caries are insufficient hygiene care and anatomic features of the patient's oral cavity.

In recent years, increased attention has been placed on the role of carbonated beverages, sports drinks, and their high sugar content in their combined chemical erosive effect on dentin. Patients who drink 4 to 6 bottles of a carbonated beverage per day, combined with poor oral hygiene, have a high risk of cervical carious lesions.

Besides that, adolescents undergoing orthodontic treatment are at risk for cervical carious lesions.

Xerostomia is also etiologic factor of cervical caries. The presence of xerostomia has been on the increase. Combined with gingival recession and exposed root surfaces, with xerostomia, teeth are at a greater risk of Class V carious lesions. Currently more than 400 medications can cause dry mouth. These medications include antihypertensives, antidepressants, analgesics, tranquilizers, diuretics, and antihistamines. Patients undergoing cancer therapy are susceptible to xerostomia. Chemotherapeutic medications can affect both the flow and composition of saliva. Also, head and neck radiation can temporarily or permanently damage the salivary glands. After radiation, the protective ability of the saliva is also impacted by a decrease in the immunoglobulin in the saliva.

Other conditions can also cause a decrease in salivary flow. Patients with endocrine disorders, depression, anxiety and stress, and nutritional deficiencies may exhibit symptoms of dry mouth. Sjorgren's syndrome, an autoimmune disease, causes both dry mouth and dry eyes. Trauma to the head and neck area due to accidents or surgery can cause nerve damage that affects salivary flow.

Question 3.Symptoms of cervical caries.

Cervical caries occurs on the gingival line. Classical symptoms like hypersensitivity to hot, cold and sweet are not observed at the initial stages of the disease progression. The signs of the disease can be noticed in external changes: that is the enamel darkens and white spots emerge. When the disease progresses painful reactions to temperature drops as well as to sour and sweet products may become frequent. On more advanced stages even tooth exposure to cold air may cause intolerable pain. Painful feelings while biting and chewing can be experienced. This disease is dangerous because of its rapid progression to the root of the tooth. Moreover, the affected tooth acquires a bad esthetic appearance. The disease may cause such complications as pulpitis, gingivitis and periodontitis. Cervical caries is transmitted from one tooth to another that is why treatment is obligatory. Moreover, advanced stages of cervical caries are accompanied by parulides,

abscesses and phlegmon occurrence. These complications are life-threatening and the patient needs immediate hospitalization.

Question4.Diagnosis of cervical caries.

To diagnose cervical caries first of all doctor have to perform clinical examination is the. Tooth surface should be cleaned before examination since plaque covering the lesion can lead to misdiagnosis.

Often **patient's complaints** are short-term pain from hot, cold and sweet, presence of carious cavity, discomfort during teeth brushing, esthetic defect and others.

Objective clinical examination. Visual-tactile method of diagnosis.

Visual examination for diagnosing dental caries is a very popular method. It is based on the criteria such as cavitation, surface roughness, opacification and discoloration of clean and dried teeth under adequate light source.

Advantage:

- Preferred over probing because of harmful effects of probing.

Disadvantages:

- -Visual examination by a skilled clinician, in some cases, can be successful, but oftentimes, a large percentage of the occurrence of decay is too small to generate a distinctive visual signature for proper detection of caries even in advanced stages.
- -Discoloration which is found in normal healthy teeth, can be mistaken for the presence of caries.
- It is difficult to assess the level of caries penetration.

Tactile examination is the diagnostic method when the examiner detects softened tooth structure with dental probe. Since demineralization is a process that does not always involve sufficient softening of the enamel to be detectable by an explorer. When an explorer sticks, it's usually a good indication that there is decay beneath; however, when it does not stick, it does not necessarily mean that decay is not present. During the past 10 years the role of probing s in caries detection has become a controversial issue. Sensitivity of these method is 62%, specificity – 84%.

Disadvantages of probing:

- It can produce traumatic defects in lesions arrested by plaque control alone.
- Does not improve accuracy of diagnosis.
- Inter operative variables.

During visual-tactile examination at the tooth cervical region we can see irregular or round or oval in shape lesion (spot or cavity) which often spread radially. The type of lesion depends on stage and disease activity.

Initial root caries:

- -White at first then may become light brown to yellow.
- Shallow, spreads laterally.

- Without patient symptoms.
- Hard on probing with moderate pressure.

Active, progressing root caries:

- Yellowish, light brown.
- Its covered by visible plaque.
- Soft or leathery on probing with light pressure.
- Its primarily detected by the presence of softness and cavitation.

Inactive root caries:

- Well-defined.
- Dark brownish or black in color.
- May be rough or smooth shiny surface but its cleanable.
- Usually not covered with plaque.
- Its seen in patients (usually older) whose oral hygiene and diet in recent years are good. Where discoloration of such areas is common and usually is associated with remineralization.

Despite the subjectivity inherent in interpreting the clinical signs of cervical caries diagnosis, good to excellent inter-examiner reliability has been reported in clinical studies. Clinical disagreement among different examiners can be attributed to several factors. Variation in an examiner's visual acuity (for example, presbyopia, color blindness) can obviously affect the interpretation of the presence or absence of cavitation and/or a color change on the root surface. Even more critical, however, is that there is frequently disagreement among examiners concerning the relative softness or hardness of the area examined due to differences in interpreting tactile sensitivity. There are no in vivo studies reported in the literature that compare clinical diagnosis with a histological assessment of the lesion. Although this is disappointing, it is not surprising because of the difficulty of conducting studies where teeth are removed and histologically examined following clinical examination.

Diagnostic tests for cervical caries. Clinicians look to diagnostic tests in the hope that they will be more reliable than clinical diagnosis and, therefore, can be used to replace clinical diagnosis. Selecting the most appropriate diagnostic test is therefore a complex matter that must take into account test characteristics, prevalence of the disorder, and the purpose of applying the test. For screening purposes, a highly sensitive test is generally preferred so that the number of false negative test results when the disorder is actually present is minimized. To assist with diagnosis, a highly specific test is preferred so that there will be few false positive test readings in the absence of disease. Test sensitivity and specificity, however, are uncalibrated measures of test performance.

Various tests have been used for the diagnosis of cervical caries. For the proximal surfaces, radiography produces good results, but the supporting evidence is weak. For cervical caries diagnosing we can use conventional or

digital or bitewing radiography. The last are performed when carious cavity is localized at the tooth contact surfaces.

Accurate radiographs can help in diagnosis but they should be free from overlapping or burnout. Radiography taken on one occasion is unable to distinguish an actively progressing from a passive lesion, or a cavitated from a noncavitated surface. Deep dentinal lesions that are visible on a radiograph are more likely to be cavitated. Demineralized, noncavitated lesions may be arrested, but the main body of the demineralized, dentin usually remains radiolucent.

A considerable loss of mineral content is mandatory before lesion becomes visible on radiograph. The actual depth of lesion is always deeper than on radiograph.

Grading:

- Grade I – Incipient,
- II – Shallow, less than 0.5 mm,
- III – Deep, pulpally involved.

Limitations of radiographs:

- Difficult to diagnose between residual and secondary caries.
- Cannot be visualized unless it reaches an additional stage.
- Cannot differentiate between activity of lesion.
- Marginal failure to be distinguished from secondary caries.

Dye penetration method. Special dyes can be useful for detecting root caries, these dyes stain the infected dentine and thus allow the clinician to detect caries.

There are two layers of decalcification can be identified in carious dentin: one layer which is soft and cannot be remineralized, a second layer, which is hard with intermediate calcification and can be remineralized. It is now clearly established that these dyes do not stain bacteria but instead stain the organic matrix of less mineralized dentin. This make them less specific because dyes do not stain bacteria nor delineate (trace an outline) the bacterial front but stain collagen associated with less mineralized organic matrix. Use of basic fuchsin in propylene glycol for the diagnosis and treatment of carious dentin has been given by Fusayama, 1980. This dye was found to be carcinogenic. To overcome this disadvantage, methylene blue was used, but methylene blue is slightly toxic.

Dye-penetration methods using nowadays

a) For enamel caries:

- Calcein: Complexes with calcium
- Fluorescent Dye: a) Brilliant blue b) ZygtoZX – 22.

Disadvantage is irreversible as dye reacts with nitrogen and hydroxyl groups of enamel.

b) For dentin caries:

- 0.5% basic fuchsin in propylene glycol,

- 1% acid red in propylene glycol.

It is known that the microorganisms most commonly associated with cervical caries are *Actinomyces viscosus/naeslundii*, *Streptococcus mutans*, *Lactobacilli*, and *Candida* species. That is why the tests determining the presence or absence of mutans streptococci and *Lactobacilli* are the known to be clinically helpful in diagnosing of cervical caries.

Fluorogenic enzyme assay estimates bacterial counts, particularly mutans streptococci and *Lactobacilli*, in plaque overlying root caries and, therefore, supports the evidence for mutans streptococci and *Lactobacilli* diagnostic tests.

Due to cervical caries is often connected with xerostomia for diagnosing we can also use such laboratory tests as salivary secretion rate test and salivary buffer effect test.

Question 5. Treatment of cervical caries.

Treatment plan for cervical caries depends on the following factors:

- Clinical examination.
- Size of the lesion.
- Type, extent, and site of the lesion.
- Esthetic requirements.
- Physical and mental condition of the patient.

Root caries lesions are difficult to restore because of their location, which is near gingival margin or subgingival. For proper restoration, sufficient access and isolation are needed.

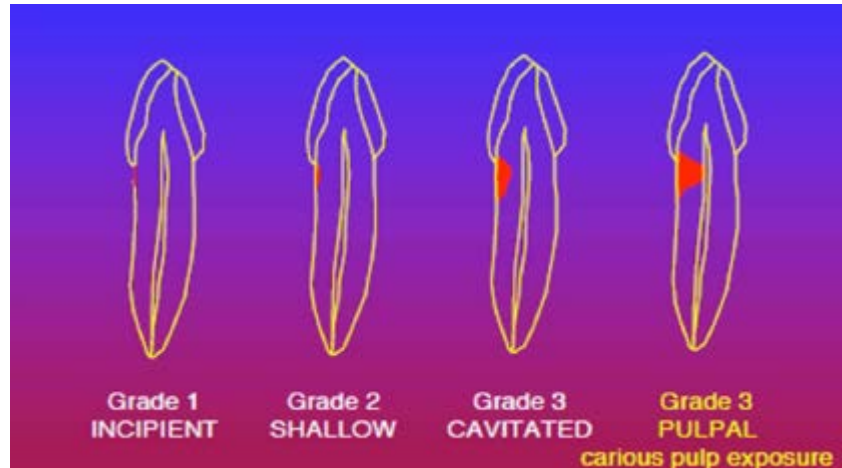
Proper access and isolation to treat root caries are very important, and ideally involve use of a rubber dam if the lesion is supragingival. To begin with it, root surface is cleaned to remove the plaque.

Then the excavation of carious tooth tissue is done and restoration walls are prepared. The margins and retention design depends on the restorative material used. For example:

- ✓ When a tooth is to be restored with amalgam, retention grooves are required occlusally and gingivally.
- ✓ For composites, beveling of the coronal margins of the preparation is required.
- ✓ If the location of the lesion is near the gingival margin or is subgingival cotton rolls and retraction cords can be used.
- ✓ If the lesion extends subgingivally and cannot be completely observed, even with the use of a retraction cord, a releasing incision may be required for completing the restorative procedure (Periodontal surgery).

There is a protocol for treatment of cervical caries that had putted by Billings in 1985 called (Index of Billings for root caries severity treatment) as following (Pic.):

- I. Grade 1: Incipient; no surface defect; need remineralizing therapy.
- II. Grade 2: Shallow; surface defect <0.5mm; need recontouring.
- III. Grade 3: Cavitation; surface defect >0.5mm; need filling.
- IV. Grade 4: Pulpal carious pulp exposure; need endodontic treatment + filling.



Picture 52. Index of Billings for root caries severity treatment.

There are different types of filling materials using for cervical caries treatment.

Amalgam. Properties:

- Easy to manipulate.
- Can be used in areas which are difficult to isolate.
- The margins are self-sealing.
- Lacks aesthetic appearance.
- No therapeutic effect.
- Cannot chemically bond to tooth structure.
- It requires the cutting of healthy tooth structure adjacent to the carious tissue for adequate retention of the restoration.

Traditional glass-ionomers. Properties:

- Biocompatible.
- It has chemical bond to tooth structure.
- Releasing fluoride over extended periods of time.
- Poor aesthetics.
- Excessive wear with time.

Resin-modified glass-ionomers. Properties:

- Biocompatible.
- Bond to tooth.
- Have thermal expansion and contraction characteristics that match tooth structure.

- Fluoride releasing feature; also it can be recharged by uptake of fluoride ions from the oral environment.
- They are aesthetic.
- Less brittle than the traditional glass ionomer.

Resin composites. Properties:

- Highly aesthetic materials.
- It bond to enamel and dentin.
- Hybrid composites possess improved strength and improved aesthetics compared with traditional resin composites.
- Microfilled composites are recommended for root surface restorations as they have lower elastic modulus than hybrid composites.
- Don't have any anti-cariogenic effect.

Resin composites are technique-sensitive materials and require proper isolation for the clinical success of the restoration.

Polymerization shrinkage associated with the curing of resin composites is another concern, since this can result in discoloration of the resin around the margins and in microleakage that leads to tooth sensitivity and secondary caries.

One of the most frequent clinical problems associated with class-II and class-V cavities in adhesive resin restorations is the weak link of restorative material to root dental structures, when the cervical margin is located below the cementum-enamel junction. In terms of cementum, the tissue-bonding properties have not been adequately elucidated. • It is well known that root surfaces exposed for a long period to the oral environment develop a superficial hypermineralized layer with limited permeability, compared with intact cementum. These surfaces may interfere in the marginal quality of root restorations, especially in elderly population. Very limited information exists on cementum-bonded restorations. Ferrari et al. in 1997 reported that cementum treated with dentine bonding systems is infiltrated by the resin, but the predictability of the bond is unclear.

Furthermore, it is still unclear (whether or not) the problem of bonding to cementum is related to the structure and properties of the tissue or to a limited effectiveness of the adhesive materials at the region.

However, the morphology of the periodontitis-affected cementum surface was highly variable, with islands of dense granular material. Based on these findings, mechanical removal of the superficial layer of the exposed cementum prior to any periodontal regenerative treatment has been advised. This treatment mode may be applied to improve adhesive bonding as well.

Modification of intact cementum surfaces to improve adhesion may include an eproteination step, prior to any adhesive treatment, in order to remove the high organic content and expose the inorganic substrate, like conditioning with aqueous solutions of sodium hypochlorite (NaOCl).

Sandwich technique is another solution to solve adhesion of composite to root surface.

Fluoride-containing resin composites. Properties:

- Fluoride-containing resin composites release only small amounts of fluoride.
- It has little ability to recharge from the oral environment.
- Therefore, they are not recommended for use with high caries-risk patients, but can be used where aesthetics is a concern.

Compomers. Properties:

- They are polyacid-modified resin composites.
- They have possess properties of both glass ionomer and resin composites.
- They leach fluoride, but to a lesser extent than glass ionomer.
- They bond to both enamel and dentin.
- They can be used in low-stress areas where esthetics is a concern.

Table 14

Characteristics of different filling materials for cervical caries treatment

<i>Material</i>	<i>Flouride release</i>	<i>Adhesive</i>	<i>Aesthetics</i>
Amalgam	None	No	Low
Glass ionomer	High	Yes	Moderate
Resin- modified glass ionomer	High	Yes	Moderately.High
Resin composite	None	Yes, with bonding	High
Flouride-containing resin composite	Little	Yes, with bonding	High
Compomer	Moderate	Yes, with bonding	Moderately. High

Treatment of root surface caries should be directed and customized to the individual case by classifying patients in risk groups to achieve maximum results.

The use of resin-modified glass ionomer materials is recommended for these restorations because of their anti-cariogenic properties in patients with a high caries risk.

Question6. Prevention of cervical caries.

1. Proper dental hygiene should be maintained: high-quality tooth brush and tooth paste must be used to clean the teeth. To brush in between the teeth it is necessary to use dental floss. Gingival massage and mouth rinsing with herbal decoctions are recommended to promote enamel regeneration.

2. Well-balanced nutrition is a key factor of dental health. Dental prophylaxis also presupposes regular dental examinations, even when nothing bothers the patient. It is necessary to remember that sweets themselves do not cause caries. It occurs as a result of poor hygiene of the

oral cavity where the microorganisms multiply. That is why parents should monitor the process of their children's teeth brushing.

3. The dentist will perform professional teeth brushing, remove dental calculus, whiten and remineralize tooth enamel.

4. Special attention should be given to root caries-prone patients who are wearing dental prostheses. This can be done by proper management of soft tissues during fixed prosthesis procedures and avoiding the placement of restoration margins apical to the surrounding tissue to avoid plaque accumulation.

5. In patients with low salivary flow, xylitol-containing chewing gum which stimulates salivary flow and decreases plaque formation has shown to decrease the caries.

6. The use of topical fluoride should be advocated because it promotes the remineralization process and reduces the rate of demineralization. There are numerous methods by which fluoride can be supplied:

- ✓ Exposure to fluoride in drinking water results in increasing resistance to root caries.
- ✓ Topical fluoride products are available as 0.05% sodium fluoride rinse, 0.12% chlorhexidine rinse, and as 1.1% neutral sodium fluoride gel in a 5 minutes tray technique, with 4 applications over 2-4 weeks.
- ✓ Other products are dentifrices containing 1100 ppm sodium fluoride.
- ✓ Fluoride chewing gum which is effective especially in patients with low salivary flow.
- ✓ Fluoride-containing varnishes have also been effective against root caries.

Test tasks

1. Cervical caries is often accompanied by:

- a. kidney diseases
- b. liver diseases
- c. *endocrine diseases*
- d. all of the above

2. Most commonly cervical caries are seen on the:

- a. incisors
- b. canines
- c. mandibular molars and premolars
- d. *all of the above*

3. What surfaces are more susceptible to the cervical caries:

- a. palatal or lingual
- b. *buccal and interproximal*
- c. all of the above

4. What etiological factors of cervical caries are intraoral:

- a. xerostomia
- b. low salivary buffer capacity
- c. poor oral hygiene
- d. periodontal disease and periodontal surgery
- e. gingival recession
- f. *all of the above*

5. What etiological factors of cervical caries are extraoral:

- a. medications that decrease the salivary flow
- b. antipsychotics, sedatives, barbiturates, and antihistamines
- c. diabetes, autoimmune disorders (e.g. Sjogren's syndrome)
- d. radiation therapy
- e. *all of the above*

6. Patient's with cervical caries complain on:

- a. prolonged ache from hot, cold and sweet
- b. *short-term pain from hot, cold and sweet*
- c. all of the above

7. What are the types of cervical carious lesions:

- a. initial root caries
- b. active, progressing root caries
- c. inactive root caries
- d. *all of the above*

8. Treatment plan for cervical caries depends on the following factors:

- a. clinical examination.
- b. size of the lesion.
- c. type, extent, and site of the lesion.
- d. esthetic requirements.
- e. *all of the above*

9. What is the protocol for the treatment of cervical caries on the initial stage:

- a. *remineralizing therapy*
- b. recontouring
- c. filling
- d. endodontic treatment + filling.
- e. prosthetics

10. What are the types of filling materials using for cervical caries treatment:

- a. amalgam
- b. *traditional glass-ionomers*
- c. *resin-modified glass-ionomers*
- d. *resin composites*
- e. *compomers*
- f. all of the above

LESSON 14. ERRORS AND COMPLICATIONS IN THE DIAGNOSIS AND TREATMENT OF DENTAL CARIES.

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

1. Errors and complications arising during carious cavity preparation.
2. Errors and complications arising during carious cavity filling.
3. Errors and complications arising after dental caries treatment.
4. Preventive measures in dental caries treatment.

Question 1. Errors and complications arising during carious cavity preparation

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.

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.

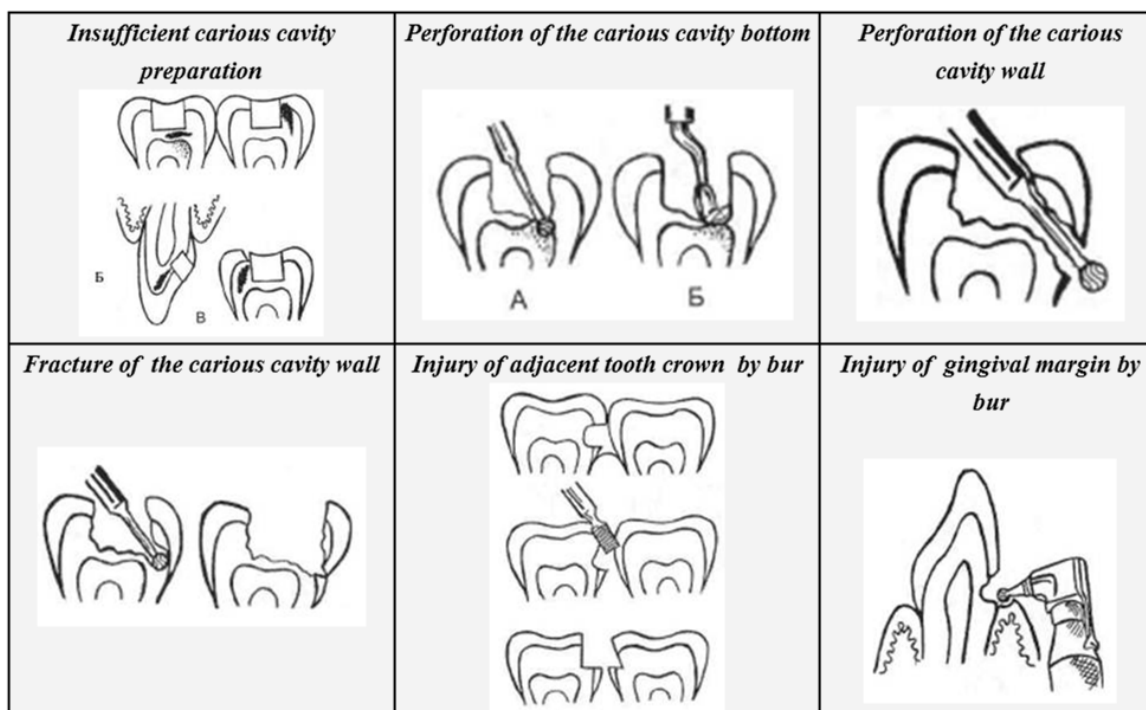
Errors and complications arising during carious cavity preparation

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 clinician 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.

4. Injury of adjacent tooth crown by bur may happen when visible control of operative field is not provided.

5. 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.

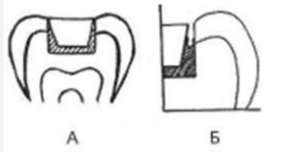
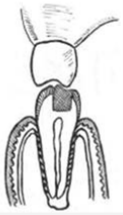
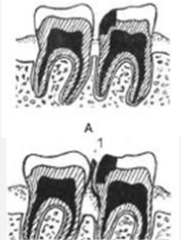




Picture 53. Errors and complications arising during carious cavity preparation

Question 2. Errors and complications arising during carious cavity filling.

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 of intradental papilla, thus causing pain to the patient and development of periodontal diseases. That's why during restoration of II class by Black (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 occlusion 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 (diagnostic feature).

<p><i>Improper choice of filling material and its mixing:</i></p> <p>Improper choice of material leads to cosmetic deficiencies, causing rapid destruction and loss of the filling due to mismatch of the material strength to occlusion pressure.</p>	<p><i>Improper placement of insulating lining</i></p> 	<p><i>Formation of high occlusion</i></p>  <p>pain may come from the periapical periodontal tissues if the restoration is left high so that the tooth is in traumatic occlusion.</p>
<p><i>Absence of a contact point</i></p> 	<p><i>Placement of a single filling in adjacent carious cavities</i></p> 	<p><i>Hanging edges of a filling</i></p> 

Picture 54. Errors and complications arising during carious cavity filling

Question 3. Errors and complications arising after dental caries treatment.

1. Inflammation (necrosis) of the pulp.
2. Secondary caries.
3. Papilitis (inflammation of an intradental papila).
4. Acute or chronic course of an apical periodontitis.
5. Colour change of the tooth crown.
6. Displacement, fracture and loss of filling.
7. Inadequate colour of filling to the colour of tooth enamel.

Question 4. Preventive measures in dental caries treatment.

Preventive measures at the stage of cavity preparation. In preservation dentistry, the preparation of dental hard tissues is based on the removal of carious lesions in dental tissue and on the ensuring of retention of the filling with regard to the prevention of dental caries. Residues of a carious lesion left on the cavity walls and the pulp wall result in secondary or recurrent caries formation with its consequences.

The identification of recurrent caries, particularly in the fillings with a good marginal closure is a diagnostic problem which cannot sometimes be solved even by using dental radiography.

The main aim of dental caries treatment is to preserve healthy dental pulp. The current choice of preparation tools considering effectiveness and the used filling material is a basis for the prevention of pain sensation during the preparation procedure.

When manual preparation instruments are used, the patient feels the instrument's pressure. If low-speed rotating preparation tools are used,

painful sensations are distributed evenly between pressure, temperature and vibration sensations (Tab.). With high-speed rotating tools, the sensation of pain due to pressure and vibrations is decreased but temperature irritation increases. With ultra-high-speed instruments, 80 – 90% of energy is converted into heat.

Table 15

The effect of the instrument's speed on pain sensation

<i>Manual preparation</i>	<i>Pressure</i>	<i>Sensation</i>
Low speed	Pressure	///
	Vibrations	///
	Heat	///
High speed	Pressure	/
Ultra-high speed	Vibrations	/
	Heat	////////

As a result, when the prepared area of the tooth is not cooled properly, thermal damage to odontoblasts may occur together with irreversible changes in the pulp.

Insufficient cooling may occur when the flow of water from the jet does not wash the prepared field due to the shape of the cavity and the angle of a preparation tool.

Unpleasant to painful sensation will arise due to the vibrations of rotating tools which are not centred properly or have been used for a longer period of time. Pain increases particularly in patients with the damaged periodontium.

Pain relief and reduction in the preparation time depend on the effectiveness of the preparation tools used (i.e. on the type, quality and the way of using the tools). When sharp tools are used, approximately one half of kinetic energy delivered to the axis of a rotating tool during drilling and polishing will change into heat. This portion increases with blunt instruments; almost all energy will change into heat when totally blunt tools are used. The effectiveness of drilling and polishing strongly depends on the cross-section of a chip and the cutting speed whose magnitude depends on the tool's diameter and the number of revolutions. An increase in the cutting speed at an ultra-high number of revolutions enables one to make a chip of a smaller diameter, with lower pressure applied on the tool and thus lesser pain during treatment. Reduction in the number of revolutions during preparation depends on the pressure on the tool. The most significant reduction in the number of revolutions is in turbine handpieces with a direct drive where pressure causes the number of revolution to decrease by 25 - 30%. Improper and damaged rotating components result in vibrations and the overheating of the handpiece. This also increases the tool's noise level.

Lege artis treatment is crucial when dental caries is found.

The cavity preparation depends on the localization of dental caries, the extent of the loss of dental hard tissue and the filling material used. Conventional preparation procedures follow basic principles.

The main principle for dental caries treatment is to remove the matter changed by carious lesion. Complications usually occur during the treatment of caries located near the pulp when the dentist failed to remove the carious dentin or when the removal of carious matter was too radical irrespective of the anatomical size of the pulp cavity, increasing the risk of perforating the pulp cavity.

When the principles of preventive extension during cavity preparation is maintained, this will ensure the extension of cavity up to the point of self-cleaning. Microbial plaque accumulates at a higher extent on irregularly positioned teeth. Treatment of dental caries for irregularly positioned teeth depends not only on the extent of loss of dental tissues, but also on a degree of seriousness of a particular irregularity. In a large number of cases, the performance of a conventional treatment is not possible and it is necessary to apply materials with adhesive bonds. At the extensive loss of hard dental tissue, there is a risk of subsequent fracture of a crown. In such cases it is therefore necessary to do prosthetic treatment or extract the abnormally positioned or markedly destructed tooth.

Through their attachment to dental hard tissues (mechanical, chemical), composite materials are able to reduce preparation procedures and save dental hard tissues (preventive fillings), achieving the best possible aesthetic effect at the same time.

The prepared cavity must ensure the sufficient retention of a filling. The preparation depends on the type of the material used (amalgam filling, cast fillings, inlays made of different materials, composite filling materials and glass ionomer cements). Non-compliance with retention principles for individual materials will result either in the release of a filling or failure to achieve its mounting. Fillings are exposed to chewing pressure. The respective kind of the material also specifies the need of sufficient depth of preparation to ensure the resistance of individual filling materials. Shallow prepared cavities fail to provide sufficient firmness for the material and will break upon biting. Damage to the filling may also occur in high fillings during the adjustment of occlusion/articulation relations. The risk of breaking the filling is highest in MOD cavities and in Class IV cavities with the insufficient adjustment of articulation relations. The broken filling may irritate the marginal periodontium causing acute papillitis or periodontal abscess.

The strength of composite materials stems from mechanical binding to the bevelled and etched enamel, and increases with chemical binding to the enamel and dentin depending on the used binding system. When composite

filling materials are used, mechanical and chemical binding shortens the preparation procedure. The depth of the prepared cavity is not governed only by the principle of the filling resistance. The extent of carious lesion and the size of pulp cavity (age factor, anatomical relationship of individual teeth) are crucial factors. When the caries is located near the pulp, it is necessary to prepare in the close vicinity of the top wall of the pulp cavity. Due to the risk of perforation, manual tools are required for performing gentle preparation and completing the preparation.

When the preparation of the cavity is carried out, it is necessary to ensure not only the sufficient stability of the filling against chewing pressure but also the proper resistance of the remaining dental hard tissues. With regard to the resistance of dental hard tissues, it is necessary to comply with the “cusp rule” during the preparation. The weakened cusps fail to provide sufficient support to the remaining dental tissue and the crown or root may fracture upon biting. The fractures expanding below the dentogingival junction and longitudinal fractures of the root are frequently an indication for the subsequent extraction of a tooth. It is therefore necessary to consider the indications for the use of plastic or cast dental fillings. In the case of a large loss of dental hard tissue, it is necessary to apply prosthetic treatment.

The extent of the preparation can be reduced in the case of small caries on the occlusion surfaces of molars with huge cusps. The use of molar composite materials is recommended.

In order to prevent secondary caries, the enamel margin of the cavity, where the amalgam filling will be placed, must be treated properly. The enamel must be underlain with dentin. The use of manual tools is recommended for the adjustment of the enamel.

The main aim of using composite materials is to ensure the greatest possible mechanical retention of a composite. The insufficient angle of the enamel or errors in subsequent phases of the procedure will lead to the failure to close the cavity, resulting in secondary caries.

When caries treatment uses cast dental fillings, the bevelling of enamel's edges is the main condition to ensure the cavity's proper closure.

Preventive measures at the stage of cavity filling

Secondary caries or poor-quality fillings can also occur as a result of the working procedure used to fill the cavity with the filling material.

Among the most common mistakes associated with amalgam fillings is the incorrect selection of matrix and its insufficient fixation in Class II cavities. The injured dentogingival junction is painful and gingival bleeding will occur. Failure to ensure the dry working field makes the *lege artis* completion of the definitive filling impossible.

The cavity has to be treated using a temporary filling and the definitive filling has to be made at a next visit.

Damage to the marginal gingiva will also occur at the improper choice and attachment of sealing wedges in the interdental space. If the matrix is sealed insufficiently, amalgam during condensation will penetrate across the cavity's margin into the interdental space. The removal of non-solidified amalgam from the interdental space using manual tools is insufficient and will always result in injury to the marginal gingiva. It is therefore necessary to make a new filling. Solidified overhanging fillings (of any material) will irritate the marginal gingiva and cause acute and chronic pathological conditions of the periodontium.

When the cavity is being filled, it is necessary to apply the amalgam into the cavity in small portions and allow it to condensate properly. Insufficient condensation is manifested by uneven filling. The risk is greatest at the filling of the gingival ledge in Class II cavities. Such fillings may break upon bite and irritate the marginal periodontium (pain upon occlusion).

One important part of the working stage in the case of cast fillings is that the cast in the cavity will be tested. Thorough examination of the marginal closure of the cavity is the main prerequisite for a proper closure. The presence of a fissure between the filling and the enamel will always necessitate the formation of a new model.

In the case of composite materials, it is necessary to comply with the working procedure for cavity filling. Shrinkage during polymerization arising due to the solidification of materials defines the suitability of using different forms of composite filling materials. Large cavities and destructed crowns are treated using photopolymerizing materials. The quality of the filling made of photopolymerizing composite material is also affected by the quality of the lamp used, the type of material and the binding system used. Failure to comply with the working procedure at each stage of the construction of a filling will result in the poor-quality filling.

Preventive measures at the stage of the final treatment of fillings

The quality of the filling also depends on the filling's final treatment. The correction of occlusion and articulation relations will prevent the elevated bite onto the filling and the overloading of the periodontium. The insufficiently adjusted height of the filling will result in the broken filling and its release from the cavity. High fillings after solidification may cause acute or chronic damage to the periodontium.

With regards to the prevention of secondary caries, the treatment of the filling's surface is important. Amalgam fillings as well as cast dental fillings must be polished thoroughly, particularly at the filling's margins.

There is currently a wide range of different polishing systems ensuring the perfect polishing of all kinds of fillings. The polishing of the solidified filling's surface is also important in composite fillings. After the adjustment of occlusion and articulation, the thorough polishing of the filling's surface is

a prerequisite for good aesthetic appearance. The polished surface of the filling will also ensure less accumulation of microbial plaque.

Test tasks

1. Errors and complications arising during carious cavity preparation:

- a. insufficient carious cavity preparation
- b. perforation of the carious cavity bottom or carious cavity wall
- c. injury of adjacent tooth crown
- d. injury of gingival margin
- e. *all answers are right*

2. Errors and complications arising during carious cavity filling:

- a. injury of gingival margin
- b. absence of a contact point
- c. formation of high occlusion
- d. injury of adjacent tooth crown
- e. *all answers are right*

3. Errors and complications arising after dental caries treatment:

- a. inflammation (necrosis) of the pulp
- b. secondary caries
- c. papilitis (inflammation of an intradental papilla)
- d. acute or chronic course of an apical periodontitis
- e. colour change of the tooth crown
- f. displacement, fracture and loss of filling
- g. *all answers are right*

4. What are the main types of errors and complications during caries treatment:

- a. Errors and complications arising during carious cavity preparation
- b. Errors and complications arising during carious cavity filling
- c. Errors and complications arising after dental caries treatment
- d. *all answers are right*

5. Insufficient carious cavity preparation may lead to:

- a. to secondary caries
- b. development of pulpitis
- c. filling loss
- d. *all answers are right*

6. Injury of gingival margin by bur may happen when:

- a. carious cavity goes deep under the gums
- b. good vision of operative field was not provided

- c. contact point is absent
- d. *all answers are right*

7. In what cavities risk of filling breaking is highest:

- a. in MOD cavities
- b. *in Class IV cavities*
- c. in Class I cavities
- d. all answers are right

8. The most common mistakes associated with amalgam fillings are:

- a. incorrect selection of matrix
- b. insufficient fixation in Class II cavities
- c. secondary caries
- d. *all answers are right*

9. The quality of the filling made of photopolymerizing composite material depends on:

- a. the quality of the lamp used
- b. the type of material
- c. the binding system used
- d. *all answers are right*

LESSON 15. CARIES PREVENTION.

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

1. Types of caries prevention.
2. Methods of caries prevention.
3. Rational feeding.
4. Using of fluorides for caries prevention.
5. Oral hygiene.
6. Fissure sealants.

Question 1. Types of caries prevention.

Primary prevention protects individuals against disease, such as immunisation, and prevention of the initiation of the disease, as in dietary advice and plaque control within dentistry. Primary prevention is aimed at keeping an individual and a population healthy and at minimising the risk of disease or injury. It is this stage that seeks to implement programmes, procedures or measures to prevent a disease, before it actually occurs. Programmes designed to prevent people from starting to use tobacco (primary prevention) or to help them quit if they have already started (secondary prevention) can help prevent oral cancer and periodontal diseases,

and can also be an effective general health promotion strategy. Additionally, plaque control and diet are effective primary prevention methods for both the prevention of dental caries and periodontal disease. Other primary prevention methods include the provision of fissure sealants, water fluoridation and routine dental examinations and diagnostic radiographs.

Secondary prevention aims to limit the progression and effect of a disease at the earliest possible opportunity after onset. It refers to the cessation of the disease process and preventing its progressive activity to more advanced stages, as well as preventing the recurrence of the disease, with further primary prevention interventions and advice. Therefore, to stop disease progression and recurrence, once a condition has been recognised, actions are needed to control and eliminate the further spread of that condition. Removing carious tooth tissue and restoring structure and function at an early stage of the caries process can prevent tooth loss or the need for more extensive treatment. This intervention may be in the form of preventive resin restorations or the placement of more extensive restorations. Secondary prevention measures to diagnose and treat periodontal diseases include periodontal probing and diagnostic radiographs, professional removal of hard and soft deposits, and the local application of antimicrobial agents. Oral examinations of the soft tissues, in addition to obtaining a comprehensive social history to assess past and present tobacco and alcohol use, are also effective measures for detecting oral cancer at its early, most treatable stages.

Tertiary prevention is concerned with limiting the extent of disability once a disease has caused some functional limitation. At this stage, the disease process will have extended to the point where the patient's health status has changed and will not return to the pre-diseased state. Tertiary disease prevention refers to the rehabilitation of an individual and, with respect to oral disease, the reestablishment and maintenance of the integrity of the oral cavity. In the dental caries process, tertiary prevention is aimed not only at restoring carious teeth but also must include further primary and secondary prevention in order to prevent further carious attack. This means that in addition to the placement of a restoration, the causes of caries must also be addressed as part of a clinically effective caries management programme. When considering periodontal disease, periodontitis can be treated by a variety of interventions and surgical procedures or by administering antimicrobial agents either locally or systemically, but again the etiology must be identified.

Question 2. Methods of caries prevention.

All measures on the caries prevention it is possible to divide into state, social, medical, hygienical and educating. The system of state preventives includes measures on the health protection mother and child, guard of environment. Realization of the state system of measures of health protection

population is directed foremost on the antenatal prevention of diseases, on forming and development of healthy child, on support of health of the grown man is basis of prophylactic direction of health protection in our country.

The system of social measures on the caries prevention is related to providing of healthy way of life is observance of the rational mode of labor, rest, scientifically grounded norms of feed, personal hygiene.

To the hygiene measures hygiene education of population from the questions of dentistry, control after the state of environment and feed, belong on the caries prevention.

Educating measures on the caries prevention are taken to diffusing learning about the healthy way of life, inoculating of skills of care of organs of cavity of mouth and support them in the healthy state. These tasks are decided by doctors, middle medical personnel.

Medical measures on the caries prevention are directed for development and introduction etiologic and the nosotropic grounded facilities and methods of influence on an organism and organs of cavity of mouth for the increase of their firmness to the caries, and also on the decline of cariogenicity of unfavorable factors of environment on the cavity of mouth.

Table 16

Teeth caries prevention (Udovits'ka, 1987)

With midications		Without medications	
<i>Exogenous</i>	<i>Endogenous</i>	<i>Exogenous</i>	<i>Endogenous</i>
Rational Hygiene	Strengthening somatic health	Medications of the fluorine, calcium, phosphorus.	Applications of or rinse with fluorine-containing solutions
The intensive mastication	Chronic diseases treatment	Vitamins	Using of remineralization medications
Minerals from water; tea	Rational diet	Immunomodulators and medications increasing general organism resistance	Pressurizing of fissures

The most important methods of caries prevention are rational feeding, using of fluoride in different forms and dosages, methods of removing plaque, fissure sealants and strengthening of somatic health.

Question 3. Rational feeding.

A balanced, nutritious diet is essential in preventing tooth decay and gum disease. Everyday food ration should include:

- *Fruits and vegetables.* Combined, these should cover half of day ration.
- *Grains.* At least half of the grains should be whole grains, such as oatmeal, whole wheat bread and brown rice.
- *Dairy.* Choose low-fat or fat-free dairy foods most often.
- *Protein.* Make lean protein choices, such as lean beef, skinless poultry and fish. Protein choices also should include eggs, beans, peas and legumes.

Foods That May Harm Dental Health

Empty calories foods such as candy (especially hard or sticky candies like lollipops, mints, taffy and caramel), sweets like cookies, cakes and muffins, and snack foods like chips are a cause for dental concern, not only because they offer no nutritional value, but because the amount and type of sugar that they contain that can adhere to teeth. The bacteria in the mouth feed off these sugars, releasing acids, and that's what leads to tooth decay. Sugar-containing drinks are soda, lemonade, juice and sweetened coffee or tea (iced or hot) are particularly harmful because sipping them causes a constant sugar bath over teeth, which promotes tooth decay.

Acidic foods like tomatoes and citrus fruits can have acidic effects on tooth enamel too, so patient should eat them as part of a meal, not by themselves. Dried fruits, including raisins, are also good choices for a healthy diet, but since they are sticky and adhere to teeth, the plaque acids that they produce continue to harm teeth long after you stop eating them.

Foods That May Benefit Dental Health

Cheese, milk, plain yogurt, calcium-fortified tofu, leafy greens and almonds are foods that may benefit tooth health thanks to their high amounts of calcium and other nutrients they provide. Protein-rich foods like meat, poultry, fish, milk and eggs are the best sources of phosphorus. Both of these minerals play a critical role in dental health, by protecting and rebuilding tooth enamel.

Fruits and vegetables are good choices for a healthy smile since they are high in water and fiber, which balance the sugars they contain and help to clean the teeth. These foods also help stimulate saliva production, which washes harmful acids and food particles away from teeth and helps neutralize acid, protecting teeth from decay. Plus, many contain vitamin C (important for healthy gums and quick healing of wounds) and vitamin A (another key nutrient in building tooth enamel).

Hands down, water is particularly fluoridated, water is the most tooth-friendly beverage.

Snacking

For dental health, it's recommended that people limit eating and drinking between meals. Of course, sometimes eating between meals must

happen. Unfortunately, most people choose foods like sweets and chips for snacks; foods that harm teeth by promoting tooth decay. Products recommended for snacking are cheese, yogurt, fruits, vegetables or nuts.

Question 4. Using of fluorides for caries prevention.

Preparations of fluorine now are basic facilities of teeth decay prevention. The mechanism of protective action of fluorine on enamel consists in the assistance to the delay of phosphoric-calcium connections in an organism and processes of remineralization of hard fabrics of tooth and also braking of activity of bacterial enzymes in the cavity of mouth and dental deposit.

Modern information shows that the favourable action of fluorine is predefined by a few mechanisms:

1. Fluorine, uniting from a hydroxide apatites enamel, substituting for OH – groups, forms a fluorapatites, doing an enamel more strong and more proof to the action of acids. This connection reduces permeability of enamel.

2. The mechanism of anticarious action of fluorides is related also to their oppressive influence on growth and exchange of matters of microflora of cavity of mouth.

3. Connections of fluorine in saliva inhibit the transport of glucose in the cages of pathogenic bacteria and formations of for cellular polysaccharidess, which form the matrix of dental deposit.

4. Fluorides violates absorption of microorganisms on-the-spot cages of tooth, absorb albumins of saliva, glycoproteins, as a result of what prevent growth of dental name-plate.

5. And finally, at internal introduction the fluorides normalize an albuminous and mineral exchange.

Fluorides present in enamel and in the dental deposit catalysis «proceeding» in the early carious defeats due to remineralization of crystals of enamel, multiplies the size of crystals of hydroxide apatites.

From modern international data days even receipts of fluorides are distributed thus:

- ✓ very low = 0,1-0,6 milligrams;
- ✓ low = 0,7-1,4 milligram;
- ✓ optimum = 1,5-4,0 milligrams;
- ✓ high (impertinent fluorosis) = 5-12 milligrams;
- ✓ ever-higher = 20 milligrams and more (at treatment of osteoporosis of bones by fluorines preparations).

The amount of fluorine in an organism depends on his maintenance in water and food products.

Endogenous fluoride prevention

A specific endogenous prevention provides for:

1. Fluorination of drinking-water

2. Fluorination milk

3. Fluorination salts

Fluorination of water. One of the acknowledged methods of caries prevention there is fluorination of drinking-water is controlled addition of connections to the fluorine to water of sources of water-supply with the purpose of leading to the concentration of ions of fluorine in a drinking-water to the level, which is sufficient for the effective teeth decay prevention and at the same time does not have an unfavorable influence on functional possibilities of organism of man, physical development and health of population.

Presently about 5% all population of earth (approximately 260 million persons) drink fluorination water. In spite of numerous objections of opponents of fluorination, the presence of undesirable effects is not well-proven, and although every objection must be explored, safety of fluorination of water can be considered set.

The optimum concentration of fluorine in a drinking-water is 1,0 mg/l. Fluorination of drinking-water allows get reduction of increase of permanent teeth decay on 40-50% — on 50-60%.

Fluorination of milk. The use of fluorinating milk is the alternative and effective method of prophylaxis. Milk a long ago brings over to itself attention of researchers on a number of reasons, so as:

- it is the necessary component of feed, especially in the first years of life;
- owns valuable nourishing properties necessary for organism;
- it is the basic source of calcium and phosphorus, fabrics of bones and teeth necessary for the structure;
- contains the high level of calcium, phosphorus and lactose which laminates carbonhydratess also.

Similar composition allows to milk to bring in the payment in the process of remineralization of enamel of teeth and in its defence.

For successful introduction of method of fluorination of milk certain terms are needed:

- high dental morbidity of population is in a region;
- low maintenance of fluorides is in a drinking-water;
- absence of other sources of system receipt of fluorides.

For fluorination of milk more frequent fluoride of sodium is used. Technology of fluorination of milk is simple and does not present difficulties.

The amount of fluoride, which must be added to milk, is guilty to take into account age of child and receipt of fluoride from other products and water.

At the use of fluorinating milk it is necessary to adhere to the followings recommendations:

- effectively to use this method at children from 3 to 12 years
- daily a child must use a 1 glass of milk from 0,5 mg of fluoride

➤ during a year a child must drink milk not less 250 days.

Fluorination of salt. Fluoride in salt is available in several countries (in Belarus too). Recommended fluorides concentration in salt is 250 – 350 mg/kg. For caries prevention it is necessary to use salt with fluorine every day.

Exogenous fluoride prevention

At the use of fluorine it is needed to take into account the following:

1. The concentration of fluorides not must exceed for a local prophylaxis 1-2% (calculating on a fluorine), as with the increase of concentration efficiency does not grow.
2. Efficiency of influencing is conditioned by their concentration in the free ionized kind.
3. It is necessary to take into account in this connection fastening possibility fluoride ions with the ions of calcium.
4. Fluorides is appointed taking into account maintenance of fluorine in a drinking-water and climatic factors.

Tooth-pastes. The most commonly used product containing fluoride is fluoride toothpaste, which dominates on the toothpaste market. The amount of fluoride in toothpaste varies between 250 ppm and 2 500 ppm. Usually recommended concentration for adults is 1 500 ppm. Strong scientific evidence shows that the use of fluoride toothpaste has an effect on the prevention of caries in the permanent teeth of children and young adults. This effect is dose dependent, ie, toothpaste with higher fluoride concentration, 500 ppm fluoride, yields better effect than toothpaste with 1 000 ppm. The effect of fluoride toothpaste on primary teeth has been insufficiently assessed, as have the effects in adults and elderly people. However, nothing suggests that preventive effects would not be found in these age groups.

Fluorine containing solutions for the independent use. The wide use in the prophylaxis of caries was found by solutions with the low concentrations of fluoride.

Amount of rinses makes:

- by a 0,05% solution -1 once on a day
- by a 0,1% solution -1 once in a week
- by a 0,2% solution – 1 one time in two weeks

For the improvement of cooperation of fluorine with an enamel preliminary it follows well to clean teeth and rinse a mouth by alkaline water for the change of pH environment. The rinse lasts 1-3 minutes. After it a mouth is rinsed by clean water.

Application of rinses by solution of fluorine sodium gives reduction to the caries 30%.

Fluorine containing varnishes. One of widespread facilities of local prophylaxis there are varnishes which use for the prolonged period of influencing of fluoride on enamel. They form tape adjoining to the enamel,

and which remains on teeth during a few hours and in fissures a few days and even weeks.

A fluorine – varnish shows itself composition of natural resins of vegetable origin. At the market presented: „Ftorlac” (Kharkiv), varnish „Duraphat”, „Belac” (Vladmiva).

A method is given recommended at the moderate or high level of intensity of caries of teeth, to the people with the high risk of origin of caries. Frequency of causing of varnish is 2-4 times per a year, depending on activity of caries.

Method: the surface of teeth is purged from the deposit and is dried out. Then by the special brush varnish is inflicted by a skim on the surface of tooth. At the same time it is possible to cover all teeth on one jaw or 3-5 teeth. For getting dry of varnish it is needed about 2-3 minutes. It is possible to dry the varnish by the compressed air. After coverage of teeth by fluorine varnish it is impossible to use the meal of 1-3 hours and in future the desired only spoon-meat. It is not recommended to clean teeth 24 hours. Varnish is contained on-the-spot tooth not less 12 hours and for this time his ions penetrate on a depth to 100 mcm of healthy enamel.

To cover teeth by varnish it follows depending on activity of cariosity: at a 1 degree — 2 times per a year, at 2 — 4 times per a year, at 3 — from 6 to 12 times per a year. Triple coverage of teeth is recommended with an interval 1-2 days.

It is set that in a year after application of fluorine containing varnish second caries of teeth goes down on the average on 50%.

Fluorine containing gels and solutions for the professional use. Gels and solutions of fluoride of sodium 1% and 2% are used for appliques and electrophoresis. A doctor-dentist conducts procedure in the conditions of polyclinic. Remineralization action of gels is based on diffusion of matters from gel in saliva and from her in the enamel of tooth.

Method: teeth preliminary clear, dry out and impose the wadding tampon well moistened by solution of fluoride of sodium on 3- 5 minutes. At first assess the masticatory surfaces of teeth, and then – labial and cheeks on both jaws. If gel is used, he is inflicted by heated by a brush and give to dry out. After procedure does not recommend eat and drink during 2 hours.

As a rule conduct 3-5 appliques by solution twice on a year and 2-6 appliques by gel on a year.

Using of fluorides by a spoon

1. To choose the spoon of the proper size. It must be the covered is all dental row, including the areas of retraction and it follows to provide access of gel to the contact with the structure of teeth. The ends of spoon (peripheral areas) must be closed in order that gel did not flow down in the cavity of mouth sick. Ideally spoons befit with coverage from the made foam

material, as they fit snugly dental row of patient and allow to gel to achieve all surfaces.

2. To place gel in a stretcher.
3. To insert a spoon in the mouth of patient.
4. To insert between the spoons of saliva ejector, making sure, that to the patient comfortably (at this method for balance of bite from opposite sides necessary wadding rollers)
5. To bring a spoon out of mouth sick.
6. To ask a patient to spit out immediately after the delete of spoon. After procedure, at a necessity the delete of superfluous fluoride, to apply the intensive sucking.
7. To warn a patient that during 30 minutes after procedure it is impossible to eat or drink.

Application of fluorine containing disks. Fluorine containing disks (paper and paraffins) are produced in packing for 10 things. The expense of material is a 1 disk on procedure. The disk of «Ftorglicofoskal» contains the followings ingredients:

- neurosin - 8-16 g,
- fluoride of sodium - 0,5-2 g,
- superficial matters - 0,5-2,0 g,
- beeswax - 4,5-6,5 g,
- paraffin.

A disk is fixed in an angular tip by a mandrel. A fluorine is rubbed in hard fabrics of tooth on minimum speed with the use of three types of motions: recurrently-forward, up-down, circles.

As usual, before treatment by fluorine containing disks the professional hygiene of cavity of mouth is conducted, whereupon by disks at first the vestibular surfaces of all teeth of maxilla are processed from left to right, then lower from right to left. After it the palatal surfaces of teeth of maxilla and languages surfaces of teeth of lower jaw are processed, farther are masticatory surfaces of teeth of overhead and lower jaws with the use of only circular motions clockwise. It is recommended 2-3 multiple treatment of teeth with an interval 1-2 days, in a year 2-4 courses.

In practice of therapeutic dentistry fluorine containing disks found large popularity not only at the prophylaxis of teeth decay but also at treatment of hyperesthesia of hard fabrics of teeth.

Question 5. Oral hygiene.

Good oral hygiene involves the removal of biofilm from the surface of the teeth, resulting in a mouth that has an absence of or few occurrences of bad breath, teeth that looks relatively clean and debris-free and healthy gums that are pink and do not bleed easily when brushing or flossing. The aim of practicing good oral hygiene is to modify the oral microflora so as to

maintain an oral environment of healthy gums and teeth, prevent dental caries, periodontal disease and halitosis.

Measures to practise good oral hygiene

Toothbrushing. There are many toothbrushing methods such as the Bass, Stillman's, Fones, Charter's, horizontal, vertical and scrub and roll method.

The main recommendations on toothbrushing:

- ✓ Brush the upper and lower set of teeth separately.
- ✓ When brushing the outer surfaces of the teeth, place the toothbrush at an angle of 45 degrees against the gumline and using a sweeping action, gently move the brush against the gumline, using a vibratory motion.
- ✓ When brushing the occlusal surfaces, use short forward and backward strokes.
- ✓ Similarly, when brushing the palatal or lingual surfaces of the teeth, tilt the toothbrush at an angle of 45 degrees against the gumline.
- ✓ An average time taken for brushing teeth is 2-3 minutes.

Frequency of brushing. Due to a variation in individual's preferences and habits, it is difficult to set the number of times one should brush his teeth. Instead, the emphasis should be placed on the effectiveness of biofilm removal from the surfaces of the teeth rather than on the number of brushings. It is recommended that a minimum of two brushings a day plus interdental cleaning is performed to remove the biofilm, hence preventing dental caries and bad breath. The longer the bacteria in the mouth is left undisturbed, the greater the pathologic potential of the bacteria. It is recommended to brush one's teeth before going to sleep because bacteria thrive in the warm, dark and moist conditions of the oral cavity. Based on research findings, the amount and rate of saliva flow changes in the 24-hour period. The normal flow rate of saliva in the day is about 0.3ml/min while at night, during sleep, the flow rate is about 0.1ml/min. Thus at night, the benefits of saliva are minimised in the controlling the bacterial population and its detrimental effects on the dentition.

Type of Toothbrush. A suitable toothbrush should be chosen based on many factors. Firstly, the size of the handle of the toothbrush should be chosen based on the age and the dexterity of the individual while the size of the head of the toothbrush should be based on the size of the individual's mouth. Toothbrushes with small heads are recommended because it enables easier access to the tooth surfaces at the back of the mouth. The bristles of the toothbrush should be end-rounded nylon or polyester filaments that are smaller than 0.009 inches in diameter and should be classified under 'soft' by the acceptable international industry standards (ISO). The toothbrush should be regularly replaced every 3 months or when it begins to show wear.

Replacement of old toothbrushes. It is recommended that toothbrushes should be replaced every 3 months. This is because toothbrush bristles that

are worn down are less effective in removing biofilm and plaque from the surface of the teeth. The frequency of toothbrush wear varies between individuals, ranging from as early as two weeks to as long as 6 months. Hence, it is recommended to replace toothbrushes every 3 months or when it begins to show wear. Indicators of toothbrush wear include spreading, bending and curling of the bristles.

Electric toothbrush. An electric toothbrush is a modified toothbrush that runs on electric power to make rotation oscillations in the head. It is ideal for individuals suffering from arthritis and carpal tunnel syndrome. Based on studies done, electronic toothbrushes with rotational oscillations remove more plaque than manual toothbrushes and reduced the occurrence of gingivitis in the long term.

Toothpaste. Usage of fluoride toothpaste can help to prevent caries to a large extent. It was estimated that brushing with a fluoride toothpaste twice a day reduced carious lesions by 90%. A combination of both cleaning with a toothbrush and using fluoride toothpaste is most optimal. It is also suggested that may be a cumulative effect of brushing the teeth with fluoride toothpaste. The concentration of fluoride present in toothpaste should be chosen based on age as well as the risk of caries. A fluoride concentration of 1100-1500 ppm in toothpaste is recommended for individuals above the age of seven. Rinsing the toothpaste with large amounts of water is also not advised because it removes a significantly larger amount of fluoride from the mouth. It is instead, recommended to clean off excess toothpaste using a wet toothbrush and spitting out as much toothpaste as possible.

Proper toothbrushing alone, without the use of fluoride toothpaste, is still effective in removing plaque and biofilm from the surfaces of the teeth and oral hygiene is not compromised. However, it is recommended that fluoride toothpaste is used because fluoride helps to prevent carious lesion formations and remineralizes initial carious lesions.

Interdental Cleaning. The removal of plaque on the interproximal surfaces is important in the prevention of caries. However, because the toothbrush is relatively ineffective in removing interproximal plaque, interdental cleaning methods, such as flossing, should be performed to maintain good oral hygiene.

Flossing is one of the most commonly used methods of interproximal cleaning and according to the American Dental Association, an estimate of 80% of the interdental plaque can be removed by flossing.

1. Using a floss that is roughly 16 inches long, twirl most of the floss around the middle fingers, leaving a short length of about 1 to 2 inches to floss.
2. Hold the floss tightly between your index fingers and thumb, slide it gently between the teeth, using a up-down movement towards the gum.
3. Curve the floss around the base of each tooth, making sure that it goes under the gumline.

4. Guide the floss back to the gum and repeat this twice.
5. Use a new section of the floss for each tooth, while winding the used sections to the middle finger of your other hand.
6. Floss systematically, for example, from left to right, top to bottom, so as to not miss out on any tooth.
7. Some bleeding may be observed initially, however, after flossing regularly for a few days, the bleeding should stop.

There are various types of floss available. The nylon or multi-filament floss is available in the waxed and unwaxed form and in a variety of flavours. Because it is composed of many threads of nylon, it may shred at times, especially through tight contact points. The polytetrafluoroethylene (PTFE) or monofilament floss is only made up of one filament and is able to slide easily between teeth and is resistant to shredding. Superfloss has a stiff end that acts as a floss threader to thread through tight areas. It also has a fuzzy tuft segment that collects plaque while flossing. The superfloss is very useful for flossing under bridges and for teeth with braces. There are also limitations in flossing in cases where there are embrasure spaces or when there is a loss of attachment. Due to the limitations in different individual's habits and ability to floss properly, some may find flossing difficult (especially through tight contact points).

Interdental toothbrush. An interdental toothbrush is a small modified toothbrush structure that can be used to clean the spaces between teeth. It is useful in cases where there are gaps between the gingiva and the teeth or when there is orthodontic treatment. Interdental brushes can be used as a substitute for flossing and they are available in varying sizes to suit the size of the gaps between the teeth. To use an interdental brush, gently push it back and forth between the teeth but do not apply too much force.

Tongue Cleaning. Tongue cleaning should be done regularly to remove food debris, fungi and bacteria build-up and dead cells from the surface of the tongue. Decaying bacteria produce volatile sulphur compounds that play a major role in halitosis/ bad breath. Tongue cleaning is not effective in controlling gingivitis or caries. However, by removing food debris and micro-organisms, tongue cleaning can help to contribute to overall oral cleanliness and reduce mouth odours. This is especially useful for patients suffering from xerostomia (dry mouth due to a lack of saliva), deep fissures and smokers. The top surface of the tongue can be cleaned using a tongue scraper/brush. Tongue scrapers are available in metal, plastic and many other materials. Tongue cleaning should be done using a proper tongue scraper and not a toothbrush.

Procedure:

1. Place the arch of the tongue cleaner towards the posterior end of the dorsal surface of the tongue.
2. Pressing gently but firmly against the tongue, pull forward.

3. Repeat this process several times and complete the surface of the tongue.
4. Wash the tongue cleaner with water.
5. Some gag reflex may be experienced at the start.

Question 6. Fissure sealants.

Fissure sealants are used on occlusal surfaces of the teeth. Occlusal surfaces have fissures that make tooth cleaning difficult and allow caries to start. The method involves applying a thin, very fluid, plastic material directly to the fissures. To retain the material to the tooth, its surface is pretreated with an acid that creates small pores in the enamel. The material fills the pores and there by mechanically bonds to the tooth surface, creating a smooth and even surface. It is essential to keep the tooth absolutely dry during treatment. If not, the pores can fill with saliva and the sealant attaches poorly. Sealants must be applied soon after the tooth emerges to prevent early caries. Intact sealants have the potential to prevent caries on the occlusal surface. The method is not invasive and causes no pain. However, sealants are technically sensitive, and require continual checking and repair, or replacement if needed. Many studies have been reviewed to determine the preventive effects of fissure sealants composed of resin-based material. Many of the studies are outdated and deficient in study design and follow-up. Hence, there is only limited evidence in the literature that fissure sealants prevent caries in the short and long term. The literature offers insufficient evidence to assess other types of material used as fissure sealants. The scientific documentation also offers insufficient evidence for determining if fissure sealants have a preventive effect against caries in populations with low and high rates of caries.

Strengthening of somatic health. It is assumed that under act of commons diseases the terms of forming and ripening of hard fabrics of tooth change in the first turn of enamel which does them less proof in relation to influencing of cariogenic factors.

Practically the defeats of any organs and systems of organism are extrapolated on hard fabrics of teeth. That is why it is needed with the purpose of prophylaxis of dental diseases, on possibility, to treat be - what somatic pathology.

Test tasks

1. What types of caries prevention do you know:

- a. primary prevention
- b. secondary prevention
- c. tertiary prevention
- d. *all answers are right*

2. What are the most important methods of caries prevention:

- a. rational feeding
- b. using of fluoride in different forms and dosages
- c. methods of removing plaque
- d. *all answers are right*

3. Everyday food ration should include:

- a. *Fruits and vegetables, grains, proteins*
- b. Cola, sweets, cakes
- c. all answers are right

4. Endogenous fluoride prevention includes:

- a. *fluorination of drinking-water*
- b. *fluorination milk*
- c. fluorine containing solutions for the independent use
- d. fluorine containing varnishes

5. Exogenous fluoride prevention includes:

- a. fluorination of drinking-water
- b. *tooth-pastes with fluorine*
- c. *fluorine containing varnishes*
- d. *fluorine containing solutions for the independent use*
- e. all answers are right

6. How many times it is recommended to cover teeth with fluorine containing varnishes:

- a. *triple coverage with an interval 1-2 days*
- b. double coverage with an interval 1-2 days
- c. one coverage with an interval 1-2 days
- d. all answers are right

7. How many times it is recommended to cover teeth with fluorine containing gels:

- a. *2-6 appliques by gel on a year*
- b. 1-2 appliques by gel on a year
- c. all answers are right

8. Usually recommended fluorine concentration in toothpastes for adults is:

- a. *1500 ppm*
- b. 1000 ppm
- c. 200 ppm
- d. 2500 ppm

9. What amount of rinses with fluorine containing solutions for the independent use is recommended:

- a. by a 0,05% solution -1 once on a day
- b. by a 0,1% solution -1 once in a week
- c. by a 0,2% solution – 1 one time in two weeks
- d. *all answers are right*

10. What are the main objects and tools using for oral hygiene:

- a. *toothpaste*
- b. *toothbrush*
- c. *floss*
- d. solutions
- e. irrigator
- f. all answers are right

LESSON 16. NONCARIOUS TOOTH LESIONS THAT OCCUR BEFORE TOOTH ERUPTION.

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

1. Classification of noncarious lesions.
2. Anodontia.
3. Supernumerary teeth.
4. Abnormalities of size and form of teeth.
5. Mottled teeth.
6. Disturbances in tooth formation.
7. Enamel hypoplasia.
8. Tetracycline Discoloration.
9. Hereditary disturbances in tooth structure, not elsewhere classified.
10. Disturbances in tooth eruption.

Question 1. Classification of noncarious lesions.

Noncarious tooth tissue loss is defined as surface loss due to a disease process other than dental caries (Pual A Brunton, Decision making in Operative Dentistry). Although decay is the usual cause of tooth destruction necessitating operative procedures, it has been estimated that 25% of tooth destruction does not originate from a carious process.

According to the International classification of diseases there are 3 types of noncarious teeth lesions:

- disorders of tooth development and eruption (**K00**),
- embedded and impacted teeth (**K01**),
- other diseases of hard tissues of teeth (**K03**).

K00 Disorders of tooth development and eruption

K00.0 Anodontia, Hypodontia, Oligodontia

K00.1 Supernumerary teeth Distomolar, Fourth molar, Mesiodens, Paramolar, Supplementary teeth

K00.2 Abnormalities of size and form of teeth

Teeth Concrecence, Fusion, Gemination

Dens: evaginatus, in dente, invaginatus, Enamel pearls, Macrodonia, Microdonia, Peg-shaped [conical] teeth, Taurodontism, Tuberculum paramolare.

K00.3 Mottled teeth

Dental fluorosis

Mottling of enamel

Nonfluoride enamel opacities

K00.4 Disturbances in tooth formation

Aplasia and hypoplasia of cementum

Dilaceration of tooth

Enamel hypoplasia (neonatal, postnatal, prenatal)

Regional odontodysplasia

Turner tooth

K00.5 Hereditary disturbances in tooth structure, not elsewhere classified

Imperfecta Amelogenesis, Dentinogenesis, Odontogenesis

Dentinal dysplasia

Shell teeth

K00.6 Disturbances in tooth eruption

Dentia praecox

Natal, Neonatal tooth

Premature:

-eruption of tooth

-shedding of primary [deciduous] tooth

Retained [persistent] primary tooth

K00.7 Teething syndrome

K00.8 Other disorders of tooth development

Colour changes during tooth formation

Intrinsic staining of teeth

K00.9 Disorder of tooth development, unspecified

Disorder of odontogenesis

K01 Embedded and impacted teeth

K01.0 Embedded teeth

K01.1 Impacted teeth

K03 Other diseases of hard tissues of teeth

K03.0 Excessive attrition of teeth:

-approximal

-occlusal

K03.1 Abrasion of teeth:

- dentifrice
- habitual
- occupational
- ritual
- traditional

Wedge defect

K03.2 Erosion of teeth

Erosion of teeth due to:

- diet
- drugs and medicaments
- persistent vomiting
- idiopathic
- occupational

K03.3 Pathological resorption of teeth

Internal granuloma of pulp

Resorption of teeth (external)

K03.4 Hypercementosis

Cementation hyperplasia

K03.5 Ankylosis of teeth

K03.7 Posteruptive colour changes of dental hard tissues

K03.8 Other specified diseases of hard tissues of teeth

Irradiated enamel

Sensitive dentine

K03.9 Disease of hard tissues of teeth, unspecified

Depending on the occurrence of non-carious lesion of dental hard tissues are divided into two groups (M.I. Hroshykov, 1985):

- 1st - those that occur during follicular development tooth - hypoplasia, hyperplasia, fluorosis and hereditary lesions (dysplasia Kapdepona, imperfect Amelie and dentynohenez, marble disease, etc.)
- 2nd - damage arising after the eruption of the tooth - pathological tooth wear, wedge-shaped defects, necrosis, erosion of hard tissue, tooth hypersensitivity, trauma.

Question 2. Anodontia.

K00.0 Anodontia. In dentistry, anodontia, also called anodontia vera, is a rare genetic disorder characterized by the congenital absence of all primary or permanent teeth. It is associated with the group of skin and nerve syndromes called the ectodermal dysplasias. Anodontia is usually part of a syndrome and seldom occurs as an isolated entity.

Anodontia is the congenital absence of teeth and can occur in some or all teeth (partial anodontia or hypodontia), involve two dentitions or only teeth of the permanent dentition (Dorland's 1998). Approximately 1% of the

population suffers from oligodontia. Many denominations are attributed to this anomaly: partial anodontia, hypodontia, oligodontia, the congenital absence, anodontia, bilateral aplasia.

Congenital absence of permanent teeth can present as hypodontia, usually missing 1 or 2 permanent teeth, or oligodontia that is the congenital absence of more than 6 teeth. The most common missing teeth are: lower third molars, upper lateral incisors, lower second premolars.

The congenital absence of at least one permanent tooth is the most common dental anomaly and may contribute to masticator dysfunction, speech impairment, aesthetic problems, and malocclusion (Shapiro and Farrington 1983).

Treatment. For the treatment of adontia are used orthodontic procedures and/or prosthetic replacement of missing teeth using dental implant technology or dentures.

Question 3.Supernumerary teeth.

K00.1 Supernumerary teeth. Supernumerary teeth (hyperdontia) is the condition of having teeth that appear in addition to the regular number of teeth. They can appear in any area of the dental arch and can affect any dental organ.

Supernumerary teeth can be classified by shape and by position. The shapes include the following:

- Supplemental (where the tooth has a normal shape for the teeth in that series);
- Tuberculate (also called barrel shaped);
- Conical (also called peg shaped);
- Compound odontoma (multiple small tooth-like forms);
- Complex odontoma (a disorganized mass of dental tissue).

When classified by position, a supernumerary tooth may be referred to as a mesiodens, a paramolar, or a distomolar.

The most common supernumerary tooth is a mesiodens, which is a malformed, peg-like tooth that occurs between the maxillary central incisors.



Picture 55. Two mesiodens: supernumerary teeth in the midline between the upper central incisors.

Fourth and fifth molars that form behind the third molars are another kind of supernumerary teeth.

Causes. Here is evidence of hereditary factors along with some evidence of environmental factors leading to this condition. While a single excess tooth is relatively common, multiple hyperdontia is rare in people with no other associated diseases or syndromes. Many supernumerary teeth never erupt, but they may delay eruption of nearby teeth or cause other dental or orthodontic problems. Molar-type extra teeth are the rarest form. Dental X-rays are often used to diagnose hyperdontia.

It is suggested that supernumerary teeth develop from a second tooth bud arising from the dental lamina near the regular tooth bud or possibly from splitting the regular tooth bud itself. Supernumerary teeth in deciduous (baby) teeth are less common than in permanent teeth.

Question4. Abnormalities of size and form of teeth.

K00.2 Abnormalities of size and form of teeth

Tooth fusion. The phenomenon of tooth fusion arises through union of two normally separated tooth germs, and depending upon the stage of development of the teeth at the time of union, it may be either complete or incomplete. On some occasions, two independent pulp chambers and root canals can be seen. However, fusion can also be the union of a normal tooth bud to a supernumerary tooth germ. In these cases, the number of teeth is fewer if the anomalous tooth is counted as one tooth.



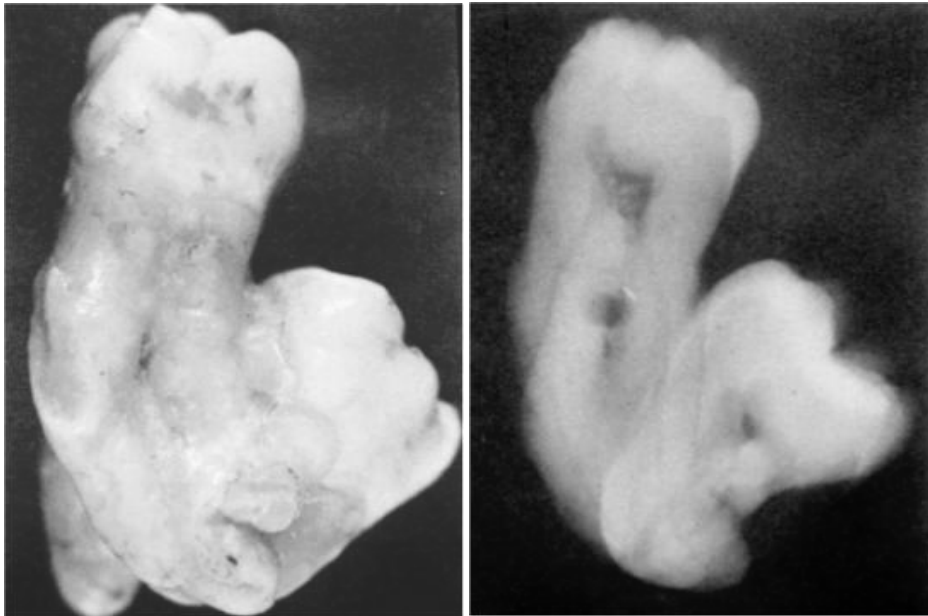
Picture 56. Fusion.

Tooth gemination is a dental phenomenon that appears to be two teeth developed from one. There is one main crown with a cleft in it that, within the cervical third of the crown, looks like two teeth, though it is not two teeth. The number of the teeth in the arch will be normal. The phenomenon of gemination arises when two teeth develop from one tooth bud and, as a result, the patient has an extra tooth, in contrast to fusion, where the patient would appear to be missing one tooth. Fused teeth arise through union of two normally separated tooth germs, and depending upon the stage of development of the teeth at the time of union, it may be either complete or incomplete. On some occasions, two independent pulp chambers and root canals can be seen. However, fusion can also be the union of a normal tooth bud to a supernumerary tooth germ. In these cases, the number of teeth is also normal and differentiation from gemination may be very difficult, if not impossible. In geminated teeth, division is usually incomplete and results in a large tooth crown that has a single root and a single canal. Both gemination and fusion are prevalent in primary dentition, with incisors being more affected.



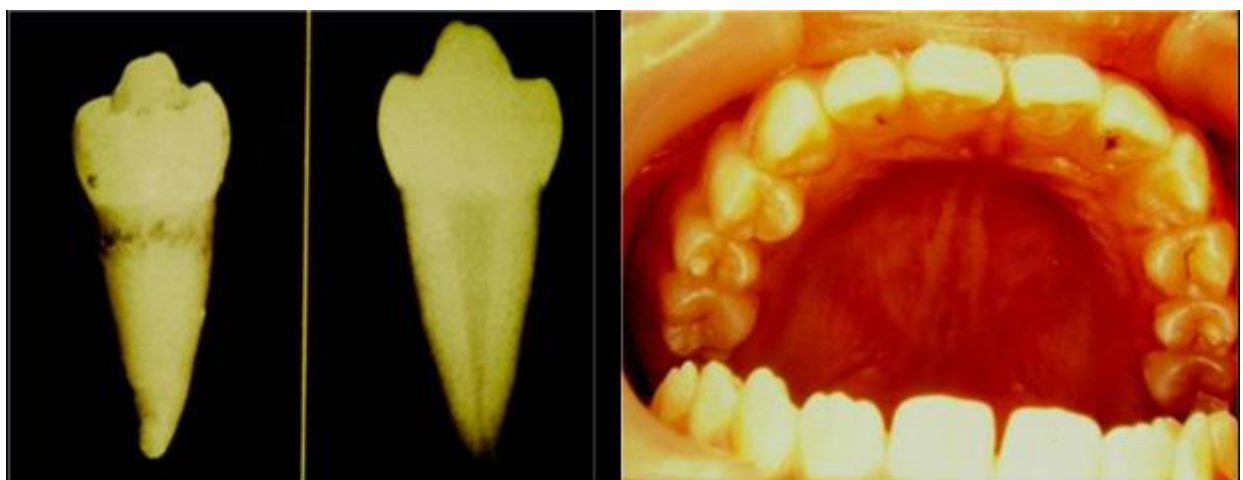
Picture 57. Gemination.

Concrescence is a condition of teeth where the cementum overlying the roots of at least two teeth join together. The cause can sometimes be attributed to trauma or crowding of teeth. Surgical separation of the teeth may be necessary if one is to be extracted.



Picture 58. Concrescence.

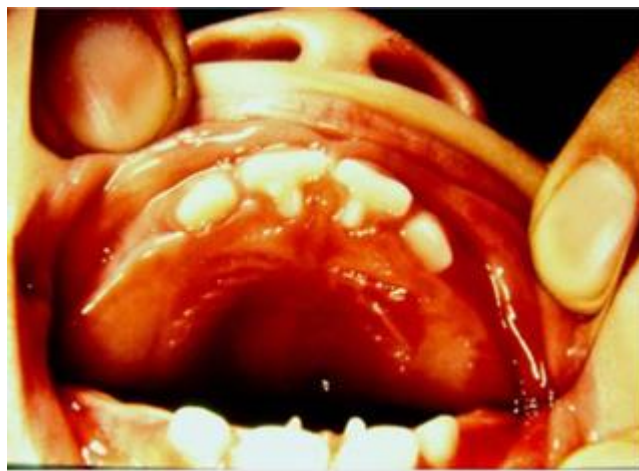
Dens evaginatus is a condition found in teeth where the outer surface appears to form an extra bump or cusp. Premolars are more likely to be affected than any other tooth. This may be seen more frequently in Asians, but almost exclusively in Down Syndrome. The pulp of the tooth may extend into the dens evaginatus. There is a risk of the dens evaginatus chipping off in normal function. Hence this condition requires monitoring as the tooth can lose its blood and nerve supply as a result and may need root canal treatment.



Picture59. Dens evaginatus.

A talon cusp, also known as an "eagle's talon", is an extra cusp on an anterior tooth. The term refers to the same condition as dens evaginatus, but the talon cusp is the manifestation of dens evaginatus on anterior teeth. The incidence has been found to range from less than 1% to 6% of the population. Of all cases, 55% occur on the permanent maxillary lateral incisor, and 33% occur on the permanent maxillary central incisor. They are found rarely in primary teeth.

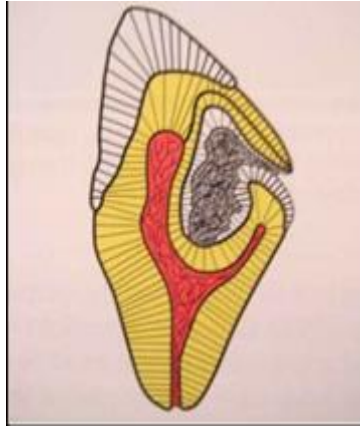
The condition is usually benign, but it can cause mild irritation to soft tissues around the teeth and the tongue, and if large enough, may pose an aesthetic problem. Talon cusps that are too large are filed down with a motorised file, and then endodontic therapy is administered.



Picture 60. Talon cusp.

Dens invaginatus, also known as dens in dente ("tooth within a tooth") is a condition found in teeth where the outer surface folds inward. There are coronal and radicular forms, with the coronal form being more common.

Dens invaginatus is a malformation of teeth most likely resulting from an infolding of the dental papilla during tooth development or invagination of all layer of the enamel organ in dental papillae. Affected teeth show a deep infolding of enamel and dentine starting from the foramen coecum or even the tip of the cusps and which may extend deep into the root. Teeth most affected are maxillary lateral incisors and bilateral occurrence is not uncommon. The malformation shows a broad spectrum of morphologic variations and frequently results in early pulp necrosis. Root canal therapy may present severe problems because of the complex anatomy of the teeth. Aetiology, prevalence, classification, and therapeutic considerations including root canal therapy, apical surgery and prevention of pulpal involvement are reviewed.



Picture 61. Dens invaginatus.

An enamel pearl is a condition of teeth where enamel is found on locations where enamel is not supposed to be, such as on a root surface. They are found usually in the area between roots, which is called a furcation, of molars. Enamel pearls are not common in teeth with a single root. The most common location of enamel pearl is the furcation areas of the maxillary and mandibular third molar roots. The Enamel pearls are formed essentially from the Hertwig's Epithelial root sheath. After the initiation of the formation of dentin in the root area of the tooth, the root sheath disintegrates and moves away from the root surface so that the cells of the dental sac can come in contact of predentin to differentiate into cementoblasts and start deposition of cementum. However, if the cells of epithelial root sheath remain adherent to predentin, they may differentiate into fully functional ameloblasts and deposit Enamel. Such droplets of enamel are called Enamel Pearls.

Macrodonia (or megadontia or megalodontia) is a type of localized gigantism in which teeth are larger than normal for the particular type(s) of teeth involved. The three types of macrodonia are true generalized macrodonia, relative generalized macrodonia, and macrodonia of a single tooth. True generalized macrodonia is rare. Macrodonia of a single tooth is more common. Some kind of macrodonia in the permanent dentition occurs in 1.1% of the total population. It should not be confused with taurodontism (bull teeth), fusion (double tooth) or the jaws being relatively small, giving the appearance of macrodonia. Males tend to have larger teeth than females, and tooth size also varies according race. Abnormal tooth size is defined by some as when the dimensions are more than 2 standard deviations from the average. Macrodonia is when the teeth are abnormally large, and microdonia when they are abnormally small. Macrodonia of a single tooth is attributed to a disturbance of morphodifferentiation. Generalized macrodonia is usually attributed to some hormonal imbalance (e.g., pituitary gigantism). It can also be associated with facial hemihyperplasia.



Picture 62. Enamel pearl.

True generalized microdontia. All the teeth are smaller than the normal size. True generalized microdontia is very rare, and occurs in pituitary dwarfism. Due to decreased levels of growth hormone the teeth fail to develop to a normal size.

Relative generalized microdontia. All the teeth are normal size but appear smaller relative to enlarged jaws. Relative generalized microdontia may be the result of inheritance of a large jaw from one parent, and normal sized teeth from the other.

Localized (focal) microdontia. Localized microdontia is also termed focal, or pseudo-microdontia. A single tooth is smaller than normal. Localized microdontia is far more common than generalized microdontia, and is often associated with hypodontia (reduced number of teeth). Females are affected more than males, and the condition occurs in permanent (adult) teeth more than deciduous (baby teeth or milk teeth). The most commonly involved tooth in localized microdontia is the maxillary lateral incisor, which may also be shaped like an inverted cone (a “peg lateral”). Peg laterals typically occur on both sides, and have short roots. Inheritance may be involved, and the frequency of microdontia in the upper laterals is just under 1%. The second most commonly involved tooth is the maxillary third molars, and after this supernumary teeth.



Picture 63. Localized (focal) microdontia.

There are many potential factors involved.

- ✓ Congenital hypopituitarism.
- ✓ Ectodermal dysplasia.
- ✓ Down syndrome.
- ✓ Ionizing radiation to the jaws during tooth development (odontogenesis).
- ✓ Chemotherapy during tooth development.
- ✓ Marshall syndrome.
- ✓ Rieger syndrome.
- ✓ Focal dermal hypoplasia.
- ✓ Silver-Russell syndrome.
- ✓ Williams syndrome.
- ✓ Gorlin-Chaudhry-Moss syndrome.
- ✓ Coffin–Siris syndrome.
- ✓ Salamon syndrome.
- ✓ Cleft lip and palate.

Others include trichorhinopharyngeal, odontotrichomelic, neuroectodermal and dermo-odontodysplasia syndromes.

Unerupted microdents may require surgical removal to prevent the formation of cysts. Erupted microdents, peg laterals especially, may cause cosmetic concern. Such teeth may be restored to resemble normal sized teeth, typically with composite build ups or crowns. Orthodontics may be required in severe cases to close gaps between the teeth.

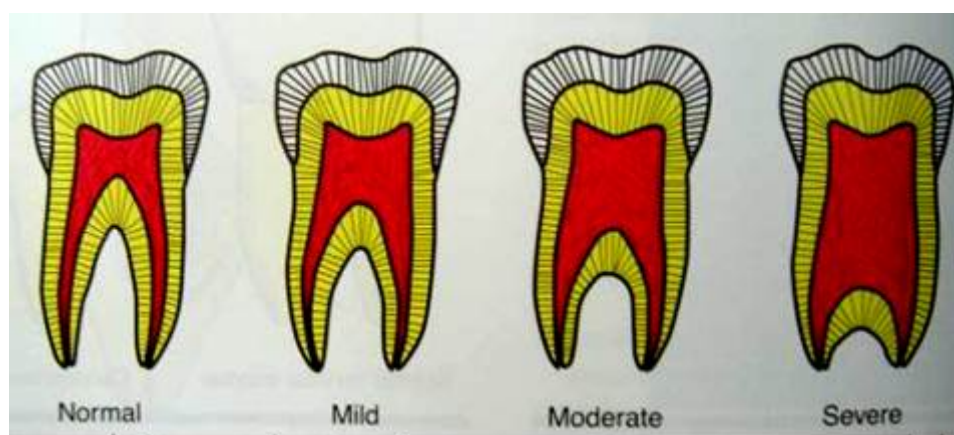
Taurodontism is a condition found in the molar teeth of humans whereby the body of the tooth and pulp chamber is enlarged vertically at the expense of the roots. As a result, the floor of the pulp and the furcation of the tooth is moved apically down the root. The underlying mechanism of taurodontism is the failure or late invagination of Hertwig's epithelial root sheath, which is responsible for root formation and shaping causing an apical shift of the root furcation.

The constriction at the amelocemental junction is usually reduced or absent. Taurodontism is most commonly found in permanent dentition although the term is traditionally applied to molar teeth. In some cases taurodontism seems to follow an autosomal dominant type of inheritance.

Taurodontism is found in association with amelogenesis imperfecta, ectodermal dysplasia and tricho-dento-osseous syndrome. The term means “bull like” teeth derived from similarity of these teeth to those of ungulate or cud-chewing animals.

It has also been reported in Klinefelter’s syndrome, XXYY and Down’s syndrome. The teeth involved are invariably molars, sometimes single and at the other times multiple teeth may be involved. The teeth themselves may look normal and do not have any particular anatomical character on clinical examination.

On a dental radiograph, the involved tooth looks rectangular in shape without apical taper. The pulp chamber is extremely large and the furcations may be only a few millimeters long at times.



Picture 64. Taurodontism.

Question5. Mottled teeth.

K00.3 Mottled teeth. Dental fluorosis, also called mottling of tooth enamel, is a developmental disturbance of dental enamel caused by the consumption of excess fluoride during tooth development. Fluorosis continues to be an endemic problem. The following countries have been identified for the problem of fluorosis: Pakistan, Bangladesh, Argentina, United States of America, Morocco, Middle East countries, Japan, South African Countries, New Zealand, Thailand etc. Children in the age group of 0 to 12 years are most prone to fluorosis as their body tissues are in formative / growth stage during this period. Expectant mothers are also to be protected, as there is growing concern about effects of fluoride on fetus. Acceptable measures of fluorine in water according to hygienic standards is 0,8-1,2(1,5)mg/l.

Many well-known sources of fluoride may contribute to overexposure including dentifrice/fluoridated mouthrinse (which young children may swallow), bottled waters which are not tested for their fluoride content, inappropriate use of fluoride supplements, ingestion of foods especially imported from other countries, and public water fluoridation. The last of these sources is directly or indirectly responsible for 40% of all fluorosis, but the resulting effect due to water fluoridation is largely and typically aesthetic. Severe cases can be caused by exposure to water that is naturally fluoridated to levels well above the recommended levels, or by exposure to other fluoride sources such as brick tea or pollution from high fluoride coal.

Fluorosis is the term given to changes in the enamel which are associated with excess ingestion of fluoride. These vary from localised white opacities to more severe brown–yellow mottling on the teeth. The precise effect depends on the dose of fluoride (from all sources), the duration for which it was taken and the age of the patient at the time of ingestion. Fluorosis when very severe (concentrations in the water supply greater than six parts per million) may result in extensive hypoplasia with brown staining. Teeth are generally composed of hydroxyapatite and carbonated hydroxyapatite; as the intake of fluoride increases, so does the teeth's composition of fluorapatite. Excessive fluoride can cause white spots and, in severe cases, brown stains, pitting, or mottling of the enamel. A tooth is no longer at risk of fluorosis after eruption into the oral cavity. At this point, fluorapatite is beneficial because it is more resistant to dissolution by acids (demineralization). Although fluorosis usually affects permanent teeth, occasionally the primary teeth may be involved.

Distinctive features of dental fluorosis

- Mottling is endemic in areas where fluorides in the drinking water exceed about 2 parts per million, i.e. it has a geographical distribution.
- Neighbouring communities with fluoride-free water do not suffer from the disorder.
- Only those who have lived in a high-fluoride area during dental development show mottling. The defect is not acquired by older visitors to the area.
- Permanent teeth are affected; mottling of deciduous teeth is rare.
- Mottled teeth are less susceptible to caries than normal teeth from low-fluoride areas.
- A typical effect is paper-white enamel opacities.
- Brown staining of these patches may be acquired after eruption

Dean's Index. H.T. Dean's fluorosis index was first published in 1934. The index underwent two changes, appearing in its final form in 1942. This form became the most universally accepted classification system for dental fluorosis. An individual's fluorosis score is based on the most severe form of fluorosis found on two or more teeth.

Table 17

Dean's Index

<i>Classification</i>	<i>Criteria – description of enamel</i>
Normal	Smooth, glossy, pale creamy-white translucent surface
Questionable	A few white flecks or white spots
Very Mild	Small opaque, paper white areas covering less than 25% of the tooth surface
Mild	Opaque white areas covering less than 50% of the tooth surface
Moderate	All tooth surfaces affected; marked wear on biting surfaces; brown stain may be present
Severe	All tooth surfaces affected; discrete or confluent pitting; brown stain present

Clinical features. Mottling ranges from paper-white patches to opaque, brown, pitted and brittle enamel. Clinically, it may be difficult to distinguish fluorotic defects from amelogenesis imperfect when the degree of exposure to fluoride is unknown. There is considerable individual variation in the effects of fluorides. A few patients acquire mottling after exposure to relatively low concentrations, while others exposed to higher concentrations appear unaffected.

Grading of mottled enamel***Picture 65. Mottled enamel.***

- *Very mild.* Small paper-white areas involve less than 25% of surface.
- *Mild.* Opaque areas involve up to 50% of surface.
- *Moderate.* The whole of the enamel surface may be affected with paper-white or brownish areas or both.
- *Severe.* The enamel is grossly defective, opaque, pitted, stained brown and brittle.

Treatment. Dental fluorosis can be cosmetically treated by a dentist. The cost and success can vary significantly depending on the treatment. Tooth bleaching, microabrasion, and conservative composite restorations or porcelain veneers are commonly used treatments. Generally speaking,

bleaching and microabrasion are used for superficial staining, whereas the conservative restorations are used for more unaesthetic situations.

Question 6. Disturbances in tooth formation

K00.4 Disturbances in tooth formation. Dilaceration is a developmental disturbance in shape of teeth. It refers to an angulation, or a sharp bend or curve, in the root or crown of a formed tooth. The condition is thought to be due to trauma or possibly a delay in tooth eruption relative to bone remodeling gradients during the period in which tooth is forming. The result is that the position of the calcified portion of the tooth is changed and the remainder of the tooth is formed at an angle.

The curve or bend may occur anywhere along the length of the tooth, sometimes at the cervical portion, at other times midway along the root or even just at the apex of the root, depending upon the amount of root formed when the injury occurred.

Such an injury to a permanent tooth, resulting in dilaceration, often follows traumatic injury to the deciduous predecessor in which that tooth is driven apically into the jaw.



Picture 66. Disturbances in tooth formation.

Regional odontodysplasia or odontogenesis imperfecta is a developmental disturbance consisting of both enamel and dentin abnormalities in several adjacent teeth. The often-added suffix “regional” emphasises this usually localised character, but a few cases have been described with involvement of more extensive parts of the dentition, the abnormal teeth being present bilaterally and in both upper and lower jaw. The condition is nonhereditary. There is no predilection for race, but females are more likely to get regional odontodysplasia. The enamel, dentin, and pulp of teeth are affected, to the extent that the affected teeth do not develop properly. These teeth are very brittle. The teeth are abnormally formed and the covering enamel layer is thin and yellow. The pulp chambers are wide and the amount of dentin is greatly reduced. The enamel is hypoplastic and

the dentin contains large areas of interglobular dentin. Also, the predentin zone is very wide. The dental pulp usually contains large and irregular aggregates of mineralised matrix, the so-called denticles. The condition may be accompanied by gingival enlargement.

On radiographs the teeth appear more radiolucent than normal, so they are often described as "ghost teeth". Most cases are considered idiopathic, but some cases are associated with syndromes, growth abnormalities, neural disorders, and vascular malformations. Permanent teeth usually show effects of regional odontodysplasia if the deciduous tooth was affected. Many of these teeth do not erupt, and those that do have an increased risk of caries and periapical inflammation.



Picture 67. Regional odontodysplasia.

Treatment and prognosis are usually based upon keeping these teeth and devitalized and restorable. For unerupted teeth, function can be restored with a removable partial denture until all major growth has been completed and a final restoration can be placed.

Question 7. Enamel hypoplasia.

Enamel hypoplasia is a defect of the teeth in which the enamel is hard but thin and deficient in amount, caused by defective enamel matrix formation with a deficiency in the cementing substance. Usually the condition involves part of the tooth having a pit in it. In some cases, the natural enamel crown has a hole in it, and in extreme cases, the tooth has no enamel, which doesn't mean the tooth doesn't exist because dentin is also a component of teeth. It can be caused by any of the following:

- Malnutrition.
- Low birthweight.
- Prematurity.
- Maternal illness.
- Smoking.

- Drug abuse.
- Liver disease.
- Other systemic diseases.

If only a single tooth is affected these are classified as localized hypoplasia, but if several teeth are affected then they are referred to as generalized.

Table 18

Hypoplasia		
	<i>Generalized (systemic)</i>	<i>Localized</i>
Etiology	Premature birth, rhesus incompatibility, diseases suffered in childhood (from 0-9 months of life), intake of medicaments (antibiotics), malnutrition, lack of vitamins. Hypoplasia of temporary teeth: diseases of expecting mother, intake of toxic medicaments, malnutrition, smoking	Infection (follicles of premolars are placed between roots of temporary molars that are very often have apical periodontitis). Trauma to the deciduous predecessor (intrusive luxation of temporary tooth leads to injure of permanent follicle). These teeth are sometimes referred to as Turner teeth.
Affected teeth	Symmetrical teeth (those that have the same period of mineralization: maxilla central incisors and 1-st molars)	Permanent teeth, usually one tooth. The most common teeth affected due to trauma are anterior teeth; due to infection of predecessors are premolars.
Clinical manifestation	It is manifested in such forms: spotted, pitted, grooved, and linear. The more pronounced defect, the more severe was disease that caused it.	Single white (yellowish-brown) spot on incisal edge or in the equator of tooth (caries-immune zones). Turner tooth – enamel could be partly or totally absent, tooth could have pits, grooves, be brown-yellowish in color.
Objectively	Enamel is shine, hard and smooth on probing and not painful.	
Complaints of the patient	Aesthetic defect.	
Diagnosis	Diagnose is set from anamnesis of patient's life, anamnesis of disease and by applying extra methods of examination – vital coloring (methylene blue solution 2%) is negative.	
Differential diagnosis	Fluorosis, white spot lesion (initial caries)	Caries in the stage of white spot (initial caries)
Treatment and prophylaxis	Prophylaxis should be aimed at: care of general health of expectant mothers and later about newborns; prophylaxis of infectious and noninfectious diseases of kids in early childhood period; effective and timely treatment of somatic diseases (acute infectious diseases,	Prophylaxis: to prevent injuries of maxillofacial region in children; timely treatment of temporary teeth to prevent progression of infection in periapical region.

	malnutrition that leads to disease, hypo-, avitaminosis, etc.); increased awareness about this disease, is done as explanation to expectants mothers about proper lifestyle during pregnancy.	
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Localised defects most commonly affect the upper incisor teeth or premolars and usually form as a result of infection (follicles of premolars are placed between roots of temporary molars that are very often have apical periodontitis) or trauma to the deciduous predecessor. This affects the ameloblasts of the developing tooth, resulting in the production of enamel which may be yellow–brown, pitted or irregular. These teeth are sometimes referred to as Turner teeth. Generalised defects are sometimes referred to as chronological hypoplasias and most are the result of a generalised or systemic infection or disturbance occurring during tooth development. The disturbance affects enamel formation and results in a linear horizontal band of hypoplasia. This may be characterised by ridging or grooving or pitting on the enamel surface and the teeth that are affected are those which were forming at the time of the disturbance. Hypoplastic defects that are localized on incisal edge of maxilla central incisors, canines and cusps of 1-st molars and lateral mandible incisors as well - could justify the disturbance in mineralisation at the age from 6 month until 1 year of life. Thus with a knowledge of the times of crown formation it is possible to predict at what age a patient was systemically unwell. The overwhelming majority of chronological hypoplasia as is the result of disturbances in the first 10 months of life and the teeth affected are:

- The first permanent molars.
- Upper central incisors.
- Lower lateral incisors and canines.

Table 19

Timing of eruption and calcification

	<i>Calcification begins (months)</i>	<i>Eruption (years)</i>
<i>Upper:</i>		
Central incisor	3-4	7-8
Lateral incisor	10-12	8-9
Canine	4-5	11-12
First premolar	18-21	10-11
Second premolar	24-27	10-12
First molar	At birth	6-7
Second molar	30-36	12-13

Third molar	84-108	17-21
<i>Lower:</i>		
Central incisor	3-4	6-7
Lateral incisor	3-4	7-8
Canine	4-5	9-10
First premolar	21-24	10-12
Second premolar	27-30	11-12
First molar	At birth	6-6
Second molar	30-36	12-13
Third molar	96-120	17-21
Root calcification is complete 2-3 years after eruption. Typical eruption sequence: upper 6 1 2 4 5 3 7 8; lower 6 1 2 3 4 5 7 8.		

Turner's hypoplasia is an abnormality found in teeth. Its appearance is variable, though usually is manifested as a portion of missing or diminished enamel on permanent teeth. Unlike other abnormalities which affect a vast number of teeth, Turner's hypoplasia usually affects only one tooth in the mouth and, it is referred to as a Turner's tooth.



Picture 68. Turner's hypoplasia.

If Turner's hypoplasia is found on a canine or a premolar, the most likely cause is an infection that was present when the primary (baby) tooth was still in the mouth. Most likely, the primary tooth was heavily decayed and an area of inflamed tissues around the root of the tooth (called a periapical inflammation), affecting the development of the permanent tooth. The tooth most likely affected by this cause is the canine tooth. The appearance of the abnormality will depend on the severity and longevity of the infection.

If Turner's hypoplasia is found in the front (anterior) area of the mouth, the most likely cause is a traumatic injury to a primary tooth. The traumatized tooth, which is usually a maxillary central incisor, is pushed into the developing tooth underneath it and consequently affects the formation of enamel. Because of the location of the permanent tooth's developing tooth bud in relation to the primary tooth, the most likely affected area on the permanent tooth is the facial surface (the side closer to the lips or cheek). White or yellow discoloration may accompany Turner's hypoplasia. Enamel hypoplasia may also be present.

Turner's hypoplasia usually affects the tooth enamel if the trauma occurs prior to the third year of life. Injuries occurring after this time are less likely to cause enamel defects since the enamel is already calcified.

One of the forms of systemic hypoplasia is Hutchinson's teeth. Prenatal syphilis, the result of maternal infection, can cause a characteristic dental deformity, described by Hutchinson in 1858. If the fetus becomes infected at a very early stage, abortion follows. Infants born with stigmata of congenital syphilis result from later fetal infection, and the permanent teeth are affected. The characteristic defects are usually seen in the upper central incisors.



Picture 69. Hutchinson's incisors.

The incisors (Hutchinson's incisors) are small, barrel-shaped, and taper towards the tip. The incisal edge sometimes shows a crescentic notch or deep fissure which forms before eruption and can be seen radiographically. An anterior open bite is also characteristic. The first molars may be dome-shaped (Moon's molars) or may have a rough pitted occlusal surface with compressed nodules instead of cusps (mulberry molars). These defects are now largely of historical interest.

Pathology. The effects are due to infection of the dental follicle by *Treponema pallidum*. The postulated consequences are chronic inflammation, fibrosis of the tooth sac, compression of the developing tooth and distortion of the ameloblast layer. *T. pallidum* causes proliferation of the odontogenic

epithelium which bulges into the dentine papilla causing the characteristic central notch.

Question 8. Tetracycline discoloration.

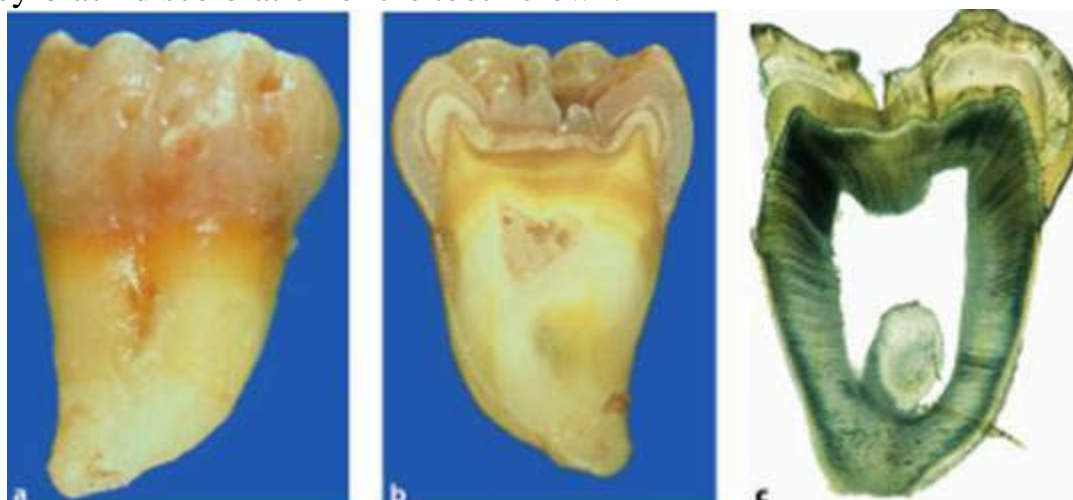
Tetracycline is taken up by calcifying tissues, and the band of tetracycline-stained bone or tooth substance fluoresces bright yellow under ultraviolet light. The teeth become stained only when tetracycline is given during their development, and it can cross the placenta to stain the developing teeth of the fetus. More frequently, permanent teeth are stained by tetracycline given during infancy. Tetracycline is deposited along the incremental lines of the dentine and, to lesser extent, of the enamel. The more prolonged the course of treatment the broader the band of stain and the deeper the discolouration. The teeth are at first bright yellow, but become a dirty brown or grey. The stain is permanent, and when the permanent incisors are affected the ugly appearance can only be disguised. When the history is vague the brownish colour of tetracycline-stained teeth must be distinguished from dentinogenesis imperfecta. In dentinogenesis imperfecta the teeth are obviously more translucent than normal and, in many cases, chipping of the enamel from the dentine can be seen. In tetracycline-induced defects the enamel is not abnormally translucent and is firmly attached to dentine. In very severe cases, intact teeth may fluoresce under ultraviolet light. It is no longer necessary to give tetracycline during dental development. There are equally effective alternatives and it should be avoided from approximately the fourth month to 12th year of childhood. Nevertheless tetracycline pigmentation is still seen.



Picture 70. Tetracycline discoloration.

Trough binding to calcium, tetracyclin is deposited together with calcium in any tissue undergoing mineralisation. After its incorporation during mineralisation, it can be demonstrated in teeth and bones in ultraviolet

light, showing up as fluorescent yellow bands. Grossly, tetracyclin causes a grey-black discoloration of the tooth crown.



Picture 71. Tetracycline discoloration.

When making ground sections of these teeth, the tetracyclin bands can be observed under UV light illumination both in dentin as well as in enamel, each band indicating a time point of tetracyclin administration. After decalcification the tetracyclin has been lost together with the calcium and therefore, in decalcified paraffin sections, this fluorescence is not present anymore.

Treatment of hypoplastic defects. Hypoplastic teeth can be disguised by restorative procedures such as veneers or jacket crowns. The latter should be delayed until adult life. The young pulp is large, is easily damaged during preparation of the tooth, and injuries are more frequent than in older persons.

Localised composite resin restorations. Defective enamel can be replaced with a tooth-coloured restoration that bonds to, and blends with, enamel. It is indicated for well-demarcated white, yellow or brown patches. The localised restoration is quick and easy to complete. Despite the removal of defective enamel down to the amelodentine junction, there is often no significant sensitivity and, therefore, no need for local anaesthesia. If the hypoplastic enamel has become carious and extends into dentine, a liner of glass ionomer cement (correct shade) prior to placement of composite resin will be necessary. In these cases, local anaesthesia will probably be required. Advances in bonding and resin technology make these restorations simple and obviate the need for a full labial veneer. Disadvantages are marginal staining, difficulty in achieving an accurate colour match and reduced composite translucency when lined by a glass ionomer cement.

Composite resin veneers. Although the porcelain jacket crown (PJC) may be the most satisfactory long-term restoration for a severely hypoplastic or discoloured tooth, it is not an appropriate solution for children for two reasons: the large size of the young pulp horns and chamber, and the immature gingival contour. Composite veneers may be direct (placed at

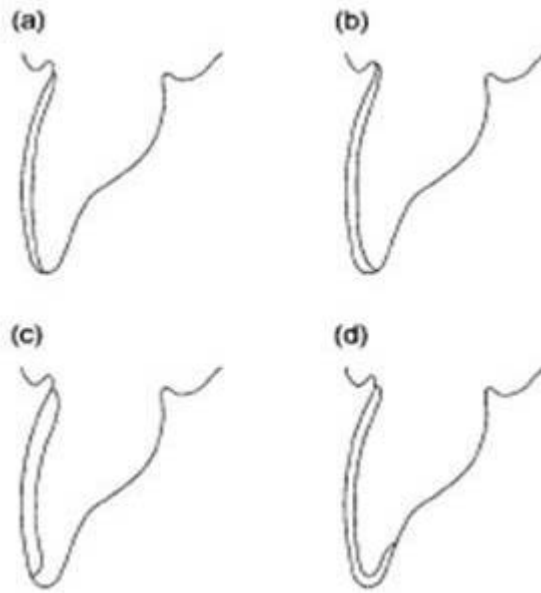
initial appointment) or indirect (placed at a subsequent appointment having been fabricated in the laboratory). The conservative veneering methods may not just offer a temporary solution but may also offer a satisfactory long-term alternative to the PJC. Most composite veneers placed in children and adolescents are of the 'direct' type as the durability of the indirect composite veneers is as yet unknown. Composite veneers are durable enough to last through adolescence. Before proceeding with any veneering technique, the decision must be made whether to reduce the thickness of labial enamel before placing the veneer. Certain factors should be considered:

- increased labiopalatal bulk makes it harder to maintain good oral hygiene; this may be courting disaster in the adolescent with dubious oral hygiene
- composite resin has a better bond strength to enamel when the surface layer of 200-300 μm is removed
- if a tooth is very discoloured, some sort of reduction will be desirable as a thicker layer of composite will be required to mask the intense stain
- if a tooth is already instanding or rotated, its appearance can be enhanced by a thicker labial veneer.

New-generation, highly polishable hybrid composite resins can replace relatively large amounts of missing tooth tissue as well as being used in thin sections as a veneer. Combinations of shades can be used to stimulate natural colour gradations and hues. The exact design of the composite veneer will vary with each patient. Usually it will be one of four types: intraenamel or window preparation, incisal bevel, overlapped incisal edge or feathered incisal edge.

Indications:

- ✓ discoloration,
- ✓ enamel defects,
- ✓ diastemata,
- ✓ malpositioned teeth,
- ✓ large restorations.



Picture 72. Types of veneer preparation

- (a) Feathered incisal edge, (b) Incisal bevel preparation,
(c) Intra-enamel or window preparation,
(d) Overlapped incisal edge preparation.

Question 9. Hereditary disturbances in tooth structure, not elsewhere classified.

K00.5 Hereditary disturbances in tooth structure, not elsewhere classified

Amelogenesis imperfecta (AI) presents with a rare abnormal formation of the enamel or external layer of the crown of teeth. Enamel is composed mostly of mineral, that is formed and regulated by the proteins in it. Amelogenesis imperfecta is due to the malfunction of the proteins in the enamel: ameloblastin, enamelin, tuftelin and amelogenin.

Mutations in the AMELX, ENAM, MMP20, KLK-4, FAM83H, WDR72, C4orf26, SLC24A4 LAMB3 and ITGB6 genes have been found to cause amelogenesis imperfecta (non-syndromic form). AMELX and ENAM encode extracellular matrix proteins of the developing tooth enamel and KLK-4 and MMP20 encode proteases that help degrade organic matter from the enamel matrix during the maturation stage of amelogenesis. SLC24A4 encodes a calcium transporter that mediates calcium transport to developing enamel during tooth development. Less is known about the function of other genes implicated in amelogenesis imperfecta.

Amelogenesis imperfecta can have different inheritance patterns depending on the gene that is altered. Mutations in the ENAM gene are the most frequent known cause and are most commonly inherited in an autosomal dominant pattern. This type of inheritance means one copy of the altered gene in each cell is sufficient to cause the disorder.

Amelogenesis imperfecta is also inherited in an autosomal recessive pattern; this form of the disorder can result from mutations in the ENAM, MMP20, KLK4, FAM20A, C4orf26 or SLC24A4 genes. Autosomal recessive inheritance means two copies of the gene in each cell are altered.

About 5% of amelogenesis imperfecta cases are caused by mutations in the AMELX gene and are inherited in an X-linked pattern. A condition is considered X-linked if the mutated gene that causes the disorder is located on the X chromosome, one of the two sex chromosomes. In most cases, males with an X-linked form of this condition experience more severe dental abnormalities than affected females.

Recent genetic studies suggest that the cause of a significant proportion of amelogenesis imperfecta cases remains to be discovered.

People afflicted with amelogenesis imperfecta have teeth with abnormal color: yellow, brown or grey; this disorder can afflict any number of teeth of both dentitions. The teeth have a higher risk for dental cavities and are hypersensitive to temperature changes as well as rapid attrition, excessive calculus deposition, and gingival hyperplasia.

The clinical features are variable and depend on which gene is defective:

- ✓ In some forms the teeth appear normal on eruption but, because the enamel is poorly mineralised, it is soft and soon wears away. The remaining enamel becomes stained and the teeth appear yellow–brown.
- ✓ In other forms the enamel is reduced in amount and the teeth are pitted and appear yellow–brown in colour.
- ✓ Some forms are carried on the X chromosome and boys are affected more severely than girls.
- ✓ The teeth are often sensitive, especially where the enamel is thin or has chipped away.

Hypomaturation amelogenesis imperfecta. The enamel is normal in form on eruption but opaque, white to brownish-yellow. The teeth appear similar to mottled fluoride effects. However, they are soft and vulnerable to attrition, though not as severely as the hypocalcified type. There are several variants of hypomaturation defects such as a more severe, autosomal dominant (type 4) of hypomaturation combined with hypoplasia.



Picture 73. Amelogenesis imperfecta, hypomaturational type.

Hypocalcified amelogenesis imperfecta. Enamel matrix is formed in normal quantity but poorly calcified. When newly erupted, the enamel is normal in thickness and form, but weak and opaque or chalky in appearance. The teeth tend to become stained and relatively rapidly worn away. The upper incisors may acquire a shouldered form due to the chipping away of the thin, soft enamel of the incisal edge. There are dominant and recessive patterns of inheritance.



Picture 74. Amelogenesis imperfecta, hypocalcified type.

Treatment. Preventive and restorative dental care is very important as well as considerations for esthetic issues since the crown are yellow from exposure of dentin due to enamel loss. Full-coverage crowns are sometimes being used to compensate for the abraded enamel. Usually stainless steel crowns are used in children which may be replaced by porcelain once they reach adulthood. In the worst-case scenario, the teeth may have to be extracted and implants or dentures are required. Loss of nerves in the affected teeth may occur.

Dentinogenesis imperfecta is a genetic disorder of tooth development. This condition is a type of dentin dysplasia that causes teeth to be discolored

(most often a blue-gray or yellow-brown color) and translucent giving teeth an opalescent sheen. Teeth are also weaker than normal, making them prone to rapid wear, breakage, and loss. These problems can affect both primary (deciduous) teeth and permanent teeth. This condition is inherited in an autosomal dominant pattern, which means one copy of the altered gene in each cell is sufficient to cause the disorder. Dentinogenesis imperfecta affects an estimated 1 in 6,000 to 8,000 people.



Picture 75. Dentinogenesis imperfecta

Types of dentinogenesis imperfecta

Type I: Type of dentinogenesis imperfecta with similar dental abnormalities usually an autosomal dominant trait with variable expressivity but can be recessive if the associated osteogenesis imperfecta is of recessive type.

Type II: Occurs in people without other inherited disorders (i.e. Osteogenesis imperfecta). It is an autosomal dominant trait. A few families with type II have progressive hearing loss in addition to dental abnormalities. Also called hereditary opalescent dentin.

Type III: Type is rare; its predominant characteristic is bell-shaped crowns, especially in the permanent dentition. Unlike Types I and II, it involves teeth with shell-like appearance and multiple pulp exposures.

Mutations in the DSPP gene have been identified in people with type II and type III dentinogenesis imperfecta. Type I occurs as part of osteogenesis imperfecta.

Clinical appearance is variable. However, the teeth usually involved and more severely affected are primary teeth in type I; whereas in type II both the dentitions are equally affected.

The teeth may be gray to yellowish brown. They exhibit translucent or opalescent hue. Enamel is usually lost early due to loss of scalloping at the

dentoenamel junction (DEJ). However, the teeth are not more susceptible to dental caries than normal ones.

However, certain patients with dentinogenesis imperfecta will suffer from multiple periapical abscesses apparently resulting from pulpal strangulation secondary to pulpal obliteration or from pulp exposure due to extensive coronal wear. They may need apical surgery to save the involved teeth.

Radiographic features. Type I and II show total obliteration of the pulp chamber. Type III shows thin dentin and extremely enormous pulp chamber. These teeth are usually known as "shell teeth".

Histology. Dentinal tubules are irregular and are bigger in diameter. Areas of uncalcified matrix are seen. Sometimes odontoblasts are seen in dentin.

Treatment. Preventive and restorative care are important as well as esthetics as a consideration. In most cases, full-coverage crowns are needed for esthetic appearance, as well as to prevent further attrition. Another treatment option is bonding, putting lighter enamel on the weakened enamel of the teeth and with lots of treatments of this bonding, the teeth appear whiter to the eye, but the teeth on the inside and under that cover are still the same. Due to the weakened condition of the teeth, many common cosmetic procedures such as braces and bridges are inappropriate for patients with Dentinogenesis imperfecta and are likely to cause even more damage than the situation they were intended to correct.

Dental whitening (bleaching) is contraindicated although it has been reported to lighten the color of DI teeth with some success; however, because the discoloration is caused primarily by the underlying yellow-brown dentin, this alone is unlikely to produce normal appearance in cases of significant discoloration.

Dentin dysplasia is a genetic disorder of teeth, commonly exhibiting an autosomal dominant inheritance. It is characterized by presence of normal enamel but atypical dentin with abnormal pulpal morphology. There are two types. Type I is the radicular type, and type II is the coronal type. In the radicular type, the roots of teeth are shorter than normal and the pulp chamber may be nearly gone. The pulp chamber is sometimes described as having a "crescent shaped" appearance. In the coronal type, the pulps are enlarged and are described as having a "thistle tube" appearance, in permanent dentition. In the deciduous dentition, coronal dentin dysplasia bears a resemblance to Dentinogenesis Imperfecta type II.

Radiographic Features:

Type I: Roots are short, blunt and conical. In deciduous teeth, pulp chambers and root canals are completely obliterated while in permanent they may be crescent shaped.

Type 2: The pulp chamber of the deciduous teeth become completely obliterated. The permanent teeth displays large pulp chamber in the coronal portion of the tooth referred to as thistle tube appearance. Pulp stones may be found.

Question10. Disturbances in tooth eruption.

K00.6 Disturbances in tooth eruption. Natal teeth are teeth that are present above the gumline (have already erupted) at birth, and neonatal teeth are teeth that emerge through the gingiva during the first month of life (the neonatal period).

- ✓ The incidence of neonatal teeth varies considerably, between 1:700 and 1:30,000 depending on the type of study; the highest prevalence is found in the only study that relies on personal examination of patients.
- ✓ Most often natal teeth are mandibular central incisors. They have little root structure and are attached to the end of the gum by soft tissue and are often mobile.
- ✓ Cause: a developmental disturbance creating intracellular activity during the first stage of tooth development (bud stage) can result in the development of extra teeth.
- ✓ Most of the time, natal teeth are not related to a medical condition. However, sometimes they may be associated with:
 - ✓ Ellis–van Creveld syndrome.
 - ✓ Hallermann–Streiff syndrome.
 - ✓ Pierre Robin syndrome.
 - ✓ Sotos syndrome.

Treatment. Natal teeth: these teeth are defective and their removal is generally recommended, particularly if mobility poses a threat of aspiration. These teeth also make feeding difficult.

Neonatal teeth: these teeth are defective and their removal is generally recommended, particularly if mobility poses a threat of aspiration. These teeth also make feeding difficult.

Test tasks

1. Abnormalities of size and form of teeth include:

- a. tooth fusion
- b. tooth germination
- c. conrescence
- d. dens evaginatus
- e. *all answers are right*

2. What types of noncarious lesions do you know:

- a. noncarious lesions that occur after tooth eruption
- b. noncarious lesions that occur before tooth eruption

c. *all answers are right*

3. What concentration of fluorine in water is optimal:

- a. *0,8 – 1,2 mg/l*
- b. 2,5 – 3 mg/l
- c. 0,3- 0,4 mg/l
- d. all answers are right

4. The main cause of fluorosis is:

- a. *excessive fluorine consumption*
- b. poor oral hygiene
- c. insufficient fluorine consumption

5. What types of enamel hypoplasia according to the etiology do you know:

- a. systemic
- b. localized
- c. general
- d. *all answers are right*

6. Grading of mottled enamel:

- a. very mild
- b. mild
- c. moderate
- d. severe
- e. *all answers are right*

7. According to the Dean`s index mild fluorosis is characterized by:

- a. all tooth surfaces affected; marked wear on biting surfaces; brown stain may be present
- b. small opaque, paper white areas covering less than 25% of the tooth surface
- c. *opaque white areas covering less than 50% of the tooth surface*

8. According to the Dean`s index moderate fluorosis is characterized by:

- a. all tooth surfaces affected; marked wear on biting surfaces; brown stain may be present
- b. small opaque, paper white areas covering less than 25% of the tooth surface
- c. opaque white areas covering less than 50% of the tooth surface
- d. *all tooth surfaces affected; discrete or confluent pitting; brown stain present*

9. What teeth are the most susceptible to the hypoplasia:

- a. *the first permanent molars*
- b. *upper central incisors*
- c. second molars
- d. *lower lateral incisors and canines*
- e. all answers are right

10. Tetracycline discoloration arises when tetracycline is given:

- a. *during tooth development*
- b. after tooth development
- c. before tooth development
- d. all answers are right.

LESSON 17. NONCARIOUS LESIONS THAT APPEAR AFTER TEETH ERUPTION.

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

1. Classification of noncarious lesions that appear after teeth eruption.
2. Excessive attrition of teeth.
3. Teeth abrasion.
4. Wedge defect.
5. Teeth erosion.
6. Teeth abfraction.
7. Pathological resorption of teeth.
8. Hypercementosis.
9. Ankylosis of teeth.
10. Trauma of teeth.

Question 1. Classification of noncarious lesions that appear after teeth eruption.

Noncarious tooth lesions is a common problem. After tooth eruption exposed dentin can result from acidic erosion, abrasion, and attrition, but most toothwear has erosion as the dominant etiological factor. Localized anterior toothwear of the upper anterior teeth is often caused by the consumption of erosive carbonated beverages, fruit juices, and citrus fruits. Regurgitated stomach acid in gastroesophageal reflux disease, hiatus hernia, and esophagitis and vomiting in bulimia, alcoholism, and psychosomatic disorders can cause erosive tooth wear of the palatal surfaces of the anterior teeth. Drugs that tend to reduce the amount of saliva in the mouth, such as antidepressants, recreational drugs (LSD and Ecstasy, which is 3,4-methylene-dioxymethamphetamine), and diuretics, also diminish the buffering capacity available to neutralize dietary or stomach acids. Users of

Ecstasy commonly complain of a dry mouth, and erosion from carbonated beverages is thought to be an important etiological factor. However, the occlusal surfaces of the molar teeth are more commonly affected than the incisor teeth, which would indicate that jaw clenching and masseter muscle hyperactivity are important (Milosevic et al. 1999).

According to International classification of diseases there are following noncarious lesions that appear after teeth eruption:

K03 Other diseases of hard tissues of teeth

K03.0 Excessive attrition of teeth:

- approximal
- occlusal

K03.1 Abrasion of teeth:

- dentifrice
- habitual
- occupational
- ritual
- traditional

Wedge defect

K03.2 Erosion of teeth

Erosion of teeth due to:

- diet
- drugs and medicaments
- persistent vomiting
- idiopathic
- occupational

K03.3 Pathological resorption of teeth

Internal granuloma of pulp

Resorption of teeth (external)

K03.4 Hypercementosis

Cementation hyperplasia

K03.5 Ankylosis of teeth

K03.7 Posteruptive colour changes of dental hard tissues

K03.8 Other specified diseases of hard tissues of teeth

Irradiated enamel

Sensitive dentine

K03.9 Disease of hard tissues of teeth, unspecified

Question 2. Excessive attrition of teeth.

K03.0 Excessive attrition of teeth. Attrition is the process of wearing away of enamel, which occurs physiologically as a consequence of mastication. This process is very slow and results in a gradual loss of enamel. Pindborg distinguished three types of attrition: physiological, excessive and

pathological. The dental pulp cavity is usually not open due to attrition. Dental attrition is a type of tooth wear caused by tooth-to-tooth contact, resulting in loss of tooth tissue, usually starting at the incisal or occlusal surfaces.

Causes. Advanced and excessive wear and tooth surface loss can be defined as pathological in nature, requiring intervention by a dental practitioner. Excessive occlusal wear may occur in the following situations:

- Patients with bruxism. These patients grind their teeth excessively and in some cases this is triggered by occlusal irregularities.
- Patients who have lost several posterior teeth may show excessive attrition of the anterior teeth, especially if these are used for chewing.
- Patients who suffer from developmental disturbances of tooth structure, such as amelogenesis and dentinogenesis imperfecta, may suffer exceptional tooth wear.

The etiology of dental attrition is multifactorial however bruxism is one of the most common causes of attrition. Bruxism is the parafunctional movement of the mandible, occurring during the day or night. It can be associated with presence of audible sound when clenching or grinding the teeth. This is usually reported by parents or partners if the grinding occurs during sleep.

In some cases dental erosion is also associated with severe dental attrition. Dental erosion is tooth surface loss caused by extrinsic or intrinsic forms of acid. Extrinsic erosion is due to a highly acidic diet, whilst intrinsic erosion is caused by regurgitation of gastric acids. Erosion softens the dental hard tissues making them more susceptible to dental attrition. When dental erosion is present in conjunction with bruxism the tooth surface loss due to attrition is accelerated due to the erosive environment. Severe attrition in young patients is usually associated with erosive factors in their diets. The different physiological processes of tooth wear (abrasion, attrition and erosion) generally occur simultaneously and rarely work individually. Therefore it is important to obtain knowledge of these tooth wear processes and their interactions to determine causes of tooth surface loss. Demineralization of the tooth surface due to acids can cause occlusal erosion as well as attrition. Wedge-shaped cervical lesions are commonly found in association with occlusal erosion and attrition.

Tooth wear is typically seen in the elderly and can be referred to as a natural aging process. Attrition, abrasion, erosion or a combination of these factors are the main reasons for tooth wear in elderly people who retain their natural teeth. This tooth wear can be pathological or physiological in nature. The influence of age on tooth wear shows that the number of teeth with incisal or occlusal wear increases with the age. Dental attrition occurs in 1 in 3 adolescents and an association has been established between dental attrition and aging.

Gender has also been determined as a contributing factor associated with occlusal tooth wear. In addition to other occlusal factors some independent variables such as male gender, bruxism, and loss of molar occlusal contact, edge-to-edge relation of incisors, unilateral buccolingual cusp-to-cusp relation, and unemployment have been identified in affecting occlusal wear. Similarly anterior cross-bite, unilateral posterior cross-bite, and anterior crowding have been found to be protective factors for high occlusal wear levels.

Clinical indications of attrition can include:

- Loss of tooth anatomy: This results in loss of tooth characteristics including rounding or sharpening of incisal edges, loss of cusps and fracturing of teeth. Enamel of molar teeth may appear thin and flat. When in occlusion the teeth may appear the same height which is particularly apparent for anterior teeth.
- Sensitivity or pain: Attrition may be entirely asymptomatic, or there may be dentin hypersensitivity secondary to loss of the enamel layer, or tenderness of the periodontal ligament caused by occlusal trauma.
- Tooth discolouration: A yellow appearance of the tooth surface may be due to the enamel being worn away, exposing the darker yellower dentin layer underneath.
- Altered occlusion due to decreasing vertical height, or occlusal vertical dimension.
- Compromised periodontal support can result in tooth mobility and drifting of teeth.
- Loss in posterior occlusal stability.
- Mechanical failure of restorations.



Picture 76. Excessive attrition of teeth.

Prevention and management. To manage the condition it is first important to arrive at a diagnosis, describing the type of tooth surface loss, the severity and location. Early diagnosis is essential to ensure tooth wear has not progressed past the point of restoration. The examination should include assessment of

- ✓ Temporomandibular joint function and associated musculature

- ✓ Orthodontic examination
- ✓ Intraoral soft tissue analysis
- ✓ Hard tissue analysis
- ✓ Location and severity of tooth wear
- ✓ Social history;
- ✓ Diet.

It is important to record severity of tooth wear for monitoring purposes, helping to differentiate between pathological and physiological tooth surface loss. It is essential to determine whether the tooth wear is ongoing or has stabilized. However where generalised one can assume the underlying cause is bruxism. In fast progressing cases there is commonly a coexisting erosive diet contributing to tooth surface loss.

When a diagnosis of bruxism has been confirmed it is recommended that the patient purchase a full coverage acrylic occlusal splint. Patients must be monitored closely, with clinical photographs 6–12 monthly to evaluate if the tooth surface loss is being prevented.



Picture 77. Occlusal splint.

Treatment. Cosmetic or functional intervention may be required if tooth surface loss is pathological in nature or if there has been advanced loss of tooth structure. The first stage of treatment involves the management of any associated conditions such as fractured teeth or sharp cusps or incisal edges. These can be resolved via restoration of and polishing of sharp cusps. At this stage desensitizing agents such as topical fluoride varnishes can be applied, and at home desensitising toothpastes recommended. There are many different restorative treatment options which have been proposed such as direct composite restorations, bonded cast metal restorations, removable partial dentures, orthodontic treatment, crown lengthening procedures and protective splints. The decision to restore the dentition depends on the wants and needs of the patient, the severity of tooth surface loss and whether tooth surface loss is active. The use of adhesive materials to replace lost tooth structure can be performed as a conservative and cost effective approach before a more permanent solution of crowns or veneers is considered.

Question 3. Teeth abrasion.

K03.1 Abrasion of teeth. Abrasion is the loss of tooth structure by mechanical forces from a foreign element. If this force begins at the cemento-enamel junction, then progression of tooth loss can be rapid since enamel is very thin in this region of the tooth. Once past the enamel, abrasion quickly destroys the softer dentin and cementum structures.

Abrasion is seen at cervical necks of the teeth, as a deep ridge on the buccal or labial surfaces. The surface is shiny rather than carious, and sometimes the ridge is deep enough to see the pulp chamber within the tooth itself. The teeth most commonly affected are premolars and canines.

Causes of abrasion :

- Traumatic occlusion.
- Unproper brushing technique.
- Occupational (Habits such as holding bobby pins in between the teeth).
- Tobacco chewing /tobacco pipe.
- Vigorous use of tooth picks between the adjacent teeth.
- Excessive mastication of coarse foods.

Iatrogenic causes:

- Dentures with porcelain teeth opposing natural teeth.
- Extremely rough occluding surface of the restoration enhancing its abrasive capability .
- Ill fitting dentures and clasps, producing a constant wear of the affected surfaces.



Picture 78. Teeth abrasion



Picture 79. Teeth abrasion.

Tooth brush abrasion results in a horizontal cervical notches on the buccal surfaces of exposed radicular cementum and dentin. Notching in right central incisor caused by improper use of bobby pins.

The clinical signs and symptoms of an abrasion are:

- The surface of the lesion is extremely smooth and polished and it seldom has any plaque accumulation or caries activity in it.
- The surrounding walls tend to make a V shape ,by meeting at an acute angle axially.
- Peripheries of the lesion are angularly demarcated from the adjacent tooth surface.
- Probing or stimulating the lesion can elicit pain.
- Hypersensitivity may be intermittent in character appearing and disappearing at occasional or frequently repeated periods

Treatment modalities

- 1.Diagnose the cause of the presented abrasion.
- 2.A detailed history is to be taken considering various factors such as:
 - Oral hygiene techniques (use of abrasive tooth cleaning techniques and materials)
 - Habits- pipe smoking, chewing tobacco, professional habits
 - Iatrogenic causes, if any.
- 3.Avoidance or counteraction of the causes which may lead to its production.
- 4.Instituting proper oral hygiene measures.
- 5.Judiciously tooth brushing with a dentifrice i.e. incorporating correct method of tooth brushing Have the habit of chewing tobacco ,toothpick , etc discontinued. If successful in breaking the habit proceed with the restorative treatment as planned.
- 6.Correcting or avoiding ill fitting metal clasps and dentures
- 7.Abrasive lesions at non-occluding tooth surfaces should be:
- 8.Evaluated critically for the need for restoring them.

9.If the lesions are multiple, shallow(not exceeding 0.5 mm in dentin) and wide → no need to restore them.

10.If there is involvement of cementum/enamel only → no need to restore .

11.If lesion is wedge (V) shaped and exceeds 0.5 mm into dentin → restoration is performed.

12.If restoration is not indicated for a lesion, then :

- edges of the defect should be eradicated to a smooth, non- demarcating pattern relative to adjacent tooth surface.
- tooth surface then should be treated by fluoride solution to improve caries resistance

13. If the involved teeth→ extremely sensitive: desensitize the exposed dentin before restoration.

Desensitization:

- 8-10% sodium/stannous fluorides for 4-8 minutes.
- Ionophoresis- -using an electrolyte containing fluorides(galvanic energy supplied to the tooth in the presence of electrolyte, drives ions deep into the dentin)

Restorative materials. High modulus restorative materials are unable to flex in the cervical regions when the tooth structure is deformed under occlusal load and ,therefore the restorative materials can be displaced from the cavity.

An intermediate material with reduced elastic modulus may function as a stress absorbing layer and improve marginal sealing.

As a result materials with low elastic modulus such as: microfilled composites, flowable resins, glass ionomer cements have been used in restoring cervical lesions ,with the aim of absorbing the stresses generated during the polymerization shrinkage of composites and mechanical loading in which the teeth are subjected during function.

Question 4. Wedge defect.

The wedge-shaped defects refers to the teeth, lips, buccal neck dental hard tissue slowly consumed due to defects. This defect was "shaped, like woodworking wedge, hence the name wedge-shaped defect. One at the junction of the wedge-shaped defects occur in the lip and cheek surfaces of the canine and premolar tooth enamel, some patients confined to unilateral disease some bilateral occurred, individual full mouth of teeth can occur. Have a greater difference in the extent of the defect in the same patient. Early there is a small amount of hard tissue defect gradually increased after the formation of tooth neck.

Defects at the tooth surface smooth, hard and without significant color change. Mild wedge-shaped defects may be no symptoms, heavy sensitive symptoms due to dentin exposure, performance eating a certain food, fruit or brushing pain symptoms, defects gradually increased, a series of lesions can

be caused by the pulp, or even because of defect caused by excessive teeth broken from the neck. The real cause of the wedge-shaped defect is unclear, may be related to the following factors.



Picture 80. Wedge defect.

Acid secretion. Chemical factors gingival sulcus often acting on the tooth neck, low pH of saliva, like to eat acidic foods, food residue accumulation of fermentation acid may cause dental hard tissue decalcification gradually dissolved and loss.

Some physical factors teeth mechanical friction often can accelerate dental hard tissue loss. Clinically found a higher incidence of horizontal brush teeth, lip and cheek surfaces using a hard bristle toothbrush, wedge-shaped defects.

Treatments of Wedge-Shaped Defects

Wedge-shaped defects early hard tissue defects and no obvious symptoms, local do not need special treatment, but to be paid to the choice of a soft bristle toothbrush with a the vertical brush or rotary brushing, and attention to weak alkaline gargle mouthwash: formation defect and allergic symptoms, do desensitization treatment or filling to repair the defect. Commonly desensitization many ways. Here are some kinds.

1. Fluoride treatment, wedge-shaped defects.Teeth topcoat fluorine may be formed - fluoride hydroxyapatite processing dentin hypersensitivity, fluoride ions can reduce the diameter of dentinal tubules and the formation of the fluorine-containing compound blocking the transmission of a small tube with the blocking stimulation, but also promote a more rigid dentin formation. Sodium fluoride treatment of wedge-shaped defects: 33% sodium fluoride paste is the most effective, with 75% sodium fluoride glycerin paste domestic. General use cotton balls dipped in paste repeatedly rubbed sensitive at 2 to 3min, 1 times / d, 10 times as a course of treatment.

2. The wedge-shaped defect stannous fluoride treatment. It is reported that a low concentration of stannous fluoride to effectively control dentin hypersensitivity. Sodium fluoride, stannous fluoride, sodium

monofluorophosphate toothpaste teeth or partial erasure, some degree of desensitization.

3. Calcium hydroxide treatment of wedge-shaped defects can accelerate dentin mineralization, reduce the permeability of dentin, generally with distilled water and transferred into a paste of calcium hydroxide paste with a brush to brush is to dry the allergy tooth surface and maintain 5min, and then paste remove and rinse 1 a day, a week for a course of treatment. Also, it was argued, paste in root surface friction or used for periodontal surgery dressings will be a good tune to the prevention or treatment of dentine hypersensitivity.

4. Strontium chloride treatment of wedge-shaped defects. Has been confirmed that the strontium can penetrate calcified tissue (dentin). Strontium is deposited in a small tube, to reduce the permeability of the dentin. Also found that the application of the calcium and strontium, the mineralization to be higher than when using calcium alone, strontium ions in the accelerating calcification, or even block the dentinal tubules works.

5. Resins and adhesives treatment of wedge-shaped defects. Without filler tooth surface coating resin (such as epoxy resin), medical adhesive, etc., can be closed dentinal tubules, immediately effective, effective time up to one year, after falling reusable has been reported light-cured dentin bonding agent to bond the dentin surface coating light 20s, generally 1 or 2 can be effective.

6. Iontophoresis treatment of wedge-shaped defects. This method is more complex and needs a long time, apply to full mouth of teeth, or the majority of the teeth sensitive the general import fluoride or calcium, or both alternating import. Has been reported, and then import with 2% sodium fluoride ionizing light cured dentin bonding agent is applied to the light, the effect is very good.

When left untreated, the wedge-shaped defect continues to deepen to the point of absurd grooves that frequently provoke the formation of secondary dentin in the root canal. An overlay of caries accelerates the process. In most cases, the progress of the formation of the wedge-shaped defect cannot even be stopped with a correction of the tooth brushing method. The bristles of the tooth brush are pressed together in the already existing wedge-shaped indentation (like in a guiding groove) and the sawing and abrasive effects can hardly be avoided, even if techniques recommended for periodontal diseases are applied.

Besides endangering the pulp, a fracture of the tooth may also occur due to the wedge-shaped defect. Up to now, there is a widespread opinion that only larger wedge-shaped defects must be treated with a filling or a crown.

After trials lasting several years, the experience gained in treating wedge-shaped defects with glass ionomer cements give rise to hope. These

materials adhere to dentin and enamel, when they are freed from organic deposits by having been processed with a cleaning compound and being dry on the surface. Whether an additional pretreatment with citric acid improves the bond cannot yet be said with the necessary certainty.

Question 5. Teeth erosion.

K03.2 Erosion of teeth. Teeth erosion – loss of surface tooth structure by chemical action in the continued presence of demineralizing agents (acids). It is one of the most predominant oral pathologic changes. There is no convincing etiology, and multiple factors have been theorized for its pathogenesis.

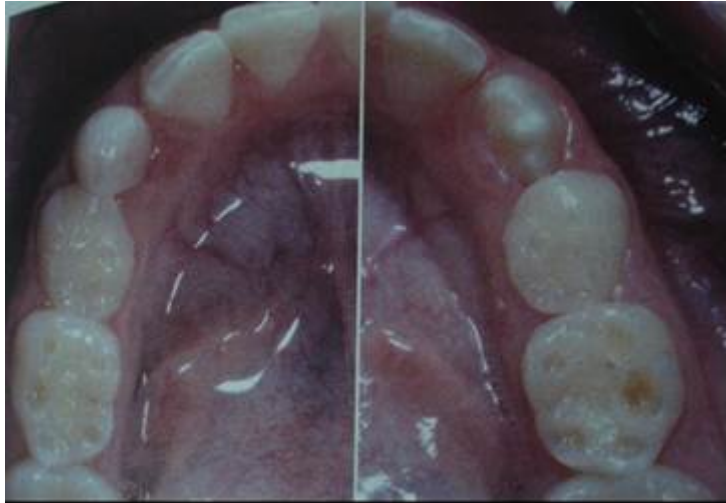
Etiology. Mechanical factors: the action of the muscles of lips and cheeks, and of tooth brush against affected surfaces.

Chemical factors:

- Ingested acids: citric acids (lemon and citrus fruits) esp. If use in large amounts , can precipitate or initiate erosive lesion
- Secreted acids: the acidity of crevicular fluid has been correlated to cervical erosion.
- Acid fumes: acid vapours from nitric acid and sulphuric acids, acting in the mouths of workers in the factories ,where these acids are largely used or manufactured
- Refused acids: as a result of chronic , frequent regurgitation ,the stomach's hydrochloric acid can hit the teeth at specific locations (atypical pattern of erosion affecting buccal surfaces of lower posterior teeth)
- The latter defective surfaces are associated with gastro esophageal reflux.



Picture 81. Occlusal view of maxillary dentition exhibiting concave dentin depressions surrounded by elevated rims of enamel



Picture 82. Multiple cupped out depressions corresponding to the cusp tip.



Picture 83. Extensive loss of enamel and dentin on the buccal surface of maxillary bicuspid. (pt had sucked chronically on tamarinds).



Picture 84. Palatal surfaces of maxillary dentition in which the exposed dentin exhibits a concave surface and a peripheral white line of enamel.

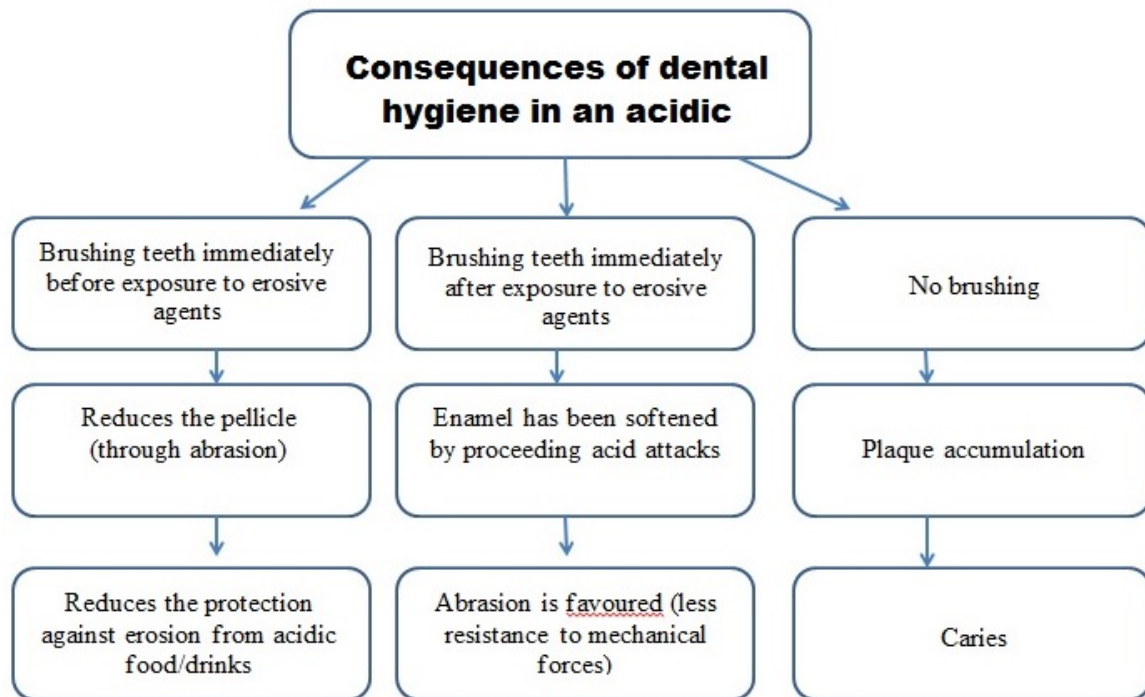
The other substances that can corrode teeth.

- chewable vitamin C tablets.
- aspirin tablets.
- aspirin powders.
- use of the amphetamine drug Ecstasy.

Protocol for the prevention of progression of erosion

- Diminish the frequency and severity of acid challenge.
 - Decreasing the amount and frequency of acidic foods or drinks.
 - Acidic drinks should be drunk quickly rather than sipped.
 - Using of straw reduces erosive potential.
- Treating the underlying medical disorder or disease.
- Enhance the defense mechanisms of body:
 - Saliva provides buffering capacity → increases with salivary flow rate.
 - Saliva supersaturated with Ca, P → inhibits demineralization of tooth structure.
 - Stimulation of salivary flow → sugarless lozenge, candy/gum is recommended
- Enhance acid resistance, remineralization and rehardening of the tooth surfaces.
 - Daily use topical flouride at home
 - Fluoride application in office- 2-4 times a year, flouride varnish recommended.
- Decrease abrasive forces.
 - Use a soft bristled toothbrush and brush gently.
 - No brushing should be done immediately after consuming acidic food and drink as teeth will be softened.

- Rinsing with water is better than brushing after consuming acidic foods and drinks.



- Improve chemical protection
 - Neutralize acids in mouth - dissolving sugar free antacid tablets 5 times a day, particularly after an intrinsic or extrinsic acid challenge.
 - Dietary components- hard cheese (provides Ca and PO_4), held in mouth after acidic challenge.
- Mechanical protection
 - By application of composites and direct bonding where appropriate – to protect exposed dentin
 - Occlusal guard /Acrylic splint in the form of stabilization splint necessary to protect dentition from further damage due to erosion.
- Monitor stability:
 - Use of casts/photos to document tooth wear status.
 - Regular recall examinations to review diet, oral hygiene methods, compliance with medications, topical fluoride and splint usage.
- Restoration
 - Metallic restorations should be the choice of material,if restoration indicated (more resistant to erosion).
 - Tooth colored materials may also be used with minimal or no tooth preparation, with the assumption that restoration may require periodic replacement.

Question 6. Teeth abfraction

Abfraction is a theoretical concept explaining a loss of tooth structure not caused by tooth decay (non-carious cervical lesions). It is suggested that these lesions are caused by forces placed on the teeth during biting, eating, chewing and grinding; the enamel, especially at the cementoenamel junction (CEJ), undergoes large amounts of stress, causing micro fractures and tooth tissue loss.

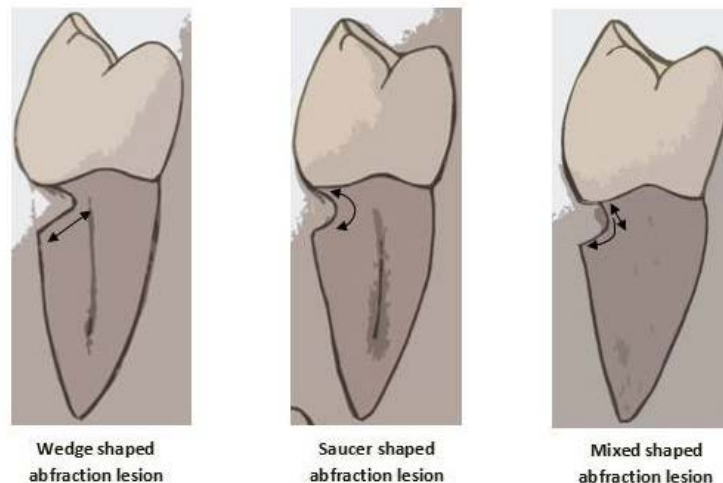


Picture 85. Teeth abfraction.

Abfraction is a form of non-carious tooth tissue loss that occurs along the gingival margin. In other words, abfraction is a loss of tooth structure that is not caused by tooth decay, located along the gum line. There has been a lot of theoretical evidence to support the concept of abfraction, but little experimental evidence exists.

The term abfraction was first published in 1991 in a journal article dedicated to distinguishing the lesion. The article was titled "Abfractions: A New Classification of Hard Tissue Lesions of Teeth" by John O. Grippo. This article introduced the definition of abfraction as a "pathologic loss of hard tissue tooth substance caused by bio mechanical loading forces". This article was the first to establish abfraction as a new form of lesion, differing from abrasion, attrition, and erosion.

Tooth tissue is gradually weakened causing tissue loss through fracture and chipping or successively worn away leaving a non-carious lesion on the tooth surface. These lesions occur in both the dentine and enamel of the tooth. These lesions generally occur around the cervical areas of the dentition.



Picture 86. Types of abfraction lesions.

Abfraction lesions will generally occur in the region on the tooth where the greatest tensile stress is located. In statements such as these there is no comment on whether the lesions occur above or below the CEJ. One theory suggests that the abfraction lesions will only form above the CEJ. However, it is assumed that the abfraction lesions will occur anywhere in the cervical areas of affected teeth. It is important to note that studies supporting this configuration of abfraction lesions also state that when there is more than one abnormally large tensile stress on a tooth two or more abfraction lesions can result on the one surface.

When looking at abfraction lesions there are generally three shapes in which they appear, appearing as either wedge, saucer or mixed patterns. Wedge and saucer shaped lesions are the most common, whereas mixed lesions are less frequently identified in the oral cavity. Wedge shaped lesions have the sharpest internal line angles and saucer/mixed shaped lesions are either smooth internally, or a variety.

Clinically, people with abfraction lesions can also present with tooth sensitivity in the associated areas. This occurs because as the abfraction lesions appear, dentine/cementum is exposed. The dentine and cementum are less dense than tooth enamel and therefore more susceptible to sensation from thermal/mechanical sources.

Causes. As abfraction is still a controversial theory there are various ideas on what causes the lesions. Because of this controversy the true causes of abfraction also remain disputable. Researchers have proposed that abfraction is caused by forces on the tooth from the teeth touching together, occlusal forces, when chewing and swallowing. These lead to a concentration of stress and flexion at the area where the enamel and cementum meet (CEJ). This theoretical stress concentration and flexion over time causes the bonds in the enamel of the tooth to break down and either fracture or be worn away from other stressors such as erosion or abrasion. The people who initially

proposed the theory of abfraction believe the occlusal forces alone cause the lesions without requiring the added abrasive components such as toothbrush and paste or erosion.

If teeth come together in a non-ideal bite the researchers state that this would create further stress in areas on the teeth. Teeth that come together too soon or come under more load than they are designed for could lead to abfraction lesions. The impacts of restorations on the chewing surfaces of the teeth being the incorrect height has also been raised as another factor adding to the stress at the CEJ.

Further research has shown that the normal occlusal forces from chewing and swallowing are not sufficient to cause the stress and flexion required to cause abfraction lesions. However, these studies have shown that the forces are sufficient in a person who grinds their teeth (bruxism). Several studies have suggested that it is more common among those who grind their teeth, as the forces are greater and of longer duration. Yet further studies have shown that these lesions do not always appear in people with bruxism and others without bruxism have these lesions.

There are other researchers who would state that occlusal forces have nothing to do with the lesions along the CEJ and that it is the result of abrasion from toothbrush with toothpaste that causes these lesions.

Being theoretical in nature there is more than one idea on how abfraction presents clinically in the mouth. One theory of its clinical features suggests that the lesions only form above the cemento-enamel junction (CEJ) (which is where the enamel and cementum meet on a tooth). If this is kept in mind, it serves as a platform for it to be distinguished from other non-carious lesions, such as tooth-brush abrasion.

Treatment. Treatment of abfraction lesions can be difficult due to the many possible causes. To provide the best treatment option the dental clinician must determine the level of activity and predict possible progression of the lesion.

It is usually recommended when an abfraction lesion is less than 1 millimeter, monitoring at regular intervals is a sufficient treatment option. If there are concerns around aesthetics or clinical consequences such as dentinal hypersensitivity, a dental restoration (white filling) may be a suitable treatment option.

Aside from restoring the lesion, it is equally important to remove any other possible causative factors. Adjustments to the biting surfaces of the teeth alter the way the upper and lower teeth come together, this may assist by redirecting the occlusal load. The aim of this is to redirect the force of the load to the long axis of the tooth, therefore removing the stress on the lesion. This can also be achieved by altering the tooth surfaces such as cuspal inclines, reducing heavy contacts and removing premature contacts. If

bruxism is a deemed a contributing factor an occlusal splint can be an effective treatment for eliminating the irregular forces placed on the tooth.

Question 7. Pathological resorption of teeth.

K03.3 Pathological resorption of teeth. Tooth resorption is a process by which all or part of a tooth structure is lost due to activation of the body's innate capacity to remove mineralized tissue, as mediated via cells such as osteoclasts.

Types include external resorption and internal resorption. It can be due to trauma, infection, or hyperplasia.



Picture 87. The maxillary left lateral incisor (right in photograph) is afflicted with internal resorption

Internal resorption is an unusual condition where the dentin and pulpal walls begin to resorb centrally within the root canal. The first evidence of the lesion may be the appearance of a pink-hued area on the crown of the tooth; the hyperplastic, vascular pulp tissue filling in the resorbed areas. This condition is referred to as a pink tooth of Mummery, after the 19th century anatomist James Howard Mummery.

The cause can sometimes be attributed to trauma to the tooth, but other times there is no known etiology. If the condition is discovered before perforation of the crown or root has occurred, endodontic therapy (root canal therapy) may be carried out with the expectation of a fairly high success rate.

The fact remains that for many afflicted by internal resorption, the cause is actually unknown as it cannot be tied to a specific injury or traumatic incident.

External resorption in dentistry, external resorption, or root resorption, is the breakdown or destruction and subsequent loss of the root structure of a tooth. This is caused by living body cells attacking part of the tooth. Severe root resorption is very difficult to treat and often requires the extraction of teeth.

Root resorption occurs as a result of differentiation of macrophages into (odontoclasts) in surrounding tissue which, if in close proximity to the root surface will resorb the root surface cementum and underlying root

dentin. This can vary in severity from evidence of microscopic pits in the root surface to complete devastation of the root surface.

Deciduous root resorption is a natural process which allows exfoliation of the primary teeth to make way for the secondary teeth. It is caused by osteoclast differentiation due to pressure exerted by the erupting permanent tooth.

Root resorption of secondary teeth can occur as a result of pressure on the root surface. This can be from trauma, ectopic teeth erupting in the path of the root; chronic inflammation; excessive occlusal loading; trauma; improper reimplantation; aggressive tumors, cysts, and/or other growths; and/or unknown causes. The most common cause in Western society is orthodontic forces.

Roots of teeth are covered with cementum, a structure that resembles bone. However, cementum is more resistant to resorption than bone. There are a number of theories as to why this is the case. The most common hypothesis is that because cementum is harder and more mineralized than bone, and has anti-angiogenic properties, blood vessels are inhibited from forming adjacent to cementum, which in turn prevents access to osteoclasts.

Question8.Hypercementosis.

K03.4 Hypercementosis

Hypercementosis is an idiopathic, non-neoplastic condition characterized by the excessive buildup of normal cementum (calcified tissue) on the roots of one or more teeth. A thicker layer of cementum can give the tooth an enlarged appearance, which mainly occurs at the apex or apices of the tooth.

Etiology. Can be caused by many things.

Local factors:

- Occlusal Trauma
- Trauma
- Non-functional tooth
- Unopposed tooth (and impacted teeth, embedded teeth, teeth without antagonists)

Systematic factors

- Idiopathic
- Pituitary Gigantism
- Paget's Disease
- Acromegaly
- Periapical granuloma
- Arthritis
- Calcinosis
- Rheumatic fever



Picture 88. Hypercementosis.

It may be one of the complications of Paget's disease of bone in the form of generalized hypercementosis. It may also be a compensatory mechanism in response to attrition to increase occlusal tooth height.

Symptoms. It is experienced as an uncomfortable sensation in the tooth, followed by an aching pain. It may be noted on radiographs as a radiopaque (or lighter) mass at each root apex.

Complications. Such deposits form bulbous enlargements on the roots and may interfere with extractions, especially if adjacent teeth become fused (concrecence). It may also result in pulpal necrosis by blocking blood supply via the apical foramen.

Question9. Ankylosis of teeth.

K03.5 Ankylosis of teeth. Tooth ankylosis refers to a fusion (ankylosis) of teeth to bone. The condition is diagnosed with radiographs (X-rays), which show loss of the periodontal ligament space and blending of the root with the bone. Clinically the tooth sounds solid when percussed (tapped) compared to the dull, cushioned sound from normal teeth. Ankylosis of teeth is uncommon, more so in deciduous teeth than permanent teeth.

Decidious (baby) teeth. Ankylosis of deciduous teeth ("submerged teeth") may rarely occur. The most commonly affected tooth is the mandibular (lower) second deciduous molar. Partial root resorption first occurs and then the tooth fuses to the bone. This prevents normal exfoliation of the deciduous tooth and typically causes impaction of the permanent successor tooth. As growth of the alveolar bone continues and the adjacent permanent teeth erupt, the ankylosed deciduous tooth appears to submerge into the bone, although in reality it has not changed position. Treatment is by extraction of the involved tooth, to prevent malocclusion, periodontal disturbance or dental caries.



Picture 89. Ankylosis of deciduous teeth.

Permanent (adult) teeth. Repair with cementum or dentin occurs after partial root resorption, fusing the tooth with the bone. It may occur following dental trauma, especially occlusal trauma, or after periapical periodontitis caused by pulp necrosis. Ankylosis itself is not a reason to remove a permanent tooth, however teeth which must be removed for other reasons are made significantly more difficult to remove if they are ankylosed.

Question10.Trauma of teeth.

Trauma to the oral region occurs frequently and comprises 5% of all injuries for which people seek treatment. In preschool children the figure is as high as 18% of all injuries. Amongst all facial injuries, dental injuries are the most common of which crown fractures and luxations occur most frequently. An appropriate treatment plan after an injury is important for a good prognosis. Guidelines are useful for dentists and other health care professionals in delivering the best care possible in an efficient manner. The International Association of Dental Traumatology (IADT) has developed a consensus statement after a review of the dental literature and group discussions. The first set of guidelines was published by IADT in 2001. Experienced researchers and clinicians from various specialties were included in the group. In cases where the data did not appear conclusive, recommendations were based on the consensus opinion of the IADT board members. The guidelines represent the current best evidence, based on literature research and professional opinion. As is true for all guidelines, the health care provider must apply clinical judgment dictated by the conditions present in the given traumatic situation. The IADT does not guarantee favorable outcomes from following the Guidelines, but using the recommended procedures can maximize the chances of success. Because management of permanent and primary dentition differs significantly.

Uncomplicated crown fracture. Fracture involves enamel or dentin and enamel; the pulp is not exposed. Sensibility testing may be negative initially indicating transient pulpal damage; monitor pulpal response until a definitive pulpal diagnosis can be made.

Treatment: If tooth fragment is available, it can be bonded to the tooth. Urgent care option is to cover the exposed dentin with a material such as glass ionomer or a permanent restoration using a bonding agent and composite resin. Definitive treatment for the fractured crown may be restoration with accepted dental restorative materials

Complicated crown fracture. Fracture involves enamel and dentin and the pulp is exposed. Sensibility testing is usually not indicated initially since vitality of the pulp can be visualized. Follow up control visits after initial treatment includes sensibility testing to monitor pulpal status.

Treatment: In young patients with immature, still developing teeth, it is advantageous to preserve pulp vitality by pulp capping or partial pulpotomy. This treatment is also the choice in young patients with completely formed teeth. Calcium hydroxide and MTA (white) are suitable materials for such procedures.

In older patients, root canal treatment can be the treatment of choice, although pulp capping or partial pulpotomy may also be selected. If too much time elapses between accident and treatment and the pulp becomes necrotic, root canal treatment is indicated to preserve the tooth. In extensive crown fractures a decision must be made whether treatment other than extraction is feasible.

Crown-root fracture. Fracture involves enamel, dentin and root structure; the pulp may or may not be exposed. Additional findings may include loose, but still attached, segments of the tooth. Sensibility testing is usually positive.

Treatment: Treatment recommendations are the same as for complicated crown fractures (see above). In addition, attempts at stabilizing loose segments of the tooth by bonding may be advantageous, at least as a temporary measure, until a definitive treatment plan can be formulated

Root fracture. The coronal segment may be mobile and may be displaced. The tooth may be tender to percussion. Sensibility testing may give negative results initially, indicating transient or permanent pulpal damage; monitoring the status of the pulp is recommended. Transient crown discoloration (red or grey) may occur

Treatment: Reposition, if displaced, the coronal segment of the tooth as soon as possible. Check position radio graphically. Stabilize the tooth with a flexible splint for 4 weeks. If the root fracture is near the cervical area of the tooth, stabilization is beneficial for a longer period of time (up to 4 months).

It is advisable to monitor healing for at least 1 year to determine pulpal status. If pulp necrosis develops, root canal treatment of the coronal tooth segment to the fracture line is indicated to preserve the tooth.

Test tasks

1. What types of attrition do you know:

- a. physiological
- b. excessive
- c. pathological
- d. *all answers are right*

2. Causes of attrition:

- a. *bruxism*
- b. *a loss of several posterior teeth*
- c. excessive fluorine consumption
- d. all answers are right

3. Causes of abrasion:

- a. traumatic occlusion
- b. unproper brushing technique
- c. tobacco chewing /tobacco pipe
- d. vigorous use of tooth picks between the adjacent teeth
- e. excessive mastication of coarse foods
- f. *all answers are right*

4. What types of filling materials are used for treatment of abrasion:

- a. microfilled composites
- b. flowable resins
- c. glass ionomer cements
- d. *all answers are right*

5. What is the main cause of erosion:

- a. *acids*
- b. fluorides
- c. consumption of tetracycline
- d. all answers are right

6. In what area of teeth abfraction occurs:

- a. *cervical*
- b. occlusal
- c. lingual
- d. all answers are right

7. What is the main cause of abfraction:

- a. improper tooth brushing
- b. acids
- c. streptococcus mutans
- d. *excessive vertical load on tooth*
- e. all answers are right

8.Types of resorption:

- a. *external*
- b. *internal*
- c. moderate
- d. severe
- e. all answers are right

9. Internal resorption is a condition which characterized:

- a. *the dentin and pulpal walls begin to resorb centrally within the root canal*
- b. the breakdown or destruction and subsequent loss of the root structure of a tooth
- c. all answers are right

10. External resorption is a condition which characterized:

- a. the dentin and pulpal walls begin to resorb centrally within the root canal
- b. *the breakdown or destruction and subsequent loss of the root structure of a tooth*
- c. all answers are right.

Educational edition

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FOR THE 5-TH TERM**

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