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PREVENTIVE DENTISTRY

Part 2

Minsk BSMU 2015

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА СТОМАТОЛОГИИ ДЕТСКОГО ВОЗРАСТА

Н. В. Шаковец, Д. Н. Наумович, Ж. М. Бурак

ПРОФИЛАКТИКА СТОМАТОЛОГИЧЕСКИХ ЗАБОЛЕВАНИЙ

PREVENTIVE DENTISTRY

Учебно-методическое пособие
В 2-х частях

Часть 2

Минск БГМУ 2015

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Ш17

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Освещены вопросы профилактики кариеса ямок и фиссур зубов, зубочелюстных аномалий, заболеваний периодонта, галитоза. Также рассмотрены особенности индивидуальной и групповой профилактики стоматологических заболеваний

Предназначено для студентов 3-го курса стоматологического факультета, обучающихся на английском языке.

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WORKSHOP № 1
TEST CONTROL OF BASIC KNOWLEDGE. THE DISCUSSION OF STUDENT HEALTH EDUCATION PROJECTS (MOTIVATIONAL PROJECTS)

Test Questions:

1. The basic rules of organizing and conducting hygiene lesson.

2. Methods and forms of health education.

The duty of the dentist is to provide age-appropriate oral health education. One of the most important components of this work is the motivation and learning of the population oral hygiene. Neglecting oral health in preschoolers can lead to dental caries. Oral health education programs can stress the importance of oral health, increase understanding of the disease process, promote healthful behaviors, and reinforce the value of regular professional care for prevention.

The subject of discussion is the student's motivational guide for health education for preschoolers (motivational project). Teacher and students discuss the advantages and disadvantages of all components of the project.

Criteria for the competition are the next:

– colorful of execution;

– visibility and accessibility of the stated information;

– the degree of disclosure topics;

– correct presentation.

Tasks for independent work of the student: prepare and bring to practice motivational guide for health education with preschoolers.

Requirements to motivational guide for health education with preschoolers:

1) in plain language, the modern concept of etiopathogenesis of dental caries is described;

2) in plain language the child nutrition recommendations is described;

3) in plain language, the toothbrushing method for preschoolers (KAI) teeth is described;

4) the amount of text is about 2 printed pages;

5) minimum 5 pictures, the size of each of at least half of the page (pictures of toothbrushing method, pictures of food useful and harmful for teeth, etc.).

LITERATURE

Marya, C. M. A Textbook in Public Health Dentistry / C. M. Marya. JP Medical Ltd, 2011. P. 156–164, 224–228, 410–412.

WORKSHOP № 2
DENTAL CARIES IN PITS AND FISSURES: ETIOLOGY AND PATHOGENESIS, RISK FACTORS, PREVENTION OPPORTUNITIES

Test Questions:

1. Localization of natural enamel grooves in teeth. Properties of molars (temporary and permanent) occlusal odontoglyphics.

2. Morphology of pits and fissures of teeth.

3. Properties of pre-eruptive mineralization of enamel pits and fissures. Optimization measures.

4. Properties of post-eruptive enamel mineralization in pits and fissures of teeth. Methods to improve enamel resistance to caries in pits and fissures.

5. Properties of formation and retention of dental plaque in the pits and fissures of teeth; control measures (prevention of primary and secondary colonization of cariogenic microflora, mechanical cleaning and chemical control; morphology modification for self-cleaning, sealing).

Pit and Fissure Caries. Bacterial plaque is the essential precursor of caries. Hence, sites on the tooth surface which encourage plaque retention and stagnation are particularly prone to progression of lesions.

These sites are:

• Enamel in pits and fissures on occlusal surfaces of molars and premolars, buccal pits of molars, and palatal pits of maxillary incisors.

Carious lesion starts at both sides of the fissure, not at the base. The enamel is thin in fissures so there is early dentine involvement. The carious lesion forms a triangular or coneshaped lesion with its apex at the outer surface and base towards the dentinoenamel junction (DEJ).

• Lesion begins beneath plaque, with decalcification of enamel.

• Pit and fissures are often deep, with food stagnation.

• Enamel in the bottom of pit or fissure is very thin, so early dentine involvement frequently occurs.

• Here the caries follows the direction of the enamel rods. In pit and fissure the enamel rods are said to flare laterally at the bottom of the pit and caries is said to follow the path of enamel rods hence a characteristic angular/inverted «V» shaped lesion is formed.

• It is triangular in shape with the apex facing the surface of tooth and the base towards the DEJ.

• When reaches DEJ, greater number of dentinal tubules are involved.

• It produces greater cavitation than the smooth surface caries and there is more undermining of enamel.

Morphologic features which may pre dispose to the development of caries are the presence of deep, narrow occlusal fissure or buccal or lingual pits. These fissure trap food, bacteria and debris leading to development of caries. Attrition on other hand makes the tooth flattened, hence less food entrapment in fissures, so less caries.

The lowest pH attained is also determined by the rates of diffusion of substrates and metabolites. Molar fissures are the most caries-prone sites because they are sheltered from saliva flow. The deepest parts are often inaccessible to toothbrushes, which mean that fissures frequently contain impacted food for extended periods of time. If the food contains carbohydrate, the plaque is likely to have a lower resting pH than that found at other sites in

the mouth. This lower resting pH will encourage the growth of aciduric microbes such as *S. mutans*. In fact, research has shown that fissure plaque has a greater proportion of aciduric microbes including *S. mutans* and lactobacilli.

CLASSIFICATION OF PITS AND FISSURES

Nagano classified occlusal fissure into five types on the basis of fissure morphology: V, U, Y, I, IK types (fig. 1).

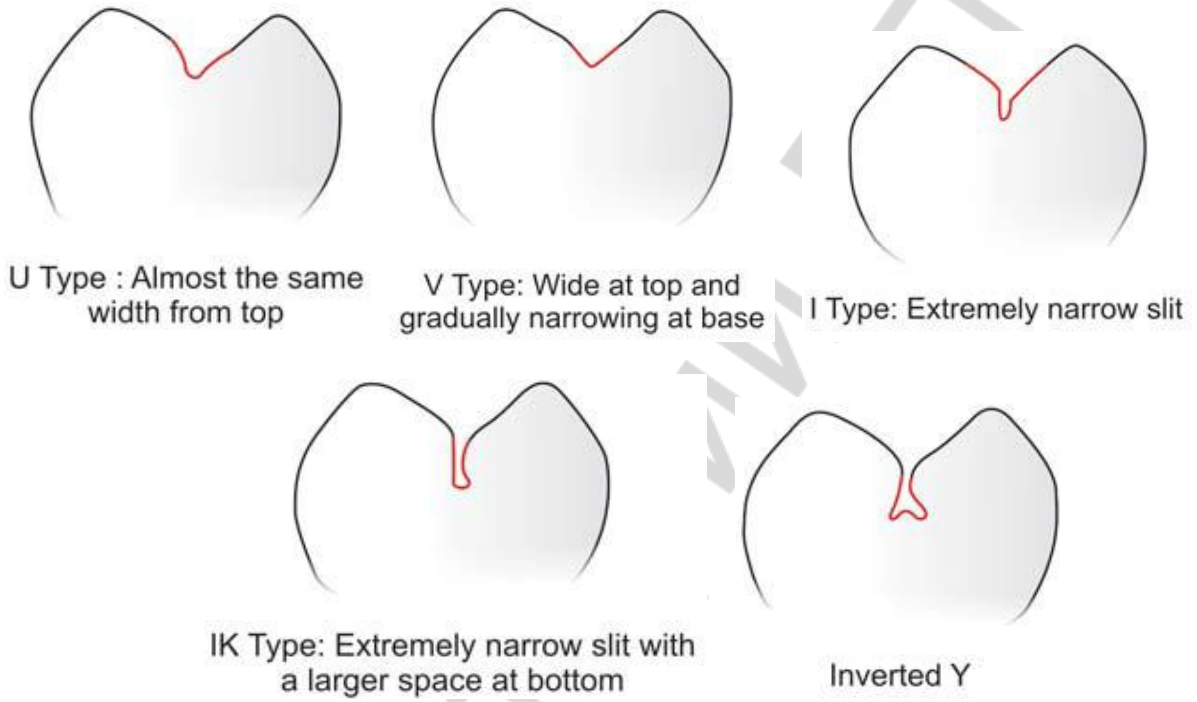

Fig. 1. Types of fissures

Pit and Fissure Sealants. Over the last few decades, several advancements have been made in caries prevention. Along with systemic and topical fluoride, the increased acceptance and use of pit and fissure sealants have
without question had an impact on the prevention of caries.

Fluorides have been found extremely effective in preventing caries on smooth surfaces of the teeth, but less effective on occlusal surfaces. Sealants protect the occlusal surfaces, inhibiting bacterial growth and providing a smooth surface that increases the probability that the surface will stay clean. It has been documented for decades that sealants are safe, effective, and

underused. The latest data available indicate that in the United States only 15 percent of children aged 6–17 years have dental sealants (G. Cherry-Peppers, H. C. Gift, J. A. Brunelle, C. B. Snowden, 1995). C. D. Gonzalez (1996) report indicates just 10 percent of the sample had sealants on their permanent molars. Why there is underusage of a proven preventive material is hard to explain. Dentists continue to identify lack of insurance coverage for sealant application as a major barrier to patients receiving the service.

A pit and fissure sealant is an organic polymer (resin) that flows into the pit and fissures and bonds to the enamel surface mainly by mechanical retention. Majority of sealants are made of Bis-GMA (Bisphenol Aglycidyl methylacrylate).

Purpose of Sealant:

1. To provide physical barrier to seal off the pit or fissure.

2. To prevent the bacteria and their nutrients from collecting within the pits or fissures to create the acid environment necessary for the initiation of dental caries.

Criteria for the Ideal Sealant:

1. A viscosity allowing penetration into deep and narrow fissures even in maxillary teeth.

2. Adequate working time.

3. Rapid cure.

4. Good and prolonged adhesion/bonding to enamel.

5. Low sorption and solubility.

6. Resistance to wear.

7. Be compatible with the oral tissues (minimum irritation to tissues).

8. Cariostatic action.

LITERATURE

Marya, C. M. A Textbook in Public Health Dentistry / C. M. Marya. JP Medical Ltd, 2011. P. 384–394.

WORKSHOP № 3
SEALING OF DENTAL PITS AND FISSURES

Test Questions:

1. Pathogenesis of caries in enamel pits and fissures. Methods of diagnosis.

2. The concept of sealing and sealants. Objectives of preventive and therapeutic sealing. Indications for the implementation of sealing.

3. Non-invasive sealing: advantages and disadvantages, indications and contraindications.

4. Invasive sealing: advantages and disadvantages, indications and contraindications. Technology of preparation of tooth tissues to sealing.

5. Materials used for sealing: adequacy requirements for sealants, technological and preventive characteristics.

6. Stages of noninvasive, invasive sealing and preventive restoration with the use of various materials.

7. Assessment of teeth after sealing. Evaluation of effectiveness. Resealing.

Types of Sealants:

A. Based on generation:

1. Generation 1 Sealant (photocured via UV light).

2. Generation 2 Sealant (auto or chemically-cured).

3. Generation 3 Sealant (photocured via visible light).

4. Fluoride containing sealants.

B. Based on fillers:

1. Unfilled.

2. Filled sealant (fillers increase abrasion resistance, bond strength). Fillers: glass and quartz particles.

3. Fluoride Releasing.

C. Based on Color.

Helps in quick identification for evaluation during maintenance assessment:

1. Clear: esthetic but difficult to detect in follow-up.

2. Tinted/opaque sealant: easy to detect.

D. BIS-GMA *versus* non BIS-GMA sealant.

Rationale for Using Pit and Fissure Sealants:

1. A very high proportion of dental decay occurs in pits and fissures The change in the pattern of caries in recent years is such that it now principally involves the pits and fissures of molar teeth in children and adolescents. Recent data shows that the relative proportion of pit and fissure lesions has increased to 84 percent of the total new caries experience.

2. Pits and fissures remain at risk of caries for long periods of time, not just within the first few years after eruption. The period of caries susceptibility has extended due to a slowing of the rate of progression of dental caries. Hence, the theory that teeth should be sealed within two years after eruption needs to be reconsidered.

3. Fluoride has limited effect in preventing pit and fissure caries. The effect of systemic or topical fluorides in preventing dental caries is noted principally on the smooth surfaces of teeth; the effect on pit and fissure caries is relatively small. Even with optional fluoride therapy, pit and fissure caries may be delayed, but not prevented, on the same scale as smooth-surface lesions. Approximately 1 mm of enamel is present on smooth surfaces, whereas the base of a fissure may be close to, or even lie within dentine. Thus in the event of fissure caries, the underlying dentine becomes rapidly involved, while on a smooth surface it may take 3–4 years for a lesion to penetrate into dentine. Hence, the inclusion of pit and fissure sealants forms an important part of any caries control program because it is intended for those caries-susceptible areas least benefited by fluoride.

4. Fissure sealants are effective at preventing pit and fissure caries and are best used as part of an overall preventive program.

The British Society of Pediatric Dentistry (2000) has stated that «Sealants are highly effective in preventing dental caries in pits and fissures of teeth when applied by trained operators in clinical trials and community health programs. When used appropriately, sealants result in improvements in oral health but their use on all occlusal tooth surfaces for preventive reasons will result in wastage of scarce resources». Sealing of pits and fissures in all patients may be considered to be ideal treatment and is justified for all patients classified as «high risk». However, financial and other constraints demand that guidelines for patient and tooth selection should be established.

PROCEDURE OF PIT AND FISSURE SEALANT APPLICATION

Sealant Placement Guidelines.

Step 1: Prepare the Teeth.

Plaque and debris might interfere with the etching process or sealant penetration:

• Clean the pit and fissure surfaces.

• Utilize a dry toothbrush, prophy cup with pumice or prophy paste, or air abrasion.

• Use an explorer to remove any debris in the pit or fissure.

• Rinse for 20–30 seconds.

• Re-evaluate surface for residual or loose debris. A widening of the fissures with rotary instrumentation is yet another type of fissure conditioning that has been recommended before etchant and sealant application. This is known as the invasive pit and fissure technique.

Step 2: Isolate the Teeth.

Adequate isolation is the most critical aspect of the sealant application process. Salivary contamination of a tooth surface during or after acid etching will have a deleterious effect on the ultimate bond between enamel and resin.

• Use cotton rolls, dry angles, and/or rubber dam. Some of the disadvantages of Rubber dam include: discomfort during clamp placement, need for local anesthetic in some instances, difficulty in securely placing a

clamp onto a partially erupted tooth, an increase in the cost and need for sterilization of the armamentarium.

Another alternative to the rubber dam is the Vac-Ejector moisture control system, which consists of a bite block and a rubber tongue shield that is connected to the high-speed evacuation line, providing a clear, dry field for sealant procedures. Clinical studies have found that sealant retention with the Vac-Ejector, either with or without a chairside assistant, is comparable to that with sealants placed under rubber dam or cotton roll isolation.

Step 3: Dry the Surfaces.

• Dry teeth with air for 20–30 seconds.

• Check to make sure there is no moisture coming out of the air syringe tip.

Step 4: Etch the surfaces.

There are various etchant materials available, but the most frequently used etchant is 37 percent orthophos-phoric acid. This is available as both a liquid solution and a gel. One should always apply the etchant onto all the susceptible pits and fissures of the tooth and extend it up the cuspal inclines well beyond (at least 2 millimeters) the anticipated margin of the sealant:

• Apply etchant as directed by manufacturer (usually between 30 and 60 seconds).

• If using a gel or semi-gel apply gel and let stand it for the allotted amount of time.

• If using a liquid continue to apply etchant throughout the etchant time.

Step 5: Rinsing and Drying the teeth.

• Rinse surfaces for 60 seconds.

• Check effectiveness of etchant by drying with air; the surface should become «chalky white».

• If not, repeat etching procedure.

• Place new cotton rolls.

• Dry teeth with air for 20–30 seconds.

Step 6: Application of Sealant Material.

During sealant application all the susceptible pits and fissures should be sealed for maximum caries protection. The sealant material can be applied to the tooth in a variety of methods. Many sealant kits have their own dispensers, some are pre-loaded, they directly apply the sealant to the tooth surface:

• Self-curing: Mix equal parts of the two components.

• Will polymerize in 60–90 seconds.

• Light-curing: Apply with syringe provided by manufacturer.

• Apply curing light to material.

• Will be polymerized in 20–30 seconds.

Step 7: Evaluate the Sealant.

The sealant should be visually and tactually inspected for complete coverage and the absence of anyvoids or bubbles. Small voids in the sealant can be repaired simply by adding new material to the void and polymerizing.

Step 8: Occlusal Evaluation.

• Check occlusion with articulating paper.

• Adjustments must be made with filled resins.

Step 9: Re-evaluation.

• Recall the patient to evaluated the sealants on the sixmonth basis.

Indications for the Use. A sealant is indicated for children and adults:

1. A person who may be at moderate or high risk of developing dental caries for a variety of reasons.

2. A person with the incipient caries (limited to enamel of pits and fissures).

3. A person who has sufficiently erupted permanent teeth with susceptible pits and fissures.

4. A person who has existing pits and fissures that are anatomically susceptible.

5. A person with a deep or irregular fissure, fossa, or pit is present, especially if it catches the tip of the explorer (for example, occlusal pits and fissures, buccal pits of mandibular molar, lingual pits of maxillary incisors).

6. The fossa selected for sealant placement is well isolated from another fossa with a restoration present.

7. An intact occlusal surface is present where the contralateral tooth surface is carious or restored.

Contraindications. A sealant is contraindicated if:

1. Patient's behavior does not permit the use of adequate dry field (isolation) techniques throughout the procedure.

2. There is an open occlusal carious lesion.

3. Caries, particularly proximal lesions, exists on other surfaces of the same tooth (radiographs must be current).

4. A large occlusal restoration is already present.

5. If pits and fissures are well coalesced and self-cleansing.

6. Life-expectancy of primary tooth is limited.

7. When patients are allergic to methacrylate.

SEALANT RETENTION

Sealants are both cost-effective and underutilized in prevention of occlusal caries. The long-term efficacy of sealants is well documented. Sealants are lost most frequently fall out from the lingual surfaces of maxillary molars and the buccal surfaces of mandibular molars.

This can be attributed to the shallower pits, which increase the difficulty of complete etching and retention. Most clinicians find that retention rates are less for primary teeth; up to 50 percent are less according to Lein. The theory behind this reduction in retention is the direction of the enamel rods in primary teeth. The ends of enamel rods in permanent teeth form an angle perpendicular

to the outer enamel surface, whereas the enamel rods in primary teeth often form an angle that does not allow for optimum retention. Initial retention failure of sealants is historically attributed to technique errors, the most common of which is moisture contamination.

Other technique errors that can affect retention are inadequate etching, incorporation of air bubbles into the sealant material (which weakens the material), and incomplete removal of debris from the pits and fissures prior to etching.

Sealant retention depends not only on proper application, but also on the eruption status of the tooth. When a tooth is not completely erupted, the retention rate is lower — possibly due to difficulties maintaining a dry tooth surface during application. Without doubt, the retention rate is lowered when an operculum is present over the distal marginal ridge of a molar. One study found a replacement rate of 54 percent on molars when an operculum was present, although no replacement was necessary on molars sealed later in the eruption process.

Undoubtedly, sealants are susceptible to occlusal wear.

This is a problem only if the seal at the margins of the sealant is not maintained.

Again, this emphasizes the importance of continued evaluation of the sealant. In the past, fluoride treatment was contraindicated prior to the sealant placement, because it was felt that the fluoride interfered with the bond between the sealant and the tooth surface. Recent research suggests that fluoride used prior to sealant placement may not adversely affect the bonding strength of enamel and sealants.

INCIPIENT FISSURE CARIES AND SEALANTS

Advantages of the invasive technique, where the fissures are widened with a small bur before the placement of sealants, are the following:

1. The ability to diagnose the extent of the carious lesion if present.

2. Higher retention rates for sealants are obtained following mechanical preparation of the fissure area.

3. The risk of microleakage is also reduced when the fissure is enlarged.

Considering these points, in cases of deep and narrow fissures that are discolored and suspected of being carious, the invasive pit and fissure sealing should be chosen.

PREVENTIVE RESIN RESTORATIONS

A logical extension of preventive sealant strategy involves the use of resin restorative materials plus attacks on a fissured surface in discrete areas of caries. This 20-year-old concept first reported by Simonsen and Stallard (1978) has gained wide approval. The procedure involves removal of those areas of teeth which were affected by caries with following filling them using resin restorative material and finally covering all restorative material and any remaining fissured anatomy with sealant. The obvious saving of tooth structure is significant. By avoiding the old philosophy «extension for prevention» the tooth preparation and replacing it with the idea of discrete removal of caries, there is a major reduction in intracoronal preparation and tooth structure loss.

The longevity of the PRR depends to a great extent on the retention and repair of the overlying sealant. This method is indicated where caries within a fissure has just reached the dentine. Under ideal circumstances the fissure sealants can successfully prevent pogression of caries, therefore sealing of the very superficial lesion is a viable option compared to the more destructive conventional restorative approach, which involves the removal of healthy tooth structure to gain good access.

FLUORIDE CONTAINING SEALANTS

The addition of fluoride to sealants has been considered since 1976 and efforts to combine the two continue today. Basically, two methods of incorporating fluoride are used. In one, a soluble salt of fluoride is added to unpolymerized resin. After sealant is applied to the tooth, the salt dissolves and fluoride ions are released. The other method involves an organic fluoride compound, which is chemically bound to resin.

However, it is questionable whether this fluoride releasing sealant will have any clinical effect on caries, since sealants usually do not penetrate to the depths of the pit and fissures, where caries usually initiates.

PUBLIC HEALTH SEALANT PROGRAMS

These programs are school based, school linked or the combination of the two. The American Association of Community Dental Programs has developed a manual «Seal America: the Prevention Invention», which provides information to the public health program administrators. In 1990, the United States Public Health Service published a national health objective for the year 2000, stating that 50 percent of children should have sealants on one or more permanent molar teeth.

Instructions to the Patient or Parent. It is necessary to receive consent from the parent or guardian of a minor or a mentally impaired patient prior to placing a sealant. The patient and/or parent must understand that sealants can only help prevent caries on the tooth surfaces where the sealants are applied; and that plaque control, fluoride therapy, and sugar discipline are still necessary to prevent decay on the rest of the tooth surfaces. Discuss the life-expectancy (the retention rate, which varies from patient to patient) of sealants with the patient/guardian. Use a mouth mirror whenever possible to show the patient and/or parent which tooth has been sealed. Explain that it may feel «high» immediately after placement, but that it should feel normal in two to three days due to normal chewing action. If it does not, the patient should return to the dental office to have the excess height reduced.

The patient or parent should be advised to check the sealant during routine oral hygiene procedures and to contact the dental office if there is any sign of sealant loss or breakage.

Inform the patient or parent of the need for six-month recall appointments to monitor sealant retention. At the recall appointment, the sealed tooth should be categorized and treated according to one of the three following categories (table 1).

Table 1

Criteria for pits and fissures management at the recall appointment

Recall status of tooth	Treatment
1. All pits and fissures covered	No treatment required
2. Sealant missing from some or all the pits and fissures, exposed surface is sound	Reseal the exposed pits and fissures (i. e. sealant replaced)
3. Sealant missing from some of all of the pits and fissures, caries is present	Restore carious pits and fissures (i. e. restorative procedures)

COST-EFFECTIVENESS

Sealant effectiveness and cost-effectiveness are dependent upon disease levels and the selection of patients and tooth surfaces to be sealed. Thus, another critical way in which sealant usefulness can be increased is by developing and applying evidence-based caries risk assessments to individual patients.

To achieve maximum benefit sealants should:

• Be used for targeted prevention in high risk children and young adults.

• Be applied to teeth such as mandibular molars that are likely to develop caries.

• Be used in connection with other preventive measures.

• Employ contemporary resin materials (second or third generation resins), or glass ionomers with appropriate viscosity and surface wetting properties.

• Be placed by dental auxiliaries (dental therapists or dental hygienists) to reduce their overall cost.

• Be monitored overtime and re-applied as needed.

Clinical Considerations. When there is an indication for placement, thensealant should be placed as soon as possible. However, susceptible sites of teeth can be sealed at any age depending on assessment of risk factors:

– Where there is a real doubt about the caries status of a susceptible site on clinical examination, e. g. a stained fissure, then a bitewing radiograph should be obtained. If there is clear evidence that the lesion is confined to enamel the surface can be sealed and monitored clinically and radiographically.

– When the evidence is unclear, the removal of the stained areas in the fissures (enamel biopsy) should be performed.

– If the lesion extends into dentine after removal of staining, then a sealant restoration («preventive resin/ GIC restoration») may be placed. A more extensive cavity will require a conventional restoration.

– Preventive resin restoration (PRR) is undertaken for more extensive lesions showing involvement of the DEJ.

– The choice between resin/composite and glass ionomer sealant should be based on adequacy of moisture control. As the resins are the most durable they should generally be preferred, while GIC should be used in cases where moisture control is difficult, e. g. in erupting or newly erupted teeth. GIC sealants in these cases are regarded more as a temporary sealant or a Fluoride release vehicle, rather than a true sealant.

Fluoride-containing sealants have not shown superiority to regular sealant. Glass ionomer sealants have failed miserably in comparison with resin-based sealants, showing very poor retention. The major benefit of resin sealants is excellent retention and thus physical blocking of the fissure system, appears much more important for caries prevention than the transient benefit of fluoride release of the short time retaining glass ionomer sealants.

Unfilled sealants perform better than filled sealants. Colored or clear resin sealant is a matter of personal preference; however, it has been shown that the ability to assess retention properly in colored sealants is much less error prone than with clear sealants. Use of an opaque color may interfere with the potential for laser fluorescent diagnosis of caries under a sealant.

Autocured sealant appears to have equivalent documentation of performance compared to visible-light-cured sealant. The use of an intermediate bonding layer, or the incorporation of the benefits of the advances of the past decade in dentine bonding agents into newly formulated pit and fissure
sealants, is perhaps the most exciting new potential development for the future of pit and fissure sealant materials.

The advantages of invasive sealants and PRR are:

1. There is minimal removal of the tooth structure, hence, greater the tooth strength.

2. There is no marginal leakage, with a reduced risk of recurrent caries.

3. Local anesthetic is not normally required.

4. The restoration can be completed in one visit and polishing is not required.

5. Caries in adjacent pits and fissures is prevented without fissure removal.

6. Pleasing aesthetics are obtained.

7. The restorations are cost-effective and can be easily repaired.

The fissure morphology and the occlusion (e. g. load bearing area) largely dictate the choice between filled and unfilled products.

Strict adherence to moisture control must be observed. Saliva control can normally be achieved by the correct placement of a sufficient number of cotton rolls. Gaining adequate control of the oral environment at the time of placement is critical for long-term success of resins, when used for PRR or as plain sealants.

SEALANT FAILURE

The success of sealants is dependent upon a strong sealant-to-enamel bond, with sufficient mechanical retention being the primary determinant of clinical success. Improper technique is the major cause of failure or early loss of sealants; therefore, it is imperative that the operator strictly adhere to proper sealant placement. The following list describes common technique errors:

1. *Contamination* may be caused by either saliva or calcium phosphate products as described earlier. The enamel surface must be re-etched if contaminated.

2. *Inadequate surface preparation* may be caused by improper cleansing prior to applying the etchant and/or the etching process itself.

3. *Incomplete or slow mixing* of self-cure sealants affects polymerization of the Bis-GMA material. If polymerization is negatively affected (e. g. starts to set-up before placement), a new mix should be made.

4. *Too slow application of the material* results in a less viscous (thicker) mix that cannot flow easily into the pits and fissures, causing an incomplete seal. Place material within the time frame recommended by the manufacturer.

5. *Air entrapment due to whipping or vigorous mixing* can occur during the mixing of self-cured sealants. It is important to replace the caps on the resin bottles since moisture can be lost through evaporation. The result is a less viscous material which does not flow properly.

6. *Overextension of the material beyond the conditioned tooth surface* results in a weakened sealant in the areas that are overextended. If the sealant margins extend beyond etched tooth structure, those areas will cause increased micro-leakage beneath the sealant and/or fracture of the sealant. The sealant should be replaced, confining the area of placement to etched tooth structure.

7. *Outdated materials* may not serve as an effective sealant.

NEW SEALANTS

1. WetBond Pit and Fissure Sealant. The difference between Embrace WetBond pit and fissure sealant and traditional sealants is that it bonds chemically and micromechanically to moist tooth surfaces. It integrates with the tooth structure to create a strong, margin-free bond that virtually eliminates microleakage. This is the first pit and fissure sealant resin that can be applied to a moist field. It forms a unique Resin Acid-Integrating Network (RAIN) that improves penetration into pits and fissures and provides superior sealing of the margins. No bonding agents are required.

2. Illuminating Pit and Fissure Sealant. Seal-N-Glo fluoresces with a blue/white color when using a UV pen light. The fluorescent glow provides clinicians with a visual verification of the sealant margins at the time of placement and offers the easiest way to verify retention and inspect margins during patient recall appointments.

3. Pit and Fissure Sealant With ACP (Amorphous Calcium Phosphate). It is a resilient and flexible, creating a strong, long-lasting sealant. This light-cure has a controlled flowability that keeps the sealant on the tooth structure while complete filling of occlusal surfaces is made. It forms a chemical and thermal barrier that protects the tooth enamel on the occlusal surface from caries. Pit and fissure sealant is a light-cured sealant that contains the «smart material» amorphous calcium phosphate (ACP) that slowly releases calcium and phosphate ions, the basic building blocks of teeth.

ACP is referred to a «smart material» because it only releases calcium and phosphate ions when the surrounding pH drops to a level where it could start to dissolve the tooth. Once the calcium phosphate is released, it acts to neutralize the acid and buffer the pH. It forms a chemical and thermal barrier that can help keep patients free from carious lesions on the occlusal surfaces.

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WORKSHOP № 4
OCCLUSION AND FUNCTION OF THE MAXILLOFACIAL AREA: FORMATION CONDITIONS, AGE NORM AND DEVIATION FROM THE NORM

Test Questions:

1. Tissues and organs of the maxillofacial area that form the occlusion.

2. Sequence analysis of the occlusion.

3. Normal occlusion, normal maxillofacial area functions in period 1 of development of the dentition.

4. The norm in period 2.

5. The norm in period 3.

6. The norm in period 4.

7. The norm in period 5.

Stages of formation of occlusion and functions of the maxillofacial region. Formation of occlusion and function of the maxillofacial region begins in utero, in close connection with the formation of tissues and organs of the head, neck and the entire musculoskeletal system. This process continues after birth, essentially complete by the age 15 years. There are five clinical periods of formation of occlusion and functions:

- predental stage or mouth of neonate (0–6 months);
- deciduous dentition stage (6 months – 6 years);
- mixed dentition (6–12 years):
  - first transitional stage;
  - second intertransitional stage;
  - second transitional stage;
- permanent dentition.

Pro-dentition Stage (0–6 Months) (Mouth of the Neonate/Gum Pads). The alveolar arches at the time of birth are called gum pads. Initially they are smooth and firm, but later get segmented corresponding to the sites of developing teeth. The basic form of the arches is determined in intrauterine life. Leighton has outlined the various factors that determine the size of gum pads as follows:

- The state of maturity of infant at birth.
- The size at birth as expressed by birth weight.
- Size of developing primary teeth.

• Genetic factors.

Maxillary arch is horse-shoe shaped and the gum pads extend labially and buccally beyond those in the mandible. Mandibular gum pads are U shaped.

Relationships of the arch. Gum pads relationship is arbitrary. They do not have definite or precise jaw relationship or bite in the neonatal jaws. The upper lip appears short. Tongue is interposed between the lips. Maxillary gum pad is wider than mandibular gum pad and there is total overlapping of maxillary gum pads transversely and anteroposteriorly. Vertical gap exists in between the upper and lower gum pads in the anterior region. The gum pads grow rapidly during the first year of life, and the growth is more in the transverse direction. Length of the gum pad also increases, mostly posteriorly to accommodate the deciduous first and second molars.

At birth, the tooth buds of all the primary teeth are present and are in different stages of development.

Functions of maxillofacial area during this stage are:

• Breathing is nasal.

• Sucking is developed. This function begins to form in utero, it reaches maximum at the age of 6 months and then begins to fade.

• Swallowing is infantile. This is such a pattern in which the tongue protrudes through the gum pads during swallowing and while the tongue is at rest. Nearly all infants exhibit a swallowing pattern involving tongue protrusion, but by six months of age most lose this reflex allowing for the ingestion of solid foods.

Primary Dentition Stage (6 Months to 6 Years). Eruption of primary tooth starts after beginning of formation of roots. The normal sequence of eruption of primary teeth is as follows: central incisors, lateral incisors, first molars, canines and second molars.

Development of Primary Occlusion

Closed dentition/Nonspaced dentition: primary teeth without any spaces in between teeth are called closed dentition. Lack of space could be eithier due to wider primary teeth or reduced arch length. Closed dentition invariably leads to crowding in the permanent dentition.

Deep bite: when the primary incisors erupt, the overbite is deep. This could be due to vertical inclination of the primary incisors. Over a period of time, this deep bite reduces due to two reasons: 1) eruption of primary molars; 2) rapid attrition of incisors. At about six years of age, there may be an edge to edge relationship.

Overjet: overjet is initially more in primary dentition The overjet decreases with the movement of the whole dental arch anteriorly. The average overjet in primary dentition is 1 to 2 mm.

***General features of deciduous dentition*:**

• Dental arches are normally ovoid in shape.
• Deep bite is present initially which changes to edge-to-edge relationship.
• Development spaces present.
• Flat curve of spee.
• Shallow intercuspal contact.
• Minimal overjet.
• Straight or vertical inclination of incisors.
• Absence of crowding.

***Functions of maxillofacial area during this stage are*:**

• Breathing is nasal.

• Chewing forms by the age of 3 years old. Normally it is active and bilateral.

• Swallowing turns to mixed after the eruption of primary incisors. This is such a pattern in which the tongue thrusting the anterior incisors during swallowing and while the tongue is at rest. This pattern of swallowing turns to somatic one by the age of 3 years old. Somatic swallowing pattern includes tongue thrusting palate near the cervices of upper incisors.

• Speech matures by the age of 5 years old.

First Inter-transitional Period. This is the period between the completion of the primary dentition and the emergence of the first permanent tooth. During this period there is marked intrabony changes but with little changes
intraorally. Vertical changes due to growth and attrition of teeth ore evident. The space for the eruption of the first permanent molars is achieved by the resorption of the anterior border of ramus in mandible and apposition of bone in maxillary tuberosity. There в deepening of bite due to attrition of deciduous incisors and there в reduction in overjet also.

Mixed Dentition Stage — the Transitional Years (6–12 Years of Age). Transition from the primary dentition to the permanent dentition begins at 6 years of with the eruption of permanent fast molars and permanent incisors. Early during the period of time, many children experience the eruption of the four permanent first molars end the exfoliation of the mandibular central incisors and the subsequent eruption of permanent incisors. It is the period during which both primary and permanent teeth are present in the mouth.

This stage of occlusal development can be divided into three stages: first transitional period, second intertransitional stage and second transitional period.

First Transitional Period. The period marks the first exchange of teeth, which begins by 6 years of age and is usually complete within two years. Two

important events take place in the period, namely the eruption of permanent first molars and the replacement of incisors.

At the end of the first transitional stage, the molars usually erupt in end on relationship and the incisors are also present with slight crowding in the mandible and with spacing in the maxilla.

Second Inter-transitional Period. This period lasts from the complete eruption of incisors until the beginning of replacement of deciduous canines and first and second molars for approximately 1½ years. The vertical dimension of face increases which allows for heightening of the alveolar ridge. Space for maxillary and mandibular second molars is gained by bone remodeling in maxillary tuberosity and mandibular ramus.

Second Transitional Period. This active stage involves replacement of primary canines and molars. This exchange normally takes place between 10 and 12 years of age.

Features of normal occlusion in permanent dentition include:

Overlap: in normally occluding dentition, the maxillary teeth are labial or buccal lo mandibular teeth.

Angulations: permanent teeth have buccolingual and mesiodistal angulations.

Occlusion: with the exception of mandibular central incisors and maxillary third molars, each permanent tooth occludes with two teeth.

Arch curvature:

• Anteroposterior curvature in the mandibular arch — Curve of spee.

• Corresponding curve in the maxillary arch is called compensating curve.

• Buccolingual curvature from one side to other side is called Monson‘s curve.

Overbite: the normal overbite expressed in terms of percentage in adult dentition is 10 to 30 percent.

Overjet: normal overjet is 1 to 3 mm.

Molar Relationship: class I Molar — Mesiobuccal cusp of maxillary first permanent molar occludes in the mesiobuccal groove of the permanent mandibular first molar.

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WORKSHOP № 5
RISK FACTORS CONTRIBUTING TO MALOCCLUSION: DIAGNOSIS, MEDICAL TACTICS

Test Questions:

1. Value of normal and impaired posture for occlusion formation. Diagnosis, causes, prevention and treatment of posture disorders.

2. Pathology of respiratory function: causes, diagnosis, pathogenic effect on the occlusion, prevention and correction.

3. Pathology of swallowing function: causes, diagnosis, pathogenic effect on the occlusion, prevention and correction.

4. Sucking reflex and related harmful habits as risk factors in formation of malocclusion: causes, diagnosis, pathogenic effect on the occlusion, prevention and correction.

5. Pathology of the chewing function as a risk factor for the formation of malocclusion: causes, diagnosis, pathogenic effect on the bite, prevention and correction.

6. Pathology of speech function as a risk factor for the formation of malocclusion: causes, diagnosis, pathogenic effect on the bite, prevention and correction.

7. Diseases of maxillo-facial area, early removal of the temporary teeth, the loss of permanent teeth, mechanical trauma as a condition of prevention normal occlusion formation: mechanism of action, prevention and correction. Index DAI.

Preventive Measures (Preventive Orthodontics):

1. Parents counseling.

2. Caries control.

3. Space maintenance.

4. Exfoliation of deciduous teeth.

5. Abnormal frenal attachments.

6. Treatment of locked permanent first molars.

7. Abnormal oral musculature and related habits.

PARENTS COUNSELING

Parents counseling is the most effective way to practice preventive orthodontics.

Parents counseling may be divided into:

a) Prenatal counseling.

b) Postnatal counseling which in turn can be associated with the clinical examination of the child at:

i. six months to one year of age;

ii. two years of age;

iii. three years of age;

iv. five to six years of age.

Prenatal Counseling. This is the most effective time to get across the expecting parents. They are open to ideas and receive the suggestions regarding better welfare of the child's well being. The gynecologists would benefit immensely on having their patients counseled on dental health.

Prenatal counseling may involve the following:

• The importance of oral hygiene maintenance by the expectant mother.

• How mother's irregular eating and hunger pangs can result in her developing decayed teeth, which can be quite painful on pulpal involvement, especially during the third trimester of pregnancy.

• Recent studies have indicated a possible co-relationship between the mother's poor oral hygiene and premature births.

• A pregnant woman with gestational diabetes mellitus is more difficult to manage during the pregnancy period especially if her oral hygiene is poor.

• To take natural foods containing calcium and phosphorus, e. g. milk, milk products, egg, etc. especially during the third trimester, as they are to allow adequate formation of deciduous teeth crowns.

Postnatal Counseling. Postnatal counseling should be advocated along with the clinical examination of the child. The same can be divided into:

Six Months to One Year of Age. This is the most important period of counseling. Parents should be aware of:

• Teething and the associated irritation, slight loose motions are possible in a mildly elevated febrile condition.

• Most of the parents are appalled on seeing the deciduous teeth erupting in rotated positions. Information about this condition should be brought that they will eventually straighten out on full eruption.

• No sugar addition to a bottle of milk, however mother's milk is preferred and it is the best thing for the TMJ development as well as for non development of tongue thrusting habits.

• Brushing with the help of a finger brush during bathing should be introduced. Cleaning the deciduous dentition with a clean, soft cotton cloth is also recommended, to prevent the initiation of nursing or rampant caries.

• The child should be initiated to drink from a glass by one year of age.

Two Years of Age:

• Bottle feeding if previously initiated should never be given during the passage to sleep. Bottle feeding should be withdrawn completely by 18 to 24 months of age. This would decrease the chances of initiation of decay and the potential for nursing caries.

• Post-breakfast and post dinner brushing should be initiated.

• Clinical examination shoul be done to assess any incipient decay and eruption status of teeth.

Three Years of Age:

• *Clinical examination.* The full complement of deciduous dentition should have erupted by now. To assess the occlusion, molar and canine

relationships and if there is the presence of any discrepancies from the normal, e. g. unilateral cross bite, supernumerary teeth, missing teeth, fused teeth, etc.

• The child should be on 3 square meals a day.

• Oral habits such as thumb sucking, lip sucking, oral breathing, etc and their effects on the development of occlusion should be considered. Parents should be informed accordingly. The use of muscle training appliances should be considered.

• To assess clinically for incomplete eruption of deciduous second molars/pericoronal flaps may lead to decay on the same.

• The child should be encouraged to begin brushing by his own at least once a day, preferably post breakfast.

Five to Six Years of Age:

• Parents should be informed about the initiation of exfoliation of deciduous teeth and that it will go up to 12 to 13 years of age.

• Clinical examination.

• The need for constant review and recall on a regular basis.

• In case of extraction of deciduous teeth due to decay, etc. the needs, advantages and importance of space maintainers should be explained.

Space Maintenance. Space maintainers in different forms may be required to maintain spaces in case of premature loss of teeth (to be discussed later in this chapter).

Exfoliation of Deciduous Teeth. Generally the deciduous teeth should exfoliate in about three months after exfoliation of the one in the contralateral arch. Any delay more than that period should be considered with suspicion and the following should be ruled out:

• Overretained deciduous/root stumps.

• Fibrous gingivae.

• Ankylosed/submerged deciduous teeth should be assessed radiographically.

• Restoration overhangs the adjacent tooth.

• Presence of any supernumerary tooth.

Abnormal Frenal Attachments. It may cause the development of diastemas/excess spacing between the teeth, which in turn may not allow the eruption of succedaneous teeth. Surgical correction of the high frenal attachments is therefore advised. The tongue should also be assessed for ankyloglossia/tongue-tie.

Locked Permanent First Molars. The permanent first molars may be locked distally to the deciduous second molars, at times. Slight distal (proximal) stripping of the deciduous second molar allows the permanent first molar to erupt in their proper place.

Abnormal Oral Musculature. Abnormal oral musculature can be prevented:

• *Tongue thrusting habits* or retained infantile swallow patterns are related to prolonged breastfeeding or bottle feeding by the mother. The same should be withdrawn by 18 to 24 months of age.

• *Hyperactive mentalis* action results in the lingual inclination of mandibular incisors which in turn decreases the arch length and increases the chance for the developing anterior crowding.

• Oral habits such as:

i. *Thumb/digit/lip sucking.* The child can be distracted it.

ii. *Mouth breathing*. The child can be given adequate medical attention, regarding recurrent upper respiratory tract infection. Oral screens and the recently introduced myofunctional appliances such as the preorthodontic trainers, train the child to breathe through the nose, thus allowing the proper development of nasal passage, regression of adenoid mass and the development of a shallow, broad palate.

SPACE MAINTENANCE (IN THE DECIDUOUS AND THE MIXED DENTITION)

Space maintenance is defined as the measures or procedures that are brought into the use due to premature loss of deciduous tooth/teeth, to prevent the loss of the arch development.

Space maintainers are defined as the appliances that prevent the loss of the arch length and which in turn direct the permanent tooth into a correct position in the dental arch.

Premature loss of deciduous tooth/teeth in the anterior or posterior quadrant results in the adjacent tooth to tip or migrate into the edentulous space. This in turn results in the decrease of the arch length and it would lead to lesser space for the premolars to erupt. The premolars will then be impacted or erupt buccally or lingually, resulting in malocclusion.

The likelihood of a child requiring corrective orthodontic treatment tends to increase in the premature loss of deciduous teeth, irrespectively of it being deciduous first or second molars.

Classification of Space Maintainers. The majority of the authors classify them into: a) fixed; b) removable.

INTERCEPTIVE ORTHODONTICS

Malocclusion, if detected as soon as possible, can be eliminated or made less severe, by initiation of interceptive orthodontic procedures. Preventive orthodontic procedures are also interceptive in nature if undertaken soon after the development of malocclusion.

An interceptive procedure undertaken at the right time can, therefore, either eliminate a developing malocclusion or make it less severe to allow corrective orthodontics to deliver a stable and conservative result in the shortest time with least discomfort to the patient. An interceptive orthodontic procedure will ensure that an oral habit does not become fixed and its deleterious effects do not affect the normal growth and development. It will also ensure that there is no loss of the arch length due to the premature loss of deciduous tooth/teeth or due to the rotated teeth or due to crowding of teeth and guide the growth of mandible by using myofunctional appliances to deliver greater benefits to the patient.

The basic undertaken interceptive procedures are:

1. Space regaining.

2. Correction of anterior and posterior cross bites.

3. Elimination of oral habits.

4. Muscle exercises.

5. Removal of soft or hard tissue impediments in the pathway of eruption.

6. Resolution of crowding.

7. Interception of developing skeletal malocclusions.

Space Regaining. If space maintenance is not carried out on the premature loss of a deciduous second molar, the permanent first molar may tip or move mesially, resulting in the loss of the arch length. The loss of the arch length may impede the second premolar to erupt.

Causes of the mesial tipping/drifting of molars are:

1. Extensive carious lesions.

2. Ectopic eruption.

3. Premature extraction of primary molars — without any space maintenance.

Correction of Anterior and Posterior Cross Bites. Cross bites, anterior or posterior should be corrected as soon as they are detected. Some authors believe that the same should be treated during the deciduous dentition. However, it may be better to treat them as the permanent teeth begin to erupt into the oral cavity.

The child may be too young and uncooperative at the deciduous dentition stage. Moreover, it is easier to bring about changes in the mixed dentition stage.

Cross bites can be unilateral or bilateral. These can also be true or functional in nature or a combination of the two.

If the cross bite is not treated in time it could lead to a skeletal malocclusion, which would require corrective orthodontic treatment later on.

Elimination of Oral Habits Leading to Interception. Habit can be defined as the tendency towards an action that has become a repeated performance, relatively fixed, consistent and easy to perform by an individual. It is necessary to record such habits which are related to the oral cavity or which affect the oral cavity.

Oral habits such as thumb/digit sucking, mouth breathing, tongue thrusting, lip sucking, etc. tend to cause malocclusion. Clinical studies have linked the development of Class II malocclusions to these oral habits. All the oral habits lead to an imbalance in the forces acting on the teeth, causing the development of dental malocclusion and if left untreated over a longer period of time, these definitely cause skeletal malocclusion. Oral habits also lead towards abnormal positioning of the tongue, aberrant lip and perioral musculature, development of unfavorable V shaped and high palatal arches as well.

Thumb or Digit Sucking. The habit of thumb or digit sucking is considered normal till the age of 3 to 4 years. Prolonged habit may lead to malocclusion.

The diagnosis of this habit can be done by relating clinical findings. The clinical findings for thumb or digit sucking are:

• Increased overjet — Labial tipping of upper anterior and/or lingual tipping of lower anterior.

• Open bite.

• Posterior cross bite.

• Callus on fingers/thumb and clean nails.

• Apart from the clinical signs enquiry the parents (for children) or directly asking the patient may provide information.

Habit breaking appliances are given to the child to prevent thumb sucking such as thumb guard and crib.

Tongue Thrusting. Tongue thrusting can be identified by the following clinical signs:

• Proclination of upper and lower anteriors.

• Anterior open bite.

• Bimaxillary protrusion.

• Indentition on tongue.

• An enquiry with patient may be necessary for confirmation.

Habit breaking appliances are given to the child to prevent tongue thrusting.

Mouth Breathing. Mouth breathing can be identified using one of the following methods.

Mirror test — a double sided mirror is placed between nose and mouth. Fogging will occur on the side of respiration.

Water test — patient is asked to fill his mouth with water. In a period of time the mouth breathers will start to feel uncomfortable.

The clinical features of mouth breathing are:

• Long narrow face.

• Gingivitis of upper anteriors.

• Increased caries pattern.

Biting Lip/Nail/Pen/Pencil

Patient will generally accept the presence of the habit. The clinical features range from:

• Wear of incisors.

• Cracking of lips.

• Notching of teeth.

Muscle Exercises. The normal development of the occlusion depends on the nature of the muscles of the face. If the oromaxillofacial musculature is in a state of balance, a good occlusion will develop and if any of the muscle groups are aberrant it will result in a malocclusion in some form or the other. Muscle exercises allow a clinician to bring such aberrant muscular functions into normal functioning, to create normal health and function, as they are important elements in aiding the growth and development of normal occlusion.

Uses:

1. To guide the development of occlusion.

2. To allow optimal growth patterns.

3. To provide retention and stability in post corrective (mechanical) orthodontic cases.

Exercises. *Exercises of orbicularis and circumoral group of muscles*:

• Upper lip is stretched in the posteroinferior direction by overlapping the lower lip. Such muscular exercises allow the hypotonic lips to form an oral seal labially.

• Hypotonic lips can also be exercised by holding a piece of paper between the lips.

• Parents can stretch the lips of the child in the posteroinferior direction at regular intervals.

• Swishing of water between the lips until they get tired.

• Massaging of the lips.

• Playing a reed musical instrument produces fine lip tonicity.

• Placement of scotch tape over the lips helps to train them to remain sealed.

• Use of an oral screen with a holder to exercise the lips.

• Button pull exercise: take a 1 inch diameter button with a thread passed through it. The patient is asked to place the button behind the lips and pull the thread while the lips try to resist the same.

• Tug of war exercise: It is similar to the button pull exercise, the difference is that two buttons are used and another individual pulls the thread gently while the same movement is resisted by the patient.

Exercises of the Tongue. Exercises of the tongue are done to correct any aberrant tongue swallow patterns:

• *One elastic swallowing*: orthodontic elastic, usually 5/16th inch, is placed on the tip of the tongue and the patient is asked to raise the same to rugae area and swallow.

• *Two elastic swallowing*: 25/16th inch elastics are used and one is placed on the tip of the tongue whereas the other is placed on the dorsum of the tongue in the midline and asked to swallow.

• *Tongue hold exercise*: a 5/16th inch elastic is used and the patient is asked to place it on a designated spot over a definite period of time with the lips closed. The patient is asked to swallow with the elastic in the designated position and lips apart.

• *Hold pull exercise*: the tip of the tongue contacts the palate in the midline and the mandible is gradually opened. This allows the stretching of the frenum to relieve a mild tongue-tie.

Exercises of Masseter Muscles. At times it is advised to strengthen the masseter muscles. The patient is asked to clench his teeth, count up to 10 in his mind and then relax them. This has to be repeated over a period of time, until the masseter muscles feel tired.

Exercises of Pterygoid Muscles. In case of disto occlusion cases, the patient is asked to protrude the mandible as much as possible and then retracted. Repeat the exercises until the muscles feel tired. The ability to keep the mandible in correct position gradually improves.

Limitations of Muscle Exercises:

1. Exercises are not known to drastically alter any bone growth pattern.

2. They are not a substitution for corrective orthodontic treatment.

3. Patient compliance is extremely important.

4. If not done correctly, can be counterproductive.

Removal of Soft or Hard Tissue Impediments in the Pathway of Eruption. The clinical conditions in which the hard and soft tissue acts as an impediment in the natural pathway of tooth eruption are:

• *Retained Deciduous Tooth/teeth.* A clinical manifestation, which has become more common today due to the shift from hard, detergent diet to a soft diet. Generally, retained deciduous teeth are observed in the mandibular anterior region, with the permanent teeth erupting lingually or in the maxillary anterior and buccal regions, with the permanent teeth erupting labially/buccally.

The unilateral presence of such retained teeth also results in a midline shift thus compounding an arch space deficiency in a quadrant. Interception by extraction of the retained deciduous teeth would resolve the malocclusion completely or decrease its severity, thus allowing easier management of the same.

• *Supernumerary Teeth.* Presence of supernumerary teeth and mesiodens would impede the eruption of the permanent teeth in their rightful place. Therefore, timely extraction of the same would go a long way in interception of a developing malocclusion.

• *Fibrous/Bony Obstruction of the Erupting Toothbud.* If the contralateral tooth fails to erupt even after three months, there should be a cause for concern and a radiographic assessment, therefore, becomes mandatory. Surgical intervention may be required.

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WORKSHOP № 6
PERIODONTIUM: AGE NORM, ETIOLOGY, PATHOGENESIS, SYMPTOMS OF DISEASE. INDEX ASSESSMENT OF PERIODONTIUM

Test Questions:

1. Structure, function, and clinical characteristics of normal periodontium in different age periods.

2. Inflammatory changes in the periodontium.

3. Methods of clinical and epidemiological assessment of the periodontal state:

a) index PMA;

b) Schiller-Pisarev test;

c) gingival index GI;

d) community periodontal CPITN;

e) complex periodontal index CPI by P. A. Leus;

f) index of gingival recession;

g) index of gingival attachment loss.

STRUCTURE, FUNCTION, AND CLINICAL CHARACTERISTICS OF NORMAL PERIODONTIUM IN DIFFERENT AGE PERIODS

Periodontium is the tissues that surround and support the teeth. Periodontium includes the gums, cementum, periodontal ligament, and alveolar and supporting bone. Sometimes periodontium is called alveolodental membrane, peridental membrane.

Gingiva (or gums) is the part of the oral mucous membrane that covers the alveolar processes and cervical portions of the teeth.

The gingiva is divided into the papillary portion, which occupies the interdental space, the marginal portion, which forms the collar of free gingiva around the neck of the tooth, and the *attached gingiva*, which is attached by fibrous tissue to the underlying alveolar bone. Marginal and papillary gingiva together constitute the *free gingiva*.

Important anatomical structure is also *gingival sulcus* (it is the space between the free gingiva and the tooth surface). The base of the sulcus is formed by the junctional epithelium — a specialized type of epithelium that attaches to the tooth surface. In case of pathology the junctional epithelium forms the base of *periodontal pocket*. A periodontal pocket is a sulcus that has deepened because of disease. The depth of a periodontal pocket is greater than 3 mm.

Healthy gums have the following features:

• Coral pink color (although the colour may be related to the race identity, the age, the complextion of the person, the thickness of the tissues and the degree of keratinization).

• Gums hug teeth tightly.

• No bleeding.

The periodontal ligament is the connective tissue structure that surrounds the root and connects it with the bone. It is continued with the

connective tissue of the gingiva and communicates with the marrow spaces through vascular channels in the bone.

The most important elements of the periodontal ligament are the principal fibers, which are collagenous, arranged in bundles. Terminal portions of the principal fibers that insert into cementum and bone are termed Sharpey's fibers. The principal fibers are arranged in the following groups: transseptal, alveolar crest, horizontal, oblique and apical. Less regularly arranged collagen fibers are found in the interstitial connective tissue between the principal fiber groups which contain blood vessels, lymphatics, and nerves. Other fibers of the periodontal ligament are the elastic fibers, which relatively few, and the so-called oxytalan (acidresistant) fibers, which are distributed mainly around the blood vessels and embedded in cementum in the cervical third of the root.

Cellular elements of the periodontal ligament are fibroblasts, endothelial cells, cementoblasts, osteoblasts, osteoclasts, tissue macrophages, and strands of epithelial cells termed the «epithelial rests of Malasser».

Cementum is the calcified mesenchymal tissue that forms the outer covering of the anatomic root.

There are two main forms of root cementum: acellular (primary) and cellular (secondary). Cellular cementum contains cementocytes in individual spaces (lacunae) which communicate with each other through a system of anastomosing canaliculi. Cementoblasts also form the glucoprotein interfibrilary ground substance. Cellular cementum is less calcified than the acellular cementum. It is present only on teeth apices and furcation regions. There are two sources of collagen fibers in cementum: Sharpey's and the fibers belonging to cementum matrix per se produced by the cementoblasts. Cementum deposition continues throughout the life.

Alveolar and supporting bone — the portion of the maxilla or the mandible that forms the dental arch and serves as bony investment for the teeth.

Functions of periodontium are the next:

1. Resistant — retaining (anatomic) — basic, it is provided by the ligament vehicle of tooth which is in the suspended state. The structure of fibres of periodontium hinders the fabrics squeezing and wedging the tooth in the bone of alveolus.

2. Regulative (to the division of pressure) — carried out due to a tissue liquid (hydraulic gasket) which in periodontium is 60 %, blood and lymph.

The rich net of blood and lymphatic vessels, operating as a hydraulic system, distributes masticatory pressure evenly on all walls of alveolus and thus improves the function of periodontium as a liaison and amortizing vehicle. Direction of bunches of collagenous fibres of periodontium answers the direction of forces which operate on teeth at their function exactly. Masticatory effort is a physiologically needed for process exchange and trophic in periodontium. At underloading there are changes in the vessels of periodontium that
results in violation of trophic functions in paradontium on the whole and, as a result of it, in the development of various pathological processes.

3. Reflex. This function is carried out due to a plenty of sensible nervous completions. The force of masticatory pressure is regulated by mechanical receptors from which signals are passed on a masticatory musculature. Liquid environment of periodontium and his fibred structures take part in this process.

4. Plastic. Fibroblasts, cementoblasts (build the second cement), osteoblasts (build an alveolar bone) carry out this function.

5. Trophic. The net of vessels and nerves provides the normal feed of tooth cement, compact plate of alveolus and correct exchange of matters in periodontium.

6. Sensory. This function is predefined by the presence of completions of nervous fibres which pierce connective tissue and pass to a peripheral irritation center. Any tooth without pathological changes can perceive the easy touching to the crown and irritation from the particles of meal, which get into interdental space.

Clinical characteristics of normal periodontium in different age periods are described in table 2.

Table 2

Features of gums in children

	Period of temporary dentition	**Period of mixed occlusion**	**Period of permanent occlusion**

	Period of temporary dentition	Period of mixed occlusion	Period of permanent occlusion
Gum	Gingival epithelium is thin, poorly differentiated, with pain-shim amount of glycogen, particularly in children under 3 years. The basement membrane is thin, delicate. Collagen-new fibers are arranged loosely, elastic fibers are absent. Rich capillary vasculature	Layer of the epithelium thickens, nipples become of more prominent shape and depth, the basement membrane thickens, collagen fibers become denser and navigate. The tendency to diffuse reactions is reduced	The gum has differentiated mature-suite structure. In the cervical area epithelium is devoid of surface-layer (stratum layer of cells)
Periodontal ligament	Ligament apparatus presents fiber bundles run parallel to the long axis of the tooth and forms an intermediate plexus	Fiber ligament apparatus begins to change its direction, lying at the angle of 45° to the long axis of the tooth	Periodontal ligament is disposed in different directions and tightly fixes the tooth in the alveolus. They mostly come at the angle of 45°, and at the neck of the tooth they are almost horizontal and form a circular bundle
Cementum	Cellular cement is found in the root tip of deciduous teeth and to the period of the change of teeth becomes more powerful	The number of cells increases, and in 10–11 years cellular cement covers about half the length of the roots of temporary and permanent teeth	2/3 of roots is covered with acellular (primary) cement, and the apical third of the roots with secondary (cell) cement

INFLAMMATORY CHANGES IN THE PERIODONTIUM. SYMPTOMS OF DISEASE. ETIOLOGY AND PATHOGENESIS

Periodontal disease is an inflammatory process involving progressive, episodic loss of the periodontal attachment apparatus, resulting ultimately in tooth loss in susceptible patients. Periodontal diseases are generally divided into two groups:

1. *Gingivitis* — is an inflammation involving only the gingival tissues next to the tooth.

2. *Periodontitis* which damages the bone and connective tissue supporting the teeth is a more serious form of gum disease. Periodontitis occurs when the gum tissues separate from the tooth and sulcus, forming periodontal pockets.

Symptoms of disease. *The common symptoms and signs* of gum disease include:

• Receding gums.

• Bleeding gums.

• Red, swollen and tender gums.

• Discoloration of gums.

• Formation of spaces between teeth and gums.

• Loose teeth.

• Changes in the way teeth fit together on biting, or the way dentures fit together.

• Continuous bad breath or bad taste in the mouth (halitosis).

Gingivitis is characterized by tender, red, swollen gums that bleed easily and may cause bad breath (halitosis).

Periodontitis is characterized by:

1. Gum inflammation, with redness and bleeding.

2. Deep pockets (greater than 3 mm in depth) are formed between the gum and the tooth.

3. Loose teeth, caused by the loss of connective tissue structures and bone.

Etiology and pathogenesis of periodontal diseases. The main risk factor for the pathology is microorganisms realizing its pathogenic potential in terms of the immune response and environmental conditions. We can say that gum disease develops as a result of a plaque build up because of poor oral hygiene — not brushing and flossing teeth regularly and visiting the dentist.

Other risk factors include host factors and environmental factors.

Microorganisms can produce disease directly, by invasion in the tissues, or indirectly by bacterial enzymes and toxins. The inflammatory response in periodontal disease includes the activation of leucocytes, neutrophils, T-lymphocytes and plasma cells, the release of antibodies, lipopolysaccharides and

chemical inflammatory mediators. The level of periodontal destruction depends on the balance between destructive and protective inflammatory mediators. While periodontal bacteria are required for infective periodontal disease, individual response determines disease progression.

Initial lesion of gums is the result of an inflammatory response to bacterial plaque. It occurs within 2–4 days. The first changes occur around the small gingival blood vessels apical to the junctional epithelium. There is migration and infiltration of white blood cells into the junctional epithelium and gingival sulcus. There is increased exudation of tissue fluid from gingival crevice. There may be no clinical signs of tissue change at this stage.

If plaque deposition persists, the bacterial plaque becomes older and thicker. The initial inflammatory changes continue with an increased flow of gingival fluid and migration of polymorphonuclear leucocytes (PMNs). There is small increase in the number of inflammatory cells, 75 percent of which are lymphocytes, a few plasma cells and macrophages. There is breakdown of collagen fiber so that the seal of the marginal cuff of gingiva is weakened. Early signs of gingivitis become apparent with slight gingival enlargement. Early gingivitis is reversible when plaque is controlled.

Progression from the early lesion leads to the establishment of clinically obvious gingivitis within 7–14 days. Clinical signs of inflammation appear and the interdental papillae may become swollen and bleed on probing. The number of lymphocytes increases and predominant inflammatory cells are plasma cells. Plasma cells are related to areas of chronic inflammation. Clinical signs of inflammation appear and the interdental papillae may become swollen and bleed on probing. Marginal gingiva becomes spongy. With the increased destruction of collagen and inflammatory swelling the gingival margin can be separated easily from the surface giving rise to «gingival» or «false pocket». There is degeneration of junctional epithelium cells and some proliferation of junctional and sulcular epithelium continues. As fibrous tissue is destroyed within the site of active inflammation there is some proliferation of fibrous tissue and formation of new blood vessels at more distant sites. Thus destruction and repair continue side by side.

Continuous plaque irritation and inflammation damages the integrity of the junctional epithelium. There is degeneration and separation of epithelial

cells and there is breakdown of their attachment to the tooth surface. Connective tissue fibers are destroyed. The junctional epithelium proliferates into the connective tissue and down the root surface as the dentogingival fibers and the alveolar crest fibers are destroyed. The epithelium migrates along the root surface. Apical migration of the junctional epithelium continues and as this epithelium separates from the root surface a periodontal or true pocket is formed. The connective tissue is edematous; vessels are dilated and thrombosed; vessel walls break down with hemorrhage into the surrounding tissues. There is a massive inflammatory infiltrate of plasma cells, lymphocytes and macrophages.

The progression of lesion is not prolongs, periods of advance and remission take place and fibrosis is a constant feature. With the destruction of periodontal ligament and alveolar crest resorption, the pocket deepens. Prolongation of the disease may lead to varying degree suppuration and abscess formation. Finally the teeth may become loose, migrate and lost.

METHODS OF CLINICAL AND EPIDEMIOLOGICAL ASSESSMENT OF THE PERIODONTAL STATE

The diagnosis of gum disease is based on a thorough examination of the patient's mouth by a dentist. Dentist looks for signs of pathology (described above) and assesses the state of periodontium using special indices. In case of periodontitis a full mouth x-ray is taken to determine the extent of disease.

The calibrated periodontal probe is a periodontal instrument that is marked in millimeter increments and used to evaluate the health of the periodontal tissues. The periodontal probe is the most important clinical tool for obtaining information about the health status of the periodontium.

1. *Design of Calibrated Probes.* Calibrated probes have blunt, rod-shaped working-ends that may be circular or rectangular in cross section. Periodontal probes come in a variety of styles and are made by many different manufacturers. There is a periodontal probe for epidemiological studies conducted by WHO on the picture. This probe has spherical tip diameter of 0.5 mm and black band (3 millimeters in length) and 2 rings. As a result the millimeter markings on this particular probe are at 3,5–5,5–8,5–11,5 mm (fig. 2). The probe is used to determine indices recommended by WHO.

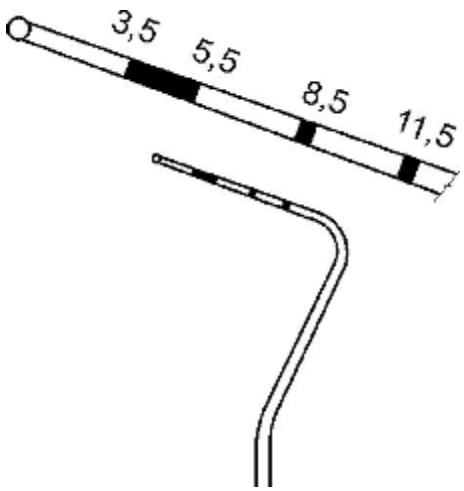


Fig. 2. The calibrated periodontal probe

2. *Function of Periodontal Probes.* The calibrated periodontal probe is used to measure sulcus and pocket depths, to measure clinical attachment levels, to determine the width of attached gingiva, to assess the presence of bleeding and/or purulent exudate (pus), and to measure the size of oral lesions. In general, the deeper the probe slips into the pockets between a tooth and gums, the more severe the gum disease in that area.

Indices. There are a number of indices for the assessment of periodontium. The indices may be divided into gingival (PMA, GI) and periodontal (CPI by P. A. Leus, CPITN, Index of gingival attachment loss) (table 3).

PMA is described: Marya C. M. A Textbook in Public Health Dentistry. JP Medical Ltd, 2011. P. 196.

GI is described: Marya C. M. A Textbook in Public Health Dentistry. JP Medical Ltd, 2011. P. 193.

CPITN is described: Marya C. M. A Textbook in Public Health Dentistry. JP Medical Ltd, 2011. P. 197–200.

Index of gingival attachment loss as part of (COMMUNITY PERIODONTAL INDEX (CPI)) is described: Marya C. M. A Textbook in Public Health Dentistry. JP Medical Ltd, 2011. P. 201–202.

Schiller–Pisarev test. Schiller and Pisarev test is based on determining the glycogen in gingiva. The glycogen content is increased during inflammation due to violation of epithelium keratinization. In healthy gingiva

there is no glycogen or there are traces of glycogen. In a few seconds after the application of iodine-containing drugs (most often the solution Schiller–Pisarev), iodine and glycogen begin to react and inflamed gums change colour. The test can be estimated as negative (straw-yellow color), weakly positive (light brown staining) or positive (dark brown color). This test can not be used for the diagnosis of periodontal disease in children younger than 6 years, because their healthy gingiva contains a large amount of glycogen.

Index of gingival recession.

Teeth examined: all teeth in the mouth.

Examination Procedure: teeth with gingival recession are determined.

Scoring:

$$\textbf{Index of gingival recession} = \frac{r}{n} \times 100\ \%,$$

r — the number of teeth with gingival recession; n — the number of teeth examined.

LITERATURE

1. *Hollins, C.* Basic Guide to Anatomy and Physiology for Dental Care Professionals / C. Hollins. Wiley 2012. P. 158–166.

2. *Marya, C. M.* A Textbook in Public Health Dentistry / C. M. Marya. JP Medical Ltd, 2011. P. 193–201.

3. *Marya, C. M.* A Practical Manual in Public Health Dentistry / C. M. Marya. JP Medical Ltd, 2012. P. 170–193.

Table 3

Simplified scheme of several indices

Index	Teeth examined	Surfaces examined	Tools	Scoring criteria	Scoring	Interpretation
PMA	All teeth except third molars	**P**apillary portion, **M**arginal portion and **A**ttached gingiva for each tooth	Blunt probe for pressing on gingiva	0 — no inflammation; 1 — inflammation of papillary portion; 2 — inflammation of marginal portion; 3 — inflammation of attached gingiva	Add scores for each tooth and divide by the number of teeth, then multiply result by 100 percent	≤ 33 % — mild gingivitis; 34–66 % — moderate gingivitis; ≥ 67 % — severe gingivitis
GI	16, 21, 24, 36, 41, 44	Buccal, lingual, mesial and distal for each examined tooth	Blunt probe for pressing on gingiva	0 — healthy; 1 — slight discoloration and mild swelling are observed, but bleeding is not observed; 2 — redness, swelling and bleeding are observed; 3 — severe redness, swelling, ulceration and spontaneous bleeding are observed	For each examined tooth add scores for surfaces and divide by 4. Add scores for each tooth and divide by the number of teeth	0 — excellent (no gingivitis); 0.1–1.0 — good (mild gingivitis); 1.1–2.0 — fair (moderate gingivitis); 2.1–3.0 — poor (severe gingivitis)
CPITN	17/16, 11, 26/27, 47/46, 31, 36/37 as representatives of the appropriate sextants. The index should not be recorded for children under	Mesiobuccal, buccal, distobuccal, mesiolingual, lingual and distolingual (6 points as a result) for	Calibrated periodontal probe	0 — healthy; 1 — bleeding is observed; 2 — calculus is detected; 4 — pocket ≤ 5 mm is detected	Each sextant evaluated on worst score of the index teeth	For each sextant: 1 — there is no need for treatment; 2 — there is need for

Index	Teeth examined	Surfaces examined	Tools	Scoring criteria	Scoring	Interpretation
	the age of 12. When examining children under the age of 15 years, pockets are not recorded although probing for bleeding and calculus are carried out as routine	each examined tooth		; 5 — pocket ≥ 6 mm is detected		improving the personal oral hygiene; 3 — there is need for professional hygiene with scaling and root planning; 4 — there is need for complex treatment
Index of gingival attachment loss	17/16, 11, 26/27, 47/46, 31, 36/37 as representatives of the appropriate sextants. The index should not be recorded for children under the age of 15	Mesiobuccal, buccal, distobuccal, mesiolingual, lingual and distolingual (6 points as a result) for each examined tooth	Calibrated periodontal probe	0 — loss of attachment 0 to 3 mm; 1 — loss of attachment 4 to 5 mm; 2 — loss of attachment 6 to 8 mm; 3 — loss of attachment 9 to 11 mm; 4 — loss of attachment 12 mm or more	Each sextant evaluated on worst score of the index teeth	Index helps to estimate the level of periodontal attachment destruction
CPI by P. A. Leus	17/16, 11, 26/27, 47/46, 31, 36/37. The index should not be recorded for children under the age of 3 and aged 5–6 years	Mesiobuccal, buccal, distobuccal, mesiolingual, lingual and distolingual (6 points as a result) for each examined tooth	Ordinary dental probe	0 — healthy; 1 — soft deposits; 2 — bleeding is observed; 3 — calculus is detected; 4 — pocket is detected;	Add scores for each tooth and divide by the number of teeth	0.1–1.0 — risk of the disease (pathology); 1.1–2.0 — mild disease (pathology); 2.1–3.5 — moderate disease (pathology);

Index	Teeth examined	Surfaces examined	Tools	Scoring criteria	Scoring	Interpretation
				5 — loose teeth are detected		3.6–5.0 — severe disease (pathology)

WORKSHOP № 7
RISK FACTORS FOR DEVELOPMENT OF PERIODONTAL PATHOLOGY, STRATEGY AND TACTICS TO MINIMIZE AND ELIMINATE THEM. PROFESSIONAL ORAL HYGIENE. INDIVIDUAL ORAL HYGIENE FOR PATIENTS WITH PERIODONTAL PATHOLOGY

Test Questions:

1. Dental deposits as a cause of inflammatory periodontal diseases: composition, structure, biochemistry, methods of diagnosis, principles of control.

2. Pathogenesis of inflammatory infectious diseases of the periodontium.

3. Noninfectious local risks factors for periodontium (occlusion pathology, anomalies of the architectonics of atrium of the mouth cavity, dental caries, iatrogenic factors, smoking): mechanisms of influence on periodontium, methods of diagnostics and control.

4. Common conditions in the human body that affect periodontal pathology (special hormonal status, aging, disease and their treatment): mechanisms
of influence on the processes in periodontium and prevention of periodontal pathology.

5. The role of environmental factors in the development of periodontal pathology.

6. Definition of professional oral hygiene: purpose, objectives and basic content. The course of professional oral hygiene for the patient.

7. Professional removal of soft dental deposits: tasks, tools and methods.

8. Removing of mineralizated dental deposits with hand and power tools and methods: tools description (scalers, hoes, chisels). Requirements for ergonomics and safety.

9. Instrumental methods of removing mineralizated dental deposits: description of the oral sandblasters, pneumatic and ultrasonic scalers,

indications and contraindications to the use, requirements of ergonomics and safety.

10. Correction of restorations.

11. Ensuring effective self-care for the patient with a high risk and with periodontal pathology:

a) topography of retention of dental deposits in presence of periodontal pathology;

b) tooth-brushing with periodontal pathology (selection of brushes, cleaning methods and hygiene management in periodontal pathology (toothpaste, mouthwashes, elixirs);

c) hygiene management in teeth proximal surfaces with periodontal pathology (selection and application of superflosses, tapes, toothpicks, electrical devices);

d) irrigation of subgingival spaces: purpose, methods, tools, devices; advantages and limitations; methods of irrigation during self-care.

The main risk factor for periodontal pathology is microorganisms realizing its pathogenic potential in terms of the immune response and environmental conditions. We can say that gum disease develops as a result of a plaque build up because of poor oral hygiene — not brushing and flossing teeth regularly and visiting the dentist.

The current concept concerning the etiology of periodontal disease includes bacterial pathogens, host factors and environmental factors.

Bacterial pathogens. The main cause of periodontal diseases is microorganisms. The oral biofilm consists mainly of microbes and host proteins that adhere to teeth within minutes of a dental oral hygiene procedure. Healthy gingival sulcus has a flora dominated by equal proportions of Gram positive cocci. Over time, the flora changes from predominantly gram-positive to gram-negative, from facultative aerobes to strictly anaerobic species, with more
motile forms present. Mature subgingival biofilm takes up to 12 weeks to develop. As biofilm accumulates, gingivitis develops over a period of several days in the presence of periodontal bacteria.

Main periodontopathogens are the next:

– Porphyromonas gingivalis (P.g),

– Prevotella intermedia (P.i),

– Bacteroides forsythus (B.f),

– Treponema dentico/u/la (T.d),

– Aggregatibacter actinomycetemcomitans/previously Actinobacillus actinomycetemcomitans (A.a),

– Fusobacterium nucleatum (F.n),

– Capnocytophaga species (C.sp),

– Tannerella forsythia (T.f),

– Campylobacter rectus (C.r).

In various forms of periodontal disease different microorganisms are prevalent:

1. In case of gingivitis main pathogens are Str. intermedius, Str. sanguis, A. odontolythicus, V. parvula.
2. In patients with mild periodontitis there are the following microorganisms: B. forsythus, Tr. dentico/u/la.
3. In patients with moderate and severe periodontitis there are the following microorganisms: A. actinomycetemcomitans, P. gingivalis, P. intermedia.

It should be noted that no individual is truly biofilm free; there is either a healthy biofilm in place or a pathogenic biofilm contributing to caries and periodontal disease. The supragingival biofilm forms a reservoir for periodontal bacteria and the development of subgingival biofilm. As the biofilm matures, the concentration and virulence of the periodontal bacteria change. Moreover, when plaque is allowed to remain in the periodontal area, it transforms into calculus (commonly known as tartar) which has a mechanical action on periodontal tissues.

Supragingival calculus formation takes place from interactions between tooth surface and plaque. Subgingival calculus involves inflammatory exudates within pockets. It forms more slowly and forms an intimate relationship with the rough root surface. Calculus is always covered with soft plaque and retains

toxic bacterial products. The surface texture of calculus promotes plaque accumulation and retention of irritant bacterial deposits. Calculus itself is not capable to initiate periodontal disease. Supra and subgingival calculus vary in their colour, composition and content.

Microorganisms can produce disease directly, by invasion in the tissues, or indirectly by bacterial enzymes and toxins. The inflammatory response in periodontal disease includes the activation of leucocytes, neutrophils, T-lymphocytes and plasma cells and the release of antibodies, lipopolysaccharides and chemical inflammatory mediators. The level of periodontal destruction depends on the balance between destructive and protective inflammatory mediators. While periodontal bacteria are the cause of infective periodontal disease, individual response determines disease progression.

Environmental factors include ecological factors, economical factors and some others. For example, people living in adverse environmental conditions are marked by the worst periodontal condition. People with low socioeconomic status often have the worst periodontal condition due to pure oral hygiene.

Host factors are divided into two groups: oral factors and general factors.

Oral factors include:

Oral Hygiene. Lack of oral hygiene, such as not brushing or flossing regularly, encourages bacterial buildup and plaque formation. It has been accepted for years that the relationship between oral hygiene status and periodontal
disease is consistent (Nagraj Rao, 1980).

Poorly Contoured Restorations. Poorly contoured restorations (fillings or crowns) provide traps for debris and plaque and can also contribute to periodontitis.

Tooth Structure. Abnormal tooth structure can increase the risk of periodontal disease.

Wisdom Teeth. Wisdom teeth, also called third molars, can be a major breeding ground for the bacteria that cause periodontal disease. Periodontitis can occur in wisdom teeth that have broken through the gum as well as teeth

that are impacted (buried). Adolescents and young adults with wisdom teeth should be checked by dentist to prevent periodontal disease.

Traumatic Occlusion. Sharp cusps act as plungers and being a derogatory to periodontal health, leading to periodontitis.

Tooth Malalignment. Gingivitis is more common and more severe around malaligned teeth because they are harder to clean.

Intraoral Distribution. Tooth surfaces most affected by gingivitis or periodontitis are the proximal surfaces (Loe et al., 1965). The teeth most severely affected by gingivitis are the molars and lower anteriors. Attachment loss
on average is greater in the maxilla than in mandible, and least in canines, mandibular 1st premolars and maxillary central incisor.

Occupational Habits. Habits like thread biting by tailors and holding nails between teeth by carpenters cause trauma to the periodontium leading to periodontitis. Miscellaneous habits like pipe smoking, pencil biting, nut biting, finger nail biting produce traumatic injury to periodontium.

The main general risk factors for periodontal disease include:

1. Age.

2. Smoking or tobacco use.

3. Female hormonal changes.

4. Illnesses such as diabetes or HIV/AIDS, and the medications used to treat some conditions.

5. Genetic factors.

Age. Periodontitis typically occurs as people get older and is most common after age 35.

Smoking. Smoking is the major preventable risk factor for periodontal disease. Smoking can cause bone loss and gum recession even in the absence of periodontal disease. The risk of periodontal disease increases with the number of cigarettes smoked per day. Smoking cigars and pipes carries the same risks as smoking cigarettes.

DEFINITION OF PROFESSIONAL ORAL HYGIENE: PURPOSE, TASKS AND BASIC CONTENT

Professional oral hygiene may be defined as the scientific care of the teeth and mouth (Xavier, 2000). Also professional oral hygiene is defined as preventive measures aimed at removing dental plaque and preventing it from recurring (Axellson, 1981).

The *purpose* of professional oral hygiene is to prevent the building up of plaque.

The *objectives* (approaches) of professional oral hygiene are the next:

1) mechanical plaque removal by individual;

2) plaque removal by dental professional.

The course of professional oral hygiene for the patient. The course of professional oral hygiene for the patient consists of at least 3 visits.

In case of professional plaque removal during only one visit in patients with poor oral hygiene we work as a cleaner. Because during the next visit teeth will be dirty again and we will see soft deposits and calculus at the same or even more amount.

During *visit 1* the items of our agenda are the next:

– oral status and oral hygiene assessment;

– the motivation for the controllable risk factors of dental diseases;

– planning prevention of oral diseases;

– selection of tooth brushing methods, oral hygiene tools and dentifrices;

– tooth brushing training of a patient on a model.

Therefore, during visit 1 we solve the first objective of professional oral hygiene — mechanical plaque removal by individual.

During *visit 2* we should do the next:

– supervised tooth brushing and correction of individual oral hygiene;

– correction of selected tooth brushing methods, oral hygiene tools and dentifrices (if it is necessary);

– plaque removal by a dental professional from 1–4 quadrants (it depends on clinical situation).

But in the case of poor oral hygiene of the patient we repeat plan of visit 1 again.

And during *visit 3* our plan is the next:

– supervised tooth brushing and oral hygiene assessment;

– checking the quality of cleansing subgingival areas in the treated quadrants;

– removal of the remaining dental plaque;

– identification of foci of demineralization, demonstration to patient and remineralisation.

We carry out professional oral hygiene usually twice a year. In case of periodontal diseases frequency of professional oral hygiene may be increased.

PROFESSIONAL REMOVAL OF DENTAL DEPOSITS

Professional removal of dental deposits (mechanical tooth cleaning) is the selective removal of supragingival and subgingival plaque from all tooth surfaces with mechanically driven instruments and fluoride polishing paste by specially trained personnel — prophy dental nurse, dental hygienist or dentist. It also includes the removal of calculus and subgingival plaque. Before starting the professional plaque removal we must do oral instillation with antiseptic and local anesthesia (if it is necessary).

PROFESSIONAL REMOVAL OF SOFT DENTAL DEPOSITS: TASKS, TOOLS AND METHODS

Professional removal of soft dental deposits is carried out with *instrumental* and *apparatus* methods. The aim of both methods is to remove dental plaque.

During *instrumental* removal of soft dental deposits the vestibular and lingual surfaces of all teeth are cleaned with such aids as rotating rubber cups

(used for gingival area) and rotating brushes (used for tuberal area). Interdental cleaning is carried out with reciprocating interproximal tips, dental tape and floss, dental plastic and metal strips. We use also polishing pasts with different abrasive level.

Apparatus removal of soft dental deposits means airflow (sandblaster, air polishing) usage. Air polishing is a procedure which uses air and water pressure to deliver a controlled stream of specially processed powder (sodium bicarbonate, calcium carbonate, glycine) into a slurry through the handpiece nozzle. Fine particles of powder are propelled by compressed air in a warm spray and directed onto the surfaces of the teeth. This pressurized jet of air, water and powder removes surface stains, plaque and other soft deposits. We use airflow only for enamel because abrasive can damage dentin and cementum of the root.

Indications for the use of airflow:

– general post-scaling prophylaxis;

– cleaning of deep fissures;

– interproximal cleaning;

– surface cleaning (stains and dental plaque removal);

– tooth cleaning before fluoridation;

– removal of temporary cement residues.

Contraindications for the use of airflow:

– respiratory tract diseases;

– if a patient is on a salt-free diet;

– allergy to flavor of powder;

– children under 12 years.

During plaque removal with airflow we must protect eyes and nose of the patient. It is necessary to work with saliva ejector and vacuum ejector. After the treatment the patient should not eat products that can stain his/her teeth, drink tea or coffee and smoke for two or three hours.

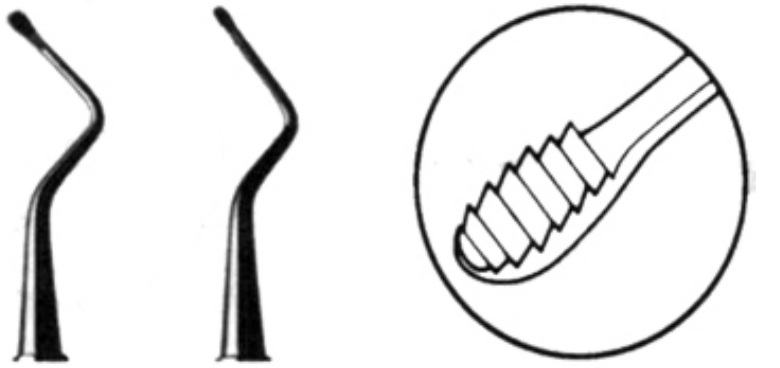
REMOVING MINERALIZATED DENTAL DEPOSITS WITH HAND AND POWER TOOLS AND METHODS

Removal of mineralized dental deposits is carried out with *chemical*, *manual* (hand), *electromechanical* and *laser* methods.

Chemical method is used as initial phase of calculus removal. Dental materials for chemical removal contain acids (mainly HCl). The time of application is from 30 till 60 seconds usually. Gums protection is necessary.

For *manual* removal of mineralized dental deposits we use scalers, curettes (fig. 3), excavators, files/rasps (fig. 4), hoes (fig. 5) and chisels (fig. 6).

Fig. 3. Curettes, working part (universal & Gracey)



a ***b***

Fig. 4. File (rasp):

a — overall view; *b* — working part

Fig. 5. Hoe (overall view & working part)

a ***b***

Fig. 6. Chisel:

a — overall view; *b* — working part

There are instruments with a non-metallic working part made of heavy-duty plastic for the patients with initial caries, patients with implants and for children.

We can take manual instrument in different ways (fig. 7). It depends on tooth area cleaned and the kind of instrument that we use. We have to keep the instrument very tightly so it can not harm the patient in case of bad situation.

Fig. 7. Different ways of manual instrument fixation

Electromechanical methods of removal of mineralized dental deposits are divided to sonic (pneumatic, air, sound) scalers and ultrasonic (ultrasound) scalers using. Ultrasonic scalers has two kinds: piezo scalers and magnetostrictive scalers.

There are four mechanisms of action of ultrasonic scalers to dental plaque: mechanical destruction and displacement, irrigation, turbulence and cavitation.

Ultrasonic cleaning may not be advised for patients with pacemakers, for patients who have highly virulent bloodborne diseases, for patient with respiratory pathology, decreased immunity and incoagulable blood.

Patients affected with such bloodborne pathogens as HIV, hepatitis B, C, D, and E should be treated with precautions. Patients with infectious diseases should also be scheduled for the last appointments of the day when any type of invasive treatment needs to be rendered. Dental personnel must thoroughly disinfect equipment and vent the operatory to eliminate microorganisms and minimize aerosols.

The first kind of ultrasonic scalers is piezo scaler. The frequency of working part vibration is 25 000–60 000 Hz. Water cooling is necessary because the instrument becomes hot quickly. The only active surfaces are side surfaces and according to it working part likes gusset. We can not use piezo scaler in the periodontal pocket, in case of artificial crowns and orthodontics constructions (risk of removal) and for patients with initial caries and children.

The second kind of ultrasonic scalers is magnetostrictive scaler. The frequency of working part vibration is 18 000–45 000 Hz. Water cooling is also necessary. All surfaces of the working part are active and therefore its form

may be various. Magnetostrictive scaler can be used for periodontal pocket that is not very deep (depth is not more than 5 mm).

There is special kind of ultrasonic scaler called system «Vector». The frequency of working part vibration is 25000 Hz. The distinctive feature of «Vector» apparatus is vertical movements of the working part. System «Vector» has the wide range of uses — from removal of biofilm in subgingival area to calculus removal in a very deep pocket. The instrument's energy is softly given to periodontal pocket through hydrocover. This process is called hydrodynamic influence. Periodontal pocket is extensively handled and washed without further appearance of aerosol. «Vector» method is much less painful than traditional methods of periodontal treatment.

Special suspension containing micro parts increases the effectiveness of root cleaning and subsequent root polishing is not necessary. The particles of the used suspension work as tooth paste and remove soft and hard dental deposits, even from the most hard-to-reach areas. We are achieving smooth surface of root so quickly.

Sonic scaler is less harmful than ultrasonic. We can use this scaler for children and for work in periodontal pocket. The frequency of working part vibration is 3000–8000 Hz. Water cooling can be used. All surfaces of the working part are active and therefore its form may be various. The only active surfaces are side surfaces and according to it working part resembles a gusset. We can not use piezo scaler in periodontal pocket, in case of artificial crowns and orthodontics constructions (risk of removal) and for patients with initial caries and children.

Laser method appeared a few years age. A laser is defined as a nondivergent, man-made, monochromatic, electromagnetic energy of one wavelength. Mechanisms of action of laser removal are vaporization and ablation. Laser method has the wide range of uses – not only in professional oral hygiene. This method is painless, harmless and very expensive. We can use laser for children and for work in periodontal pocket.

The *features* of professional hygiene *in children* are the next:

– in case of tartar manual instruments are preferred;

– hygienist uses manual instruments with a rounded end (curettes);

– if scaler using is necessary pneumatic scaler is used;

– plastic instruments are preferred;

– hygienist avoid contact the tip of the scaler and enamel;

– airflow is used in children older than 12 years.

CORRECTION OF RESTORATION

Marginal restorations are appropriate places for plaque accumulation and bacteria reproduction causing periodontal disease. Restorative materials and cements can affect periodontium tissues in different ways and result in periodontal pathology. In most cases these destructive responds are due to superficial roughness formed by dental materials. Other way of harm is mechanical action of restoration for example due to overhang. Dental materials can also chemically damage mucosa of gingival sulcus.

In case of improper gingival-adjacent restoration, overhang, unsuitable contour, lack of restoration margin fit dental professional has to correct or change the restoration.

ENSURING EFFECTIVE SELF-CARE FOR THE PATIENT WITH A HIGH RISK AND WITH PERIODONTAL PATHOLOGY

The main principle for preventive dentistry is the preventive measures will give the most significant effect if we concentrate them on «key-risk age groups», «key-risk individuals», «key-risk teeth» and «key-risk surfaces». Self-care for the patient means the sum effect of motivation, knowledge, oral hygiene instruction, oral hygiene aids and manual (motor) skill. To inform patients about the causal relationship that lead to the disease process is important as it encourages patients to take responsibility for their own oral health. Toothbrushing instruction for a patient includes teaching of «what», «when», «where» and «how».

Dental plaque is mainly localized in the subgingival and interdental areas. Home care includes *mechanical* and *chemical* plaque control by individuals.

Mechanical Plaque Control includes using various kinds of toothbrushes for vestibular and oral surfaces cleaning: manual, electrical, ionic, sonic and ultrasonic. A soft bristled brush is more effective in removing plaque with less harm to soft and hard tissues than a brush with hard bristles because soft

bristles are more flexible and thus can reach subgingival and proximal areas. Round ended bristles are recommended because they have been shown to cause 30 to 50 percent less soft tissue trauma than course-cut bristles.

The duration of brushing was found to have a greater influence on plaque removal than either its frequency or pattern. The brushing techniques for the patient with periodontal pathology are:

1. Bass method.

2. Modified Bass technique.

3. Stillman and Modified Stillman's technique.

4. Charter's Method.

Tooth brushing alone can not effectively control interproximal plaque. Interdental oral hygiene aids include:

– dental floss;

– dental floss holder;

– toothpicks and toothpick holder;

– interproximal brushes;

– single tuft brushes;

– knitting yarm;

– gauze strip;

– pipe cleaner;

– wedge stimulators.

Patients with tight teeth contact areas need thin unwaxed floss that can be slipped easily between the contact areas, whereas in patients with crowded teeth, heavy calculus deposits, or defective and overhanging restorations, a bonded unwaxed floss or waxed floss is the dental floss of choice because they do not fray as easily as unwaxed floss. Dental tape is recommended when there is considerable interdental space resulting from gingival recession and bone loss. Toothpicks, or the equivalents, are used for open interproximal

spaces. In individuals with advanced periodontal disease we find a relatively large number of wide interdental spaces and partially exposed root surfaces. The interdental brushes usually have the greatest cleaning effect in wide interproximal spaces. If there are exposed root surfaces in the interproximal area, caution should be used with abrasive toothpaste.

Irrigation devices (water-pik) are the adjunctive and very effective aids of self-care for the patient with a high risk and with periodontal pathology. Irrigation devices may be used with water or with antimicrobial agents. Using the device on full strength may be hazardous. It is possible for the impact of the fluid to drive pocket bacteria into the tissues and produce a periodontal abscess.

Among the *chemicals* with plaque and gingivitis inhibiting potential, chlorhexidine digluconate has shown the greatest promise. Triclosan and cetylpyridinium are also widely used. These chemicals may be included in mouthwashes. Other substances for chemical plaque control are the next:

– antibiotics;

– enzymes;

– phenolic compounds and essential oils;

– natural products;

– metal salts;

– amine alcohols;

– oxygenating agents;

– fluorides.

Female hormones affect the gums, and women are particularly susceptible to periodontal problems. Hormone-influenced gingivitis appears in some adolescents, in some pregnant women, and is occasionally a side effect of birth control medication.

Medical Conditions Associated with Periodontal Disease.

Diabetes. There is a strong association between diabetes (both type 1 and 2) and periodontal disease. Diabetes causes changes in blood vessels, and high

levels of specific inflammatory chemicals such as interleukins, that significantly increase the chances of developing periodontal disease.

Other Medical Conditions. A number of medical conditions can increase the risk of developing gingivitis and periodontal disease. They include conditions that affect the immune system such as HIV/AIDS, leukemia and possibly autoimmune disorders (Crohn's disease, multiple sclerosis, rheumatoid arthritis, lupus erythematosus).

Genetic Factors. Periodontal disease often occurs in members of the same family. Genetic factors play a role in making some people more susceptible to periodontal disease.

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WORKSHOP № 8
HALITOSIS

Test Questions:

1. Halitosis: definition, biochemical basis (odorants), classification.

2. Etiology and pathogenesis of physiological oral halitosis.

3. Etiology and pathogenesis of genuine pathological oral halitosis.

4. Etiology and pathogenesis of genuine pathological extra-oral halitosis.

5. Pseudohalitosis and halitophobia: etiology and pathogenesis.

6. Methods of differential diagnostics of halitosis.

7. Prevention and treatment of physiological oral halitosis.

8. Prevention and treatment of pathological oral halitosis.

HALITOSIS: DEFINITION, BIOCHEMICAL BASIS (ODORANTS), CLASSIFICATION

Halitosis is a medical term for bad breath or an offensive, foul breath odor.

Halitosis is a widespread problem in the general population. Prevalence of halitosis is:

– 50–65 % in Europe;

– more than 30 % in USA;

– 30–65 % in Russia.

Main sources of halitosis (*odorants*) are:

– *volatile sulfur compounds (VSC)* such as methyl mercaptan, hydrogen sulfide and dimethyl sulfide;

– *amines* (their representatives are putrescine and cadaverine);

– *short-chain fatty acids* such as butyric acid, valeric acid and propionic acid;

– *indoles* (their representatives are indole and skatole).

Speaking of halitosis we use the following terms:

Real (genuine) halitosis — obvious malodor with intensity beyond socially acceptable level and/or affecting personal relationships. Real halitosis includes physiological and pathological halitosis.

Physiological halitosis — the patient does not have pathology in oral cavity or outside the mouth.

Pathological halitosis — the patient has pathology within or outside the mouth.

Temporary halitosis — malodor caused by food and dietary factors such as garlic or morning bad breath.

Intra-oral halitosis — the source of bad breath lies within the mouth.

Extra-oral blood-borne halitosis — the source lies outside the mouth.

Extra-oral non-blood-borne halitosis — the malodor originates from nasal, paranasal, or laryngeal regions, or the pulmonary or upper digestive tract.

Psychogenic halitosis — obvious malodor is not perceived by others but the patient complains of its existence.

Pseudo-halitosis — no objective evidence of malodor, but the patient thinks they have it.

Halitophobia — the patient persists in believing they have halitosis despite firm evidence for the absence of objective evidence. The patient usually has embarrassment and problem with interpersonal social communication.

Classification of halitosis is shown on figure 8.

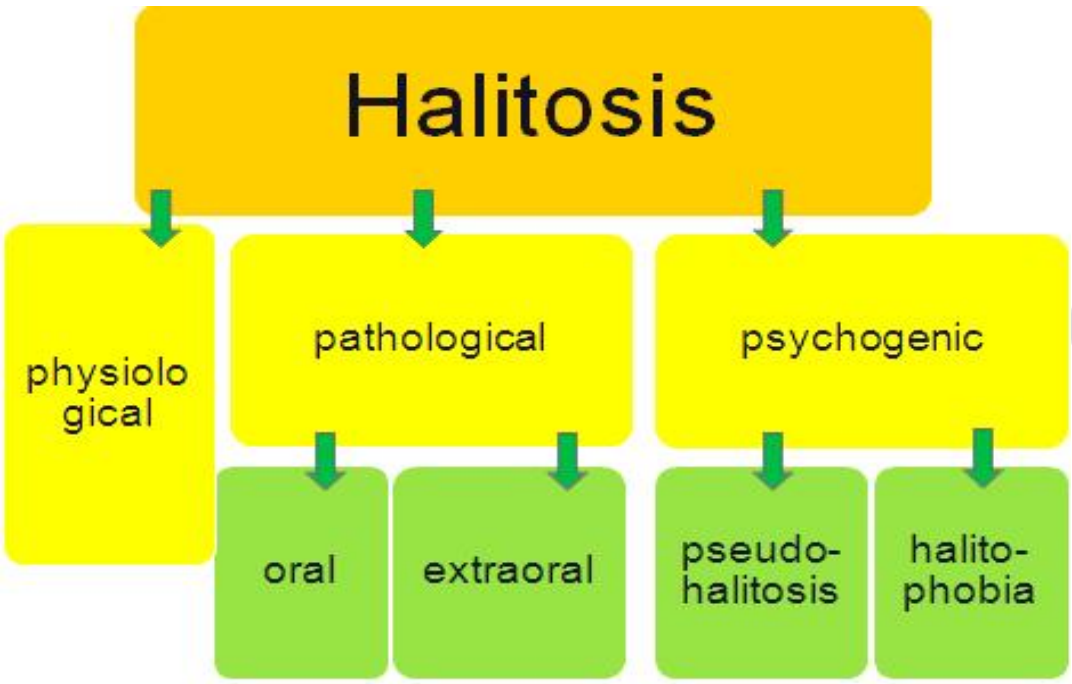

Fig. 8. Classification of halitosis

PHYSIOLOGICAL ORAL HALITOSIS

The origin is often a coating on the dorso-posterior region of the tongue and/or dental plaque in interproximal areas of molars. Anaerobic microorganisms of tongue coating produce odorants which dissolve in saliva. When there is too much of odorants in saliva they convert in gas and go to the air and we can smell it.

Malodor at other times may be the consequence of lifestyle. Halitosis as a result of the ingestion of certain food and drinks, such as spices, garlic, onion, durian, cabbage, cauliflower and radish, or of habits such as smoking tobacco or drinking alcohol, is usually transient, often caused by sulfur-containing volatile agents and is considered to arise both from intra-oral (food debris, decrease in production of saliva) and extra-oral (respiratory) origins.

Treatment of physiological oral halitosis is the next:

– tooth brushing and flossing training of a patient;

– mechanical removal of tongue coating;

– professional oral hygiene;

– use of mouth rinses;

– dietary recommendations: cleansing the mouth after eating dairy products, fish, meat, garlic, onions, coffee, tobacco;

– regular visits to the dentist.

There are many different tools for mechanical removal of tongue coating on the market. A tongue scraper can only remove the upper surface layer of the biofilm, the effect of a tongue scraper is shorter in duration than the effect of a tongue brush. Moreover, cleaning too hard with a tongue scraper, is a risk for tongue injury. Any kind of electrical device for professional tongue cleaning is not recommended because of the risk of traumatic injury. Animal experiments have shown that mechanical injuries of the tongue may be carcinogenic. Therefore, detailed and comprehensive tongue cleaning instructions are necessary. Tongue cleaning should be carried out gently with low force at the posterior part of the tongue dorsum. The lateral borders should not be cleaned because of the risk of traumatic injury.

The patient may use antiseptics and dentifrises with deodorizing effect:

– chlorhexidine (0,05–0,2 %);

– triclosan (0,03–0,05 %);

– chlorine dioxide;

– hydrogen peroxide;

– zinc;

– cetylpyridinium (0,025–0,2 %);

– urea (in pastes and gels);

– mint and essential oils.

A combination of low concentrations of zinc and chlorhexidine seems to be the most efficient way to remove the volatile sulfur compound that causes bad breath.

GENUINE PATHOLOGICAL ORAL HALITOSIS

The main causes of pathological oral halitosis are the next:

– periodontal pathology;

– carious teeth and poor-quality restoration;

– dental plaque under the orthodontic and orthopedic constructions;

– xerostomia (dry mouth).

Therefore, the treatment of pathological oral halitosis may include:

– professional oral hygiene;

– periodontal surgery;

– removal of destroyed teeth;

– correction of xerostomia;

– using of oral irrigators for individual oral hygiene.

GENUINE PATHOLOGICAL EXTRA-ORAL HALITOSIS

The first source of bad breath is the respiratory pathology (such as sinusitis, antral malignancy, cleft palate, tonsillitis). In this case we are speaking of extra-oral non-blood-borne halitosis.

The malodor also may be emitted via the lungs and originates from disorders anywhere in the body (e. g., hepatic cirrhosis) — extra-oral blood-borne halitosis.

The main causes of pathological extra-oral halitosis are the next:

– pathology of upper respiratory tract;

– pathology of the gastrointestinal tract;

– liver disease;

– renal failure;

– diabetes;

– helminthosis;

– radiation therapy;

– some metabolic disorders.

In some cases, depending on the smell of the patient's breath, the dentist may suspect a likely cause for the problem. For example, «fruity» breath may be a sign of uncontrolled diabetes. An urine-like smell, especially in a person who is at high risk of kidney disease, can sometimes indicate kidney failure.

There are some drugs that predispose to the occurrence of halitosis in patients:

– antibiotics;

– corticosteroids;

– antihypertensive;

– antihistamines;

– antidepressants;

– anticholinergic;

– diuretics;

– anticonvulsants.

The treatment of pathological extra-oral halitosis is the care of extra-oral pathology.

PSEUDOHALITOSIS AND HALITOPHOBIA

Pseudohalitosis and halitophobia are psychogenic kinds of halitosis.

Not all persons who believe they have halitosis actually have any malodor, and this pseudo-halitosis can be a real clinical dilemma. Interestingly, anxiety itself increases the levels of volatile sulfur compounds.

In patients without the evidence of halitosis, even if they are examined with objective testing, and in patients who always complain, halitosis may be attributed to a form of delusion or monosymptomatic hypochondriasis. But also the causes may be latent psychosomatic illness and mentally immature.

The treatment of pseudohalitosis and halitophobia includes:

– empathy and trust relationship with the patient;

– investigation of exhaled air;

– reasoning of the patient;

– if necessary — consultation with other professionals (neurologist, psychologist or psychiatrist).

DIAGNOSTICS OF HALITOSIS

A person may not always know that he or she has bad breath. This phenomenon is because odor-detecting cells in the nose eventually become accustomed to the constant flow of bad smell from the mouth. Other people may
notice halitosis and tell to this person. A dentist or physician may notice the patient's bad breath while the patient is discussing his or her medical history and symptoms.

Clinical assessment of oral malodour is important and should be performed by 2 different examiners. The assessment is usually based on the clinician sniffing the air exhaled from the mouth and nose, and subjectively assessing the presence or absence of malodour.

A series of organoleptic tests that can be performed during self-diagnostics or during clinical examination have been suggested. Both the patient and the clinician should refrain from drinking coffee, tea, or juice, as well as from smoking and using scented cosmetics, before the assessment. These tests include:

– *Sniffing test*: the clinician sniffs the exhaled air from 10 cm distance after the patient's mouth has been closed for 2 minutes.

– *Count-to-twenty test*: the clinician sniffs the exhaled air from 10 cm distance while the patient counts up to twenty.

– *Wrist-lick test*: the patient is asked to lick his/her wrist, whereupon the clinician (or the patient) sniffs the wrist from 5 cm distance.

– *Spoon test*: a plastic spoon is used to gently scratch the lingual dorsum to collect epithelial cells and microbial film. The clinician (or the patient) sniffs the spoon from 5 cm distance.

– *Flossing test*: the patient is asked to floss his molar teeth, and the clinician (or the patient) sniffs the floss from 3 to 5 cm distance.

If no malodor can be found during the initial examination, the assessment for halitosis should be repeated in two or three days. Thereafter, if halitosis is still not present, the patient can be considered to be affected by psychogenic halitosis.

It is important to understand whether a given complaint of bad breath is justified and whether the odor originates in the mouth, nose or elsewhere. A full history and an oral examination will be required.

The medical history focuses on current medications and systemic diseases. The presence of nasal obstruction, mouth breathing, report of snoring and sleep apnea, postnasal drip, allergy, tonsillitis, tonsilloliths, dysphagia, previous ear, nose and throat encounters, types of food typically eaten, as well as vitamin A, B, C, D, and zinc-containing food intake are recorded. The dental history includes questions assessing the frequency of dental visits, the presence and maintenance of dental prosthesis as well as the frequency and the instruments used for tooth brushing, interdental cleaning, or tongue brushing/scraping and other dental products being used. The oral examination focuses on the predilection sites of intraoral halitosis.

More objective measurements of halitosis are available but they are not used in routine clinical practice, as they are expensive and time consuming. These measurements are:

– assessment of concentration of volatile sulfur compounds using a gas analyzer;

– gas chromatography of saliva;

– microbiological studies to identify the bacteria associated with the development of halitosis.

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WORKSHOP № 9
INDIVIDUAL AND GROUP PREVENTION OF PRINCIPAL DENTAL DISEASES

Test Questions:

1. Risk-factors for oral diseases.

2. Methods of determination of individual risk level for dental caries (T. F. Vinogradova; F. Courson, M. Landru; P. A. Leous).

3. Individual dental diseases prevention: goal, objectives and strategy. Principles and stages of individual programs compiling, efficiency evaluation, adjustments.

4. Variety of individual programs for dental caries prevention (E. Newbrun, D. Ericsson).

5. Prevention of dental diseases in groups: recipients, methods and implementers.

Risk may be defined as the probability of incidence of an event within a certain period of time. The risk of oral diseases, therefore, is the risk of an individual developing an oral pathology. In case of caries increased risk may be the result of several caries-producing factors coinciding or of insufficient defense mechanisms leading to different caries prevalence. By definition, risk is aimed at assessing developments in the future.

According to *T. V. Vinogradova* children with the initial caries and children with decompensated form of tooth decay have greatest risk of dental caries.

Method of determination of individual risk level for dental caries by *P. A. Leous* allows us to estimate the risk of caries in the next 1–5–6 years with 80–90 % reliability and to develop appropriate prevention program. The determination of the level of risk is based on: a) clinical examination data (determined by the index of hygiene, caries intensity); b) history data collected by means of questionnaires containing questions about the level of dental knowledge, oral and dietary habits, preventive activity and other risk and protective factors for appropriate age group.

Method of determination of individual risk level for dental caries was described by F. Courson, M. Landru (2001) (table 4).

The main task of therapeutic dentistry is to prevent oral diseases. Prevention programs may work on 3 levels: individual, group, communal.

Individual program for dental caries prevention are described in tables 5, 6.

Table 4

Method of determination of individual risk level for dental caries
(F. Courson, M. Landru, 2001)

The intensity of dental caries	Risk factors	Risk level for dental caries
dmf (DMF) = 0	☐ no caries increment in the past year; ☐ pits and fissures in permanent teeth are mild; ☐ good oral hygiene with twice daily cleaning; ☐ constant application of fluoride (toothpaste); ☐ regular visits to the dentist; ☐ balanced diet; ☐ somatic anamnesis is not burdened; ☐ fluorosis within 0–3 points (TF)	Low risk
dmf (DMF) ≤ 3, new carious lesion in the past year	☐ new carious lesion in the past year; ☐ white spots (initial caries) on smooth surfaces and/or radiological detection of proximal caries; ☐ expressed pits and fissures are not sealed; ☐ poor oral hygiene (brushing irregular, the presence of plaques in certain areas; ☐ irregular visits to the dentist; ☐ inadequate fluoride exposure; ☐ fluorosis stages 4–5 (TF); ☐ malocclusion; ☐ orthodontic treatment by fixed devices	Moderate risk
dmf (DMF) > 3, two or more new carious lesion in the past year	☐ multiple new cavities in the past year; ☐ minimum or no fluorides; ☐ poor oral hygiene, dental plaque on all surfaces; ☐ uncontrolled irregular tooth brushing; ☐ unbalanced diet with poor eating habits; ☐ 6–9 stages of fluorosis (TF); ☐ orthodontic treatment with multiple fasteners to the teeth; ☐ visit to a doctor only about the pain; ☐ complicated somatic history; ☐ adverse social conditions	High risk

Table 5

Individual programs for dental caries prevention by E. Newbrun, 1992

Caries risk level	Preventive measures	
	professional	Home-measures
Low risk	☐ use of fluorinated polishing pastes for professional hygiene; ☐ sealants	☐ daily use of fluoride toothpaste; ☐ daily use of fluorine-containing mouthwashes
Moderate risk	☐ use of fluorine-containing gels or varnishes twice a year; ☐ sealants; ☐ modification of diet; ☐ use of teeth restoration materials preventing the	☐ use of fluoride toothpaste 2–3 times a day; ☐ daily use of fluorine-containing gels (1,1 % NaF or 0,4 % SnF_4)

Caries risk level	Preventive measures	
	professional	**Home-measures**
	development of secondary caries	
High risk	□ use of fluorine-containing gels or varnishes twice a year; □ sealants; □ modification of diet; □ bacteriological monitoring; □ use of teeth restoration materials, prevents the development of secondary caries	□ the use of fluoride toothpaste; □ daily use of fluorine-containing gels (1,1 % NaF or 0,4 % SnF_4); □ mouthwash contains chlorhexidine (daily for two weeks, the course is repeated 2–4 times during the year); □ the use of chewing gum with xylitol; □ in case xerostomia to use of «artificial saliva»

Table 6

Individual program for dental caries prevention by D. Ericsson, 1994

Risk factors	DMF=0	Low caries intencity	Moderate caries intencity
Acute caries		фторлак	(←) the same + chlorhexidine
The high number of lactobacilli	□ diet counseling; □ panoramic radiography; □ sealants	(←) the same + polishing of; Lb control	(←) the same
The high number of streptococci	□ risk information; □ St. m. control; □ sealants	(←) the same + professional hygiene	(←) the same + chlorhexidine
Low buffering capacity of saliva	□ control of saliva secretion; □ saliva sediment examination; □ fluoride varnish	(←) the same + stimulation of salivation	(←) the same
Low rate of salivation	□ risk information; □ clarification of medication; □ stimulation of salivation; □ Lb. & St. m. control	□ council for replacement drugs; □ diet consultation	(←) the same + fluoride varnish
Poor oral hygiene	□ risk information; □ oral hygiene training □ professional hygiene	(←) the same + oral hygiene control	(←) the same
Unbalanced diet	risk information	□ diet consultation; □ Lb control	(←) the same + diet correction

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CONTENT

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